Anaesthesia Emergencies

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INDEX:

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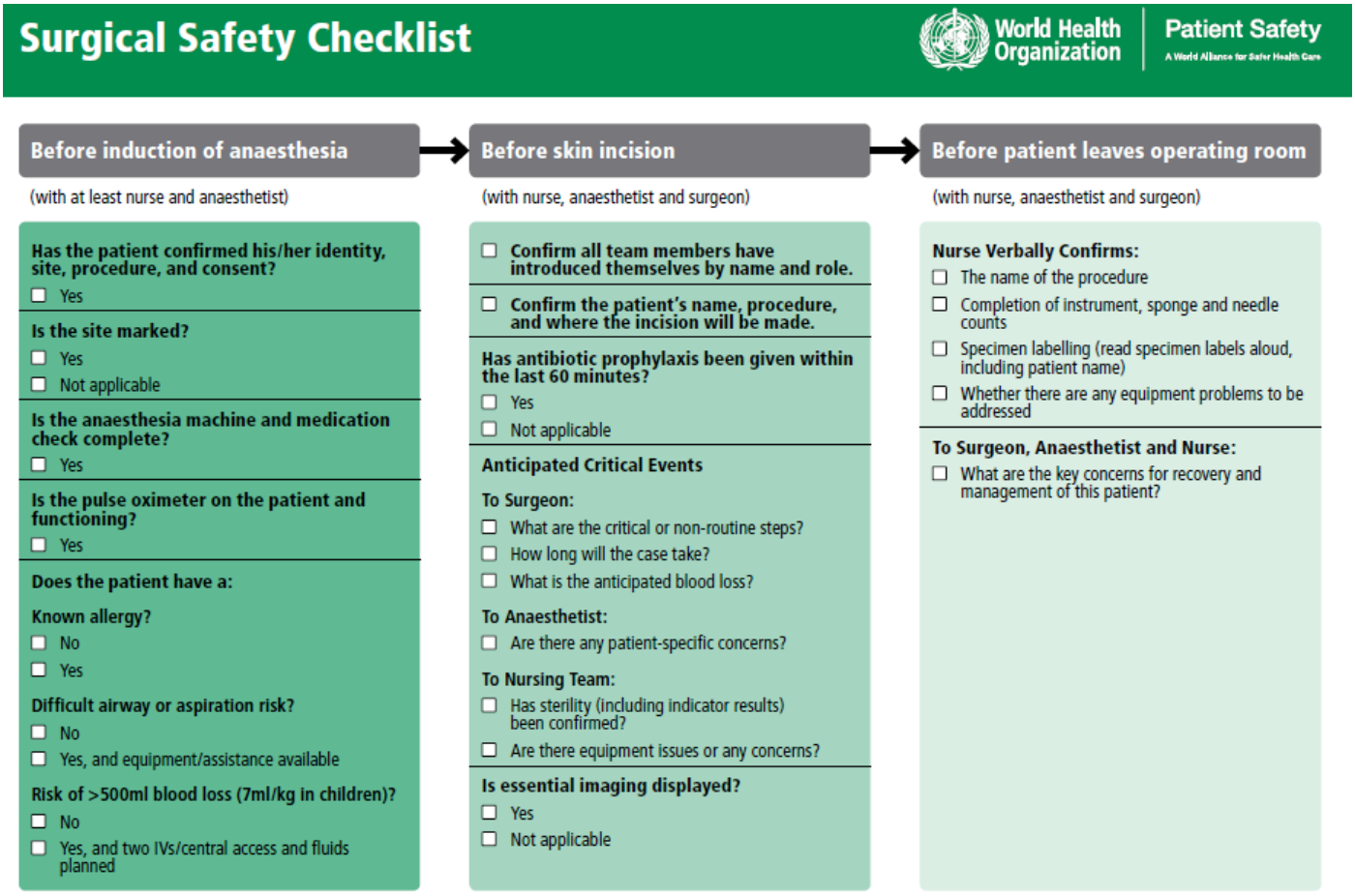
AN APPROACH TO COMMON INTRA-OPERATIVE PROBLEMS

Anaesthesia and surgery can be a time of great physiologic instability. Intra-operative problems occur secondary to the patient's medical or surgical condition, surgical manipulation and bleeding, or may be related to anaesthesia.

Anticipating and avoiding problems

Patients undergoing surgery under regional or general anaesthesia can encounter intra-operative problems that require prompt diagnosis and treatment by a physician (anaesthetist) dedicated to the care of the patient.

Avoiding problems requires the anaesthetist to pay attention to detail. Many problems occur due to a lack of adequate preoperative assessment and checking of drugs and equipment. The plan for the anaesthetic is verified with all concerned with the anaesthetic care, particularly the assistant to the anaesthetist. The (World Health Organization) WHO requires that a checklist of many factors including patient identity, correct site of surgery and other anaesthetic and surgical parameters, precede each surgical intervention. (1) This check list may be modified to suit local requirements.



Good anaesthetic practice involves the continuous presence of the treating anaesthetist or a delegate who is trained to detect and manage instability or a new problem in the patient.

Regular observation and charting of the patient's vital signs is required. The Australian and New Zealand College of Anaesthetists (2) recommends monitoring of the cardiovascular parameters including arterial pulse and blood pressure, respiratory parameters including continuous monitoring of ventilation and oxygenation (via oximetry and observation of the patient's colour). The frequency of observation needs to be at least 5 minutely or more frequently if there is instability.

Equipment that should be available includes an oxygen analyser , breathing system disconnection alarm, pulse oximeter, electrocardiograph, non-invasive and invasive blood pressure monitor, carbon dioxide monitor, volatile anaesthetic agent concentration monitor, temperature monitor, neuromuscular function monitor and equipment to monitor the anaesthetic effect on the brain.

Managing interoperative problems

When a problem is detected, the things to consider include: the significance of the problem, the safety of the patient and the need for help. Detecting the cause of the problem is a priority so that treatment can be initiated promptly before a minor problem causes serious consequences.

A systematic approach is required to detect the cause of the intraoperative problem. This can be done in two ways. The ABCDE approach that is part of the advanced life support philosophy or the COVER ABCD A SWIFT CHECK (4) algorithm, used by many anaesthetists in Australia. This approach will detect 95% of incidents. This second approach is more applicable to anaesthesia. A review of all systems is required unless it is apparent early on what the cause of the problem is. Life threatening problems are treated first and help is called for early if the situation is serious or beyond the expertise of the treating anaesthetist. Call for experienced help whenever the cause of a problem is not identified or if the situation is deteriorating. Treatment of some problems will require help from a colleague or other members of the team, particularly if the situation escalates into a crisis. It is important to communicate effectively with the surgeons and assistants so that everyone is aware of the potential for the situation to evolve into a crisis.

ABCDE/ Advanced Life Support approach:

A= Airway. Ensure a patent airway at all times.

B=Breathing. Provide adequate oxygenation and ventilation (increase the inspired oxygen concentration to 100% and hand ventilate the patient to check for chest wall movement. Auscultate the chest.

C=Circulation. Ensure the circulation is adequate to perfuse the vital organs.

D = Disability or neurologic state of the patient. Check the depth of anaesthesia (too much or too little anaesthetic agent), any look for any causes of reduced consciousness in a patient having a spinal or regional technique.

E= Exposure. Look at the patient from head to toe (as far as possible during surgery). Check for blood loss and surgical manipulation or positioning that may account for changes in blood pressure or heart rate. Look at your equipment and drugs to check that everything is connected and re-check what medications have been administered.

COVER ABCD A SWIFT CHECK (9) **Approach** (for an intubated patient under general anaesthesia):

C= Circulation and colour (saturation)

O= Oxygen supply and Oxygen analyser

V= Ventilation and vaporizer

E= Endotracheal tube and eliminate machine

R= Review monitors and equipment

A= Airway

B= Breathing

C= Circulation

D= Drugs

A= Awareness

A=Air Embolus

A=Anaphylaxis

A=Air in the pleura

S= Surgeon Situation

S=Sepsis

W=Wound

W=Water Intoxication

I=Infarct

I=Insufflation

F="Fat syndrome" obesity or abdominal distension causing problems with ventilation and hypotension

F=Full bladder causing sympathetic nervous stimulation

T=Trauma

T=Tourniquet down (release of a tourniquet will cause vasodilation, bleeding or systemic absorption of local anaesthetic in a Bier's block)

C= Cement (methylmethacrolate will cause haemodynamic changes)

C=Catheter, intravenous cannula, chest drain problems

H=Hyperthermia

H=Hypoglycaemia

E=Embolus

E=Endocrine

C=Check the correct patient, correct operation, correct surgeon

C=Check case notes, preoperative status and preoperative medications

K=K+ (potassium)

K=Keep (keep the patient asleep)

Respiratory problems

Respiratory complications during anaesthesia will eventually cause hypoxaemia and hypercarbia with serious cardiac and neurologic consequences. Oxygen saturation monitoring, monitoring of ventilatory pressures, end tidal capnography, inspired oxygen concentrations and oxygen supply monitoring will help to detect and avoid tissue hypoxia.

Low oxygen saturations:

Low oxygen saturation is significant as it reflects low oxygen content in the blood. (Recall that most oxygen is carried bound to haemoglobin, and it is the saturation of this haemoglobin that is measured by a pulse oximeter). A saturation of less than 90% reflects an arterial oxygen partial pressure of 60mmHg. Delivery of oxygen to the tissues may begin to be compromised, particularly if it is accompanied by hypotension.

The detection of hypoxaemia is best with the use of a working pulse oximeter, but the colour of the patient will be another indicator. Cyanosis occurs at a saturation level of less than 85% (or PaO2 of 45-50 mmHg) assuming a normal haemoglobin concentration. Detection of cyanosis is only possible in the presence of greater than 5g/100ml of deoxygenated haemoglobin, and this may not occur if there is severe anaemia. Hypoxaemia is associated with blood pressure, heart rate and mental state changes, as well as ischaemia and cardiac arrhythmias.

The potential causes of hypoxaemia include:

- Reduced inspired oxygen concentration, which may be due to a failure of oxygen supply to the patient circuit or a breathing system fault or leak.

- Airway obstruction, which may be due to upper airway, glottic or lower airway obstruction, needs to be addressed rapidly. The artificial airway needs to be checked for correct positioning and patency. Bronchospasm may be detected by listening to the chest. Lower airway obstruction may result from blockage of the conducting airways by secretions or aspirated gastric material and foreign objects.

- Hypoventilation will occur if there is inadequate artificial ventilation or depression of spontaneous ventilation due to drugs or respiratory muscle weakness from high spinal blockade.

- Inadequate ventilation may occur if the patient has high oxygen consumption or if there is a ventilation/perfusion mismatch in the lungs.

- Inadequate cardiac output can create an increase in dead space (that is, areas of lung that are ventilated but not perfused leading to hypoxaemia).

- Physiologic shunt will cause hypoxaemia due to areas of lung that are perfused but not ventilated. Anatomic shunt occurs when part of the cardiac output bypasses the alveoli and therefore does not receive oxygen. The patient will be hypoxaemic and may appear cyanosed. (5)

The management of hypoxaemia requires a rapid diagnosis and simultaneous treatment. The fraction of inspired concentration is increased and vital signs checked as well as confirming the saturation readings and end tidal carbon-dioxide concentrations. Hand ventilating the patient may reveal reduced compliance or stiff lungs. The chest wall should move bilaterally. Ventilation with a few high volume breaths may open areas of collapsed lung. Care should be taken to avoid excessive airway pressures that may cause a pneumothorax. Auscultation of the chest may reveal crepitations or bronchospasm or the silent chest of a pneumothorax.

If the cause of hypoxaemia is not immediately apparent, the endotracheal tube can be suctioned and consideration given to bronchoscopy. The addition of positive end expiratory pressure and restoration of circulating blood volume can help treat persistent hypoxaemia. The arterial blood gases need to be measured and a chest x-ray taken to assess severity and determine a cause. Surgery may need to be terminated and arrangements made to transfer the patient to intensive care post operatively.

High airway pressures: (5)

Airway pressures are measured in the anaesthesia circuit and reflect pressure in the patient's airway. The measured pressure in the circuit in a patient receiving positive pressure ventilation depend on lung and chest wall compliance, resistance in the conducting airways, flow of gas, tidal volume and pressure generated by the ventilator.

High airway pressures may cause difficulty with ventilation, trigger an alarm (if it is in use), cause hypoxia and circulatory collapse, and may present with an upsloping trace on the end tidal carbon dioxide monitor.

A sudden increase in the airway pressure or a feeling of "stiff lungs" needs to be addressed promptly as it could indicate a problem with the patient's lungs, obstruction of the airway or a problem with the ventilator circuit. If high pressures are present in the airway, there is the potential for a pneumothorax, barotrauma to the lungs or reduced venous return due to increased intrathoracic pressure. If the circuit or upper airway is occluded, ventilation will not be possible and hypoxia will occur.

The initial approach to management includes a quick check of ABC (Airway Breathing and Circulation) and checking of the monitors (saturations, capnography), looking at the breathing circuit and anaesthetic machine and checking to see what the surgeon is doing. One of the first things to do it to excluded the machine, that is, to take the

patient off the ventilator and manually ventilate with 100% oxygen. Some causes will be obvious, so they should be looked for initially. They include: the level of muscle relaxation, a kinked circuit or closed expiratory limb, or excessive tidal volumes during ventilation.

A more systematic approach is then used if the cause is not immediately apparent. Check from the source of gas/oxygen, machine, circuit, ventilator, endotracheal tube, then to the patient to identify the cause.

The anaesthetic circuit and gas supply should be checked. There may be a fault with the oxygen flush button and it could be stuck in the on position, there may be a high pressure gas source, the circuit could be connected incorrectly or the volumes set on the ventilator could be excessive. A foreign body could block the patient circuit or the airway filter could be wet, which obstructs the flow of gas.

The endotracheal tube needs to be checked. It could be kinked, obstructed or in the right main bronchus. A suction catheter can be passed to check the patency of the tube and the cuff deflated to ensure that the problem is not cuff herniation. If these manoeuvres do not solve the problem, the tube may need to be changed.

Patient pathology can cause an increase in airway pressures. The causes can be thought of in terms of a reduction in chest wall compliance, a reduction in lung compliance or an increase in airway resistance. The causes of reduced chest wall compliance include inadequate paralysis, increased intra-abdominal pressure, obesity, prone or lithotomy positioning, large doses of opioids and malignant hyperthermia. A reduction in lung compliance occurs with pneumothorax or haemothorax, atelectasis, pulmonary oedema, lung fibrosis and adult respiratory distress syndrome. The causes of increased airway resistance include bronchospasm due to airways disease or anaphylaxis, aspiration of gastric material, foreign body inhalation and amniotic fluid embolism.

Cardiovascular Problems

Disturbances of cardiac function and the circulation are common during anaesthesia due to the side effects of drugs, regional anaesthesia (that may cause sympathetic nervous system blockade) and surgery. There may also be pre-existing disease that will cause intra-operative disturbances of cardiovascular function.

Cardiovascular disturbances can be classified as problems with heart rate (either bradycardia or tachycardia), cardiac contractility, and blood pressure (either high or low).

Hypotension

Hypotension is defined as a drop in blood pressure of more than 20% below baseline. The reading needs to be validated and then a decision should be made as to whether the

fall in blood pressure is serious and likely to cause end organ ischaemia (including drowsiness, confusion, agitation, nausea, angina or ST segment changes on the ECG). If there is evidence of low perfusion to end organs, the hypotension needs to be treated and assessed as an emergency. The inspired oxygen concentration is increased to ensure tissue oxygenation and if a cardiac arrest ensues, chest compressions need to be commenced and the cardiac rhythm checked as per the advanced life support algorithm.

Severe intra-operative hypotension can occur due to acute blood loss, anaphylaxis, high spinal block, cardiac dysfunction (from ischaemia or depressant effects of drugs) compression of the inferior vena cava, gas or fat embolism, and release of a tourniquet or vascular clamp. Arrhythmias will cause hypotension and may need specific treatment.

Assuming the cardiac rhythm is normal, the first priority is to ensure circulatory support with intravenous fluid, increasing venous return (by raising the legs if possible) and administration of vasopressors such as ephedrine, metaraminol, phenylephrine, noradrenaline or adrenaline.

If the hypotension is not severe, there is time to identify and treat the cause and to consider rare causes such as endocrine causes, drug error, pericardial tamponade, septic shock, transfusion incompatibility and anaphylaxis.

Hypertension

Hypertension is defined as a rise in blood pressure of over 20% above normal blood pressure. Under anaesthesia, it can be due to a sympathetic response (to pain, intubation and surgery), pre-existing hypertension, hypoxaemia and hypercarbia, drugs, cerebral ischaemia, volume overload or sudden increases in afterload (as in cross clamping of the aorta, pneumoperitoneum and hypothermia). Some rare endocrine and metabolic causes can cause severe hypertension and need to be considered if the hypertension is severe or resistant to routine treatment. They include thyroid storm, phaeochromocytoma and malignant hyperthermia.

If it occurs, hypertension should be validated with another blood pressure reading. The severity is assessed and causes considered before commencing treatment. If treatment of the presumed cause is not effective alone, medications are administered. The choice of treatment includes vasodilators (such as hydralazine, glyceryl trinitrate and sodium nitroprusside), alpha-blockers (clonidine, phentolamine) and beta-blockers (metoprolol, atenolol, esmolol, labetolol). (5)

Disturbances of heart rate and rhythm and cardiac ischaemia will be considered elsewhere in this booklet.

Conclusion

Cardiac and respiratory problems are of most concern to anaesthetists as they occur frequently to some degree with administration of anaesthesia. Good preparation and assessment of the patient can help to predict some problems and the use of adequate monitoring along with intra-operative vigilance by the anaesthetist will allow early detection and correction of problems before they are allowed to become serious and evolve into critical events. Serious disturbances require a rapid and systematic response. Call for help early in this situation. Treatment may need to occur simultaneously with assessment.

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PERI-OPERATIVE TACHYCARDIA/BRADYCARDIA

The management of patients with cardiac arrhythmias is driven by clinical assessment and the need to make timely decisions:

- Is the situation immediately life threatening?
- Does the patient need cardio-pulmonary resuscitation?
- Is the rhythm slow or fast?

If the patient is unstable with serious signs or symptoms, then urgent and invasive therapy is indicated. Serious signs and symptoms include hypotension (systolic blood pressure less than 90mmHg in a conscious patient - a lower pressure is usually tolerated in anaesthetised patients), heart rate >150 or <40 beats per minute, reduced level of consciousness, chest pain or congestive heart failure.

There are 3 basic questions to address when managing cardiac arrhythmias: What is the rhythm, what is the underlying cause and what is the treatment?

What is the rhythm? There are two basic possibilities - bradycardia and tachycardia. When looking at the ECG check the following points. Is there a P-wave, if so what is its relationship to the QRS? Is the QRS morphology normal, what is its width, and is the rhythm regular?

What is the underlying cause? Peri-operative arrhythmias generally occur in patients who have structural heart disease and some factor that initiates the arrhythmia. The factor may be:

1. Acute ischaemia
2. Sympathetic stimulation
3. Drug effects
4. Electrolyte imbalance (especially hypokalaemia or hypomagnesaemia)
5. Hypoxia, hypercarbia
6. 4H's and 4T's (Hypoxia, hypovolaemia, hyper/hypothermia, hyperkalaemia or electrolyte imbalance, toxins, tension pneumothorax, tamponade and thrombosis - cardiac or pulmonary).

What is the treatment? This is determined by the clinical urgency and the availability of equipment (such as a pacemaker for bradycardia). **Always treat the contributory factors as well as the arrhythmia.**

A simplified approach is shown below:

Urgency	Rhythm	Initial Therapy
Life-threatening	Bradycardia	Most readily available of electrical therapy (pacing) or drugs
	Tachycardia	Electrical therapy - cardioversion
Unstable but not immediately life threatening	Bradycardia	Reverse cause Consider Drugs
	Tachycardia	Reverse cause Consider Drugs

Bradycardia

Bradycardia may be absolute (<40 beats per minute) or relative (inappropriately low in the physiological context). The treatment for symptomatic bradycardia, irrespective of the cause, includes stopping vagal stimulation and then the critical decision is whether to pace or to use drugs.

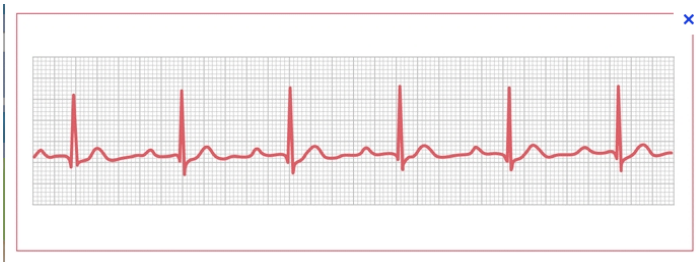
Initial drug therapy is atropine 600 mcg repeated to a total of 3 mg. If initial response is satisfactory, re-evaluate to consider the risk of asystole. The risk of asystole is higher if: there has been an episode of recent asystole, Mobitz II Atrio-Ventricular block, complete heart block with wide QRS, or ventricular pauses for longer than 3 seconds.

In the absence of a response to atropine, adrenaline is the recommended second line medication. Third-line drug therapies include aminophylline, isoprenaline, dopamine, glucagon (if there has been a β-blocker or Calcium channel blocker overdose) and glycopyrrolate. Unstable symptomatic patients should have transcutaneous cardiac pacing, atropine and/or adrenaline, as a bridge to transvenous pacemaker insertion. Pacing should be available for stable patients where there is a perceived risk of asystole.

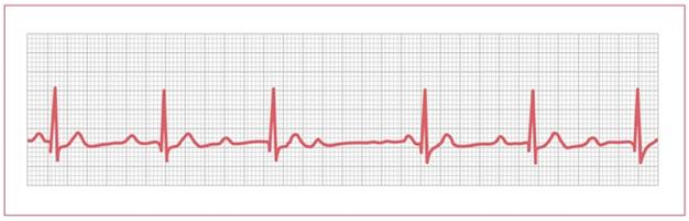
Perioperative bradyarrhythmias are usually caused by vagal stimulation, medications, electrolyte disturbances, hypoxaemia or ischaemia. Supportive therapy should be concurrent with identification of, and therapy for, underlying causes.

Sinus bradycardia, First Degree Block & Second Degree Heart Block:
All of these may be caused by excessive vagal stimulation, especially if the patient is receiving digoxin, β-blocker or verapamil. Second-degree block involves intermittent failure of atrio-ventriulcar (A-V) nodal conduction.

Mobitz Type I block is generally benign and asymptomatic. The block is usually at the A-V node with a normal His-Purkinje System. There is a progressive increase in delay between the P and QRS until a QRS complex is missed.



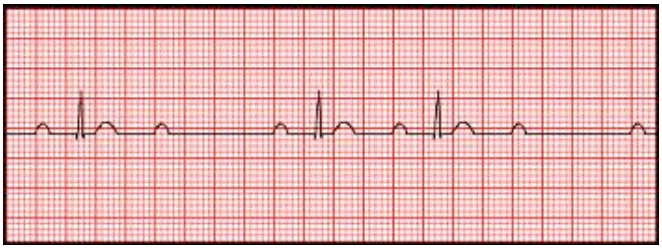
First-degree heart block



Mobitz Type I

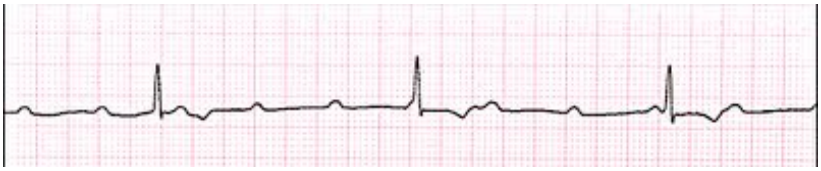
Sick Sinus Syndrome involves an alternating bradycardia and tachycardia. The treatment includes a combination of antiarrhythmics and permanent pacemaker insertion.

Second Degree Block – Mobitz Type II heart block is more ominous than Mobitz Type I. There is an intermittent failure of AV conduction with loss of QRS complexes; without progressive increase in delay between P waves and the QRS. There is an irregular QRS rhythm. It is usually caused by myocardial infarction or chronic degeneration of the conduction system and it may progress unexpectedly to third degree heart block. Symptomatic patients should be referred to a cardiologist for permanent pacing.



Third Degree Block:
In third degree block there is a total failure of A-V conduction. The block is usually below the A-V node and involves total block through both bundles, hence there is a wide QRS complex on the ECG. There is a regular but very slow QRS rhythm.

This is an unstable rhythm that is associated with extreme bradycardia and episodes of ventricular asystole. It is usually caused by myocardial infarction or chronic degeneration of the conduction system.



3^{o} heart block

Emergency Pacing:
The indications for external pacing are-

- Haemodynamically unstable bradycardia (systolic blood pressure less than 90mmHg, altered mental state, angina, pulmonary oedema). Especially if the bradycardia is unresponsive to drug therapy.
- Bradycardia with pause dependent ventricular rhythm (risk of ventricular tachycardia or ventricular fibrillation).
- Cardiac arrest secondary to drug overdose, acidosis, electrolyte disturbance or other reversible process.
- After cardiac surgery

The relative contraindications are:

- Severe hypothermia (because of the risk of triggering ventricular tachycardia and ventricular fibrillation)
- Brady/asystolic arrest >20 minutes (patient is already dead).

Technique for Transcutaneous Cardiac Pacing
External pacing is the first choice in emergency cardiac care. Modern defibrillators should have transcutaneous cardiac pacing capability. The recommended output is approximately twice the output of a standard peripheral nerve stimulator and there is no significant bystander risk (in contrast to cardioversion/defibrillation).

Large diameter (8cm) stick-on electrodes are applied. The anterior electrode is placed to the left of sternum at the cardiac apex. The posterior electrode is placed immediately behind the anterior electrode, to the left of the spine. The standard placement of electrodes as used in defibrillation is also acceptable if there is no access to the chest.

Pacing is initiated by selecting the demand mode and increasing the ECG gain. The rate is set to 60-90 per minute. The output is gradually increased until capture is achieved (most transcutaneous cardiac pacing systems have an output current of 0-200 mA). Pacing is set at 10 mA above the capture threshold.

Check that the pacing current is triggering the ventricle to depolarize. You should see a wide QRS complex and a broad T wave. Ensure mechanical capture, which is a palpable pulse synchronous with ECG.

Complications of Transcutaneous Pacing
The pacemaker current has a duration of 20 - 40 milliseconds and this current may

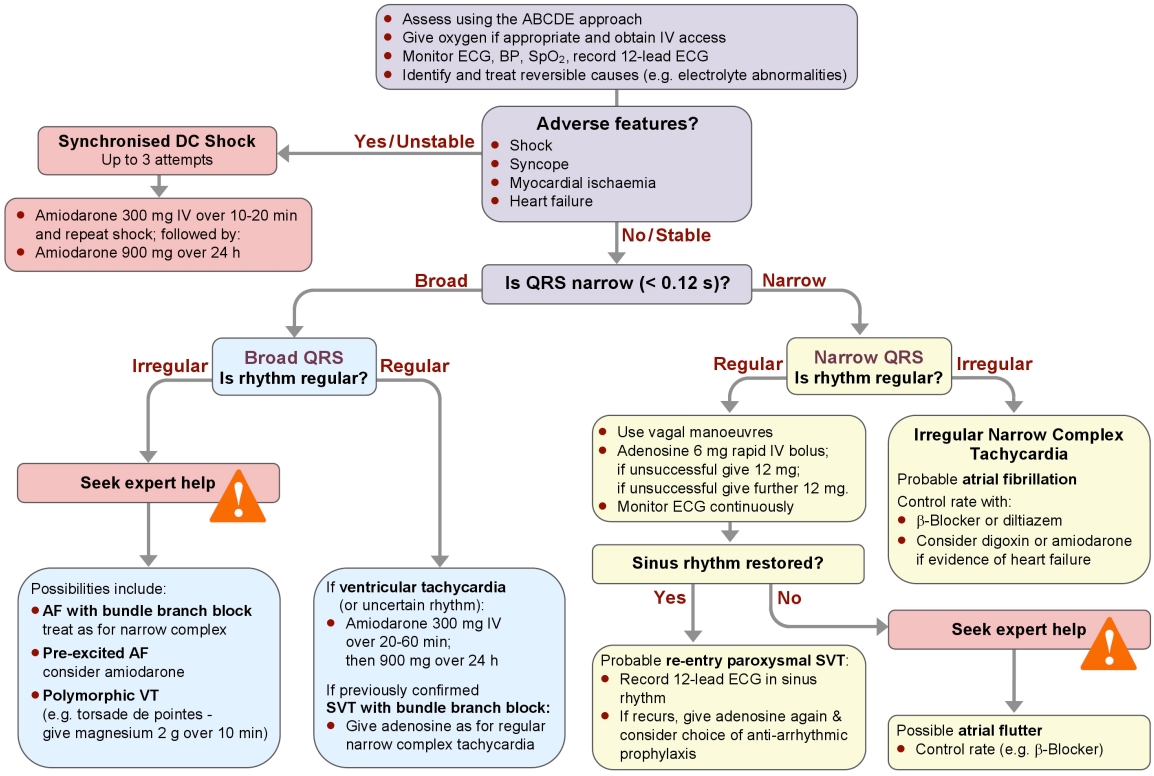
conceal the underlying rhythm. This may cause the operator to fail to recognize either non-capture or underlying ventricular fibrillation. A special “blanking” facility that conceals the pacemaker current must be incorporated in the equipment.

Pain from electrical stimulation of skin or muscle may make this difficult in the conscious patient; hence analgesia and sedation are required. Tissue damage will occur with prolonged use.

Tachyarrhythmias

The algorithm for the management of tachycardia is shown below. Broad-complex tachycardia is tolerated less well than narrow complex tachycardia, and most wide complex tachycardias are ventricular in origin.

Tachyarrhythmias are usually differentiated on the basis of site of origin (supraventricular or ventricular). This distinction is important because ventricular tachycardia may degenerate into ventricular fibrillation (VF), whereas supraventricular tachycardia (SVT) is less hazardous. In addition the pharmacological treatments are different.



Most patients with wide-complex tachycardia will have ventricular tachycardia (VT) and should be treated as such in first instance, even though some will have supra-ventricular tachycardia (SVT) with Bundle Branch Block. Most patients with narrow-complex tachycardia can be assumed to have supraventricular tachycardia. Both VT and SVT reduce the diastolic period and thus may reduce myocardial perfusion and precipitate myocardial ischaemia. A cardiology opinion should be sought, although emergency treatment should not be delayed.

It can be difficult to decide if the tachycardia is due to hypotension or the cause of hypotension. Contributory factors should be sought and corrected. Failure to do so reduces the likelihood of sustained cardioversion. The factors that contribute to tachy-arrhythmias include:

- High circulating catecholamines.
- Hypokalaemia - if K<3.6 mmol/L give K at rate of 20 mmol per hour and then check it.
- Hypomagnesaemia - assume a low magnesium if the potassium is low. Treat with 8 mmol of Magnesium Sulphate (4 ml 50%) slowly over 1-2 minutes, and repeat if necessary.

Haeodynamically unstable patients with sustained supraventricular or ventricular tachyarrhythmias should be cardioverted. The shock should be synchronised with the R wave to minimize the risk of inducing ventricular fibrillation.

Contributory factors should be corrected in all patients (for example, treat hypomagnesaemia in torsades de pointes).

Antiarrhythmic drug therapy is indicated if the patient is haemodynamically stable, or has failed cardioversion; or to facilitate rhythm stabilization after successful cardioversion or defibrillation.

Vagal stimuli will terminate about 25% of episodes of paroxysmal SVT. If the patient is conscious ask him or her to perform a vagal maneuver such as to blow the plunger up an empty 20 ml syringe.

Atrial Fibrillation:

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered. Irregular rhythms are usually atrial fibrillation. Because of the risk of thromboembolus, patients should not be cardioverted without prior anticoagulation or transoesophageal echocardiogram exclusion of atrial thrombi, unless the duration of atrial fibrillation is less than 2 days.

In patients with no adverse signs and duration of AF longer than 2 days, the immediate goal is rate control, with consideration of anti- coagulation and delayed cardioversion. Ventricular rate control in atrial fibrillation is most effective with beta-blockers, followed by calcium channel blockers, and lastly digoxin. The target rate is 60-80 at rest or 90-115 with moderate exercise.

In patients with AF of longer than 48 hours duration (or unknown duration) and requiring immediate cardioversion, concurrent anticoagulation with heparin is indicated because of atrial hypokinesia and risk of thrombus formation after cardioversion. This applies to both to synchronised DC shock and pharmacological conversion (with flecainide or amiodarone). There is a clustering of stroke risk at the time of onset of AF.

Antiarrhythmics:

Drug therapy is based on the proposed mechanism of the arrhythmia: Increased automaticity, triggered activity or re-entry in the conduction system.

Every drug that is administered unsuccessfully will add to myocardial depression and can be pro-arrhythmic (a classic example is quinidine causing torsades de pointes).

Simplified antiarrhythmic choices are given in the table below:

	Wide Complex tachycardia	Narrow Complex tachycardia	Atrial fibrillation
First Choice	Amiodarone	Adenosine for SVT	Esmolol (or metoprolol) for rate control Amiodarone and flecainide for rhythm
Second choice	Lignocaine	Amiodarone, Esmolol (or metoprolol), digoxin	Calcium channel blocker, amiodarone, digoxin for rate control

Amiodarone is effective in a broad range of supra-ventricular and ventricular tachyarrhythmias. Its predominant action is as a Class III anti-arrhythmic. It prolongs action potential duration and the refractory period of all cardiac cells by blocking the repolarising K+ current, thus inhibiting re-entry. Amiodarone also blocks sodium channels, α-receptors and calcium channels.

Vasodilatation (α-blockade, Ca++ blockade, and direct histamine release by diluents) may cause hypotension, but cardiac output is generally preserved.

In unstable patients, when VF/VT persist after three shocks, one can consider the administration of 300mg of amiodarone as a bolus and a further bolus of 150 mg may be given for recurrent or refractory VF/VT.

In stable patients, with VT or SVT, administer 300mg amiodarone over 20-60 minutes. This may be followed by an additional infusion of 900 mg over 24 hours to load the patient with amiodarone.

Lignocaine is a Class 1b antiarrhythmic. It suppresses ventricular arrhythmias by decreasing the slope of phase 4 depolarization of the cardiac action potential (thus reducing automaticity) and by reducing slope of phase 0 rapid depolarization (thus slowing conduction through ischaemic areas). It acts preferentially on ischaemic tissue and blocks fast sodium channels. At the usual concentration it has no significant effect at atrial, that is sino-atrial (SA) or atrio-ventricular (AV) node tissue.

Lignocaine causes less reduction in myocardial contractility than amiodarone. When used in conjunction with other antiarrhythmic agents lignocaine may cause a reduction in contractility and blood pressure. It is recommended for VF/VT only if amiodarone is

unavailable: one should not use both. The initial intravenous dose 1-1.5 mg per kg followed by an infusion at a rate of 15-50 mcg per kg per minute.

Magnesium. 8mmol of magnesium is recommended for refractory VF and VT if there is suspicion of hypomagnesaemia, as occurs with the use of potassium losing diuretics. It can be given for ventricular rate control in atrial fibrillation and is indicated for torsades de pointes and digoxin toxicity.

Bicarbonate is only recommended if cardiac arrest is associated with hyperkalaemia or tricyclic antidepressant poisoning the dose is 50 – 100 mmol or 1mmol/kg.

Adenosine acts via adenosine receptors on the cell surface to reduce automaticity and slow conduction at the AV node. It activates potassium channels and hyperpolarizes the cells. It inhibits adenylate cyclase and thus reduces intracellular cAMP, leading to inhibition of the inward Ca++ and pacemaker currents.

The effect is limited to the sino-atrial (SA) and AV nodes, thus causing a reduction in SA node rate and a decrease in AV node conduction, thus interrupting re-entrant pathways. It has little effect on atrial tissue, accessory pathways, and the His-Purkinje or Ventricular cells (because they lack the adenosine responsive K+ channel).

Adenosine is used primarily to terminate paroxysmal supraventricular tachycardia by blocking re-entrant pathways.

Paroxysmal SVT has different mechanisms, with 90% due to either AV nodal re-entry (60%), or AV re-entry mediated by an accessory pathway (30%). Adenosine is indicated for both, with the knowledge that in AV re-entrant tachycardia, such as Wolff-Parkinson-White syndrome, conduction across the accessory pathway may be facilitated and may precipitate a rapid ventricular response.

In non-re-entrant arrhythmias (such as flutter and atrial tachycardia) adenosine may cause transient AV block and slowing of the heart rate, allowing the atrial rhythm to be detected visually, thus enabling a diagnosis to be made. Because of transient vasodilatation and hypotension it is no longer recommended as a method to allow VT and SVT to be differentiated.

Xanthines competitively inhibit adenosine receptors, therefore one may need to increase the dose of adenosine if the patient takes caffeine or theophylline. A lower dose may be required if the patient is treated with carbamazepine.

Adenosine has a half-life of 10-15 seconds due to rapid sequestration by red cells. This is important because it means that it needs to be administered as a rapid bolus and its effects are short-lived, including side effects (headache, chest pain, flushing, and broncho-constriction).

Administration of adenosine involves an initial rapid bolus 6 mg followed by a 20 ml saline flush. After administration, a brief period of asystole up to 15 seconds duration is common. If there is no response to the adenosine in 2 minutes, then administer 12 mg of adenosine**.**

Failure to terminate a narrow complex tachycardia with adenosine or vagal maneuvers, suggests an atrial tachycardia such as atrial flutter.

*Verapamil***.** Although verapamil is very effective in narrow complex tachycardia, its use can be potentially dangerous. Like adenosine, it can increase the ventricular rate in patients with Wolff- Parkinson-White syndrome. It is not usually the first choice for most anaesthetists because it can reduce myocardial contractility in patients with depressed ventricular function, and can cause gross bradycardia in patients treated with β-blockers or inhalational anaesthetics.

Synchronised Cardioversion:
Cardioversion implies a synchronized shock as opposed to the unsynchronized shock of defibrillation. Its use is preferred over antiarrhythmics if there are serious signs or symptoms, such as a heart rate over 150, hypotension, myocardial ischaemia or failed drug therapy.

Broad complex tachycardia and atrial fibrillation require large energy shocks: Monophasic 200J or biphasic 120-150J Atrial flutter and supraventricular tachycardia require lower energy: Monophasic 100J or biphasic 70-120J

Pulseless VT is treated the same as VF (asynchronous defibrillation).

Cardioverter Defibrillators:
A defibrillator is a device that delivers a controlled electric shock to terminate a cardiac arrhythmia. This requires the passage of a sufficient current through the heart to depolarize all myocardial cells simultaneously, with the expectation that normal electrical activity will resume.

Cardioversion uses the same principle, but with the use of a synchronised shock. The shock is synchronized to the R wave of the rhythm. It cannot be used for ventricular fibrillation. Cardioversion requires less energy and 100J is the most common initial energy, except for atrial fibrillation where a larger initial shock (200J) is recommended.

A variety of automated devices are now available.

Defibrillator Features and Operation

- A capacitor that stores the current
- Control switches to allow charging and discharging by the operator
- Controls that allow the operator to select a delivered energy level (Joules)
- A choice between a synchronized or non- synchronized shock. The unsynchronized mode is usually the default setting.

Modern defibrillators deliver their energy as a biphasic waveform. They have a greater first- shock efficacy for long duration VF/VT than mono-phasic defibrillators, and do so with lower delivered energy. Biphasic energy recommendations are manufacturer-specific. This is because the required energy varies depending upon the specific waveform of discharge.

When using a defibrillator, the operator needs to optimize trans-thoracic impedance by ensuring there is good electrode contact with the chest wall, use of the appropriate sized electrodes and conductive gel, timing the delivery of the shock to coincide with the end of expiration (because air in the chest increases impedance), and avoiding placement of electrodes over bone (because bone is a poor conductor).

The electrodes are placed with the anterior electrode in the right parasternal area below the right clavicle and the apical electrode at the midaxillary line below the left nipple.

Synchronization is used to avoid the risk of inducing VF. The shock is synchronized relative to QRS, so that the shock is delivered after the relative refractory period. Many defibrillators re-set to the asynchronous mode after delivering a shock and need to be re-set to the synchronized mode before further attempts at cardioversion. If there is a delay in synchronization (for example a problem sensing the QRS complex) then use an unsynchronized shock.

The potential hazards of cardioversion include:

- Damage to the heart. Choosing the minimum effective energy can minimize this. The initial shock energy reflects a compromise between probability of success and risk of harm. The shock energy should be increased only if a shock fails to terminate the rhythm. If the defibrillation is effective but the arrhythmia recurs, then the problem is recurrence, not failure to defibrillate and so re-shock with the same energy. Address the underlying cause and add an antiarrhythmic drug. Be sure to differentiate failure to defibrillate from rapid reversion to VF.

- Electrical induction of VF may occur with asynchronous shocks.
  Insufficient or wrong gel, including metallic glyceryl trinitrate patches can cause arcing and burns or fire risk.

- Damage to implanted pacemakers or defibrillators - try and avoid defibrillation directly over implanted devices.

- Hazards to healthcare workers. Give clear warning of impending shock.
  Modern defibrillators require less than 5 seconds to charge.

Suggested energy levels for cardioversion of arrhythmias:

	Wide complex or Atrial Fibrillation	Narrow complex or atrial flutter
Biphasic	120-150 Joules	70-120 J
Monophasic	200 J	100 J

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PERIOPERATIVE ISCHAEMIA AND MYOCARDIAL INFARCTION (PMI)

Perioperative cardiac ischaemia ranges from myocardial infarction, cardiac arrests and cardiac death. This chapter will only focus on perioperative myocardial infraction.

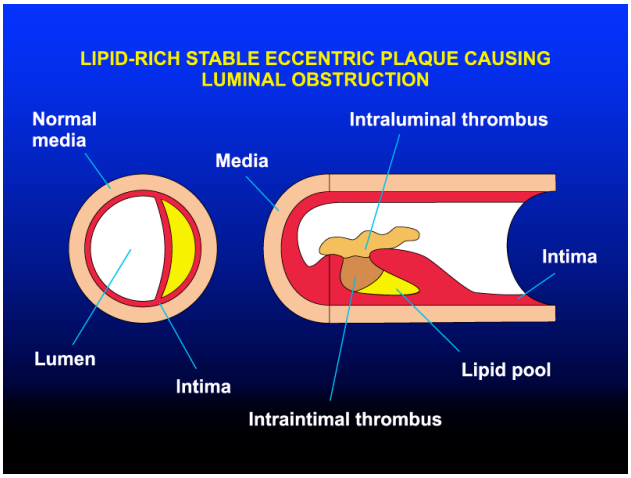
Incidence:

The actual incidence of perioperative ischaemia is difficult to determine, but ranges from 1.4% to 30%[1]. Postoperative events are five times more common than intra operative events. The hospital mortality of perioperative infarction after non-cardiac surgery ranges from 15 -25%.

Pathophysiology:

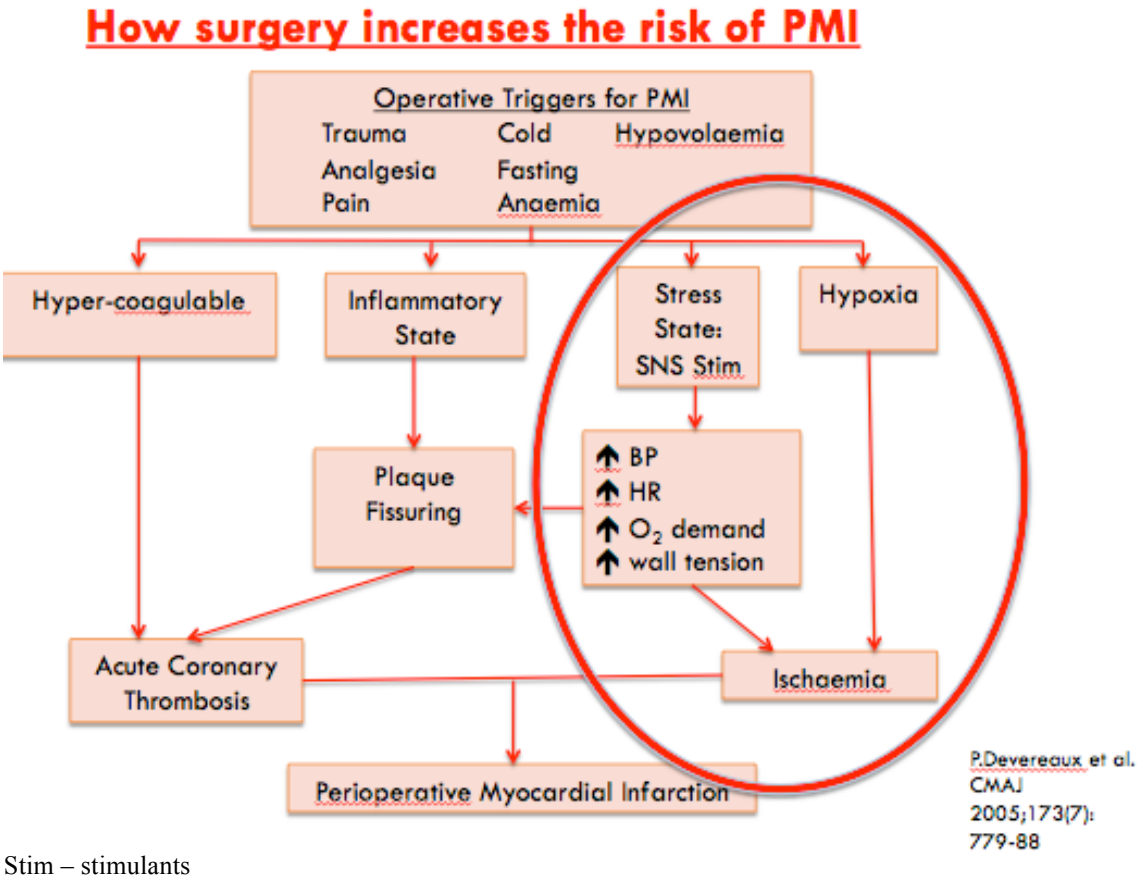
There are 2 main mechanisms for postoperative myocardial ischaemia.

1. The Acute coronary syndrome:
    a. An atherosclerotic plaque ruptures with smooth muscle constriction leading to a thrombus formation.
    b. These patients often have multiple cardiac risk factors
    c. Can be a critical or non critical lesion



2. Myocardial Oxygen supply-demand imbalance:
    a. Severe BUT stable stenosis
    b. Prolonged ST depression type ischaemia is the most common cause of PMI

(1) [1] H.Priebe BJA 2004;93:9-20



Stim – stimulants

The ischaemia tends to start at the end of surgery or on emergence from anaesthesia. This period is characterized by increased heart rate, blood pressure, sympathetic tone and pro-coagulant activity. Pro coagulant activity is related to: increased number and reactivity of platelets, increased fibrinogen, increased VIII and vWF (Von Willebrands Factor), reduced red blood cell deformability and reduced Protein C & Antithrombin 3.

Risk factors:

The risk factors for perioperative myocardial infarction include high risk surgery especially moderate to high risk vascular surgery and patients with co-morbidities such as:

- History of myocardial ischaemia (high risk is recent evidence of ischaemia in the last one month)
- Diabetes mellitus
- Renal failure
- Heart failure
- Cerebrovascular disease.

Clinical Features:

Importantly a majority of ischaemia is silent (no pain). It often begins at the end of surgery or during emergence. ECG changes can vary:

Ischaemia:

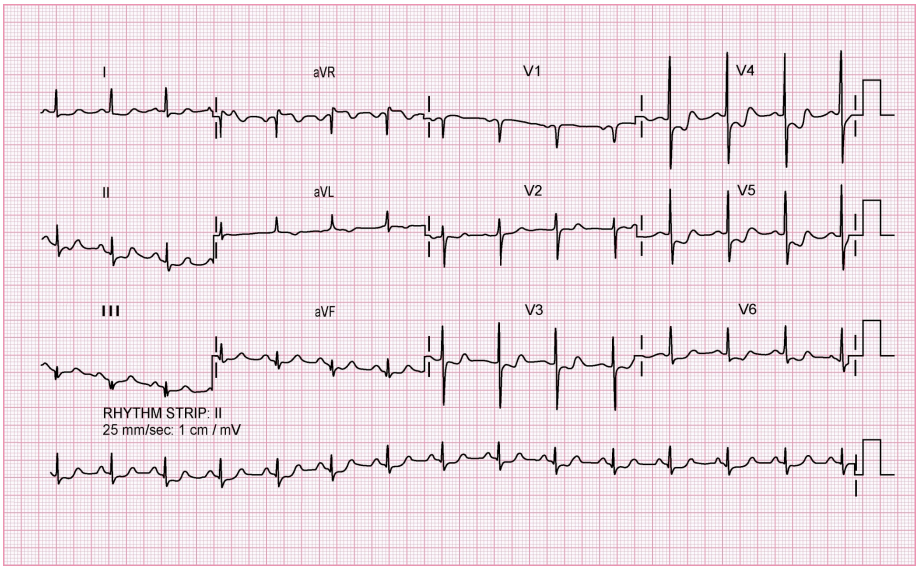
Features:

- ST depression
- Elevated cardiac enzymes
- Persistent ischaemia greater than 30 minutes, or repetitive episodes of ischaemia increases the risk of myocardial infarction

Non ST elevation MI:

Features:

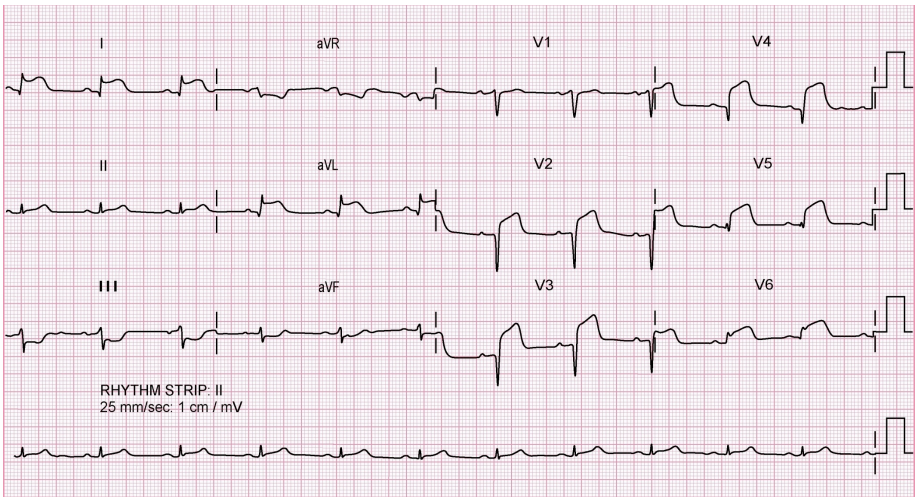
- ST depression, T wave inversion
- Elevated cardiac enzymes



ST elevation MI:

Features:

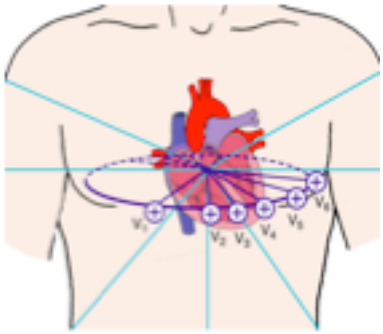
- ST elevation, T wave inversion, Q waves develop
- Elevated cardiac enzymes



Pattern for infarction on ECG:

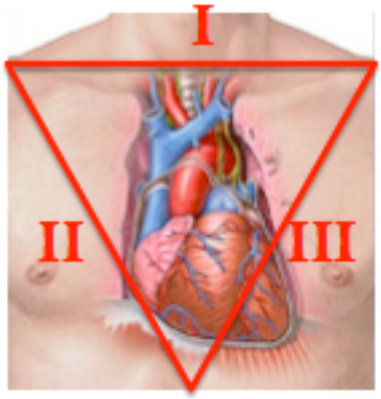
Leads:

- Inferior infarction: ST elevation in leads II, III and aVF
- Anterior infarction: ST elevation in leads V2-5
- Antero-lateral infarction: ST elevation in leads 1, aVL, V5-6



ECG monitoring for ischaemia:

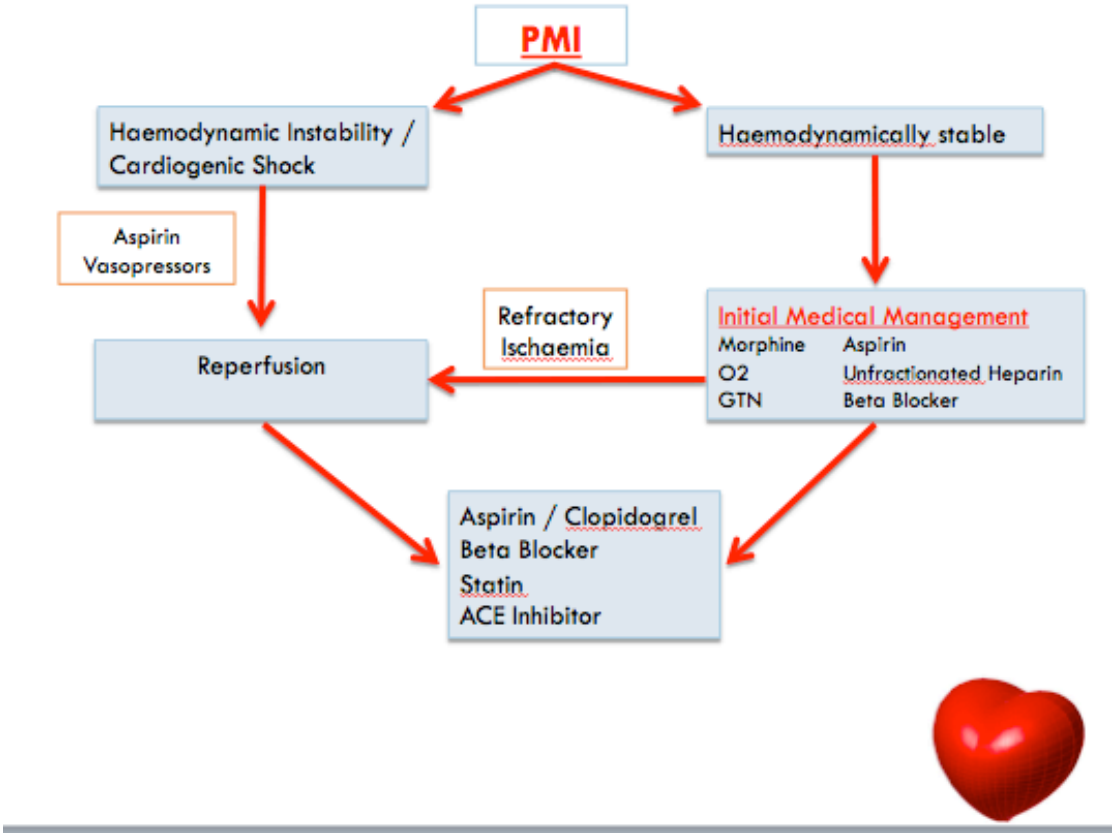
5 lead ECG monitoring is more appropriate for monitoring for cardiac ischaemia. Precordial leads V4/5 has been show to be the most sensitive for detecting cardiac ischaemia, as it represents the left ventricle.



Management:

- Early recognition – monitoring high risk patients.
- Optimising the myocardial oxygen balance:
    - Increase oxygen supply – 100% oxygen, avoid hypotension, ensure patient's haemoglobin is appropriate (7-10g/dL).
    - Decrease oxygen demand – avoid hypercapnia, tachycardia
- Ensure pain is appropriately managed
- Maintain temperature (avoid hypothermia)

- Anaglesia – Intravenous morphine titrated to patients pain and conscious state.
- Nitrates – glyceryltrinitrate (GTN) spray or sub-lingual tablets, if the blood pressure is not low
- Aspirin – 300mg



O2 – oxygen
ACE = Angiotensin Converting Enzymes

Drug Therapy Specific:

Anaglesia:

- Venodilation with reduced preload and blood pressure helps to cause a reduction in oxygen demand.
- Reduction in heart rate associated with pain
- Morphine is the drug of choice

Anti-platelets:

- Aspirin:
  - Give as soon as possible.
  - Cardiac outcomes improve with early administration.
- Clopidogrel
  - When used in combination with aspirin will cause a 50% increase in relative risk of perioperative bleeding.

Anticoagulation:

- Increased thrombin activity associated with acute myocardial infarction
- Thrombin generation occurs at site of plaque rupture
- Unfractionated Heparin
  - Anticoagulant of choice due to reversibility.
  - 60 IU/Kg [Max 4000 Units] then infusion
  - Hospital Specific protocol

Beta Blockers:

- Reduce heart rate, blood pressure and contractility
- Increase duration of diastole: period of left ventricular perfusion
- Central reduction in sympathetic nervous system.
- Anti-arrhythmic
- Aim to improve myocardial supply & demand balance. Titrate to heart rate 50-60 beats per minute but avoid hypotension

Calcium Channel Blockers:

- Act on both vascular and cardiac muscle
- Reduces heart rate and contractility, vasodilation, increases atrioventicular block.
- Beware left ventricular dysfunction and reflex tachycardia.
- May be useful when ischaemia is ongoing despite beta blocker and GTN or if beta blocker is contraindicated.
- verapamil and diltiazem have the most evidence for its use in this setting.

Nitrates:

- Reduces preload
- Modest effects on reducing afterload
- Dilation of large and collateral coronary arteries
- Titrate Dose: Aims for systolic blood pressure appropriately 110 if normotensive or 25% below starting blood pressure, if hypertensive.
- Avoid in hypotensive, bradycardia, tachycardia

Vasopressors and Iontropes:

- Aim:
  - Maintain coronary perfusion pressure
  - Inotropic support in cardiac failure
- Potential Pitfalls
  - May increase heart rate and oxygen requirements.
  - Arrhythmogenic
  - Needs central access

Fibrinolysis

- Streptokinase, tPA - Tissue plasminogen activator

Indication:

- In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and ST elevation greater than 0.1 mV in at least 2 contiguous precordial leads or at least 2 adjacent limb leads.
- In the absence of contraindications, fibrinolytic therapy should be administered to STEMI patients with symptom onset within the prior 12 hours and new or presumably new left bundle branch block.

Absolute contraindications

- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion
- Known malignant intracranial neoplasm
- Ischemic stroke within 3 months
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head trauma or facial trauma within 3 months

Relative contraindications

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (systolic blood pressure >180 mm Hg or diastolic blood pressure >110 mm Hg)
- Traumatic or prolonged (>10 min) cardiopulmonary resuscitation or major surgery less than 3 weeks
- Recent (within 2-4 weeks) internal bleeding
- Non-compressible vascular punctures
- For streptokinase - prior exposure (more than 5 days ago) or prior allergic

reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulant (e.g. warfarin sodium) that has produced an elevated international normalized ratio (INR) >1.7 or prothrombin time (PT) >15 seconds

Post Operative:

- Serial 12 lead ECGs
- Cardiac Enzymes
- Analgesia
- Monitoring
- Referral to a cardiologist

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Uptodate (website)

INTRAOPERATIVE HYPOTENSION AND HYPERTENSION

Maintenance of blood pressure intraoperatively is an important means by which to avoid cardiac, neurological and renal complications and optimise surgical outcomes. Hypotension during anaesthesia is common. It may be mild and self-limiting however sustained hypotension will cause decreased organ perfusion with irreversible ischaemic damage.

The anaesthetist should aim to maintain the patient's blood pressure to within 25% of the patient's normal resting blood pressure. That said, the absolute minimum tolerable intraoperative mean arterial pressure for a well adult patient in the supine position should be 60mmHg. In hypertensive patients this threshold is reduced, and in patients with atherosclerosis of the cerebral arteries, higher blood pressure may also be required to maintain flow. Patients with fixed cardiac output, for example with aortic stenosis, are particularly susceptible to hypotension during anaesthesia.

There are also other systemic conditions which may predispose to intraoperative hypotension. Examples include Addison's disease, hypothyroidism and carcinoid syndrome.

Hypertension is also common during anaesthesia. Once hypertension is identified and confirmed, its rapid control by the careful use of a volatile anaesthetic agent, intravenous opioids, or rapidly acting antihypertensives will usually avoid serious morbidity.

This chapter will look at the following:

- Physiology of blood pressure.
- Intraoperative factors that can affect the patient's blood pressure
- The treatment of intraoperative hypotension and hypertension

Physiology

Blood Pressure (BP) $\propto$ Cardiac Output (CO) x Total Peripheral Resistance (TPR)

BP $\propto$ CO x TPR

CO $\propto$ Heart Rate (HR) x Cardiac Contractility (CC) x Stroke Volume (SV)

SV $\propto$ Venous Return (VR) x End Diastolic Volume (EDV)

$\propto$ (is proportional to)

With these factors in mind and when confronted with intraoperative blood pressure fluctuations, the anaesthetist can assess what is causing changes in TPR, HR, CC, VR or EDV and what the treatment is to correct the blood pressure while also addressing the underlying aetiology.

Intraoperative Factors Affecting Blood Pressure and Treatment

Hypotension

Decreased Total Peripheral Resistance vasodilatation and increased vascular permeability

-Anaesthetic drugs: these dugs have a direct affect on smooth muscle tone (inhalational and intravenous anaesthetic agents)

-Regional anaesthesia: causes vasodilatation secondary to sympathetic nervous system blockade

-Sepsis: cytotoxins cause vasodilatation

-Immune mediated: mast cell degranulation in the setting of anaphylaxis causes vasodilatation and increased permeability

-Metabolic: Addisonian crisis (primary or secondary to long term steroid therapy) resembles hypovolaemic shock with loss of TPR

Altered Heart Rate Bradycardia

-Drugs including beta blockers

-High sympathetic block in the setting of regional anaesthesia

-Vasovagal effect

-peritoneal stretch at laparoscopy

-bowel dilatation at colonoscopy

-Heart block

Tachyarrhythmia

-Ventricular tachycardia/Ventricular fibrillation

-Rapid atrial fibrillation

-Supraventricular tachycardia

Decreased Cardiac Contractility

-Ischaemia

-Fluid overload

-Valvular heart disease (especially aortic stenosis)

-Sepsis

-Drugs including local anaesthetic toxicity

Decreased Venous Return

-Hypovolaemia

-Air embolism/pulmonary embolism (thrombus)/fat embolism

-Tension pneumothorax

Decreased End Diastolic Volume

-Restrictive cardiomyopathy

-Pericardial disease

Treatment of Intraoperative Hypotension

-Confirm correct measurement, is severe call for assistance and inform the surgical team and discuss possible causes

-Airway/breathing/circulation (including consideration to reversible causes of cardiac arrest). Increase inspired oxygen concentration

-Optimise preload with intravenous fluids: 10ml/kg (colloid/crystalloid/blood) and check surgery (for example, gas insufflation or caval compression) and blood loss

-Reduce anaesthetic agent, volatile/TIVA whilst avoiding awareness(depth of anaesthesia)

-Treat bradycardia (atropine)

-Administer a systemic vasoconstriction (alpha agonist or ephedrine initially and if there is no response, use adrenaline)

-Increase contractility (noradrenaline/adrenaline infusion)

-Further assessment and treatment of underlying causes as listed above. Investigation to consider: arterial blood gas, electrocardiograph, cardiac enzymes, chest x-ray.

In the event of anaphylaxis, major haemorrhage, cardiac arrest and any other emergency for which a treatment protocol exists, ensure you are familiar with local hospital or national guidelines.

Further management may therefore include:

Further intravenous fluid boluses and/or blood products
Vasopressors / inotropic support
Establishing second large bore intravenous access
Sending bloods for full blood count / coagulation / cross-match if necessary
Discussion with haematologist if major haemorrhage suspected
Drug therapy specific to the cause e.g. anaphylaxis
Invasive haemodynamic monitoring This will include frequent re-assessment and review of any corrective interventions.

Hypertension

Increased Total peripheral resistance	Vasoconstriction
	-Drugs: always consider inadvertent administration of vasoconstrictors by the anaesthetist (sometimes at above recommended dose). Local anaesthetics containing adrenaline may also cause hypertension, especially with adrenaline at high dose or if inadvertent intravenous injection takes place.
	-Pre-eclampsia
	-Metabolic: phaeochromocytoma
	-Surgical: cross clamping of the aorta for example
	(In the setting of raised intracranial pressure, intracerebral vasodilatation is exacerbated by hypercapnia and hypoxia further increasing systemic hypertension and leading to loss of auto regulation of cerebral perfusion. Secondary bradycardia then occurs)
Heart Rate	Tachycardia
	-Drugs
	-Pain
	-Awareness
	-Thyroid storm
Cardiac Contractility	Increased cardiac contractility
	-Drugs
	-Pain
	-Awareness

-Raised intracranial pressure

-Metabolic: phaeochromocytoma

Increased Venous Return

-Drugs (a reactive baroreceptor bradycardic response may be observed)

-Hypervolaemia – iatrogenic or Conn's syndrome

EDV (not applicable)

Treatment of Intraoperative Hypertension

-Consider ceasing surgery until hypertension controlled and confirm measurement

-Airway/breathing/circulation (addressing hypoxia and hypercapnia)

-Check depth of anaesthesia (inhalational or total intravenous anaesthesia)

-Provide analgesia

-Vasodilator therapy (inhalational agents/glyceryl trinitrate/ magnesium sulphate/hydralazine)

-Beta blockers if tachycardia (esmolol/labetolol)

-Alpha blockers if normal or low heart rate (phentolamine)

-Diuretics: especially if suspected raised intracranial pressure (mannitol) and ensure good superior vena caval drainage with head-up tilt and low airway pressures

-Further assessment and treatment of underlying causes as listed above. Investigation to consider: electrocardiograph, cardiac enzymes, thyroid function tests, CT brain, 24 hour urine catecholamine)

-If pregnant check for pre-eclampsia (proteinuria/liver function tests/platelets)

INTRAOPERTIVE MONITORING

Dr. Roni Krieser

All forms of anaesthesia result in changes to a patient's physiology. These can be desired pharmacological responses such as unconsciousness or paralysis, but others, such as hypotension or hypothermia may be detrimental to the patient. Even small changes in a patient's "normal physiology," may result in life threatening events due to existing pathology for example tachycardia or hypotension in a patient with aortic stenosis.

Constant **vigilance** by the anaesthetist is the most important monitoring available, however other monitors add to the physiological picture unfolding during surgery. Ideally, all patients undergoing anaesthesia should have continuous monitoring of their blood pressure, oxygenation, ventilation [Capnography] and electrocardiogram. Other monitors, should also available, for example temperature monitoring devices.

The invasiveness and choice of monitoring must take into account:

- The patho-physiology of the patient.
- The anaesthetic technique to be used.
- The type and risks of the surgery being performed.
- The availability of monitoring devices.

Understanding the basics of how a monitor works ensures accurate interpretation of the information provided. Failing to recognize a false or 'nonsense' reading or trace may result in the wrong treatment being given and harm to the patient.

This chapter will examine:

1. Oxygen Saturation Monitoring
2. Electrocardiogram
3. Capnography
4. Blood Pressure
5. Temperature

Oxygen Saturation Monitoring.

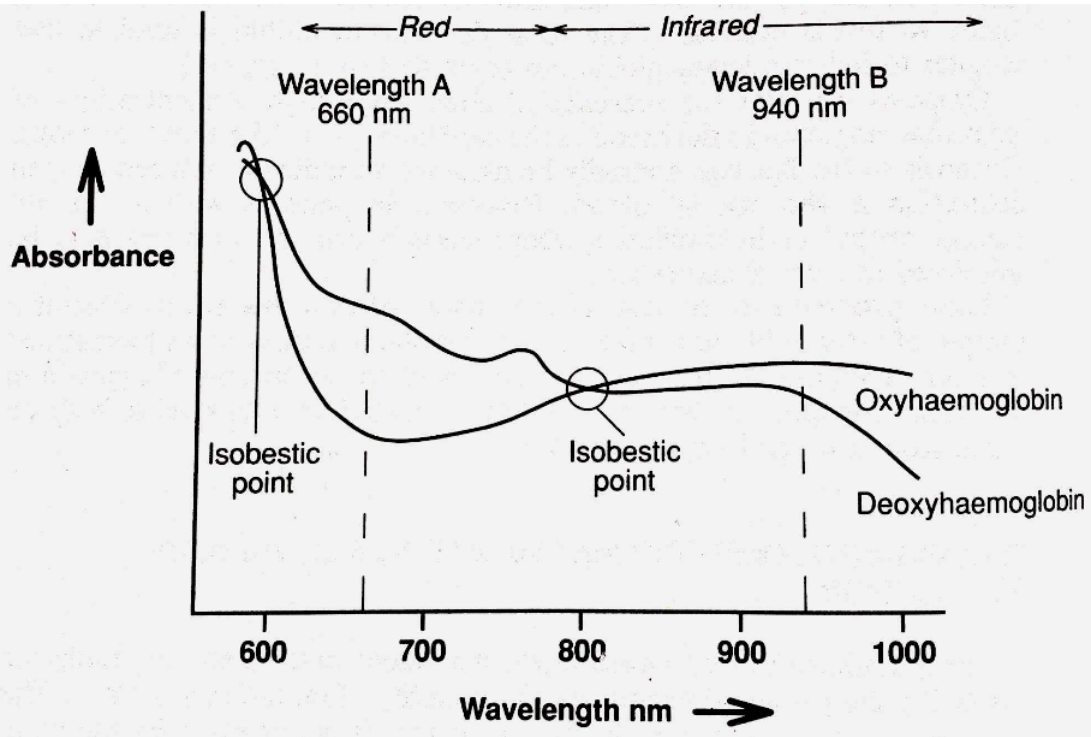
[Also called: Pulse oximeter or Sats]

Greater than 99% of all oxygen transported is bound to haemoglobin. Only a small amount of oxygen is transported dissolved in the blood.

Oxygen saturation monitors are a reusable, non-invasive method of accurately and rapidly determining what percentage of arterial haemoglobin is bound to O_2. The sensitivity and accuracy of pulse oximeters, allows early diagnosis of falling oxygenation, well before the clinical signs of cyanosis, which occurs at O_2 Sats of around 80%. The device also measures heart rate. Importantly, most oximeters also provide an auditory tone with the pitch proportional to the saturations, that is low tone= low saturations and high tone=high saturations. The auditory tone is important

and should not be turned off, as it provides an indication of oxygenation, heart rate and rhythm, even when not looking at the monitor. Pulse of oximetry is considered mandatory monitoring for all anaesthesia.

The sensor works by shining 2 wavelengths of light [infrared 940nm and red 660nm] through tissue that can be trans-illuminated. The toe, finger, ear, or nose. Oxygenated-Hb absorbs more infra-red light, whilst deoxy-Hb absorbs more red light.

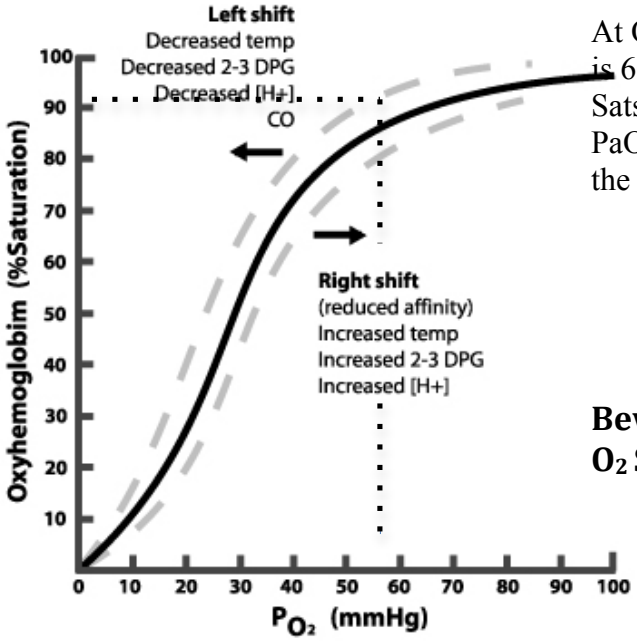


The ratio of light absorbance is analyzed to provide a reading of oxygenation. The original experiments to determine what ratio of light absorbance corresponds to what O_2 Saturation, was performed on human volunteers given increasingly hypoxic mixtures to breathe. The experiments were limited to O_2 saturations of 70%. As a result, any reading below 70% is an extrapolation, and may not be accurate. In practice, the management of a hypoxia, whether it is an O_2 sat of 70% or 50% is usually the same: GIVE THEM OXYGEN!

By examining the pulsatile light absorbance, the arterial blood oxygenation is targeted. The monitor ignores non-pulsatile light absorbance, as this is due to venous blood and tissue. The saturation reading is averaged over about 5-20 seconds, whilst the heart rate is measured by examining the time between successive pulsatile signals.

Haemoglobin-Oxygen Dissociation curve.

The interaction of Hb and O_2 can be described in a sigmoidal Hb-O_2 dissociation curve.



At O_2 Sats of ~91%, the $P_{arterial}O_2$ is 60mmHg. Any small fall in O_2 Sats will significantly drop the PaO_2 due to the steep gradient of the O_2 dissociation curve.

Beware the patient with O_2 Sats of 91-92%!

Limitations of Pulse Oximetry

1. In low output states, or where peripheral perfusion is poor, the monitor may be unable to pickup an adequate signal. This can sometimes be overcome, by placing the probe at a more central site like the ear, nose, or cheek. Most oximeters will give a visual pulsatile trace indicating the "strength" of the signal. A flat trace may mean that the reading is unreliable.

2. Although the device measures oxygenation, it does not provide information on the adequacy of ventilation. (That is, tidal volume times respiratory rate). For example, a patient who is hypoventilated with a high inspired O_2 concentration may have normal oxygen saturations, but the $P_{arterial}CO_2$ will climb to high levels, resulting in a respiratory acidosis. [Tidal volume 300ml. Respiratory rate 5, Inspired O_2 concentration 80% may have a saturation of over 98%, but have a high arterial CO2]

3. CarboxyHb and HbO2 absorb red light [660nm] in the same way. Patients with carbon monoxide poisoning will have falsely high oxygen saturation readings but by hypoxaemia.

4. Methaemoglobin has the same absorption of red and infrared light. This results in an O_2 Sat reading of 85%. Depending on the clinical situation, this reading may be falsely high or low, and is unreliable.

5. Dyes, such as Methylene blue or Indocyanine green, also absorb light affecting the O_2 saturations reading.

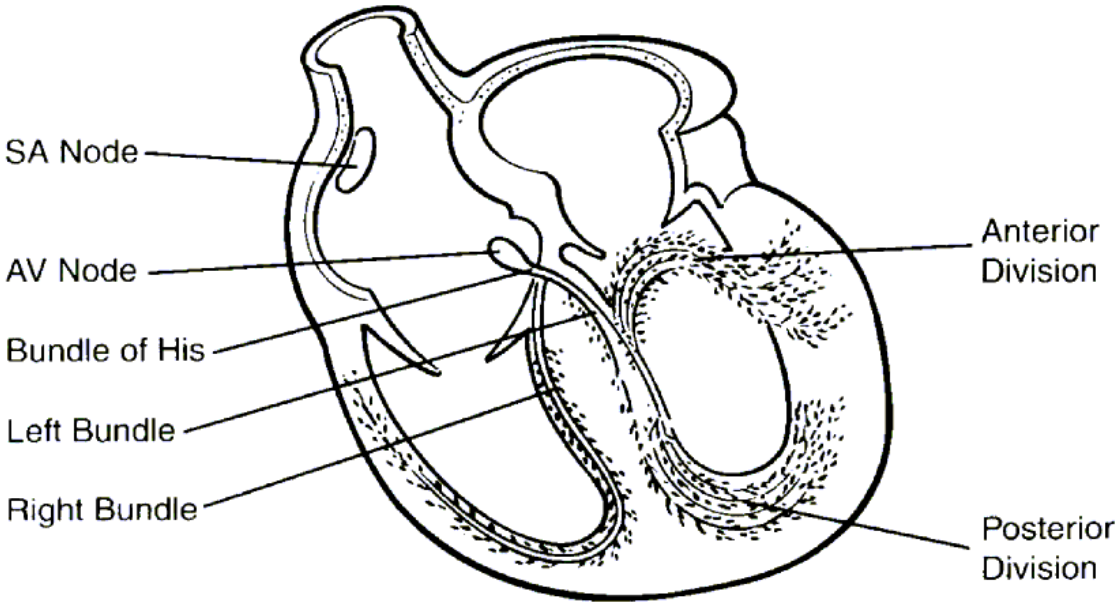
6. Excessive ambient light & movement also interfere with oximetry.

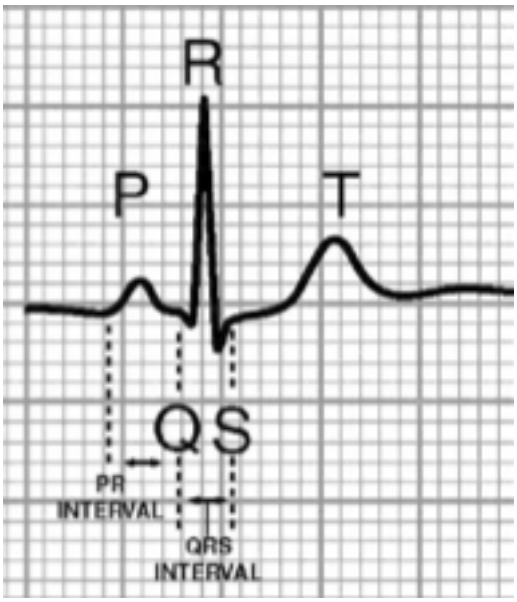
7. The presence of severe anaemia *and* hypoxia, may result in unreliable readings.

Electrocardiogram ECG

The ECG is a recording of the electrical potentials of the myocardium. It is a cheap, non-invasive monitor that provides information on:

- Heart Rate
- Heart arrhythmias
- Myocardial Ischaemia and Infarction
- Electrolyte abnormalities
- Diathermy interference with Pacemakers
- Drug toxicity -Local anaesthetic and Digoxin toxicity produce characteristic changes

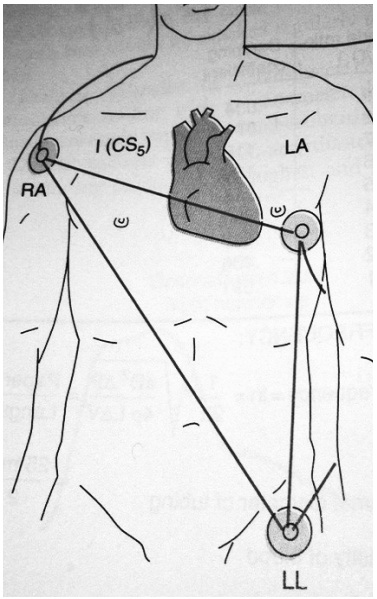




The electrical signal starts in the Right Atrium's sino-atrial node, and spreads through the atria to the atrio-ventricular node. The depolarization of the atria results in the P-wave of the ECG. The AV node slows the electrical signal, giving time for the blood to fill the ventricles. This delay is represented by the PR interval, and is normally between 0.12 - 0.2 seconds. As depolarization moves through the ventricles, the QRS complex forms. Normally the QRS last no longer that 0.12 seconds. A widened QRS represents an electrical signal moving slowly through the ventricular muscle, and may be a marker of underlying pathology. such as Bundle Branch Block. The T wave, which is normally upright, is the graphical representation of ventricular repolarisation.

The position of the ECG electrodes, and the selection of which lead to monitor is important. The simultaneous use of leads V5 and II, has been shown to have a higher sensitivity for diagnosing ischaemia than lead II alone. If only a 3 lead ECG is available, then a CS5 arrangement simulates a V5 precordial lead with a sensitivity for picking up ischaemia of 75%.

Even patients with a low risk of cardiac pathology, benefit from the regular use of an intra-operative ECG. Electrolyte abnormalities or drug toxicity (for example local anaesthetics) may be diagnosed on ECG and allow for early recognition and management of potentially life threatening conditions.



CS5: Right arm lead in the standard position and Left Arm lead placed at Apex. Monitor Lead I: good for lateral and anterior ischaemia.
Monitoring Lead II will examine the inferior part of the heart.

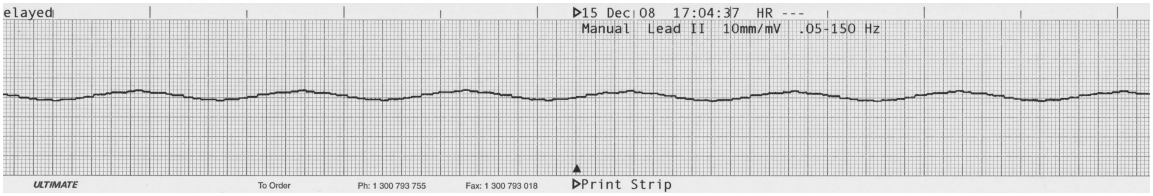
To diagnose an arrhythmia, a systematic approach to interpretation of a rhythm strip is important. The Advanced Life Support approach to interpretation of an ECG is as follows.

1. Is there any electrical activity?
2. What is the Ventricular Rate?
3. Is the QRS rhythm regular or irregular?
4. Is the QRS complex width normal or prolonged?
5. Is there atrial activity?
6. Is atrial activity related to Ventricular activity, and if so how?

The vast majority of life threatening rhythms will be rapidly diagnosed with this approach.

Here are some ECG strips to practice on

1.



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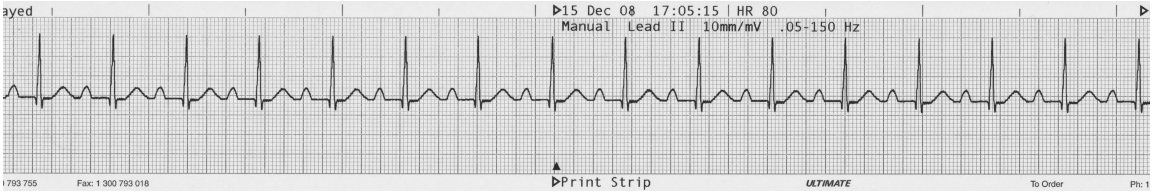

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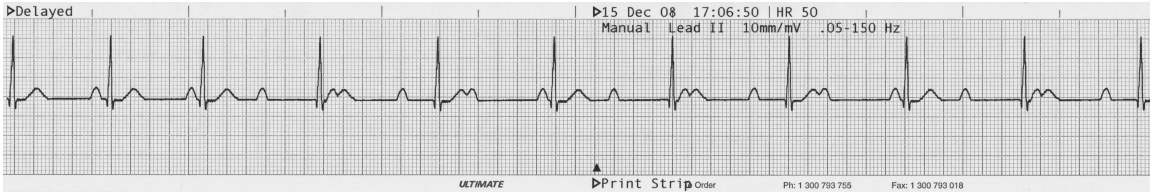

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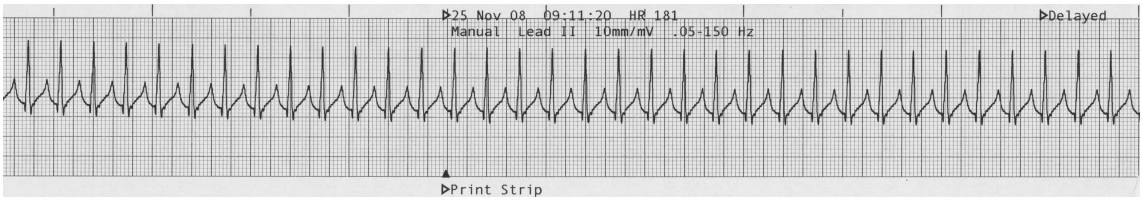

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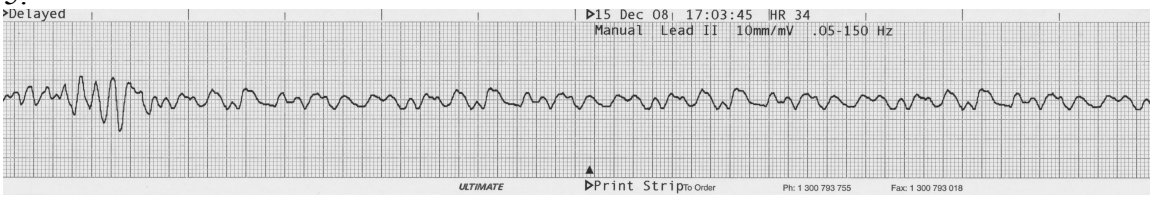

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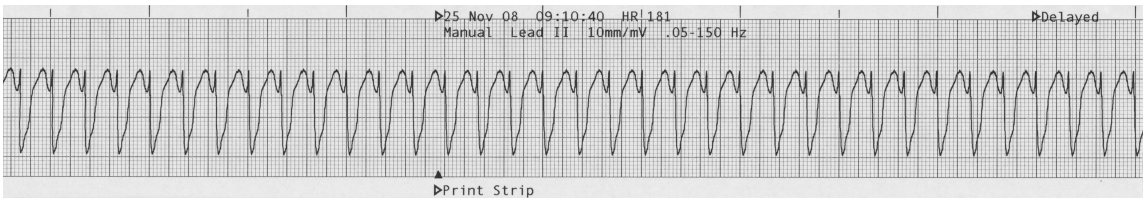

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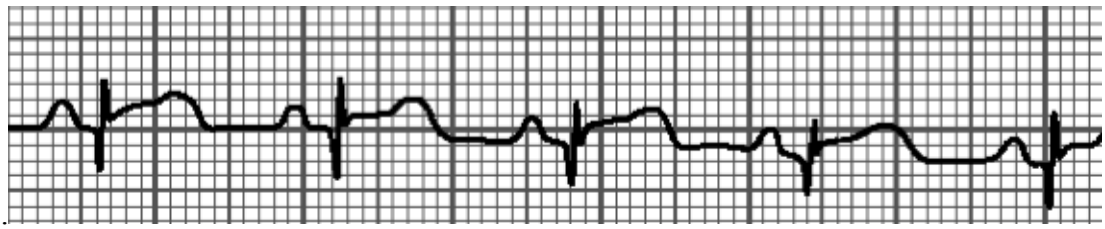

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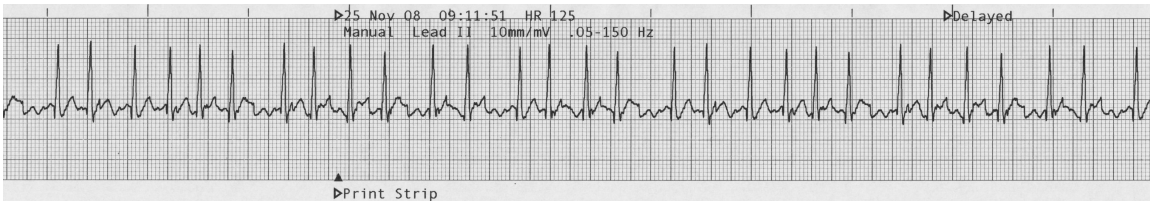


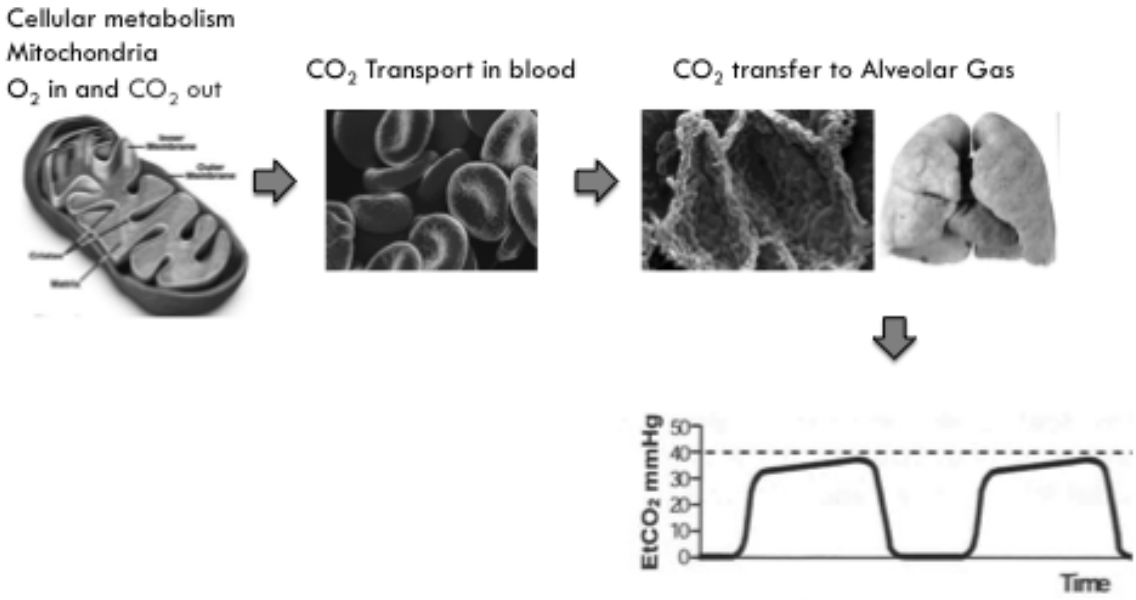
Answers: 1. Asystole 2. First Degree Heart Block 3. AV dissociation with junctional escape rhythm 4. Supra-Ventricular Tachycardia 5. Ventricular Fibrillation 6. Ventricular Tachycardia 7. ST elevation 8. Rapid Atrial Fibrillation

Capnography

Measuring the End Tidal CO_2 [$ETCO_2$] provides a sensitive monitor of the respiratory and cardiovascular system, as well as the integrity of the anaesthetic circuit.

CO_2 is produced in the mitochondria, then transported to the lungs in 3 ways:

- HCO_3^-
- Bound to proteins [mainly haemoglobin]
- Dissolved in blood.



There are 2 main types of capnography:

Side stream: A sample of the end tidal gas is aspirated via a long narrow tube into the analyser. Around 150ml/min is drawn from the circuit using this technique and represents a consistent leak from the circuit.

Main Stream: The CO_2 sensor is incorporated into the circuit and directs an infrared beam through the exhaled gas.

Uses of Capnography

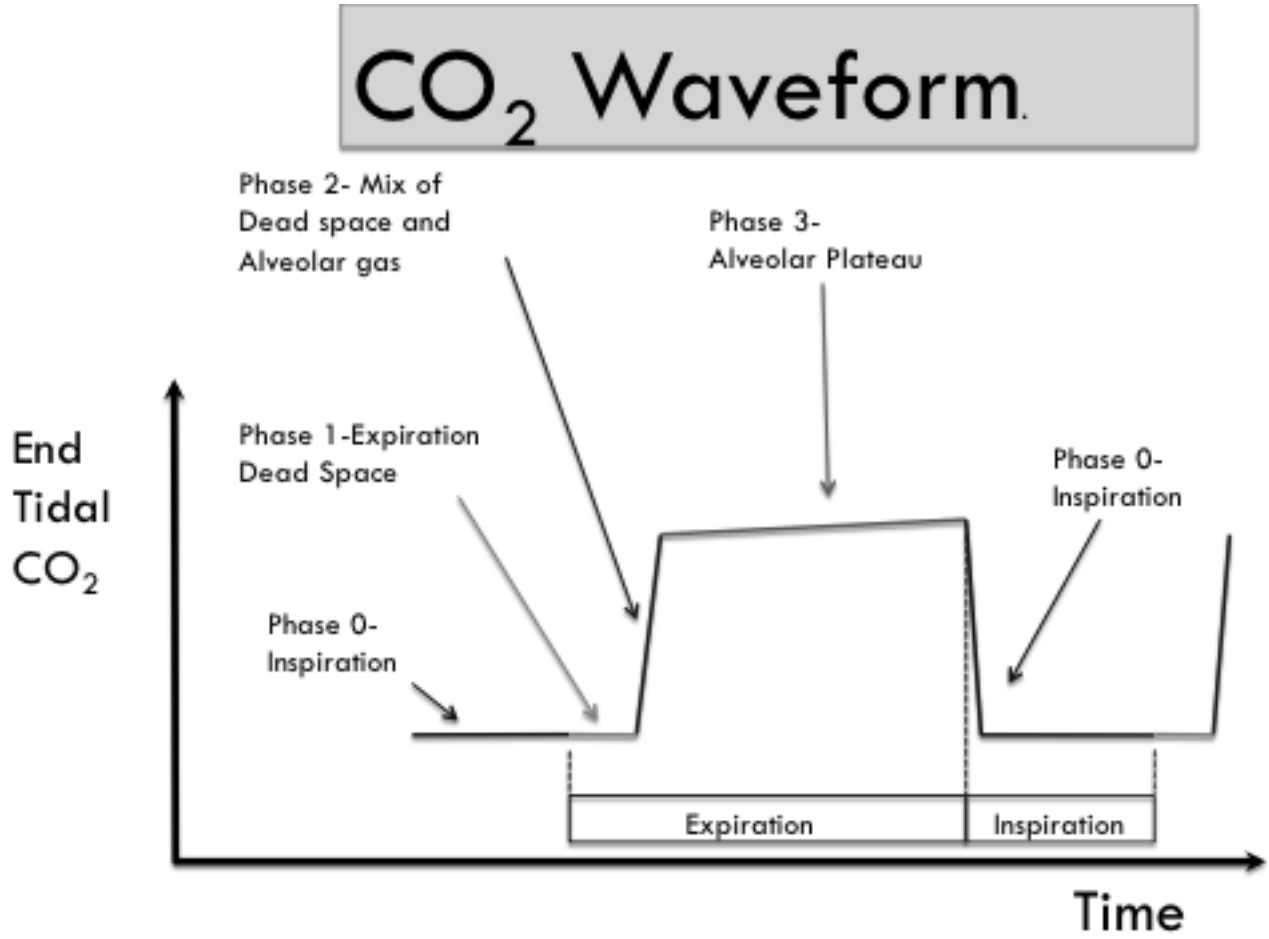
Capnography reliably detects oesophageal intubation. The rapidly diagnosis of a misplaced endotracheal tube, will assist rapid repositioning of the tube, or the introduction of another form of oxygen delivery to prevent hypoxia. Capnography is the GOLD standard for confirming placement of the tube in the airway. Capnography will not diagnose endobronchial intubation.

In addition capnography is used to determine the adequacy of ventilation and to detect circuit disconnection or ventilator failure. Circulatory collapse will result in reduced blood flow through the lungs and hence diminished delivery of CO_2 to the lungs. A sudden drop in $ETCO_2$ can be a marker of cardiac arrest or a profound decrease in cardiac output. The cause of the circulatory failure may be linked to the surgery (Air embolus in neurosurgery), or due to an anaesthetic complication like anaphylaxis [Note anaphylaxis may also cause a trace consistent with bronchospasm.] Successful resuscitation may be demonstrated by a return of $ETCO_2$.

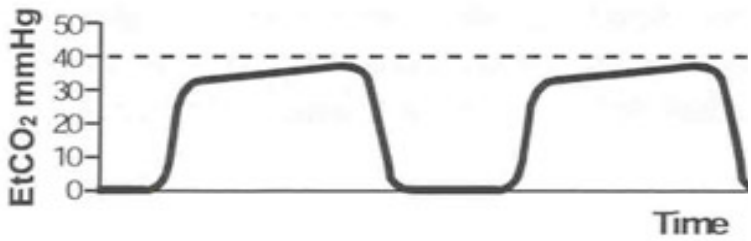
A rising $ETCO_2$, despite adequate ventilation, may be an early indicator of developing Malignant Hyperthermia.

ETCO$_2$ provides information about:

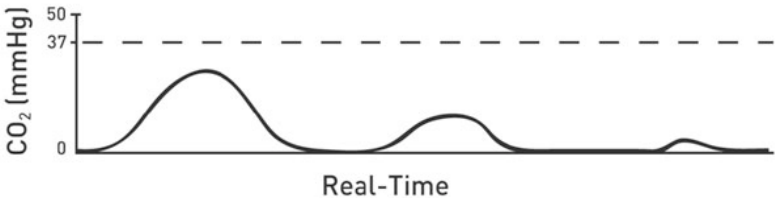
1. CO_2 production
2. Pulmonary perfusion and cardiac output
3. Alveolar ventilation and Endotracheal tube position
4. Respiratory pattern and rate
5. Ventilator and Soda Lime function
6. The presence of obstructive airways disease (Asthma, Emphysema)
7. Circuit integrity



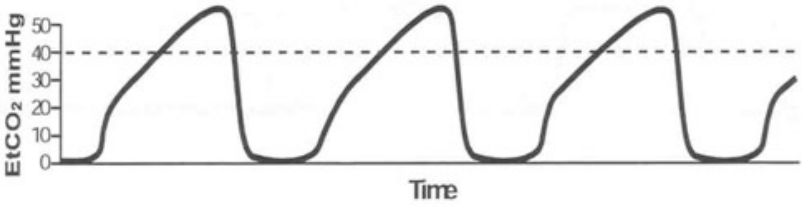
The different capnograph waveforms are all based on physiology and the physics of the circuit. Simple pattern recognition however can be useful.



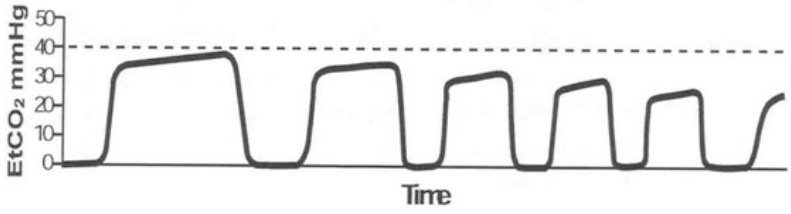
Normal Trace



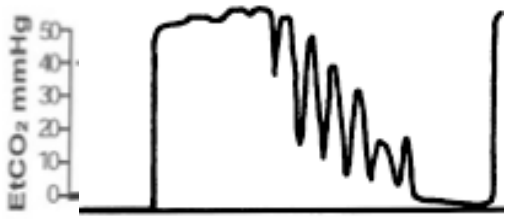
Oesophageal intubation: Note initial small, abnormally shaped rise due to gastric CO_2 content. Alternatively, an oesophageal intubation may also produce no rise $ETCO_2$.



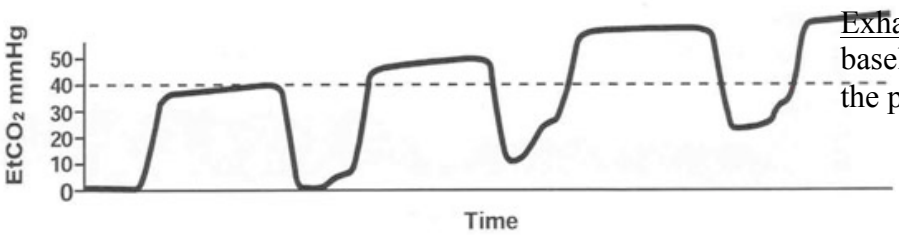
Obstructive Airways Diasease: Asthma or COAD. Slopped phase 3 that doesn't reach a plateau.



Hyperventilation: progressive decline in ET CO_2. Will result in respiratory Alkylosis



Cardiac Oscillations: notches correspond to cardiac movements pressing on the lungs and causing displacement of gas in the trachea.



Exhausted Soda Lime: Rising baseline and peak $ETCO_2$. In effect the patient is re-breathing.

Patient breathing against ventilator: dip in plateau phase 3.

Blood Pressure [BP] Monitoring

Maintaining an adequate blood pressure is required to ensure organ perfusion. Many anaesthetic techniques and pathological states can reduce BP. Untreated; hypotension can result in organ hypoxia and ischaemia.

The use of inotropes or vasopressors also demand vigilance over a patients BP. Excessive BP, increases the afterload on the left ventricle, increasing wall tension and raises myocardial oxygen requirements. At a very high BP, there is a risk of stroke and in the surgical setting, increased bleeding may occur.

Monitoring of BP can be achieved in a number of ways.

- Clinically
- Non- Invasive BP monitoring
- Invasive BP monitoring [Beyond the scope of this chapter]

Clinical observation of BP is qualitative, and should be used in conjunction with objective measurements. Clinical markers include:

- Palpation of a pulse.
- Urine output
- Capillary refill time >2 seconds suggests inadequate perfusion. Cold skin may be a marker of poor perfusion. Note: septic patients can be peripherally warm, but significantly hypotensive.
- Conscious state in patients having regional anaesthesia

Non-invasive BP measurement: Uses a sphygmomanometer, which can be manual or automatic.

Manual sphygmomanometer involves inflating a cuff to a pressure that occludes the brachial artery. The cuff pressure is then slowly reduced at ~3-4mmHg/second. Korotkoff, in 1905, first described the sounds heard over the brachial artery as the cuff pressure is released.

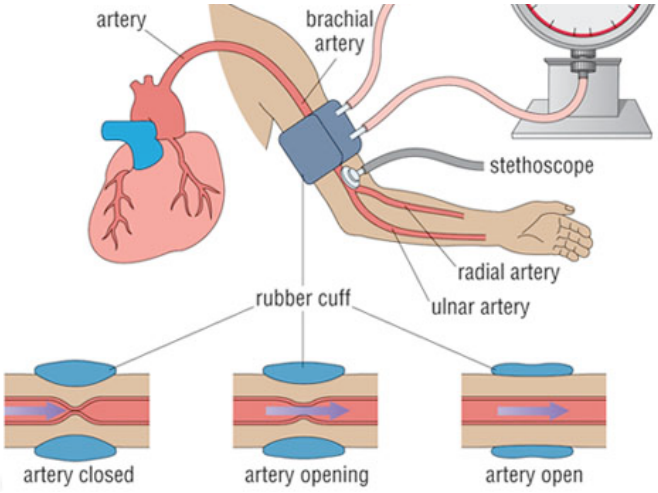
Korotkoff Sounds

Phase 1: Initial tapping sound-**Systolic pressure [SBP]**
Phase 2: Sounds quieten
Phase 3: Sounds become louder
Phase 4: Sounds suddenly become muffled
Phase 5: Sounds disappear.

The Diastolic reading can be either Phase 4 or 5. Occasionally, Phase 5 may not occur until a very low pressure & in such patients use the onset of Phase 4 for **Diastolic Pressure. [DBP]**

When an O_2 Saturation probe is used distal to the BP cuff, the return of the oximeter trace corresponds to the systolic pressure.

Mean Arterial Pressure [MAP] can be calculated using: MAP=DBP + 1/3[SBP-DBP]

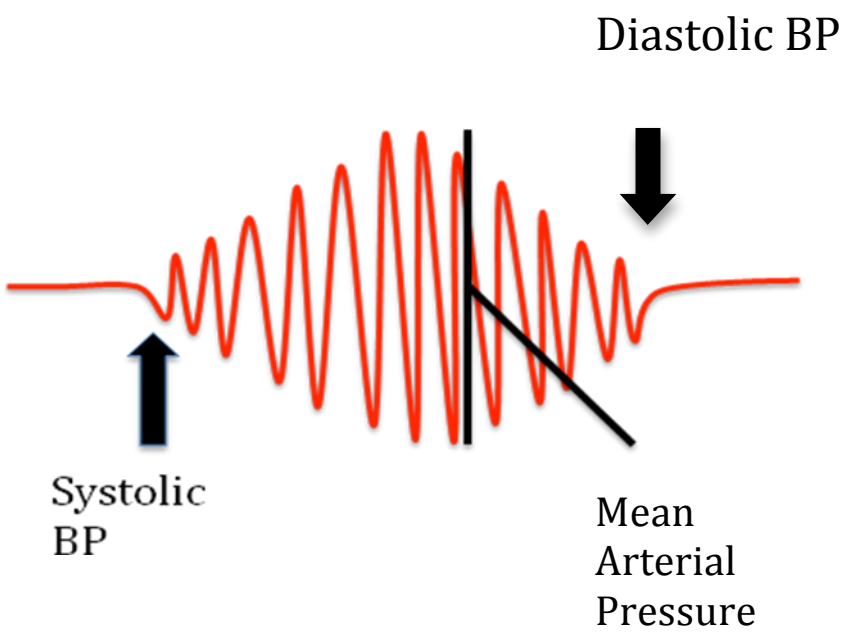


Choosing the appropriate cuff width is important to ensure an accurate reading. The cuff width must be 20% greater that the arm diameter or >40% of its circumference. A too small cuff will give falsely high readings and vice versa. Cuffs can also be placed on the lower leg, but attention to appropriate cuff size remains important.

Automated Non-invasive BP measurements [Oscillometry]

A pneumatic pump inflates the cuff to above systolic pressure. The cuff is gradually deflated, with sensors measuring tiny oscillations in the cuff pressure caused by the pulse in the underlying artery. The onset of fluctuations corresponds to the systolic blood pressure. The point of maximal fluctuations is the Mean BP, and cessation of the fluctuations is the Diastolic BP. Diastolic BP can also be derived by some devices using MAP=DBP +1/3[SBP-DBP]

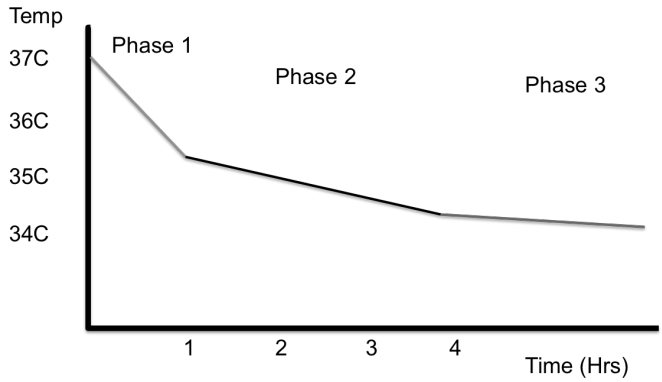
An automatic device can be set to cycle regularly, providing frequent "snapshots" of the BP. Being automatic allows the anaesthetist to do other activities. A minor complication of the automated device includes localized bruising and rarely underlying neuropraxia. At extremes of BP, the device loses accuracy. An irregular heart rate may also be a source of error.



Temperature

Temperature should be monitored during all but brief surgery. Core temperature drops by 1-2C during the first hour, due to anaesthesia induced vasodilation. This results in redistribution of core heat to the peripheries. This is Phase 1.

Phase 2, is a more gradual reduction in temperature, due to loss of heat to the environment. Finally a new plateau is reached, Phase 3.



Graph of Temperature [Celsius] vs. Duration of Anaesthesia

Hypothermia can have a number of adverse effects:

1. Reduce wound healing and increased infection
2. Left shift of the O_2 dissociation curve
3. Coagulopathy
4. Increased Peripheral Vascular Resistance.
5. Shivering: increases pain and O_2 consumption
6. Cardiac Arrhythmias
7. Reduced Drug Metabolism
8. Impaired Renal Function

Active and passive methods must be used to minimize a patient's heat loss, and prevent hypothermia.

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2. Clinical Anesthesiology 4th Ed. Morgan. Mikhail.
3. The Physiology Viva Book. Kerry Brandis.
4. Review of Medical Physiology 21st Ed. William F. Ganong.
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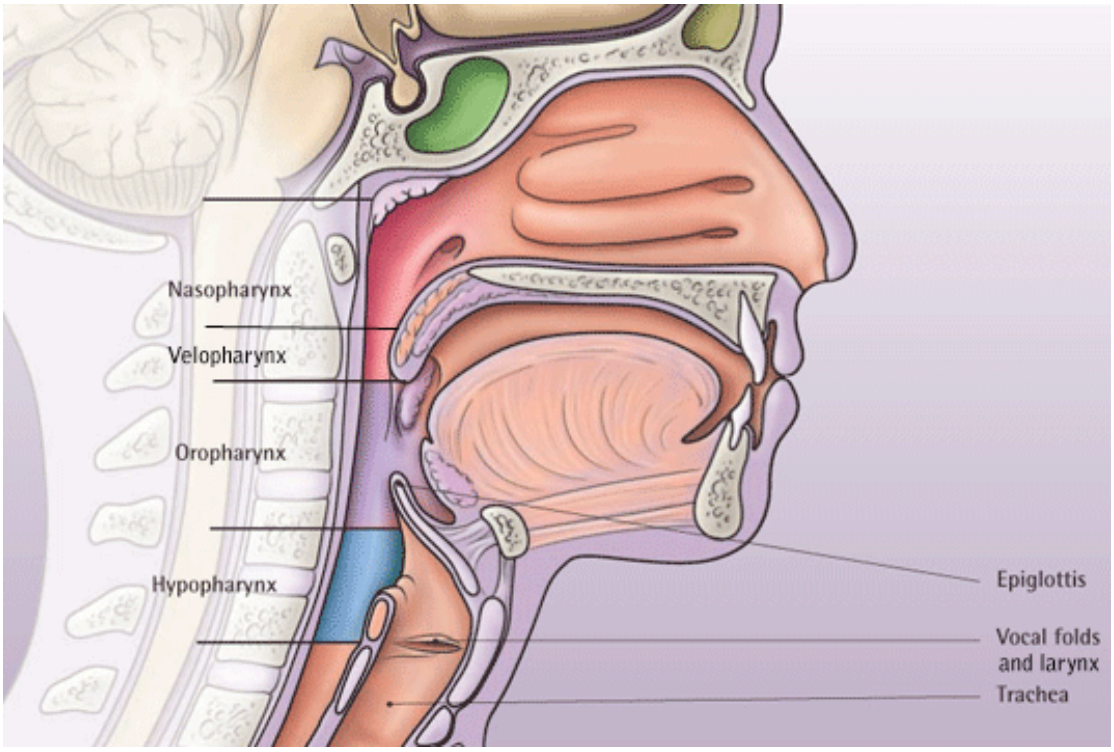
AIRWAY OBSTRUCTION

Obstruction of the airway results in the decreased ability or the inability to get oxygen to the lungs. The consequences are hypoxia, hypercapnoea, cardiac arrest and death. There are a variety of causes. It may present in dramatic fashion in the awake patient, or it may only occur after a patient is anaesthetised. Either way, it is the job of the anaesthetist to safely manage an obstructed airway.

Airway Anatomy

The airway can be viewed as a passage that starts at the lips or at the nares, and continues down past the trachea. From the nares, the airway passes via the nose into the nasopahrynx, the velopharynx, the oropharynx, into the larynx and then the trachea. From the lips, the passage passes the tongue into the oropharynx, then larynx and trachea.



Source: http://www.vocalclinic.org/larynxandvocalcords.htm

Obstruction of the airway can occur at any point along the passage – in the upper airway above the larynx (supraglottic), at the larynx (glottic), or below the larynx in the trachea (infraglottic).

Obstruction of the Normal Airway

Even in the anatomically normal airway, obstruction occurs commonly during anaesthesia. The 2 most common sites of obstruction are in the upper airway, at the velopharynx and the oropharynx.

Structurally, the upper airway consists of a framework of bone and cartilage, with attached muscle and soft tissue. The bone and cartilage provide rigidity to the airway (in the nose, and the trachea), but the soft tissues (the pharynx, the tongue) rely on muscle tone to maintain airway patency. When muscle tone is diminished during sleep or anaesthesia, the soft tissue segments become flaccid and collapsible, and hence cause airway obstruction.

The velopharynx is found just behind the soft palate and is often the narrowest and most compliant segment in the upper airway, making it very vulnerable to collapse and obstruction from loss of muscle tone. Obstruction at the oropharynx is due to retrolingual collapse - the tongue falls back against the oropharynx due to loss of muscle tone and the effect of gravity.

In patients with normal airway anatomy, these sites of obstruction can be relieved with simple airway manoeuvres (discussed later).

Pathological Airway Obstruction

Pathology in and around the airway can affect the airway by causing abnormal narrowing of the airway, increasing pressure around the airway, decreasing pressure in the airway, or making the airway more compliant and easily collapsible. These conditions all increase the risk and severity of airway obstruction, especially during anaesthesia. The worst-case scenario is airway obstruction in the awake patient.

Causes of pathological airway obstruction are listed in Table 1.

SUPRAGLOTTIC	
• Obstructive sleep apnoea	Obesity Tonsillar and adenoidal hypertrophy Nasal obstruction Craniofacial abnormalities: Down's syndrome Pierre-Robin Treacher-Collins Acromegaly Neuromuscular disorders
• Infections	Epiglottitis Parapharyngeal abscess Retropharyngeal abscess Ludwig's angina
• Tumours	Pharyngeal wall Tongue Tonsillar Mandibular
• Foreign bodies	Loose teeth, dentures Throat pack
• Neck haematoma	Post-surgical: Carotid endartercetomy Thyroidectomy Cervical Spine Post-procedure: CVC insertion
• Angioedema	Allergic Hereditary Drug induced: ACE inhibitors
• Burns • Trauma • Blood/vomit/mucous/secretions	

GLOTTIC	
• Laryngospasm • Vocal cord palsy • Tumours • Infection • Angioedema • Burns	Recurrent laryngeal nerve palsy Laryngeal Croup (laryngotracheitis) Allergic, hereditary, drug-induced
INFRAGLOTTIC	
• Compression from neck structures	Thyroid gland- goitre Haematoma Neck tumour
• Foreign body • Trauma • Tracheal stenosis • Tracheomalacia	Small objects, food, teeth
OBSTRUCTION WITH ENDOTRACHEAL TUBE IN SITU	
• Problem with anaesthetic circuit/filter/ventilator	
• Problem with tube	Tube position : oesophageal endobronchial Obstructed tube: kinked patient biting on tube cuff herniation foreign matter
• Problem with patient	Inadequate muscle relaxation Bronchospasm Chest wall rigidity from opiods Pneumothorax Pulmonary oedema Foreign body aspiration

A detailed discussion of each of these causes of airway obstruction and their specific management is beyond the scope of this chapter. Instead a brief overview of the conditions along with general principles of safe airway management will be provided.

Supraglottic Airway Obstruction:

Obstructive sleep apnoea (OSA) is a disorder characterized by sleep-induced collapse and obstruction of the pharyngeal airway, leading to hypoxaemia and hypercapnia, with airway patency only restored after arousal from sleep. The primary defect is a collapsible or anatomically small pharyngeal airway. Risk factors for OSA are therefore obesity (increased adipose and pharyngeal tissues), and in non-obese patients tonsillar and adenoidal hypertrophy, neuromuscular disorders (decreased muscle tone in airway), and craniofacial features such as macroglossia, micrognathia and maxillary hypoplasia. Certain disease syndromes can have obstructive airways – Down's syndrome (large tongue), Pierre-Robin (large tongue, small mandible), Treacher-Collin's (small mandible), acromegaly (large tongue).

Infection of the airway causes inflammation and swelling of soft tissue structures. Potentially ominous infections include bacterial epiglottitis, retropharyngeal and parapharyngeal cellulitis or abscess, and Ludwig's angina (bacterial infection of the floor of the mouth). These infections can be severe and progressive, causing changes in voice, painful swallowing, difficulty swallowing, drooling, stridor, orthopnoea, and complete airway obstruction in the awake patient. Abscesses also carry the risk of inadvertent rupture with soiling and further obstruction of the airway.

Airway tumours can arise from any site – tonsils, tongue, pharyngeal wall, mandible and larynx. The patient may not show airway compromise when awake, but once anaesthetised large tumours can compromise the airway after loss of muscle tone. They may also interfere with direct laryngoscopy, and make visualisation of the larynx difficult if not impossible. Tumours can also be friable, with risk of bleeding if traumatised.

Foreign bodies in the mouth like dentures, partial plates and tongue jewellery should be removed before/during anaesthesia to prevent the risk of dislodgement and aspiration. Likewise throat packs inserted for surgery need to be removed prior to extubation of the patient.

Significant neck haematoma may follow any procedure in the neck (such as after carotid artery surgery, thyroidectomy, cervical spine surgery, CVC insertion) especially in patients with a bleeding tendency or on anticoagulation therapy, or with high systolic blood pressure. Airway obstruction is caused by direct compression from the haematoma, as well as oedema and blood spreading along the tissue planes of the neck. This is why airway obstruction is not always completely relieved by opening the neck and evacuating the haematoma.

Angioedema is an immunologically-mediated, rapid onset of swelling in the dermal, subdermal, mucosal and submucosal tissues, especially in the head and neck. It can involve the lips, face, soft palate, tongue and larynx, and cause rapidly progressive life-threatening airway obstruction. Causes are allergic, drug-induced (associated with angiotensin converting enzyme inhibitors) and hereditary (rare).

Burns to the upper airway can result in progressive swelling and airway narrowing for up to 36 hours, with resolution in 3-5 days. Signs that may point to a significant airway burn are burns to the face and neck, singed hairs, hoarse voice, productive cough and soot in the sputum.

Glottic Airway Obstruction:

Laryngospasm occurs commonly under anaesthesia and is discussed separately later in the chapter.

Laryngeal tumours rarely produce airway compromise until advanced. However they may make the larynx difficult to visualize, and may cause obstruction after induction of anaesthesia. Another issue to consider is radiotherapy for airway tumours, which can cause tissue oedema, necrosis and fibrosis.

Vocal cord palsies from recurrent laryngeal nerve injuries can cause adduction of the vocal cords and obstruction. This can happen from tumour invasion into the nerve, as a complication of thyroid surgery due to the proximity of the nerve to the thyroid gland, or from direct trauma.

Croup (or laryngotracheitis) is a viral infection in children up to 5 years of age that causes laryngeal and tracheal oedema. Symptoms are often worse at night and include a runny nose, barking cough, stridor, accessory muscle use, cyanosis and obstruction. This condition is normally easily treated with oral steroids with no airway intervention required.

Angioedema and burns can also directly affect the larynx causing swelling and airway obstruction.

Infraglottic Airway Obstruction:

Obstruction of the trachea can occur from foreign body inhalation, usually in small children (e.g. peanuts, small toys, buttons). External neck structures can compress the trachea e.g. thyroid goitre, haematoma, neck tumour. The trachea itself may be stenosed (subglottic stenosis) or floppy (tracheomalacia) – the causes either being congenital or acquired (e.g. after prolonged intubation). Direct external trauma to the larynx and/or trachea can occur with penetrating injuries, strangulation, and blunt trauma.

Obstruction Of An Endotracheal Tube:

This is covered in detail later in the chapter.

Pre-operative Assessment Of Airway Obstruction

Pre-operative assessment with a thorough history, examination and investigations is fundamental to identification of risk factors for airway obstruction under anaesthesia.

Airway obstruction may present as an obvious clinical problem such as an inhaled foreign body, where the patient has airway obstruction when awake. Usually, however, there are no obvious signs of obstruction when the patient is awake, so one must seek out the potential for airway obstruction when anaesthetised, by history, examination and investigations. Assessment will be individualized depending on the cause of airway obstruction.

On history, ask about snoring, or apnoea during sleep. This history is sometimes best obtained from another family member! If a patient is presenting for surgery for neck or airway pathology, it is prudent to enquire about difficulties with breathing. Voice changes or a hoarse voice or stridor may indicate pathology involving the larynx. Ask for difficulties with swallowing. Tumours and masses can cause positional shortness of breath as the tumour impinges on the airway, with patients preferring to be in a particular position to breathe. If the cause is infective the features of the illness may help to differentiate the cause and site of infection. For example, to differentiate between epiglottitis and croup, epiglottitis affects children 2-6 year old, has a sudden onset, with high temperature >38 degrees. The patient looks anxious, distressed and toxaemic, swallowing is difficult with drooling, cough is muffled and guttural, and they will be in a sitting position leaning forward. Compare this with croup where the child is often younger (6 months to 3 years), onset is gradual, pyrexia is mild, cough is barking, they may have stridor, swallowing is normal and the patient is happy to lie down with no anxiety. It is important to differentiate between the two, as a patient with epiglottitis is likely to require airway intervention, whereas croup is normally treated medically.

On examination, a regular airway assessment should be undertaken. Take note of obesity, increased neck circumference, limited head and neck extension, crowded pharyngeal appearance (high mallampati score, large tongue, enlarged tonsils), limited mouth opening, small and recessed mandible, decreased thyromental distance, and presence of any loose or false teeth. Note also any syndromic appearance or craniofacial abnormalities.

Size and position of abscesses/tumours/masses should be noted. For thyroid goitres assess for size and for retrosternal extension by percussion.

For allergy note the appearance of rash, swelling of lips/tongue, itching of the palate or external auditory meatus, dyspnoea, stridor, wheezing and haemodynamic instability.

In burns note any facial and neck burns, soot in the nostrils, burns of the tongue and pharynx, stridor or hoarseness.

Other signs of severe airway obstruction include tracheal tug, intercostal indrawing, increased respiratory rate, cyanosis and decreased oxygen saturation.

Further information about the airway can be obtained from radiographic and endoscopic evaluation. CT scans allow the full calibre of the airway to be examined. Pathology (tumours, abscesses, goiters, haematomas) in or around the airway can be assessed for size and extent, and any displacement or constriction of the airway quantified. X-rays of the neck can show tracheal deviation or compression. Fibreoptic naso-endoscopy allows awake visualization of the airway, and may be useful to determine the exact location and size of a laryngeal tumour, or asses laryngeal oedema in a burns patient.

Management of anticipated airway obstruction

When likely airway obstruction has been identified pre-operatively, there are generally 3 broad options when it comes to securing the airway. These are:

1. awake surgical airway
2. awake intubation
3. asleep intubation via a spontaneous ventilation technique

To decide the best approach, one must take into account the ability of the patient to co-operate, and the pathology and its site and severity. For example, a child with epiglottitis would not be co-operative with an awake intubation or surgical airway, so the best plan under these circumstances would be an inhalational induction and securing the airway once asleep – either by intubation, or by surgical means if intubation fails.

The difficulty of securing the obstructed airway should not be underestimated, and the plan for securing the airway should be discussed with a senior colleague as well as an airway surgeon. Even if choosing intubation (either awake or asleep) as the first plan, it is still mandatory to have an airway surgeon present and immediately ready to perform a surgical airway if intubation fails.

Awake intubation requires topicalisation of the airway with local anaesthetic. This should be carried out in standard fashion. The author prefers nebulised lignocaine 5ml of 0.4%. Sedation should be avoided. Awake intubation methods include fibreoptic intubation (oral or nasal), retrograde intubation and direct laryngoscopy. Blind nasal techniques have also been used. If the airway becomes obstructed during awake intubation, it is necessary to proceed directly to a surgical airway.

For an asleep intubation, it is best to keep the patient spontaneously breathing, so an inhalational induction is preferred. No muscle relaxant should be given, as loss of muscle tone may cause complete loss of airway patency, with inability to ventilate with positive pressure. Halothane or sevoflurane should be used, starting with low concentrations and gradually increasing to maximum concentration. Induction will be slow, especially with the partially obstructed airway. Oro- or nasopharyngeal airways

can be used to maintain the airway (avoid insertion during light anaesthesia as it may precipitate laryngospasm). Laryngoscopy is attempted when the patient is deeply anaesthetised. If the airway is lost midway through gas induction, hand ventilation with maximal manoeuvres and oral and nasal airways should be employed (see section on intra-operative airway obstruction). Ventilation via a laryngeal mask airway (LMA) should be attempted, and then intubation. If unsuccessful a surgical airway will be immediately required.

The rapid sequence induction is not normally used in acute airway obstruction, due to concern about loss of muscle tone and inability to bag and mask ventilate if intubation is unsuccessful. However in some circumstances with an unco-operative patient it may be necessary to proceed with this option, with a surgeon present prepared to perform an emergency tracheostomy as the alternative plan.

Management of impending airway obstruction

If airway obstruction is impending, whilst deciding how to secure the airway, the patient should be sat up and kept calm. If available, a helium-oxygen mixture (helium 60-80%) can be used to decrease the resistance to breathing by reducing turbulent flow. This however will give a lowered inspired oxygen concentration. Nebulised adrenaline 1mg diluted in 5ml normal saline can help. Steroids can be given (especially good for croup). Continuous Positive Airway Pressure (CPAP) given with a bag and mask can help to splint the upper airway open.

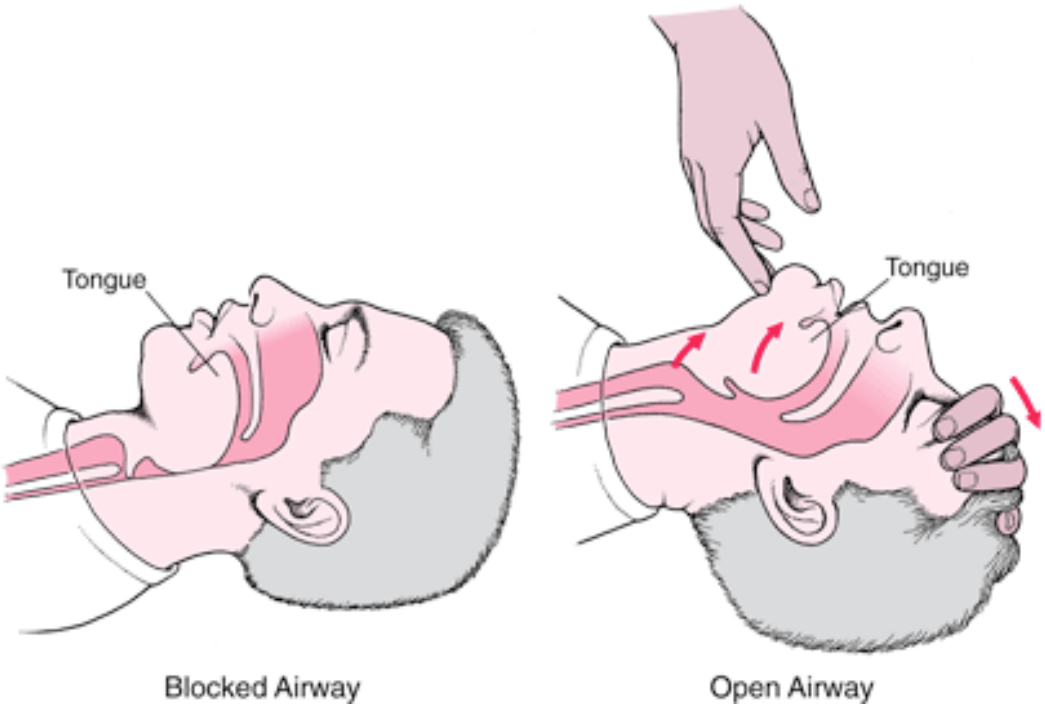
Management of intra-operative airway obstruction

Unanticipated airway obstruction can occur during any stage of anaesthesia – at induction, during surgery and during awakening.

The signs of airway obstruction occurring when a patient is asleep will depend on whether the patient is spontaneously breathing or being mechanically ventilated.

If the patient is spontaneously breathing, a partial airway obstruction can cause increased respiratory efforts, tracheal tug, snoring (if obstruction is supraglottic), or stridor (if obstruction is peri-laryngeal). Complete airway obstruction is silent as there is no air movement, and paradoxical chest and abdominal movements will occur as the patient tries to breathe, due to development of large negative intra-thoracic pressure swings. Airway obstruction during mechanical ventilation will present as poor or complete inability to ventilate. There will be loss of the end tidal carbon dioxide (ETCO2) trace.

Once airway obstruction is identified, any stimulation to the patient should cease, and 100% oxygen should be applied via the facemask. A chin lift and jaw thrust should be applied in an attempt to bring the tongue forward clear of the oropharynx, and to tighten the soft tissue structures in the airway.



Source http://www.merckmanuals.com/home/injuries_and_poisoning/first_aid/cardiac_arrest.html

Request immediate assistance if the airway is still obstructed. The pharynx should be visualised and suctioned for secretions, blood, gastric content, or a foreign body. If laryngospasm is suspected anaesthetic depth should be deepened (see section on laryngospasm). Then an oropharyngeal and/or nasopharyngeal airway should be inserted to create a passage that bypasses the tongue, and jaw thrust and chin lift reapplied, and mask ventilation re-attempted. If this is not successful, use 2 hands to do chin lift/jaw thrust/hold facemask on, and have your assistant squeeze the bag. Reducing any cricoid pressure may also help.

These steps represent optimised bag and mask ventilation. If ventilation is not achieved by these methods, then insertion of a laryngeal mask airway (LMA) or intubation of the trachea will be required to achieve ventilation, depending on clinical circumstances. If intubation is required, one attempt should be made under direct vision. A dose of succinylcholine 1mg/kg IV prior to the intubation may be appropriate if the patient is not already paralysed. If intubation fails, an LMA should be inserted for ventilation.

If unable to ventilate, and unable to intubate, this becomes a “Can’t Intubate, Can’t Ventilate” scenario, and emergency oxygenation is required via a needle cricothyroidotomy or a surgical cricothyroidotomy. The “Can’t Intubate, Can’t Ventilate” scenario is covered in another chapter.

Management of Obstruction with Endotracheal Tube Insitu

Causes of being unable to ventilate through an endotracheal tube can be related to:

- Ventilator/Circuit/Filter - The circuit may have a leak, the filter may be blocked, or the ventilator not functioning properly.

- The Endotracheal Tube (ETT) – The ETT may be blocked by secretions or kinked. The patient may be biting on the tube, or there may be cuff herniation causing blockage at the distal end of the ETT. The ETT may also be misplaced in the oesophagus.

- Patient factors – Possible causes may include chest wall rigidity from high dose opioids or inadequate muscle relaxation, pneumothorax, bronchospasm from asthma or anaphylaxis, pulmonary oedema, and foreign body aspiration.

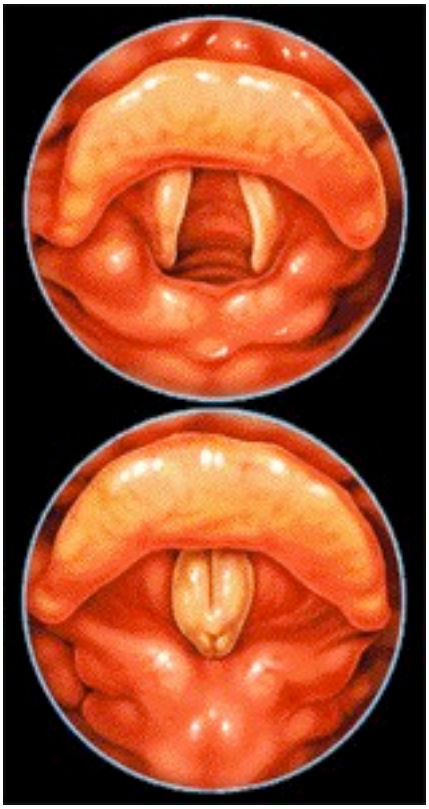
To establish what the cause is, the first step is to exclude the ventilator, circuit and filter by disconnecting the all of these from the ETT, and connecting the ETT directly to a self-inflating bag, and testing ventilation. If unable to ventilate, then the problem is at the level of the ETT or the patient.

Next the ETT is inspected for any obvious kinking or the patient biting on the tube. Tube placement through the larynx, and not into the oesophagus, should be checked with laryngoscopy. A suction catheter should be passed down the tube to identify any blockages. The cuff should be deflated to exclude cuff herniation over the end of the tube. The ETT should be replaced if there are any concerns.

If the ETT seems fine then patient factors must be considered. Check that adequate muscle relaxant has been given. Auscultate the chest for wheeze or crepitations or absence of air entry, look for tracheal deviation suggesting pneumothorax, look for signs of anaphylaxis including rash, hypotension and tachycardia. If an obstruction distal to ETT is suspected such as an inhaled foreign body, a small ETT could be pushed past the obstruction, or an attempt can be made to push the obstruction down one bronchus and ventilate the other lung.

Management of Laryngospasm

Laryngospasm is the sudden acute spasm or closure of the vocal cords, which blocks the passage of air to and from the lungs.



Top: Normal laryneal opening

Bottom: Laryngospasm

Source: http://anestesiologia.fullblog.com.ar/laringoespasmo-811220930995.html

It can occur at any point during anaesthesia, unless an endotracheal tube is insitu splinting the vocal cords open.

Precipitating factors include:

- blood or secretions in the airway
- regurgitation and aspiration
- excessive surgical stimulation
- "light" anaesthesia
- irritant volatile anaesthetic agents
- airway stimulation or extubation during the excitation phase of anaesthesia
- highly irritable airway (as with a recent upper respiratory tract infection or smoking)

A partial laryngospasm will present as an inspiratory stridor with increased inspiratory efforts and a tracheal tug. If laryngospasm is complete, the patient will have airway obstruction. If spontaneously ventilating there will be paradoxical chest/abdominal movements as they try to breathe against a closed glottis. If mechanically ventilated there will be an inability to ventilate. Desaturation, bradycardia (especially children) and central cyanosis can follow, depending on the delay in breaking the laryngospasm. Negative pressure pulmonary oedema can also occur from breathing against a closed glottis.

When laryngospasm occurs, any stimulation to the patient needs to cease (such as airway manipulation, surgical stimulation). 100% oxygen should be given, and a chin lift/jaw thrust should be applied. The cricothyroid muscle is the only tensor of the vocal cords, and gentle stretching of this muscle by applying jaw thrust and pressure on the angle of the mandible may overcome moderate laryngospasm. Immediate assistance should be requested if laryngospasm persists. The airway should be

visualized and any secretions/blood/vomit suctioned away. Continuous positive airway pressure (CPAP) should be applied via a face mask and can sometimes break the spasm, especially in children. Often increasing the depth of anaesthesia will break the spasm - an intravenous agent such as propofol should be given. If the laryngospasm is persistent, give succinylcholine (unless contraindicated) – 0.5mg/kg IV up to 50mg for an adult to break the spasm, or 1 mg/kg IV if intubation is required (if you suspect the cause is regurgitation, or if the patient is hypoxic requiring mechanical ventilation). If the patient has no intravenous access for example a paediatric patient on induction, then succinylcholine 4mg/kg IM can be given. Mask intermittent positive pressure ventilation (IPPV0 should now be possible, and the patient should be ventilated until muscle function returns and oxygenation improves.

After management of the acute event, the patient should be reviewed to exclude any pulmonary aspiration, as well as post-obstructive pulmonary oedema.

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CAN'T INTUBATE, CAN'T OXYGENATE, SURGICAL AIRWAY

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In every clinical situation in which airway management is required, the prime objective is to prevent hypoxia. When faced with a patient who cannot be ventilated by mask or via a laryngeal mask and who cannot be intubated, the priority is to get oxygen to the brain. Ventilation, with carbon dioxide removal, and securing a cuffed airway, are secondary. Thus, in the "can't intubate" scenario, oxygenation rather than ventilation is the key to preventing death or brain damage from hypoxia. It is important that every anaesthetist has a plan for the situation in which the airway is occluded and intubation fails.

Approximately 5% of patients are difficult to ventilate and 2% are difficult to intubate. This rises to 5% in the obstetric population. The situation of being unable to intubate and ventilate (or oxygenate) occurs in about 1 in 10,000 cases. It is rare but it must be considered particularly if there is a concern that airway management may be difficult.

Causes of airway obstruction

There are many causes of airway obstruction. Often they are obvious with the preoperative assessment. Causes include:

- Oedema- secondary to infection, post surgery, allergy/angioedema, pre-eclampsia, burns
- Intra-oral masses – tumour, abscess, haematoma
- Trauma to the head, neck or chest
- Foreign body
- Obesity
- Deformity, scars, radiation

Any patient presenting for surgery with any of the above features must be considered at risk for difficulty with ventilation. Other patients may prove unexpectedly difficult to ventilate and intubate. Anaesthetists must always be prepared to secure percutaneous emergency airway access in the event of a "can't intubate, can't oxygenate" (CICO) scenario.

This means that every anaesthetic department must have:

- CICO equipment

  This equipment must be rapidly accessible, dedicated to the CICO situation, easy to assemble.

- Staff trained in the CICO drill

  The decision to perform an emergency percutaneous airway must be performed without delay and then the procedure executed without delay. The anaesthetist performing the procedure needs to be trained and maintain his or her skills. Skills can only be maintained through regular practice. He or she will be reliant on other staff to provide practical assistance obtaining and handling equipment.

- A CICO algorithm

  A number of algorithms have been described. These are constantly being modified based on experience and audits such as the National Audit Project, Major Complications of Airway Management in the UK (NAP4). It is important that an anaesthetic department commits to an algorithm that is simple to follow, is linked to the available equipment and includes airway access as well as subsequent oxygenation.

Prior to any general anaesthetic it is essential to always have a complete airway management strategy. This means that there is a plan if the first attempt at securing the airway fails and a further strategy if that fails also. It is equally important for the anaesthetic assistant to understand the proposed plan.

Recognition of a CICO situation

Except in the situation of a Rapid Sequence Induction (RSI) the patient is ventilated prior to endotracheal tube (ETT) insertion. If ventilation proves to be difficult several methods may be employed including placement of an oropharyngeal airway and two handed mask ventilation with the assistant squeezing the bag. If ventilation is not possible, laryngoscopy should be performed. Intubation may then be attempted with a number of devices. If this fails, oxygenation should be attempted with a laryngeal mask airway. There should be no more than 3 attempts at intubation as there is a significant risk of airway trauma. If the patient has not been able to ventilated the oxygen saturation will be falling.

Thus there should be recognition of a CICO situation if:

- There have been 2 or more attempts at intubation
  AND
- There is a failure to oxygenate with a supraglottic device
  AND
- Oxygen saturation is persistently below 85%

Importantly, as the anaesthetist is so focused on trying to secure the airway another member of the team may be in a position to recognize that a CICO event is evolving and time is passing. The department should foster a culture in which any member of the team, nursing or medical, can ask, "is this a CICO situation?" If the answer is YES and the patient is not starting to wake up then there must be rapid implementation of the CICO plan as cerebral hypoxic damage is time dependent.

If the patient is starting to wake up and suxamethonium has been used and the oxygen saturations are not profoundly low, it may be appropriate to maintain positive pressure by face mask and wait for spontaneous ventilation. Similarly if sugammadex is available to reverse rocuronium it may be appropriate to administer this and attempt to awaken the patient. However, existing airway pathology may mean that even return of muscle tone and spontaneous ventilation may not result in oxygenation because the airway is obstructed.

Conversely, in the anticipated difficult airway some anaesthetists choose to avoid muscle relaxants until the airway is secured with an ETT. Most commonly, an inhalation induction with volatile agents is used. If the airway then becomes obstructed prior to intubation and immediate wake up is not possible a muscle relaxant should be given to exclude the possibility that obstruction is at the level of the vocal cords before proceeding to a surgical airway.

Cricothyroidotomy versus tracheotomy

Anaesthetists are taught to access the airway in an emergency situation through the cricothyroid membrane. This is because it is readily identifiable, generally more superficial and less vascular than the trachea. Access through the trachea is more difficult, associated with increased risk of bleeding, posterior tracheal wall perforation and lung damage.

If landmarks are difficult to identify, needle placement anywhere in the subglottic airway is acceptable. It is important not to waste precious time trying to accurately locate the cricothyroid membrane but it is critical to attempt to stabilize the trachea and stay in the midline.

Unfortunately, the very pathology that causes the airway to be obstructed may mean that the anatomy of the anterior neck is difficult. Extreme obesity, haematoma, oedema, burns and trauma may mean that the normal landmarks are obscured.

Cannula versus scalpel cricothyroidotomy

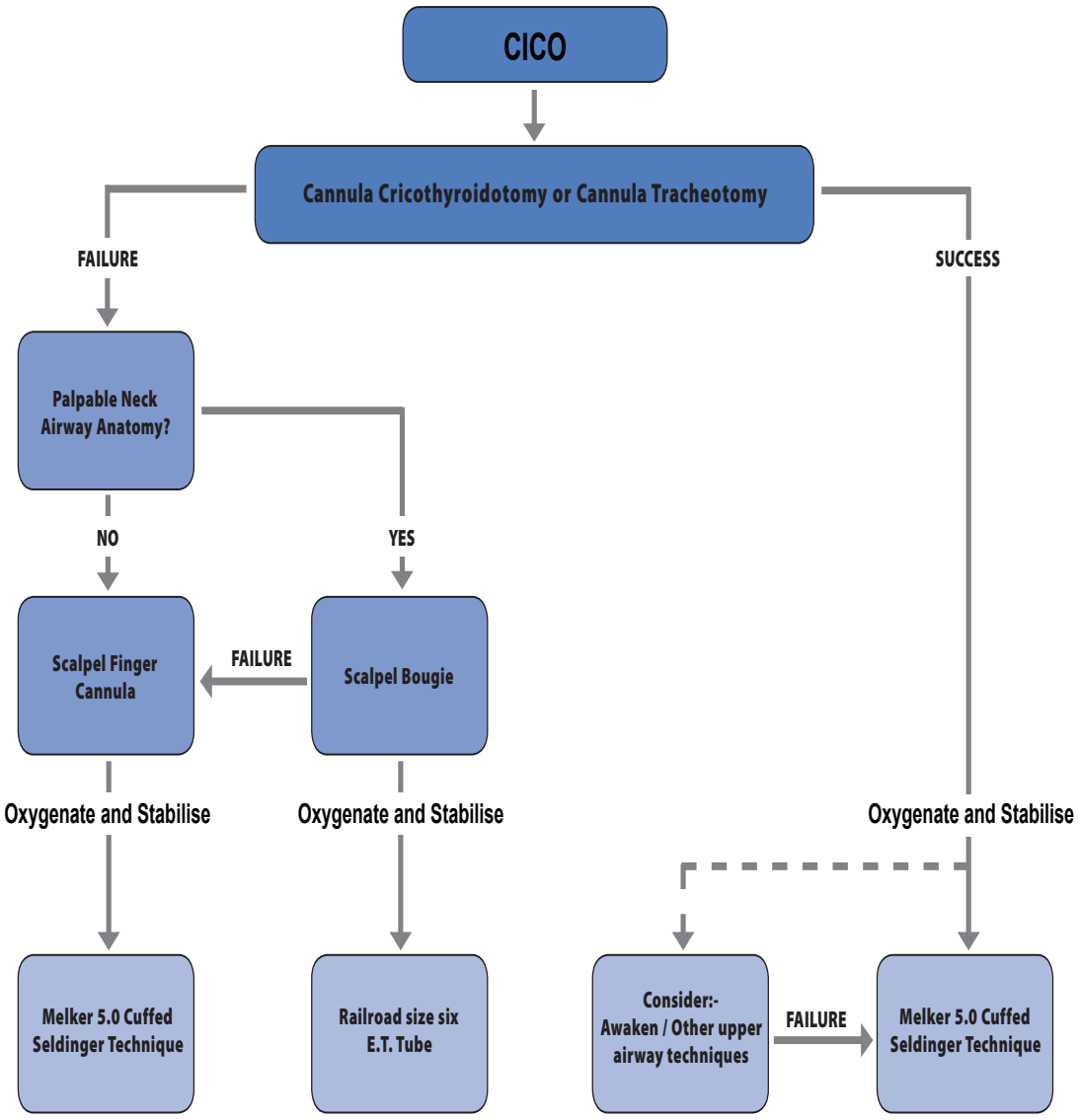
In the CICO situation rapid access to the airway is either via a cannula or using a scalpel with passage of a bougie or finger dissection down to the trachea and insertion of a cannula. It has been shown that scalpel access to the airway has a higher success rate but the skills of a surgeon with a scalpel blade are greater than those of an anaesthetist. The cannula technique is simpler and an easier psychological step to take for most anaesthetists. It has definite disadvantages but because it is considered less invasive it is generally undertaken earlier in the event than a scalpel technique. Ideally anaesthetists would be trained to perform a surgical technique as well as cannula cricothyroidotomy.

The CICO algorithm

A number of expert bodies including the Difficult Airway Society in the UK and the American Society of Anesthesiologists have produced algorithms for management of the difficult airway. In Australia an algorithm for the Can't Intubate Can't Oxygenate situation has been developed by Dr Andrew Heard and others. It is illustrated below.

CAN'T INTUBATE CAN'T VENTILATE

EMERGENCY PROTOCOL



Cannula cricothyroidotomy

Equipment

- 14 gauge intravenous cannula
- 5 ml syringe with 1ml saline
- System for oxygen delivery
  -Jet insufflator if available

  -Oxygen tubing connected to 3-way tap and flow meter

Method

- Remove the patient's pillow, extend the neck, identify cricothyroid membrane and stabilize the trachea with non-dominant hand
- Hold 5ml syringe with 2mls of water attached to a cannula in the dominant hand
- Angle the needle in a caudal direction and pass through skin at 45°
- Aspirate as the needle is advanced
- Bubbles and free flow of air into syringe indicates that the trachea has been entered
- Move the non-dominant hand to hold and stabilise the cannula hub. Do not let go at any stage.
- Immobilise the trochar with the dominant hand and slide the cannula over needle
- Remove the trochar
- Check that air can be aspirated through the cannula and release the syringe barrel to exclude a vacuum effect.
- Attach the oxygen delivery system but do not let go of the cannula

A right-handed operator should stand on the left of the patient so the needle can be inserted caudally with the dominant hand.

The main disadvantages of the cannula are that it can kink, is difficult to fixate, offers no airway protection, lacks a conduit for suction, is associated with the risk of barotrauma and requires a special attachment for jet ventilation if it is to be used for oxygen delivery. If neck anatomy is obscured, there is the risk that the airway will not be entered and vascular trauma if there is deviation from the midline.

Oxygen delivery

The simplest way to deliver oxygen is to turn the oxygen flow meter to maximum (15 l/min), attach oxygen tubing and hold firmly over cannula for a few seconds for each breath. Alternatively a three -way tap can be attached to the cannula hub and the oxygen tubing and left open in all directions. Intermittent occlusion of the open channel will direct oxygen into the patient and allow a breath.

Barotrauma is a significant risk even with these techniques.

The most effective way to ventilate is with a jet insufflator but this has the highest risk of barotrauma. The first "breath" should be for 4 seconds, the next breath should not be delivered until the saturation starts to fall and should be only 2 seconds. Whatever mode of oxygen delivery is chosen, it is critical to prevent kinking of the cannula and maintain its position securely.

Cannula cricothyroidotomy only allows for oxygenation not ventilation. Hypercarbia will occur. The risk of barotrauma is greatest if there is complete airway obstruction. Every effort should be made to open up the upper airway with jaw thrust, oropharyngeal airway or laryngeal mask insertion. An upper airway opening of greater than 4mm will dramatically reduce the chance of air trapping in the lungs.

After cannula placement an attempt should be made to wake the patient. If this is not possible or waking the patient will not resolve the airway issue, a more definitive airway is required.

The options are:

1. Attempted intubation via direct laryngoscopy – higher tracheal pressure may open a closed glottis facilitating visualization of glottis structures

2. Formal surgical tracheostomy

3. Use of a seldinger technique to secure the airway with a cuffed Melker™tube or similar

Scalpel + bougie

Equipment

- Scalpel blade – size 10 or similar
- Bougie or Frova™intubating stylette
- Size 6 endotracheal tube
- oxygen delivery system

Method

- Remove the pillow, extend the neck, identify the cricothyroid membrane and stabilize with the non- dominant hand
- Make a horizontal stab incision through the cricothyroid membrane with the dominant hand
- Rotate blade through 90° without removing it so that blade points caudally
- Pull scalpel towards you so that a triangular space is opened
- Switch hands so that the non-dominant hand stabilizes scalpel
- Hold the bougie parallel to floor at right angles to the neck and insert curved tip into hole along scalpel blade
- Rotate bougie to align with trachea, lift and pass it into trachea
- Oxygenate via bougie if it has hollow core (eg Frova)
- Railroad lubricated 6.0 ETT (15mm connector removed), rotate continuously to help passage
- Remove bougie
- Reattach connector and ventilate via circuit
- Secure tube and check bilateral ventilation

The Frova™intubating stylette has the significant advantage over a regular bougie of being more rigid and hollow with a connector that allows oxygenation by insufflation or jet ventilation.

The main complications of this technique other than failure to establish an airway, are bleeding, creation of a false passage, barotrauma and damage to other structures such as thyroid and cricoid cartilage, oespohagus, posterior tracheal wall, thyroid gland and other structures in the neck. Nevertheless, it has a high success rate in practiced hands and the potential complications should not deter someone from performing the procedure in a patient with life-threatening hypoxia.

Scalpel + finger dissection + cannula

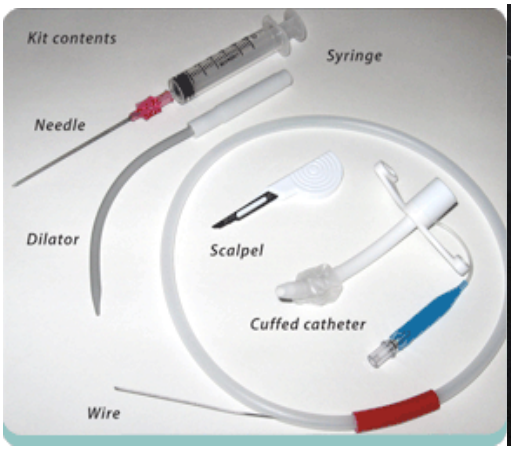
When it is difficult to identify landmarks in the neck, most often due to oedema, surgical emphysema, trauma or gross obesity it may be necessary to perform a larger vertical midline incision with a scalpel and use blunt finger dissection down to the cricothyroid membrane. This is an invasive procedure associated with bleeding and would be daunting for most anaesthetists. However the risks of trauma and bleeding are outweighed by the need to establish oxygenation. In the absence of a more skilled surgeon the alternative may be profound cerebral hypoxia and death.

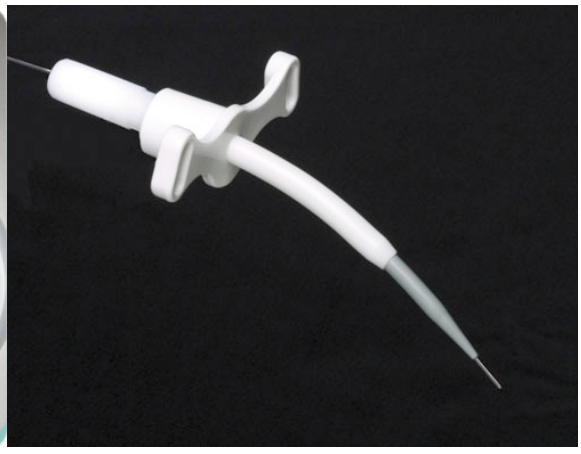
Method

- Stabilise neck in midline with non-dominant hand
- Make a vertical midline incision of at least 6 cm long in a caudal to cranial direction through skin and subcutaneous tissue
- Insert fingers of both hands to separate strap muscles by blunt dissection
- Identify airway structures with non-dominant hand and stabilize with index and middle fingers
- Insert 14G cannula with dominant hand and aspirate as you advance looking for free flow of air
- Slide off cannula, secure and attach to oxygen source

Seldinger based emergency airway techniques

A number of kits have been produced that involve passage of a wire through a needle or cannula, dilation of the tract and passage of a tracheal tube over the dilator. This can then be connected to a regular circuit. In general, time to oxygenation is increased with this technique unless oxygenation is performed after initial cannula placement. Thus these techniques are considered more appropriate as second line procedures, that is, to upgrade the airway after cannula placement.





One such kit is the Cook Melker™ cricothyrotomy kit illustrated above.

Method

- Insert wire through in situ cannula (preferably) or through needle passed into cricothyroid membrane after making a small stab incision
- Carefully withdraw cannula over wire ensuring plenty of wire is left in trachea
- Make a stab incision caudally with scalpel along wire
- Pass lubricated dilator and tube assembly over wire
- Ensure dilator is seated completely within airway
- Grip assembly device firmly preventing backward movement of dilator
- Advance assembly over wire, through skin and into airway (moderate force required)
- Remove wire and dilator
- Inflate cuff
- Ventilate via circuit or self-inflating bag

Summary

The "can't intubate, can't oxygenate" event is a crisis with potentially devastating consequences for the patient. It is traumatic for the clinicians involved, particularly if there is a poor outcome.

A good outcome is more is more likely if:

All staff members are familiar with the location and use of CICO equipment

Equipment is simple to assemble and maintain

All staff are familiar with CICO activation criteria

All staff are familiar with the steps of the chosen algorithm

The primary proceduralist is trained and mentally prepared to establish an emergency airway

The first step in any CICO event is to recognize it as such and the second step is to institute a simple, safe memorized plan that that enables oxygen delivery in the shortest possible time.

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ASPIRATION OF GASTRIC CONTENTS

Dr Richard Clarke

Aspiration of food or fluids has been recognized for many centuries as a cause of morbidity and mortality. In modern times this has also been associated with the administration of anaesthesia. Soon after the introduction of anaesthesia in 1846, reports surfaced regarding the aspiration of gastric contents as a cause of anaesthesia-related death.

Aspiration can be defined as the inhalation or ventilation of matter from the alimentary tract into the trachea and bronchial tree.

Aspiration is most likely to occur either during induction/intubation or emergence/extubation, but can occur at any time in the peri-operative period. Both fluid and solid matter from the stomach or elsewhere can be inhaled or ventilated into the lungs (using positive pressure ventilation) with potentially fatal results.

Incidence

Whilst severe cases of aspiration are rare in modern anaesthesia, the true incidence of aspiration is difficult to define, as many episodes will either not be detected or not be reported. Some studies indicate a similar incidence in adults and children (around 3 – 4 per 10,000 anaesthetics) whilst others quote up to 10 per 10,000 anaesthetics.

Regurgitation of small amounts of gastric contents without overt signs of pulmonary aspiration or respiratory embarrassment may be as frequent as 25% of all cases involving general anaesthesia.

Morbidity & Mortality

The likelihood of major morbidity or death resulting from aspiration varies widely with the volume, acidity and particulate nature of the material aspirated. Morbidity includes hypoxaemia, respiratory difficulty, pulmonary infiltrates on X-Ray, pneumonitis, pneumonia, lung abscess, Adult Respiratory Distress Syndrome (ARDS), the need for mechanical ventilation, myocardial infarction and renal failure.

Mortality from aspiration is uncommon and varies from 0 - 5% of reported episodes of aspiration. Overall, considering all causes of anaesthesia-related mortality, aspiration accounts for up to 20% of all anaesthesia-related deaths.

Physiology

Protective Airway Reflexes

Four separate upper airway reflexes that protect the lungs from aspiration have been described. These are

- Apnoea with laryngospasm – closure of both false and true vocal cords,
- Cough – forceful expiratory effort after a brief inspiration,
- Expiration - forceful expiratory effort without a preceding inspiration,
- Spasmodic panting – shallow breathing at 1 breath per second for up to 10 seconds.

These reflexes are impaired to varying degrees in unconscious patients. A normal cough reflex however doesn't correlate with the depth of unconsciousness. Two hours after recovery from general anaesthesia for day surgery, upper airway reflex sensitivity has not returned to pre-operative values. The elderly have less active airway reflexes and are at greater risk of aspiration of any pharyngeal matter.

Swallowing

Swallowing begins as a voluntary action of the tongue moving food and fluid upwards and backwards to the pharynx.

Further activity is completely involuntary, with pharyngeal receptors causing -

- The soft palate to move up to protect the nasal passage,
- The palato-pharyngeal folds form a sagittal slit in the midline allowing the passage of well-chewed food but not larger objects,
- The epiglottis covers the laryngeal opening, and
- The larynx moves up and forward to open the oesophagus.

The oesophageal phase begins with the upper oesophageal sphincter (UOS) relaxing as the superior constrictors of the pharynx contract, pushing food into the oesophagus. The peristaltic action of the oesophageal wall carries food into the stomach.

Sensory impulses travel via the 5th and 9^{th} cranial nerves to the medulla and pons, while motor impulses are carried via the 5^{th}, 9^{th}, 11^{th} and 12^{th} cranial nerves.

Stomach function

The stomach has a storage function, a mixing function (with digestive secretions) and a propulsion function. The stomach will usually hold 1 – 1.5 litres of food, but can dilate markedly, holding up to 6 litres. The different waves of stomach contractions occur every 20 seconds mixing food and moving it from fundus to antrum.

Gastric pH

Gastric pH varies greatly but is commonly between 1.5 and 3.5. Gastric acid is produced from the parietal cells in response to a number of stimuli. Gastrin is the primary hormonal stimulant. Histamine and Acetylcholine are paracrine stimulants. Inhibition of gastric acid secretion is via the hormones cholecystokinin, secretin, neurotensin and glucagon-like peptide.

Gastric emptying

Many factors influence gastric emptying, making it impossible to accurately predict when someone's stomach will be empty. Fluid passes through the stomach more quickly than solids. Any fasting guidelines must be simple and easy to understand, but also have an inbuilt margin of safety.

It has been shown that healthy children can safely drink clear fluids until 2 hours pre-operatively, and healthy adults 3 hours, without an increase in gastric volume at the time of operation. Healthy infants can take breast milk until 4 hours pre-operatively. All patients should fast for simple solid foods (such as bread) for 6 hours.

However cow's milk and formula milk as well as fruit juices containing particulate matter (such as orange juice containing pulp) behave differently. Non-breast milk separates out in the stomach to a liquid component and a solid curd. The liquid 'whey' proteins are eliminated quickly, whilst the curds are digested more slowly. Orange juice pulp behaves more like solid food and is cleared more slowly than clear liquids. It is therefore recommended that healthy patients fast from milk and juices containing particulate matter for 6 hours – the same as for a light meal (bread/toast).

In normal, calm, pain-free patients clear fluids will pass into the duodenum within 1 hour (clearance half-time 12 minutes) and simple foods within 4 – 6 hours. Basal gastric secretion rates continue at 1 ml/minute in the inter-digestive phase and 3 - 4 mls/minute in the digestive phase. So even with fasting we can never be guaranteed that the stomach will be empty.

Delayed Gastric Emptying

High caloric solids move more slowly than low caloric solids. Hyper- and hypo-osmolar foods stay in the stomach for long periods to achieve iso-osmolarity.

Fatty foods may still be in the stomach 8 or more hours after ingestion.

Gastric emptying is also delayed by pain, anxiety, many drugs including opioids, as well as labor. Diseases including diabetes mellitus, myoedema, peptic ulcer disease, inflammatory bowel disease and electrolyte disturbances will reduce gastric motility. In these conditions, a patient may have been fully fasted for 24 hours and still have

significant solid material as well as acid liquid in the stomach. This is particularly true of children suffering pain.

In all such situations these patients need to be treated as though they have a full stomach! The absence of hunger despite a long period of fasting may be a reasonable indicator that the patient's stomach should be considered to be 'full'.

Table 1. The Effect of Drugs on Lower Oesophageal Sphincter Tone

INCREASE	DECREASE	NO CHANGE
Metoclopramide	Atropine	Propranolol
Domperidone	Glycopyrrolate	Oxprenolol
Prochlorperazine	Dopamine	Cimetidine
Cyclizine	Sodium Nitroprusside	Ranitidine
Edrophonium	Ganglion Blockers	Atracurium
Neostigmine	Thiopentone	? Nitrous Oxide
Succinylcholine	Tricyclic Antidepressants	
Pancuronium	β Adrenergic Stimulants	
Metoprolol	Halothane	
α Adrenergic Stimulants	Enflurane	
Antacids	Opioids	
	? Nitrous Oxide	
	Propofol	

Regurgitation

The lower oesophageal sphincter (LOS) is the primary protection against regurgitation. This is a functional sphincter rather than a clearly defined anatomical structure. Lower oesophageal pressure is usually 20 – 30 cm H_2O whereas intragastric pressure is 5 – 10 cm H_2O. This difference in pressure is referred to as the "Barrier Pressure" and is the primary protection against regurgitation. Regurgitation is a passive process where stomach contents flow cephalad across the lower oesophageal sphincter to the oesophagus and up to the pharynx. "Silent regurgitation" is said to occur when there are no overt signs or symptoms and no stomach contents are visible in the oropharynx.

The competence of the LOS is maintained by the diaphragm, and interfered with by anatomical distortion (as with pregnancy, hiatus hernia).

Coughing and straining can create intra-gastric pressures greater than 60 cmH_2O. Reflux of gastric contents is also more likely with an obstructed breathing pattern as intra-abdominal pressure rises.

Vomiting

Vomiting is an active process that results in forceful expulsion of gastric contents. It may be initiated by impulses from either the intestinal tract or the chemoreceptor trigger zone in the medulla. Motor responses are carried in the 5^{th}, 7^{th}, 9^{th}, 10^{th} and 12^{th} cranial nerves. Many drugs, as well as rapid body motion can stimulate the chemoreceptor trigger zone, whereas unpleasant smells or tastes, disturbing thoughts or sights can stimulate cortical centres which then act on the vomiting centre.

The vomiting process starts with a deepening of breathing; the hyoid bone and larynx are raised and pull the upper oesophageal shincter (UOS) open; the glottis closes; the soft palate is lifted; the diaphragm and anterior abdominal wall muscles contract simultaneously and very forcefully to squeeze the stomach and increase intragastric pressure dramatically; the lower oesophageal sphincter then relaxes allowing gastric contents to be expelled.

PATIENTS AT INCREASED RISK OF ASPIRATION

- Patients requiring emergency surgery who are inadequately fasted, or who have gastric stasis from an acute surgical condition (for example, bowel obstruction)

- Pregnant patients are at greater risk of aspiration due to increased gastrin secretion resulting in a lower gastric pH, hormonal (progestagenic) effects that reduce muscle tone and relax the LOS (both first trimester effects), increased intra-abdominal pressure from the enlarging uterus, reduced gastric emptying both during pregnancy and labour and either physical distortion of the oesophago-gastric junction or interference with the neuromuscular control of the LOS inducing gastro oesophageal reflux disease (GORD)

- Patients with obtunded consciousness, regardless of cause, are all at increased risk of pulmonary aspiration. Possible causes include head injury, stroke, encephalopathy, epilepsy, anaesthesia, drug overdose, alcohol intoxication, cardiac arrest, diabetic ketoacidosis or hypoglycaemic coma

- Patients with oropharyngeal or gastrointestinal bleeding, hiatus hernia, Gastro-Oesophageal Reflux Disease (GORD), pyloric stenosis, scleroderma (reduces the effectiveness of the LOS) or naso-gastric tubes in place

- Patients treated with opioids for pain

- Patients with pain and/or anxiety will have significantly reduced gastric motility

- Patients with autonomic neuropathy causing delayed gastric emptying (diabetes, renal failure)

- Patients who have an inadequate depth of anaesthesia, causing coughing and gagging, and promoting regurgitation/vomiting

- Patients having particular types of surgery including laparoscopic surgery or surgery in the lithotomy, prone or Trendelenberg (head down) positions

- Patients with airway difficulties (including the obese), abnormalities, tracheostomies or trauma

- Elderly patients have less active protective laryngeal reflexes

- Patients who suffer from a variety of neuromuscular diseases including Parkinsons, Guillain-Barre and Muscular Dystrophy

Types of aspirate

Acidic liquids used in animal studies cited gastric fluid volumes of 0.3 – 0.4 ml/kg and gastric pH of < 2.5 instilled directly into the lungs of monkeys for the development of aspiration pneumonitis. This 'critical level' of pH < 2.5 is no longer considered necessary to create major pulmonary dysfunction, with aspiration of higher pH level fluids (or solids) still causing significant injury. Bile aspiration for example with a pH of 7.19 can cause a major chemical pneumonitis.

Non-acid liquids may be associated with initial hypoxaemia and significant shunting of blood. This usually returns to normal within 4 – 6 hours, and hypoxaemia corrects within 24 hours.

Particulate or solid food aspiration will cause an acute airway obstruction leading to hypoxaemia and rapid death if large airways are blocked. Prompt removal by suctioning of the upper and conducting airways (often with bronchoscopic assistance), is required.

Non-acid solids cause a rapid inflammatory response in bronchioles and lung tissue, with oedema and haemorrhages. After this initial reaction a foreign body reaction occurs with lymphocytes, macrophages and granuloma formation. The accompanying hypoxaemia is often more severe than that following acid liquid aspiration but not quite as severe as aspiration of acidic particulate matter.

Acidic solid particle aspiration is associated with the most severe tissue damage, with more extensive haemorrhage, pulmonary oedema and alveolar necrosis. Hypoxaemia, hypercarbia and acidosis are most severe, with systemic hypotension and pulmonary hypertension being common. Mortality is high and often early.

Clinical signs and symptoms

In the most severe cases, a dramatic onset may occur with the presence of gastric contents in the oropharynx. Cough, bronchospasm, hypoxaemia, cyanosis, pulmonary oedema, circulatory shock and radiographic changes quickly follow. Not all of these signs will be present, and diagnosis may be delayed until the post-operative period.

Radiographic findings may be absent, or obvious with bilateral peri-hilar and/or basal infiltrates. In moderate to severe cases the X-Ray appearance with worsen over a few days with improvement often within a week.

After an initial improvement, bacterial pneumonia, lung abscess, adult respiratory distress syndrome (ARDS) or pulmonary embolism may have occurred.

Prevention

The average length of stay following aspiration is between 21 – 28 days. Prevention is of great importance in the circumstance where general anaesthesia cannot be avoided.

- Fasting is a simple method of minimizing the risks of aspiration
- Pharmacological Gastric pH Modification
    - Antacids rapidly increase gastric pH. However particulate antacids can cause their own chemical pneumonitis. Sodium Citrate (0.3M solution) is the preferred antacid and is used extensively prior to Caesarean Section, but is effective only for 1 - 3 hours.

    - Histamine-2 Receptor Blockers (H2RBs) (such as ranitidine) have been used for many years. They are rapidly absorbed (1 – 1.5 hours) and act for at least 8 hours. (Best response if given the night before and the morning of surgery.)

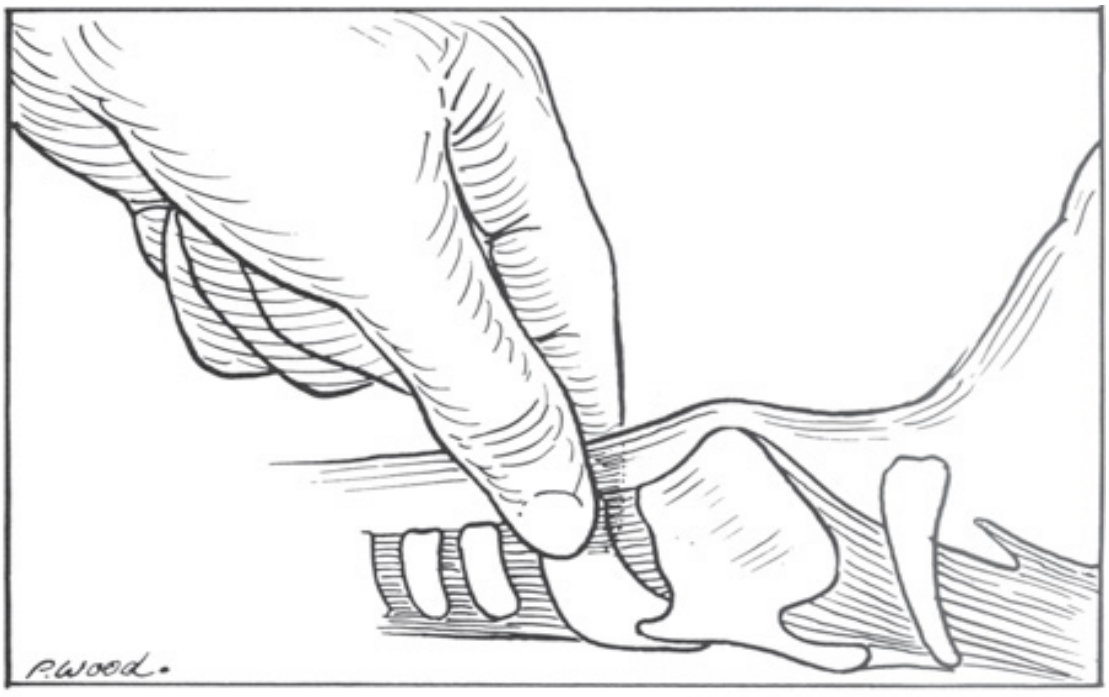
    - Proton Pump Inhibitors (PPIs) give greater acid suppression than H2 blockers, but have a slower onset of action. (Best response if given the night before and the morning of surgery.)

    - Anticholinergics (Atropine, Glycopyrrolate) can reduce gastric acidity but to a lesser extent than H2RBs or PPIs

    - Metoclopramide acts both centrally and peripherally to stimulate gastric emptying and to increase LOS pressure

Ideally either H2RBs or PPIs should be given the night before as well as the morning of surgery for the greatest effect on gastric pH.

- Airway Protection
  - Sitting or Semi-sitting position will maintain good barrier pressure due to gravity
  - Endotracheal Intubation isolates the lungs completely from the gastrointestinal tract, and can be completed if necessary in the semi reclining position.
  - Rapid Sequence Induction with Cricoid Pressure involves pre-oxygenation of the patient with 100% oxygen for 3 minutes, an induction dose of intravenous anaesthetic agent, the application of cricoid pressure and paralysis with Succinylcholine or an intubating dose of a rapidly-acting non-depolarising neuromuscular blocking drug (such as rocuronium), prior to endotracheal intubation.
  - Awake Fibreoptic Intubation should be considered if there is a likelihood of a difficult intubation
  - Cricoid Pressure (Sellicks Manoeuvre) is a simple and effective means of preventing aspiration of gastric contents. It is important that the anaesthesia assistant is experienced in the correct application of cricoid pressure. As the name implies, the pressure is applied with 2 or 3 fingers directly over the cricoid cartilage. Once applied, cricoid pressure should not be released until endotracheal intubation has been completed, the ETT cuff inflated, the seal checked to ensure that there is no leak and the anaesthetist has formally requested the assistant to release cricoid pressure.



Source: Tobin MJ: *Principles and Practice of Mechanical Ventilation, 2nd Edition*: http://www.accessanesthesiology.com

PLEASE NOTE – Cricoid Pressure is designed to prevent the passive regurgitation of gastric contents. Cricoid Pressure should be released and the patient turned into the left lateral position with 30^0 head-down tilt immediately if active vomiting occurs.

OESOPHAGEAL RUPTURE can occur if Cricoid Pressure is maintained during Vomiting.

- Nasogastric Tube Insertion with suction or drainage of as much gastric content as possible should be considered intraoperatively, thus reducing the risk of post-operative aspiration.

- Tracheal Extubation is ideally undertaken when the patient is awake, with an intact cough reflex. Studies have shown that a train-of-four ratio of 0.8 is still associated with significant reduction in UOS and pharyngeal muscle tone. Previously a ratio of 0.7 was considered safe. More recent evidence suggests that a target of 0.9 indicates a return of normal neuromuscular function.

- Patient Positioning in the lateral decubitus position prior to extubation allows any regurgitated fluid to be expelled through the mouth rather than pooling in the laryngopharynx from where it could be aspirated.

- A well-staffed Post-Anaesthesia Care Unit (PACU) with close supervision of patients emerging from anaesthesia is very important. Observation of the recovering patient for signs of regurgitation or aspiration is necessary as Laryngeal Competence may be impaired for up to 8 hrs after extubation.

Table 2. Recommendations for Fasting Times For Elective Surgery

Food or Fluid	Fasting Time
Preoperative fasting solids and non-human milk	6 hours
Preoperative fasting juices containing pulp (solid particles)	6 hours
Preoperative fasting infant formula	6 hours
Preoperative fasting breast milk	4 hours
Preoperative fasting clear fluids	2 hours
Patients must be allowed to take most of their usual medications before surgery with 30 mls of water	

Table 3. Recommendations for Pre-operative Drug Treatment

<table>
<tr><th>ASPIRATION RISK</th><th colspan="2">DRUG TREATMENT</th></tr>
<tr><td>LOW RISK</td><td colspan="2">NO SPECIFIC DRUG TREATMENT RECOMMENDED</td></tr>
<tr><td rowspan="3">INCREASED RISK</td><td>Non-Particulate Antacids (e.g. Sodium Citrate)</td><td rowspan="3">RECOMMENDED</td></tr>
<tr><td>Gastrointestinal Stimulants (e.g. Metoclopramide)</td></tr>
<tr><td>Histamine-2 Receptor Antagonists (e.g. Ranitidine)
OR
Proton Pump Inhibitors (e.g. Omeprazole)</td></tr>
</table>

Diagnosis

Despite appropriate preventive measures aspiration still occurs. Diagnosis is often difficult. History is very important and patients will commonly develop tachypnoea and tachycardia. Initially the chest X-ray may show no change, but later it may show a diffuse infiltrative pattern. Auscultation may reveal bronchospasm, crepitations and rhonchi

Testing of suctioned liquid for pH is often not helpful as pH changes rapidly. Arterial Blood Gas analysis is more helpful, with PaO_2 reduced relative to FiO_2. $PaCO_2$ and pH are less helpful except to judge severity of lesion. Patients will commonly have an increase in intrapulmonary shunting, decreased functional residual capacity (FRC) and compliance with an increased resistance to breathing.

Treatment

Regardless of cause, early and aggressive treatment favours better outcomes.

Immediate Management

- Application of cricoid pressure to limit further regurgitation and remove any supraglottic airway device.

(Note – DO NOT apply Cricoid Pressure if the patient is actively vomiting. Instead turn the patient into the lateral position with a 30^0 head-down tilt.)

- Suctioning of the pharynx with a rigid sucker
- Intubation of the trachea with a cuffed endotracheal tube
- Immediate suctioning of the trachea with a soft suction catheter before any positive pressure ventilation is started

A decision regarding cancelling or proceeding with planned surgery is required. Factors to consider include the severity of the aspiration event, the patient's respiratory status, the planned duration of surgery and whether it is an emergency operation.

Subsequent Management

- Post-operative respiratory observation is undertaken for 2 or more hours if a minor aspiration has occurred. If there are no symptoms, lung signs or hypoxaemia there is little chance of deterioration
- If there is moderate to severe aspiration or particulate matter has been aspirated, admit the patient to the intensive care unit
- Therapeutic bronchoscopy is used if there is evidence of particulate aspiration, lung or lobar collapse or foreign body on chest x-ray.
- Pulmonary lavage has not been shown to be helpful in non-particulate aspiration
- Mechanical ventilatory support is used where required. The ARDSNet trial approach, using low tidal volumes (6ml/kg ideal body weight) and limitation of plateau airway pressures to less than 30cm H_2O reduced mortality by 22% compared with the control group who received 10 – 12 ml/kg tidal volumes. Up to 24 cm H_2O positive end-expiratory pressure was used in this study if the patients were requiring 100% O_2.
- Prophylactic antibiotics are not helpful. (The exception to this is where the patient has aspirated frankly contaminated material.)
- Targeted antibiotic therapy is used if bacterial pneumonia develops on the basis of microscopy results. Otherwise broad-spectrum antibiotics should be employed only where there is good evidence of infection.
- Corticosteroids have not been shown to be effective treatment and should be avoided
- Cardiovascular and Renal support may be required in severe cases.

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CRISIS MANAGEMENT

Definition

A “crisis” in health care is “a time of great danger or trouble whose outcome decides whether possible bad consequences will follow”. (AProf David M Gaba)

A crisis requires an active response to prevent injury or harm to the patient and it is unlikely to resolve on its own.

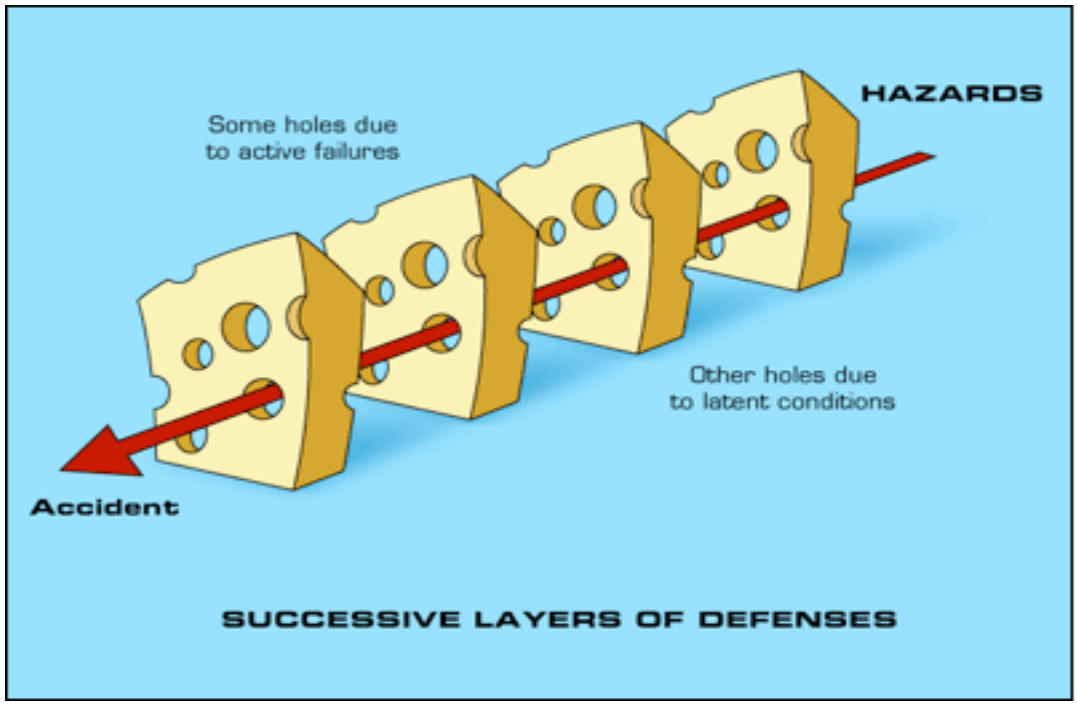
Even the most skilled anaesthetists can find themselves challenged in the operating theatre. Even for routine elective surgery in ASA I patients there is an ever-present (although small) risk of catastrophe (death, brain damage, other permanent injury). The complexity and dynamism of the environment make crises more likely to occur in fields like anaesthesia, intensive care and emergency medicine.

To manage a crisis successfully medical knowledge and skills are essential but the anaesthetist must also manage the entire situation, including the environment, the equipment and the patient care team.

How do crises arise?

In retrospect the evolution of a crisis can usually be identified from underlying triggering events.

JT Reason's “Swiss Cheese Model” shows how accidents require latent failures and active failures to bypass all layers of defense and lead to an accident.



When such accidents occur it is uncommon for any single action or 'failure' to be responsible. It is far more likely that a series of seemingly minor events all happen consecutively and/or concurrently so that one day, at that one time, all the 'holes' line up and a serious event results.

Safety Strategies

There are several successful safety strategies that can be incorporated into anaesthesia:

- Written checklists to prevent crises from occurring, for example anaesthesia machine checklist
- Established procedures for responding to crises, for example algorithms, written or memorized
- "Precompiled Responses": plans for dealing with specific types of events
- Training in crisis management, especially decision making and operating team coordination (including simulation)

Gaba's Seven Key Points for preventing and managing critical events

Humans are not very good at decision-making under pressure, so knowing these principles may help manage a crisis more effectively:

1. Know, modify and optimize your environment
   Familiarise yourself with your work environment, ensure you know how to operate the available emergency equipment and its location, introduce yourself and make sure you know who you are working with.

2. Anticipate and plan
   The best way to avoid a crisis is not to have one. Ensure you have enough information about the patient/the procedure/the equipment/the staff. The best use of resources requires advance planning; always have a Plan B and C ready. Plan for the worst-case scenario.

3. Ensure leadership and role clarity
   In most emergency situations it is best for the anaesthetist to take the role of the leader. Make sure your role is clear to the rest of the team. The leader must have good technical knowledge and must remain calm and organised, maintaining control of the situation with full participation of the team. ***Decide*** what needs to be done, ***prioritize*** the necessary tasks, ***and assign*** them to specific individuals. Leadership is aided by good "followership", getting information and feedback from other members of the team.

4. Communicate effectively
   Notify surgeons and nurses of the arising problem and tell them what you need them to do (or NOT do). Do not raise your voice unless absolutely necessary and state your requests clearly and precisely. Address a specific person, call people by their names, use eye contact and gesturing; don't say, "can someone please…" Close the communication loop: request acknowledgement of critical

communication, ensure the person addressed confirms that they understand and are capable of what you expect them to do. Create an atmosphere of open exchange; listen to what others have to say regardless of their status. Concentrate on **what** is right for the patient, not **who** is right!

5. Call for help or a second opinion early enough
   Anaesthetists have a tendency to put off calling for help, often due to denial or fear of appearing weak or incompetent or upsetting the surgeon. Declaring an emergency mobilizes needed resources quickly and communicates to the team that a crisis is at hand. Have a low threshold for asking for assistance or a second "pair of hands". An additional "brain" may see things that the initial person might have missed. Help may take some time to arrive, so call **early**!

6. Allocate attention wisely and use all available information
   As stress levels increase an individual's breadth of attention narrows (attention or cognitive tunneling). This means that fixation error is more likely under stress – an important reason to call for help: you are probably missing something! Pay attention to the activities of surgeons and nurses, demand to be informed about anything unusual happening with the surgery. Concerns of others may not be addressed to you but may give early warning of an impending problem, so listen carefully.

7. Distribute the workload and use all available resources
   Humans are not good at performing more than one task at a time, so designate tasks to those who can best do them. You have many human resources: yourself, your anaesthetic assistant, the surgeon, other nurses, and other doctors in the hospital. Utilize monitors and their alarms as resources as well as flowcharts or task-cards.

Human Factors in Healthcare

Human involvement in complex systems like healthcare or specifically anaesthesia is both necessary and beneficial, considering our ability to adapt quickly and to be flexible. On the other hand humans are unpredictable and unreliable, especially in emergency situations, and our ability to process information is limited by our memory.

The term "human factors" can be very hard to define: "Ergonomics (or human factors) is the scientific discipline concerned with the understanding of interactions among humans and other elements of a system, and the profession that applies theory, principles, data and methods to design in order to optimize human well-being and overall system performance." (International Ergonomics Association Council, August 2000) In simple terms: "human factors" means trying to understand why humans don't behave predictably, and therefore finding ways to reduce error.

Fixation Error

Fixation error is very common in dynamic situations. It means a *persistent* failure to revise a diagnosis or plan in the face of plenty of evidence that a revision is necessary.

There are three main types of fixation error:

This and only this

Persistent failure to *revise* a diagnosis. Often the available evidence gets interpreted in a way to fit the initial diagnosis or attention gets allocated to a minor aspect of a major problem.

Everything but this

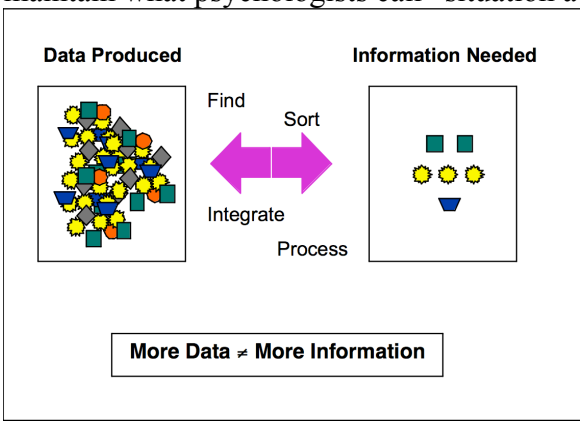
Persistent failure to *commit* to the definitive treatment of a major problem, more information is sought for without addressing potentially catastrophic conditions.

Everything is OK

Persistent belief that *no problem* is occurring despite plenty of evidence that there is. Abnormalities are attributed to artifacts or to being transient. It's a failure to declare an emergency or to accept help when facing a major crisis.

Situation Awareness

Some experts seem to have "eyes in the back of their head" because they are able to maintain what psychologists call "situation awareness". This has been defined as "the perception of elements in the environment within a volume of time and space, the comprehension of their meaning, and the projection of their status in the near future". (MR Endsley, 1995) Basically this means filtering out the important information and maintaining the "big picture".



The best way of maintaining situation awareness during an evolving crisis is to delegate tasks as much as possible and to free you up to keep an eye on all of what is happening.

Performance-shaping Factors

It is important to recognise that the abilities of even highly trained personnel can be severely influenced by internal and external performance shaping factors. The responsibility rests with the anaesthetist to ensure their performance level is sufficient for the task.

Ambient noise

The operating theatre is a noisy work environment. Some of that noise is controllable (conversation, music) whereas some is inevitable (equipment, surgical drills, alarms etc). Noise can negatively influence human performance. It can interfere with speech discrimination, short-term memory, the detection of audible alarms and effective communication.

Reading

Reading during the administration of anaesthesia should not be allowed if it impairs vigilance or patient safety, but is probably a good way of avoiding boredom (a distractor in itself) during low workload periods. Of course the anaesthestist should have a very low threshold for abandoning any potential distractions should a problem arise.

Fatigue and sleep deprivation

Research has shown that the effect on performance of being constantly awake for 24 hours equals a blood alcohol level above 0.05%. It has also been demonstrated that there is a higher incidence of medical incidents and performance failures during nighttime. It is the anaesthetist's responsibility to ensure they get enough sleep when not rostered for work. Even minimal levels of sleep loss (2 hours) can lead to lapses in performance, increased sleepiness and altered mood. The only way to pay back sleep debt is by SLEEP! Fatigue is caused by excessive physical or cognitive work, chronic fatigue and sleep deprivation can result in depression, anxiety, irritability, anger and depersonalization.

Alcohol

There are no formal studies of performance of anaesthetists under the influence of alcohol, however it seems obvious given the known negative effects of alcohol on judgement, motor function and reaction time that performance would be severely impaired.

Hazardous Attitudes

There are five particularly hazardous attitudes that can affect the anaesthetist's performance negatively:

- *"Don't tell me what to do."* (Anti-authority)
- *"Do something quickly- anything."* (Impulsivity)
- *"It won't happen to me – it's just a routine situation."* (Invulnerability)
- *"I'll show I can do it. I can deal with anything."* (Macho)
- *"What's the use? It's out of my hands."* (Resignation)

Especially hazardous are the "invulnerability" and "macho" attitudes.

Production Pressure

There are economic and social pressures on the anaesthetist to pursue efficiency and throughput and not patient safety as the primary priority. When giving in to these pressures the anaesthetist may be prone to haste, skipping appropriate preoperative evaluation and planning, or proceeding with cases despite medical concerns- doing things that are unsafe. In the end the anaesthetist has to ensure that the patient's safety is the primary criterion for their decisions.

Teamwork Issues

Formal training in team management and communication skills can produce major improvements in human performance as well as reduce critical errors.

Every anaesthetist is part of an operating room team, which involves surgeons, nurses and various other technical personnel, and equally they are part of an anaesthetic team involving anaesthetic nurses and sometimes a trainee. Everybody should introduce himself or herself including their role to the rest of the team. It is advisable to discuss important aspects of the patient and the surgery with both of these teams in a briefing before starting the procedure and the anaesthesia.

In most cases the senior anaesthetist will be best suited to be the team leader, should a crisis arise and they must ensure the rest of the team understands who is in charge.

- The ultimate goal is a good outcome for the patient
- Establish a team leader!
- Ensure good communication
- Ensure role clarity
- Create an atmosphere which encourages (junior) staff to speak up
- Listen and accept help
- **It's not *who* is right but *what* is right that counts.**

References

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THE MANAGEMENT OF MASSIVE HAEMORRHAGE

Trauma is the leading cause of death in all ages from 1 to 44 years in the UK. Haemorrhagic shock accounts for 80% of deaths in the operating theatre and 50% of deaths in the first 24 hours after injury. (1)

Massive transfusion may be defined as:

· In adults, as a transfusion of half of one blood volume in 4 hours, or more than one blood volume in 24 hours (adult blood volume is approximately 70 mL/kg)

· In children, as a transfusion of more than 40 mL blood/kg (blood volume of children older than neonates is approximately 80 mL/kg).

Development of a Massive Transfusion Protocol (MTP)

Every hospital should endeavour to develop a Massive Transfusion Protocol. An MTP includes clinical, laboratory and logistical responses. The Australian National Blood Authority (ANBA) has produced templates Figs 1 and 2.

Fig 1

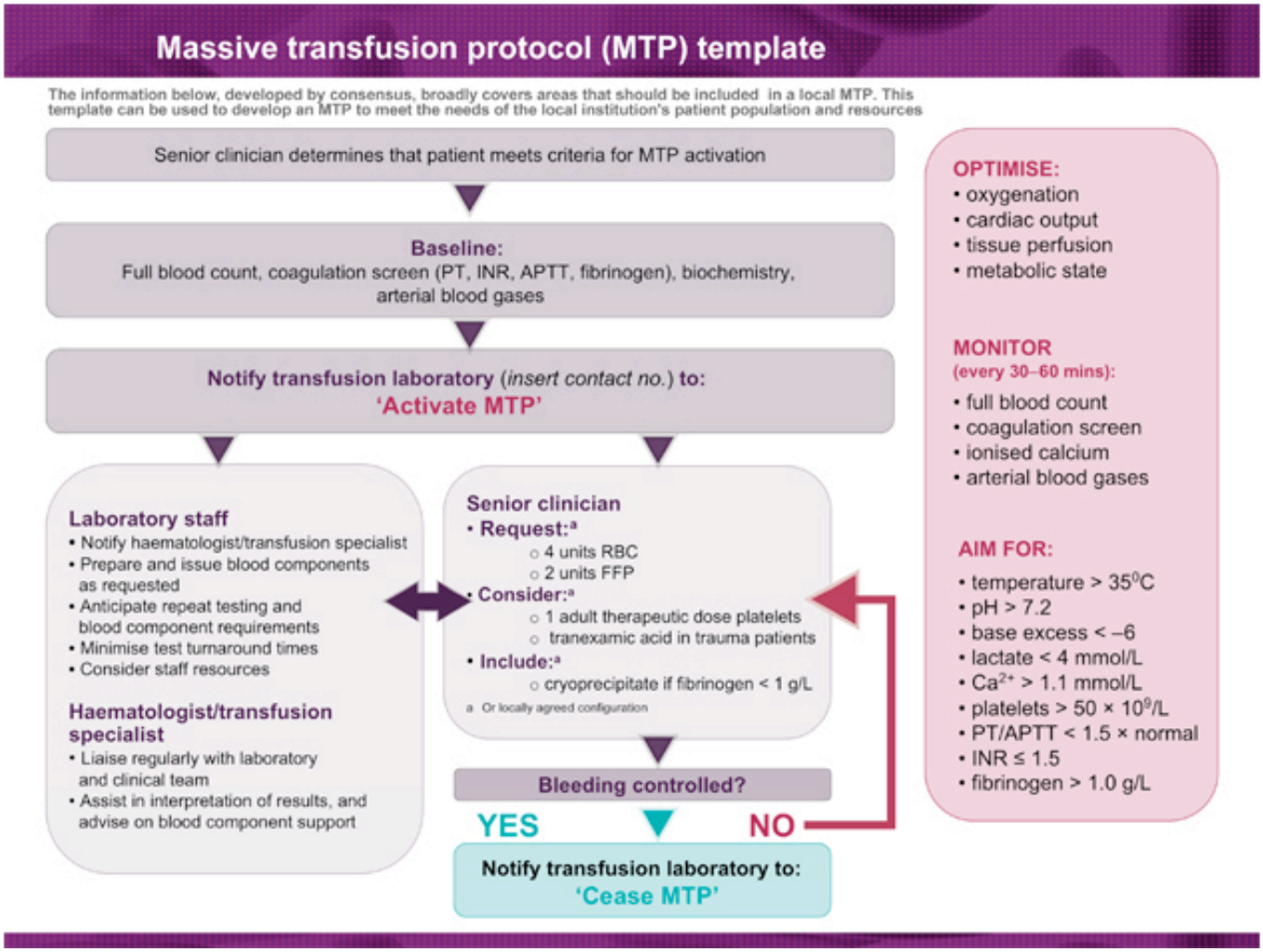
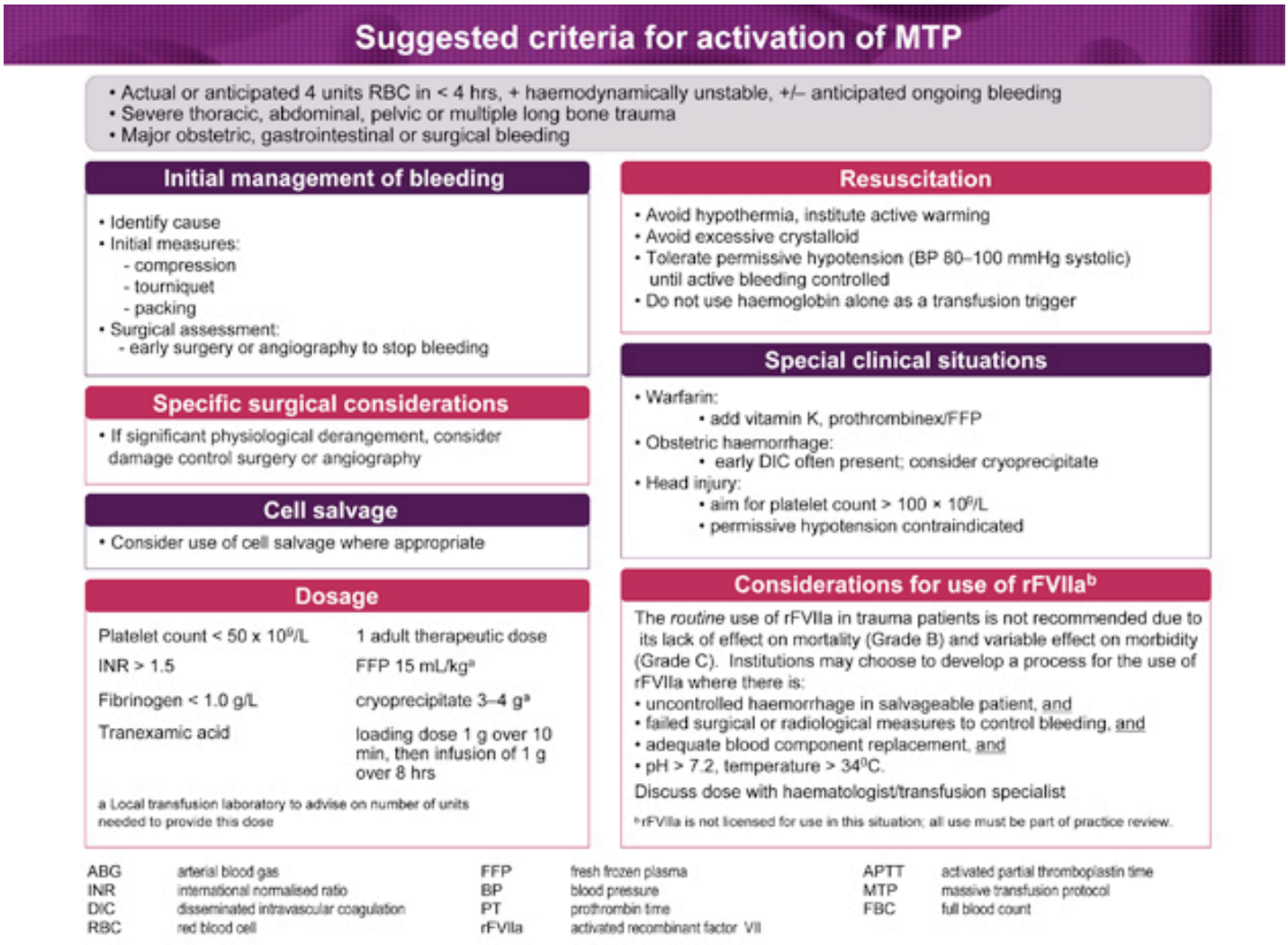


Fig 2



Individual hospitals may wish to adapt this template to:

- Take into account local resources (such as access to blood components)
- Provide details of how components will be delivered to the correct patient and location
- Specific populations such as obstetric patients, (given their potential for concealed haemorrhage and early development of disseminated intravascular coagulation) or children (with age dependent blood volumes, red blood cell mass and ability to tolerate blood loss)
- Include supporting information that explains how the clinical, laboratory and support staff will communicate. Highlight the need for early communication with a haematologist or transfusion specialist.

It is equally important that the local facility develops materials to accompany the Massive Transfusion Protocol (MTP), clarifying the roles and responsibilities of the team members (perhaps with task cards). Roles to be defined include those of

- Team Leader
- Communications
- Collection of blood samples and components
- Securing intravenous and central access
- Switchboard personnel for alerting key clinical support

It is essential to develop an effective method of triggering the appropriate major haemorrhage protocol. The need to activate an MTP should consider the cause and rate of haemorrhage, the patient's current physiological state and the likelihood of ongoing blood component treatment.

Though definitions of major haemorrhage vary, the Australian National Blood Authority (ANBA) suggests the following triggers for the activation of an MTP:

- Actual or anticipated transfusion of 4 units of red blood cells in < 4 hrs, + haemodynamically unstable, +/– anticipated ongoing bleeding or
- Severe thoracic, abdominal, pelvic or multiple long bone trauma or
- Major obstetric, gastrointestinal or surgical bleeding

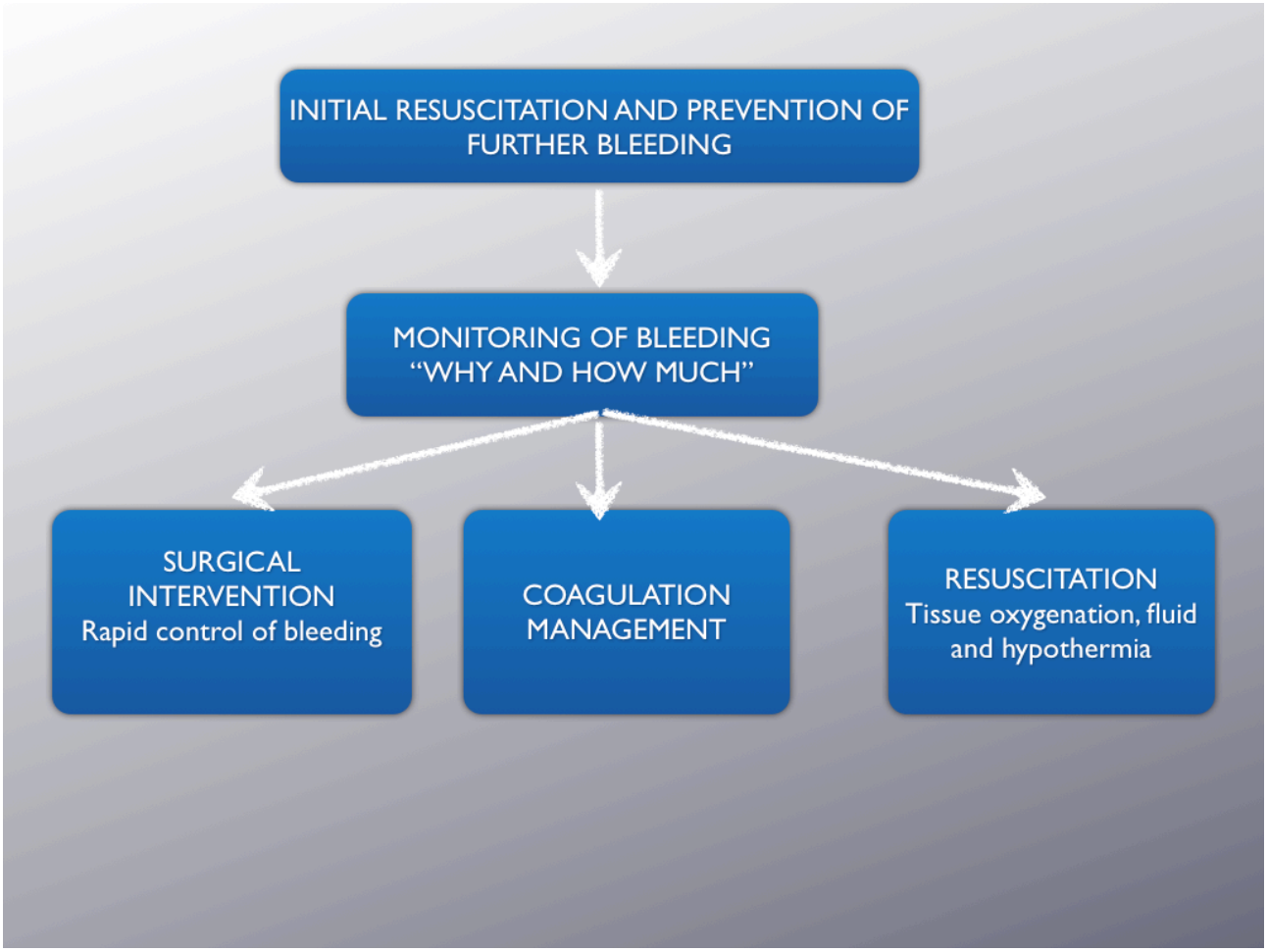
Management of massive haemorrhage

Patient blood management optimizes the use of donor blood and reduces transfusion-associated risk. It comprises optimizing the patient's blood volume and red cell mass, minimizing blood loss and optimizing the patient's tolerance of anaemia while avoiding transfusion-related adverse outcomes.

Importantly, most studies of critical haemorrhage and transfusion found that hypothermia, low pH, coagulopathy and low platelet count were associated with increased mortality. In patients with massive transfusion, the patient's temperature, acid base status, ionized calcium, haemoglobin, platelet count, INR, APTT and fibrinogen level should be checked early and frequently. With successful treatment values should trend towards normal.

The management of massive haemorrhage can be divided into four actions:

1. Initial resuscitation and prevention of further bleeding
2. Ongoing assessment: Monitoring of bleeding "Why and how much?"
3. Further management: Resuscitation, surgical and coagulation management
4. Venous thromboprophylaxis



Initial resuscitation and prevention of further bleeding.

Management of critical haemorrhage must focus on early recognition, rapid control of the bleeding source and restoration of the blood volume. The initial assessment should include the history, systolic blood pressure, pulse pressure, peripheral perfusion, mental state, respiratory rate, urine output, haemoglobin concentration, coagulation profile, acid base state and patient's temperature. Importantly, relying solely on the systolic blood pressure may delay identification of significant blood loss.

Class of haemorrhagic shock				
	I	II	III	IV
Blood loss (mL)	Up to 750	750–1500	1500–2000	> 2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	> 40
Pulse rate (per minute)	< 100	100–120	120–140	> 140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mm Hg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14–20	20–30	30–40	> 35
Urine output (mL/hour)	> 30	20–30	5–15	Negligible
Central nervous system/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic

Source: Adapted from American College of Surgeons (ACS) Committee on Trauma (2008)[19]
Reproduced with permission from ACS
Note: Values are estimated for a 70 kg male

The actions in initial resuscitation include controlling obvious bleeding points (pressure dressings, tourniquets), administer high inspired-oxygen concentrations, establish large bore intravenous access and take basic blood investigations (Haemoglobin, haematocrit, coagulation profile, blood cross matching). Actively warm the patient and all transfused fluids.

It is important to restore organ perfusion, but it is not necessary to achieve a normal blood pressure at this stage. If the patient is conscious, talking and has a peripheral pulse, then the blood pressure can be considered adequate.

Permissive hypotension and minimal volume resuscitation are generally preferable to aggressive volume resuscitation, which may cause oedema, compartment syndrome, acute lung injury, and exacerbate anaemia, thrombocytopenia and coagulopathy secondary to haemodilution. The safe systolic blood pressure and safe duration of permissive hypotension is not known. Such permissive hypotension aiming for a systolic pressure of 80-100mmHg date back to World War 2 and several more recent studies have shown a survival benefit.

Permissive hypotension is contraindicated in patients with traumatic brain injury because reduced perfusion pressure and oxygenation can lead to secondary brain injury

Fluid resuscitation – in the case of massive haemorrhage, this means warmed blood and blood components. In terms of time of availability, blood group O is the quickest, followed by group specific, and then cross- matched blood. For emergency issue blood, O Rhesus negative is the blood group of choice. It is acceptable to give O Rhesus

positive to male patients. In an emergency, group specific only blood can be issued following identification of the patient's blood group without knowing the result of an antibody screen. Group specific blood takes 10 minutes to arrive. Patients with massive bleeding will have minimal levels of circulating antibodies, although they may develop antibodies later. Cross-matched blood may take more than 45 minutes to arrive.

Patients require rapid access to imaging (ultrasound, radiography, computer tomography -CT), with appropriate use of focused assessment with sonography for trauma scanning and/or early whole body CT if the patient is sufficiently stable.

The theatre team should be alerted about the need for urgent surgery and cell salvage auto-transfusion (if available).

Ongoing assessment and monitoring of bleeding

What is the source and the extent of the bleeding ? ("why and how much?")

Look at injury patterns. Look for obvious blood loss (on clothes, on the floor, in drains). Look for indications of internal blood loss. Assess the patient's physiology (skin colour, heart rate, blood pressure, capillary refill, conscious level, urinary output, respiratory rate) and repeat laboratory investigations.

Some patients compensate well despite significant blood loss, especially if they are young and fit. A rapid clinical assessment will often give very strong indications of those at risk.

Further management: Surgical intervention, Resuscitation, Coagulation Management

Compression bandages, tourniquets, surgery, embolization or a combination of techniques, may control bleeding. Surgery must be considered early. 'Damage Control' surgery may be used for patients with severe haemorrhagic shock. With damage control surgery, limited surgery is performed to control bleeding and to prevent further contamination (for instance in the case of bowel disruptions). Once bleeding and contamination is controlled surgery ceases and the patient has aggressive ongoing resuscitation with the aims of restoring tissue oxygenation, acid-base status and circulating fluid volume before undergoing definitive surgery.

Coagulopathy should be anticipated and, if possible, prevented. If present, it should be treated aggressively. (See below.)

Following treatment for massive haemorrhage the patient should be admitted to a critical care area for observation, monitoring of coagulation, haemoglobin and blood gases, together with wound drain assessment to identify covert bleeding

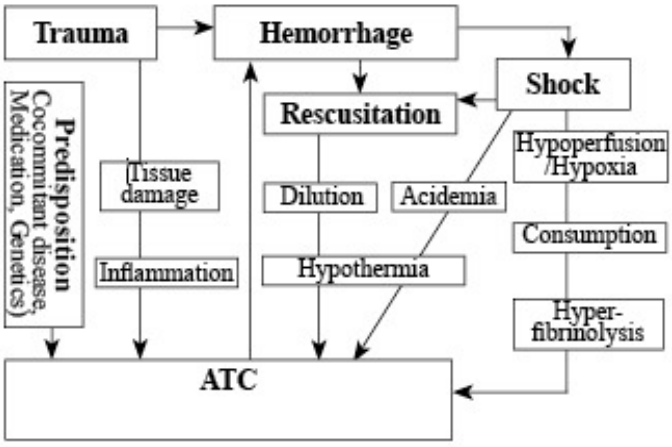
Venous thrombo-prophylaxis

Standard venous thrombo-prophylaxis should be commenced as soon as possible after bleeding has been controlled, as patients rapidly develop a pro-thrombotic state. Temporary inferior vena-cava filtration may be necessary.

Dealing with the coagulation problems

The coagulation disturbance will vary depending on the amount and cause of the bleeding, underlying patient related factors and management. It is likely to evolve rapidly. Most important in the management of these patients is regular assessment of the efficacy of replacement therapy using clinical assessment of microvascular bleeding and ongoing monitoring of coagulation parameters. The clinical scenario should lead patient management because by the time defects are detected in laboratory testing haemostatic failure may already be significantly present.

The aim of management is to try and prevent the development of a coagulopathy by the administration of fresh frozen plasma (FFP), platelets, and cryoprecipitate or fibrinogen with blood as soon as the MTP is activated by a senior clinician's assessment of the clinical scenario.



The ratios suggested are 2:1:1 (RBC:FFP:platelets) with the 1:1:1 ratio used in military protocols for battlefield injuries being reserved for the most severely traumatised patients.

Fig 5 provides an overview of the contributing factors that may be present.

Coagulopathy in massive haemorrhage is the often the result of a combination of mechanisms including dilution, consumption, platelet dysfunction, anticoagulant drugs, activation of anticoagulant pathways and hyperfibrinolysis. Dilutional coagulopathy should be prevented by the early infusion of FFP and platelets. Consumptive coagulopathy is commonly seen in obstetric haemorrhage, particularly with placental abruption and amniotic fluid embolism, following massive trauma especially involving head injury, in the setting of sepsis and with cardio-pulmonary bypass (CPB). Platelet dysfunction is associated with CPB, renal disease and anti platelet medication. Anticoagulant drugs should be reversed if possible- warfarin is reversed with Vitamin

K (5-10mg). Patients receiving heparin both unfractionated and low molecular weight should be given protamine.

A recently published randomized-controlled trial, recommended that tranexamic acid be given to *trauma* patients with or at risk of significant haemorrhage. The recommended dose is 1g over 10 minutes followed by an infusion of 1g over 8h

The aims of therapy to prevent coagulopathy should be to maintain

- temperature > 35^{o}C
- pH > 7.2
- base excess < – 6
- lactate < 4 mmol/L
- Ca2+ > 1.1 mmol/L
- Platelets > 50 x 109/L (some authorities recommend maintaining platelet count at 75 x 10^{9}/l)
- PT/APTT < 1.5 x normal
- INR <1.5
- Fibrinogen > 1.0 g/L

In those patients with clinical widespread microvascular oozing or with coagulation tests that demonstrate coagulation failure (fibrinogen<1g/l or PT/APTT>1.5 times normal), 30 ml/kg of FFP is a reasonable first line therapy. Hypofibrinogenaemia unresponsive to FFP should be treated with cryoprecipitate or fibrinogen concentrate if available.

Paediatric guidelines

Paediatric doses

Red cell concentrates	Vol (ml) = desired Hb rise (g/dl)1) · weight (kg) · 3
Platelets	Children < 15 kg (10–20 ml/kg) Children > 15 kg (1 adult bag)
FFP (MB treated)	10–20 ml/kg
Cryoprecipitate	5–10 ml.kg (usually maximum 10 units or 300 ml)

Equipment to aid transfusion.

All blood components should be administered using a blood component administration set, which incorporates a 170-200µm filter. If red cell salvage is being used, a 40µm filter may be indicated. All fluids should be warmed.

If giving platelets use a clean 170-200µm giving set as one that has been previously used for RBCs may cause platelets to stick to the red cells and therefore reduce the effective platelet dose.

External pressure devices (for administration of fluid) should only be used in an emergency in conjunction with a large bore cannula. They should exert pressure evenly over the entire bag, have a gauge to measure pressure, not exceed 300mmHg pressure and must be monitored at all times during use.

Intra operative cell salvage (ICS)

The use of cell salvage can be very effective at both reducing the demand for allogenic supplies and providing a readily available red cell supply in massive haemorrhage. The guidelines published by the Association of Anaesthetists of Great Britain and Ireland is available at the following web address

http://www.aagbi.org/sites/default/files/cell%20_salvage_2009_amended.pdf

The guideline reports successful use of ICS in malignancy, obstetric haemorrhage and bowel surgery associated with catastrophic haemorrhage.

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Westerman RW, Davey KL, Porter K. Assessing the potential for major trauma transfusion guidelines in the UK. Emergency Medicine Journal 2008; 25: 134–5.

Patient Blood Management Guidelines: Module 1. Critical Bleeding Massive Transfusion.
http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/patient_blood_guidelines_module_1.pdf

The 3 source materials for this chapter are

Association of Anaesthetists of Great Britain and Ireland (AAGBI) SAFETY GUIDELINES:
"Blood transfusion and the Anaesthetist: The Management of Massive Haemorrhage." Nov 2010. http://www.aagbi.org/sites/default/files/massive_haemorrhage_2010_0.pdf

"Blood transfusion and the Anaesthetist: Intra-operative Cell Salvage." September 2009."
http://www.aagbi.org/sites/default/files/cell%20_salvage_2009_amended.pdf

National Blood Authority Australia (NBA)
"Patient Blood Management Guidelines: Module 1. Critical Bleeding Massive Transfusion"
http://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/patient_blood_guidelines_module_1.pdf

Management of bleeding following major trauma:
an updated European guideline: Rossaint et al. Critical Care 2010, 14:R5
http://ccforum.com/content/pdf/cc8943.pdf

All are available as free downloads on the internet.

ANAPHYLAXIS

Definition:

Anaphylaxis is defined as a severe, life-threatening, generalized or systemic hypersensitivity reaction. It can be divided into allergic/immune-mediated anaphylaxis (a reaction mediated by an immunological mechanism such as immunoglobulin E (IgE), IgG or complement activation and non-allergic/non-immune-mediated anaphylaxis. The term anaphylactiod reaction is often used to refer to the non immune-mediated reaction. The clinical picture of the immune-mediated and non-immune-mediated anaphylaxis is almost identical.

Epidemiology:

The incidence of anaphylaxis is approximately 1 in 10,000 to 20,000 general anaesthetics. The most common causative agents in the general population are antibiotics, while in theatre it is muscle relaxants. Other common allergens include:

- Latex
- Intravenous colloids (5%)
- Aspirin, non-steroidal anti-inflammatory drugs, induction agents,
- Opiates, aprotinin, protamine, oxytocin, chlorhexidine, radiological
- Contrast media, dyes (Patent Blue V)

The risk factors for anaphylaxis include a history of allergy, intravenous administration, being female, young and having previous exposure to the allergen. Anaphylaxis to non-depolarizing muscle relaxants (NDMR) is three times more likely if the patient has previously had a serious allergy to penicillin. The most common muscle relaxants to cause anaphylaxis are suxamethonium and rocuronium (1 in 5000). The rate of anaphylaxis to atracurium is 1 in 50,000.

Pathophysiology:

Immune-mediated (IgE, Ig G or complement):

This is the classical allergic reaction, where a specific allergen interacts with allergen-specific IgE bound to the receptor Fc epsilon RI (FcεRI) on mast cells and/or basophils. This interaction results in a release of a large amount of chemical mediators including histamine, tryptase, leukotrienes, platelet activating factor and many others.

The histamine causes the vasodilation, bronchospasm and increased vascular permeability.

Non-immune-mediated:

Agents or events that induce sudden, massive mast cell or basophil degranulation in the absence of immunoglobulins cause non-immune-mediated anaphylaxis.

Presentation:

Symptoms	Incidence	Sole Feature
CVS depression	75-90%	1-10%
Bronchospasm	30-50%	3%
Erythema	45%	
Angiooedema	25%	1%
Rash	13%	
Urticaria	8.5%	
Pulmonary Oedema	3%	0.3%
Gastrointestinal Symptoms	7%	

Bronchospasm is the hardest symptoms to treat.

Management:

This is an emergency. Management requires rapid assessment and management.

- Call for help
- Remove the potential cause
- Stop surgery as quickly and safely as possible
- 100% oxygen
- ADRENALINE

Adrenaline: is the treatment of choice and should be given in all cases of suspected anaphylaxis. Failure to treat anaphylaxis promptly with adrenaline may result in biphasic or protracted anaphylaxis or in a fatal outcome.

Dose:

- Adrenaline – 500mcg IM (intra-muscularly)
  - Intravenous (IV) adrenaline is only recommended if the patient is monitored with continuous blood pressure, ECG and pulse oximetry. The dose should be titrated with an initial bolus of 20-50mcg, and repeated if required. Note that if the patient is arrested (no pulse or blood pressure), then the standard 1mg of IV adrenaline should be given.

Other management:

- IV fluids
- Antihistamine
- Steroids – hydrocortisone 100mg or Prednisolone 50mg
- If bronchospasm persists, use inhaled or intravenous salbutamol.

Postoperative management:

The patient should be monitored in the High Dependency or Intensive Care Ward for 24 hours becausee the anaphylaxis can recur. It is important to maintain an adrenaline infusion if hypotension persists. Calcium, beta-blockers and Angiotensin Converting Enzyme inhibitors should be avoided since they make it hard to treat hypotension.

Follow up:

Inform the patient and give a letter to the patient and their local doctor. Advise the patient that they must inform their doctors of this reaction for all subsequent medical procedures.

Testing for cause:

Blood tests can be used to test for a specific agent, namely trptase. This blood test is done during resuscitation, and then one hour and 6-24 hours post reaction. Mast Cell Tryptase is useful to confirm that a reaction has taken place, but cannot be used to identify the agent that caused the reaction.

Intradermal skin testing:

This procedure is often done 4-6 weeks following the initial reaction, which allows the Ig E stores to regenerate. A skilled person must do the test in a monitored area with all the equipment and drugs needed to treat anaphylaxis if it does occur.

The drugs that the patient was exposed to are diluted to 1:500 or 1:1000 and 0.02-0.05mls of this solution is injected into the intradermal area. This area is monitored for a period of time to check for a reaction.

Reference:

1. Stephen Robinson, Indu Sivanandan : **Anaphylactic and anaphylactoid reactions** Original Research Article *Anaesthesia & intensive care medicine*, *Volume 5, Issue 9*, *1 September 2004*, *Pages 298-300*

2. Coralie Carle, Nigel J.N. Harper **Anaphylactic reactions associated with anaesthesia** Review Article *Anaesthesia & Intensive Care Medicine*, *Volume 11, Issue 10*, *October 2010*, *Pages 391-393*

3. Uptodate website

PERIOPERATIVE NERVE INJURY

Perioperative nerve injury, loss of vision, and positioning-related problems, continues to be a significant source of injury for the anesthetized patient. Meticulous care must be taken while moving a patient into the desired surgical position. An understanding of anatomy and physiologic changes is important in prevention.

The incidence of perioperative nerve injuries is estimated to be about 15% of ASA (American Society of Anesthesia) closed claims in 1990. Of these, ulnar neuropathy comprised 34% (of which 75% were male), brachial plexus injury 23%, and lumbosacral neuropathy 16%.

Most Frequent Claims for Nerve Injury by Gender				
Nerve	**# Claims**	**% of 445**	**% Female**	**% Male**
All nerve damage claims	445	100%	49%	51%
Ulnar Nerve	113	25%	21%	79%
Brachial Plexus	83	19%	57%	43%
Spinal Cord	73	16%	49%	51%
Lumbosacral				
Nerve Root	67	15%	70%	30%
Sciatic Nerve	23	5%	61%	39%

The causes of nerve injury are many and varied, and may include

- Section
- Compression
- Traction
- Ischemia
- Type of surgery (sternotomy)
- Prolonged placement (> 4 hrs lithotomy)
- Prolonged tourniquet (>2 hrs)
- Congenital anomalies (cervical rib)

Anaesthesia may be considered as a significant contributor to nerve injury. Changes in respiratory and cardiovascular physiology as a result of anaesthesia, positioning and

surgery, may compromise perfusion and oxygenation of many structures, including peripheral neural tissue. Combined with the loss of normal protective reflexes during anaesthesia (loss of sympathetic tone, inability to protect limbs from painful or compromised positions, inability to detect and protect from ischaemia), we have placed our patients in a uniquely vulnerable position.

Neurovascular compromise, or neural ischemia, may occur as a consequence of compression or stretching of intraneural vasa nervorum (the small vessel blood supply to nerves). Nerves that have a long or superficial course between two points of fixation are at particular risk of both stretching and compression. Tissue oedema from fluid overload, hypoalbuminaemia or sepsis may contribute to neurovascular compression.

Coexisting medical problems which may contribute to injury include:

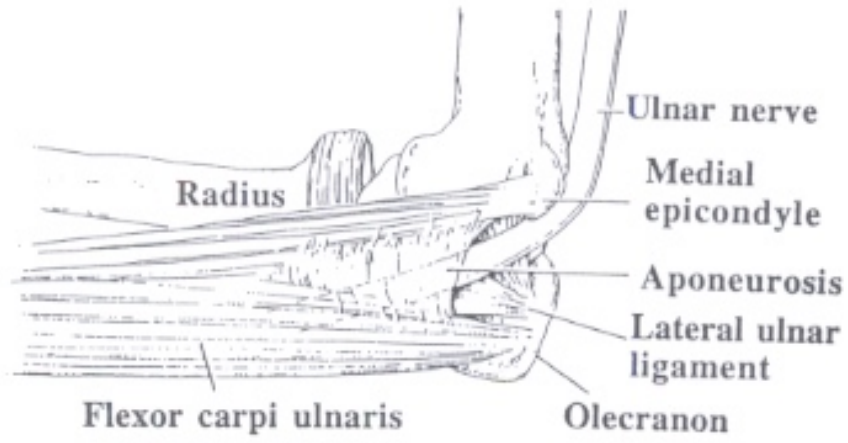
- Diabetes mellitus
- Alcohol abuse
- Vitamin deficiency
- Coagulopathy/ Hypothermia
- Uremia
- Polycythemia vera
- Acromegaly
- Hypothyroidism

Equipment is a major cause of injury, especially tourniquets, blood pressure cuffs, infusion pumps, arm boards and leg supports.

Upper extremity neuropathies

Ulnar Neuropathy
Ulnar neuropathy is the most common reported perioperative neuropathy, and has been since the 1890's (5,6). Anaesthesia-related ulnar nerve injury accounts for 30% of all nerve injuries, and 85% of all nerve injuries that occur under general anaesthesia

Anesthesia-related ulnar nerve injury is thought to be associated with external nerve compression (12) or stretch caused by malpositioning during the intraoperative period. The ulnar nerve can be compressed against the posterior aspect of the medial epicondyle of the humerus. Forearm rotation (pronation) can increase pressure in the postcondylar groove (13). The nerve may also be more easily compressed distal to the medial epicondyle, where the nerve and its associated artery are superficial in the postcondylar groove (14).



Stretching of the ulnar nerve can be induced by abducting the arm between 60 and 90 degrees, such as on an arm board in the supine or prone position. Elbow flexion to greater than 100 degrees can stretch the ulnar nerve as well as tighten the cubital fossa retinaculum, directly compressing a stretched nerve.

Other factors play a role in the development of ulnar neuropathy. A prospective study found that both surgical and medical patients develop ulnar neuropathy in both the inpatient and outpatient settings (7). Associations include being male (70-90% of perioperative ulnar neuropathies) (3,8,9,10), high body mass index (>38 kg/m^2) and prolonged bed rest. Many patients with ulnar neuropathy demonstrate contralateral ulnar nerve conduction dysfunction (10), suggesting they are an "at risk" subset. Many patients do not notice or complain of ulnar nerve dysfunction until more than 48 hours after a surgical procedure. A prospective study of 1502 surgical patients found that none developed symptoms of ulnar neuropathy in the first two postoperative days (11).

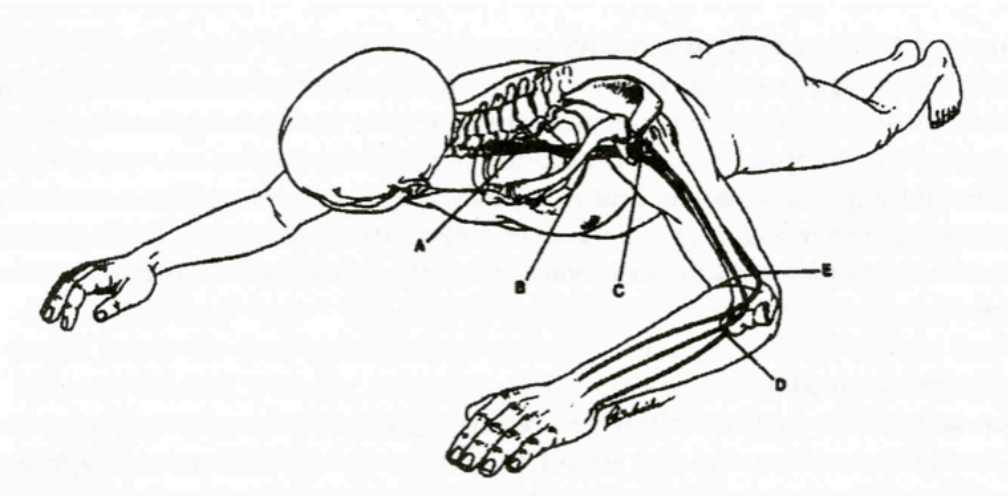
A 1994 retrospective study (8) examined over 1 million consecutive surgical cases. Ulnar neuropathy persisting for more than 3 months occurred in 414 cases, or 1 per 2729 patients. Of these, 9% demonstrated bilateral neuropathy. Just over half of the affected patients had resolution of the neuropathy by 12 months.

Brachial Plexus Neuropathies

Stretch-induced neuropathy of the brachial plexus and median nerve remains a frequently preventable complication. They may masquerade as ulnar neuropathies or be associated with symptoms that suggest injuries to other nerve structures (15, 16, 17). Brachial plexus neuropathies are associated with cardiac surgery and median sternotomy, where stretch or compression of the brachial plexus occurs during sternal separation. Injury may also occur via direct trauma from fractured first ribs (18).

Stretch of the brachial plexus can occur when the arm of an unconscious patient falls from the table or armboard. This may also be accompanied by significant soft tissue injury (rotator cuff), and lead to a marked loss of upper limb function.

The brachial plexus is also vulnerable to stretch in a patient who is positioned prone. Stretch of the brachial plexus, especially its lower trunks, is most likely to occur when the head is turned to the contralateral side, the ispilateral shoulder is abducted, and the ipsilateral elbow flexed (19).



Sources of potential injury to the brachial plexus and its peripheral components in the pronated patient:

- Head rotation stretching plexus against anchors in shoulder.
- Closure of the retroclavicular space by chest support with the arms at the side; neurovascular bundle trapped against the first rib.
- Head of the humerus thrust into the neurovascular bundle if the arm and axilla are not relaxed.
- Compression of the ulnar nerve in the cubital tunnel.
- Area of vulnerability of the radial nerve to compression above the elbow.

Similar to ulnar neuropathy, a brachial plexus injury may be diagnosed by pain, numbness and decreased movement of the arm, which may be noted immediately

postoperatively and up until forty-eight hours post surgery. Careful neurologic examination will help define the lesion.

Prevention of brachial plexus injuries requires consideration of patient positioning:

Avoid abduction of the arm greater than 90 degrees from the body

Minimise combination of arm abduction, external rotation and dorsal extension

Avoid downward pressure on the head of the humerus

Avoid placing humerus behind the plane of the body

Keep arms at patient sides wherever possible (prone or supine)

Radial Nerve Injury

This accounts for less than 3% of all nerve injuries. The nerve is susceptible to compression in its path along the spinal groove of the humerus, and is ideally placed for injury beneath a tourniquet or inappropriately-sized blood pressure cuff. The distal radial nerve at the wrist may be injured by venous cannulation.

Median Nerve Injury

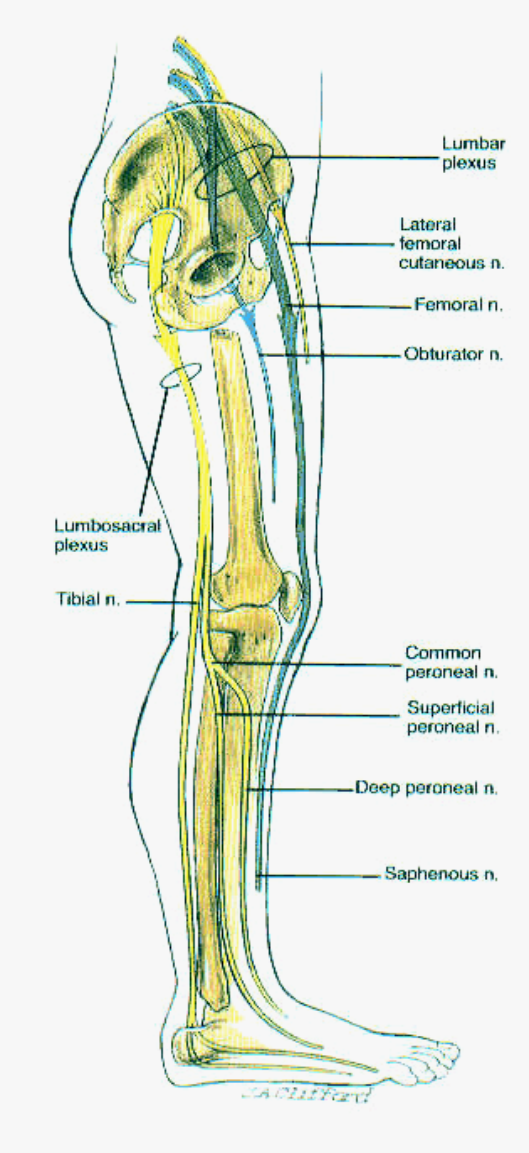
Although rare, the median nerve may be impinged by intravenous cannulation in the cubital fossa, and extreme wrist dorsiflexion.

Lower extremity neuropathies

Although neuropathies of the lower extremities may occur in a variety of patient postures, most of these occur in patients who are undergoing procedures in a lithotomy position. Traditionally these neuropathies have been considered preventable, blamed on poor intraoperative care (improper positioning or padding) or judgment (such as excessively prolonged use of lithotomy position).

A number of studies have suggested there are many factors that may contribute to the development of a lower limb neuropathy (20,21,22).

A 1994 retrospective review of patients in lithotomy positions (23) found that the most common lower extremity neuropathies were common peroneal (81%), sciatic (15%) and femoral (4%) neuropathies.



Obturator and Lateral Femoral Cutaneous (LFC) Neuropathies

Cadaver studies demonstrate that neither hip flexion nor abduction increased strain on the LFC nerve. However, abduction to greater than 30 degrees without concomitant hip flexion dramatically increased strain on the obturator nerve (24).

Common Peroneal Neuropathy

The common peroneal nerve is the most frequently damaged lower extremity nerve. A branch of the sciatic nerve, it runs a very superficial course around the head of the fibula where it may be easily compressed and injured by leg holders, resulting in loss of foot dorsal extension. The superficial peroneal nerve may be affected distal to the fibular head by compressive stockings or wraps (25).

Sciatic Neuropathy

Stretch of the sciatic nerve can occur with hyperflexion of the hip and extension of the knee. This can also occur in the lithotomy position. A patient in a lithotomy position may slide down toward the caudal end of an operating table if placed in a head-up position, or be actively pulled down the table by a member of the operating team in an attempt to obtain increased exposure of the perineum. This movement may increase the hip flexion and leg extension if the legs are fixed within leg holders. Always check that the flexor muscles of the knee (hamstring group) are not tight after placing a patient's legs into any lithotomy position.

Femoral Neuropathy

Unlike most other neuropathies, those involving the femoral nerve and its cutaneous branches are often considered to result from surgical causes, such as the improper placement of abdominal wall retractors and direct compression of the nerve. It is assumed that retractors may place continuous pressure on the iliopsoas muscle and either stretch the nerve, or cause it to become ischemic by occluding the external iliac artery or penetrating vessels of the nerve as it passes through the muscle (26).

Practical Considerations for Neuropathies

- Position and pad exposed peripheral nerves.
- Prevent their stretch beyond normally tolerated limits when awake.
- Avoid direct nerve compression, if possible.
- Distribute any necessary compressive forces (such as the tourniquet) over as large an area as possible.

What to Do if Your Patient Develops a Neuropathy

Is the neuropathy sensory or motor?

Sensory lesions are more frequently transient than motor lesions. If the symptoms are numbness and/or tingling only, inform the patient that many of these neuropathies will resolve during the first 5 days (11). Most will resolve within 6 weeks, while a small number may persist for up to 6 months. Ask the patient to avoid positions that might compress or stretch the involved nerve. Keep in frequent contact with the patient and monitor the outcome. A neurological review would be appropriate, and should be mandatory for those who have symptoms beyond 1 week.

If the neuropathy has a motor component, a neurologist should be consulted immediately. Electromyographic (EMG) studies are required to assess the location of the lesion, which may direct an appropriate treatment plan. EMG studies may also demonstrate chronic abnormalities of the nerve (for example, fibrillation potentials in denervated muscles) or, if applicable, the contralateral nerve.

Blindness

Perioperative blindness has been associated with cardiac surgery, and with prolonged spinal surgery in the prone position (28).

Most cases involve anterior or posterior ischaemic optic neuropathy (AION & PION), central retinal artery occlusion, or undefined ischaemia to the cerebral cortex. There are very few cases reported in the past two decades in which direct pressure to the globe is implicated in perioperative blindness. Blindness in cardiac patients is approximately balanced between AION and PION. In contrast, PION appears to be the predominant problem in prone-positioned patients.

The etiology of PION is unknown. Patients in the prone position develop an increase in intraocular pressure (29, 30, 31), related in part to gravity and increased central venous pressure (29, 32). Posture induced changes in the function of the anatomy or iris may contribute (33), as may anaemia, hypotension (34,35) and periorbital oedema. Venous engorgement around the optic nerve may cause compression of the optic sheath and limit arterial perfusion (36).

There are sufficient numbers of cases in cardiac surgical patients to retrospectively determine risk factors for perioperative blindness (37):

Risk factors found in cardiac surgical patients

- Patient factors (advanced age and arteriosclerosis)
- Procedural issues (prolonged pump perfusion and surgical disruption of particulate matter)
- Practice patterns (deliberate postoperative anemia and intraoperative hypotension)

There are insufficient numbers of cases in any series to evaluate risk factors in noncardiac surgical patients. The ASA Closed Claims Postoperative Visual Loss Registry suggests that most cases of vision loss is associated with prolonged spinal surgery (greater than 6 hours) in the prone position, accompanied by substantial blood loss (38).

Other Head and Neck

Pressure alopecia

Although not a neuropathy, the etiology of prolonged compression of hair follicles gives us an insight, and a warning, as to the causes or likelihood of pressure necrosis and neuropathy in our practice. It often begins 2-3 days post-operatively. There is an increased incidence when accompanied by hypotension and hypothermia, and may be prevented with padding and turning of the head.

Supraorbital nerve compression

Beware of straps over the forehead, or of airway and other equipment that might cause pressure on the patient.

Lingual nerve compression

A rare but increasing neuropathy seemingly due to the use of laryngeal masks. The lingual nerve is thought to be compressed at the base of the tongue, and may result in loss of sensation in the tongue, generally at the tip. It usually recovers well.

Spinal Cord Ischemia or Infarction From Lumbar Hyperextension

Patients undergoing pelvic procedures using an abdominal approach are positioned supine, and may have their lumbar spines hyperextended in an attempt to increase

surgeon visibility into the lower pelvis. This is reasonable when limited to the operating room table mechanism for hyperextending the lumbar spine (such as raising the kidney rest). Tables manufactured within the United States limit lumbar hyperextension to 10 degrees. When excessive padding is placed under the lumbar spine to for additional hyperextension, the spinal cord may become ischemic and infarct (27). There are no reports of anterior spinal cord ischemia when patients are positioned using only the table mechanisms to induce lumbar hyperextension.

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RECOVERY ROOM PROBLEMS

The recovery room is an area adjacent to the operating rooms that is staffed by nursing and medical practitioners who are experienced in the care of unconscious patients who are recovering from anaesthesia. It is an acute care area where patients are observed frequently in order to detect and manage problems that arise after surgery and anaesthesia until it is safe to transfer the patient to an ordinary ward or another acute care area of the hospital (such as a high dependency unit or critical care unit). Recovery room (or Post Anaesthesia Care Unit PACU) complications are common and can occur in up to 24% of patients. (1) The most common problems include pain, postoperative nausea and vomiting (PONV), airway and cardiovascular problems. Neurologic problems, such as delayed emergence and delirium can occur in the immediate postoperative period and are best managed in a dedicated recovery room.

Anticipating and avoiding recovery room problems

The postoperative period is a time of physiologic instability as the patient emerges from anaesthesia after surgery. It is expected that surgery will cause pain an inflammatory and sympathetic “stress” response. If there is intraoperative bleeding or fluid loss, it will cause haemodynamic disturbance unless the anaesthetist replaces these losses with intravenous fluids or blood. Many of the medications used during anaesthesia will have significant side effects (including sedation, nausea and vomiting) and interactions with other medications that the patient is taking. Some medications will need their actions reversed (particularly the neuromuscular blockers) in order for the patient to recover from anaesthesia safely. Intraoperative events will also affect the quality of recovery. They include exposure that induces heat loss and abnormal positioning that can cause pressure areas, joint and soft tissue injury and oedema in dependent areas (for example, airway and head and neck oedema in the steep head down or prone position).

A patient who has not regained full consciousness will require airway and cardiovascular management as well as careful positioning, just as during the intraoperative period.

Surgical complications will not be covered in this review, but the staff caring for a patient in recovery need to be aware of them and be able to offer immediate management and call for surgical assistance if required. The commonest surgical problem is bleeding, so the operative site should be examined frequently.

Regular observation and charting of the patient’s vital signs and conscious state is required. This includes respiratory rate, oxygen saturation, pulse rate, blood pressure

and temperature. The frequency of observation needs to be at least every 10 minutes or more frequently if there is instability. (2)

A recovery room needs to be staffed adequately and be well equipped, within easy access of the operating rooms. A trained nurse should supervise the patients in a ratio of not less than one nurse for every 3 patients or one nurse to one patient if the patient is unable to protect and maintain his own airway or remains unconscious. There needs to be an anaesthetist available to help the nurse looking after the patient if a problem occurs.

The Australian and New Zealand College of Anaesthetists (ANZCA) (2) recommends that each bed space in recovery have the following:

- Adequate lighting
- An oxygen outlet and oxygen delivery systems
- Medical suction and appropriate hand pieces and catheters
- Monitoring including a pulse oximeter, facilities for blood pressure monitoring and a thermometer
- A stethoscope
- Power outlets

Within the recovery area, ANZCA (2) recommends that there be a means for manual ventilation (self inflating bag or similar), equipment and drugs to manage the airway and endotracheal intubation, emergency and other drugs, intravenous fluids and equipment, drugs for pain management, patient warming devices and a device for measuring expired carbon dioxide. There should be access to a 12 lead electrocardiograph, defibrillator, neuromuscular function monitor (nerve stimulator), chest drains, warming cabinet, refrigerator for drugs and blood, basic surgical tray, blood gas and electrolyte measurement, diagnostic imaging (x-ray), mechanical ventilation and monitors for invasive blood pressure and central venous pressure measurement.

The most important aspect of the recovery room is the presence of a nurse or doctor who can monitor the patient and manage life-threatening problems, particularly airway obstruction and hypoventilation and cardiovascular instability.

Pain in recovery

Pain after surgery is common. Acute severe postoperative pain has many deleterious effects and treatment of pain will minimize these effects. It is now recognized that poorly treated acute pain will predispose the patient to developing long term effects, including persistent postoperative pain that can interfere with recovery and the ability to return to normal function.

As with all pain, the approach to postoperative pain is its Recognition, Assessment and Treatment. (RAT) Patients in pain will demonstrate physiologic and behavioural effects. There may be tachycardia, hypertension, poor cough and shallow breathing (leading to hypoventilation and hypoxia), anxiety, restlessness and distress. Assessment of the pain includes addressing the cause of the pain, its site, severity and character. It is most commonly caused by the surgical incision and tissue trauma associated with surgery, but it is important to be aware that pain may be due to a pre-existing condition, sore throat from endotracheal intubation, neurologic injury, joint pain from extreme positioning during the intraoperative period, acute surgical bleeding into a confined space, compartment syndrome, as well as other acute medical emergencies such as ischaemic chest pain or intracranial pathology.

Treatment of pain involves a multimodal approach, including finding a position of comfort (elevation of a swollen limb), local and regional anaesthesia (such as local anaesthetic infiltration intraoperatively), strong opioids, non-steroidal medications, ketamine, tramadol and clonidine, as well as other classes of analgesics. It is preferable to commence analgesia during the intraoperative period before the patient emerges from the anaesthetic, so that the patient does not awake with severe untreated pain.

The immediate management of acute severe surgical pain in recovery usually involves the administration of a strong opioid via the intravenous route in small repeated doses until there is control. A patient who is elderly (over 70 years of age) will require less opioid to achieve analgesia without encountering the side effects of sedation and respiratory depression. One approach for the patient under 70 is to give morphine in a dose of 4mg intravenously for pain scores of over 6 out of 10 immediately, followed by smaller doses of 2mg or 1mg every 3-5 minutes to achieve patient comfort or a pain score of less than 3 out of 10 on a Verbal Numeric Rating Scale. The maximum dose given is 20 mg in the patient under 70 years of age. (3) The administration of any opioid intravenously needs to be done with close observation of the patient. Respiratory depression is a potential adverse effect and is closely linked to the level of sedation experienced by the patient. If the patient is drowsy most of the time, do not administer extra doses of opioid until the patient is more alert and consider an alternative agent for treating pain.

In addition to morphine (or equivalent doses of fentanyl), paracetamol 1g per oral or per rectum, diclofenac (or other non steroidal if there are no contraindications) are given. If the patient is agitated, clonidine (1-2 micrograms per kilogram intravenously) can be considered. Hypotension and sedation are significant side effects of clonidine. (4) Ketamine in a single intravenous bolus dose of 10-15 mg intravenously can also be considered if there is intractable pain or the patient is experiencing opioid tolerance. (4)

Postoperative Nausea and Vomiting (PONV)

Nausea and vomiting are common after surgery and anaesthesia. There are many contributing factors including hypoxia, pain, hypotension and hypovolaemia, surgical handling of the intestine, parasympathetic outflow, opioid administration, and patient factors such as being female, a non smoker or having a past history of postoperative nausea and vomiting.

The management of PONV includes identification of patients at risk, administering prophylactic antiemetics and minimizing intraoperative factors known to increase the risk, addressing contributing factors and finally, administration of treatment, both pharmacologic and non pharmacologic.

As with pain, a multi-modal approach is used to treat PONV. The classes of drugs available to treat PONV include, serotonin receptor antagonists (5-Hydroytryptamine-3 antagonists) such as ondansetron, glucocorticoids intraoperatively such as dexamethasone as a single dose, anti-dopaminergic drugs such as low dose droperidol, metoclopramide and prochlorperazine, anti-histamine 1 agents such as promethazine, cholinergic agents such as transdermal scopolamine, and more recently the neurokinin-1 receptor antagonist, aprepitant has become available.

Respiratory Emergencies

Airway complications account for about 10% of recovery room complications overall. (1) Management of an acute airway emergency can present a challenge. Postoperative oedema, previous intraoperative airway manipulation, prolonged head down or prone positioning can turn even a previously straightforward airway into a challenging airway in the recovery room. Respiratory events include obstruction, apnoea, hypoxia and hypercarbia. The clinical presentation of a respiratory problem may include: no (or reduced) respiratory effort, obstructed breathing (paradoxical chest and abdominal wall movement), airway noise (snoring, stridor or wheeze), low oxygen saturation, cyanosis, cardiac arrhythmias and impaired consciousness.

Many patients with airway obstruction after anaesthesia simply have pharyngeal obstruction by the tongue due to impaired consciousness. Simple manoeuvres such as lateral (recovery) positioning, stimulation of the patient, jaw thrust and chin lift or oral and nasal airway insertion is all that is required to establish airway patency.

Stridor represents obstruction at or just above the larynx. It may represent airway narrowing secondary to oedema, foreign material in the airway, tracheomalacia or incomplete laryngeal spasm. Initial management involves ensuring a patent upper

airway, continuous positive pressure with tight fitting face mask and assisted ventilation. If there is a high suspicion of oedema, it may be treated with racemic adrenaline via a nebulizer. The patient may need to be re-intubated if this does not solve the problem. Obstruction secondary to laryngeal spasm may require positive pressure ventilation or small doses of suxamethonium (20-40mg). If a patient makes respiratory effort against an obstructed airway, he may develop negative pressure pulmonary oedema.

Airway obstruction can result from oedema secondary to surgery on the neck and upper airway, and prolonged head down or prone positioning that can cause macroglossia as well as oedema due to a reduction in venous return from the head and neck. At the end of these high risk operations, the anaesthetist needs to determine whether it is safe to extubate the trachea. It may be prudent to wait until oedema has subsided.

Hypoxaemia (defined by oxygen saturation of less than 90%) that persists after airway patency is restored may be due to an increase in right to left shunting, commonly secondary to atelectasis but may also occur secondary to other lung conditions after anaesthesia including pulmonary aspiration, pulmonary oedema and pneumothorax. Other causes to consider include hypoventilation, airways disease (asthma, chronic obstructive pulmonary disease), pneumonia and pulmonary embolism. Oxygen should be applied to the patient and the cause of the hypoxaemia sought and treated.

Hypoventilation and apnoea after anaesthesia can be due to central nervous system depression by residual effects of anaesthetic agents, respiratory muscle weakness secondary to inadequate reversal of neuromuscular blockade and pain that causes splinting of the diaphragm. The recovery room nurse and anaesthetist needs to encourage the patient to take deep breaths, treat pain where necessary and ensure that the patient has adequate neuromuscular function. (Test grip strength, head lift, eye opening and if available, use a neuromuscular monitor.)

Cardiovascular Emergencies

Cardiovasular problems in recovery account for approximately 4% of recovery room problems and can cause major morbidity if not treated promptly. (1) They include high and low blood pressure, arrhythmias, cardiac ischaemia, cardiac failure and pulmonary embolism.

Hypertension:

Hypertension in recovery is defined as a blood pressure of more than 20% above baseline or a systolic blood pressure (BP) of greater than 140 mmHg or diastolic BP greater than 90 mmHg. It occurs in 4-35% of recovery room patients. (1)

The presentation is usually asymptomatic, but sometimes can present with a headache, encephalopathy, chest pain (representing myocardial ischaemia), cardiac failure or bleeding from the surgical site.

The approach to the management of hypertension includes the following steps: (1, 4)

1. Confirmation of the severity of hypertension, check the pre-operative blood pressure and determine whether the patient is already being treated for hypertension.

2. Identify a potential cause for acute hypertension including, pain, anxiety, hypothermia, hypoxaemia, hypercarbia, bladder distension, hypervolaemia, antihypertensive agent withdrawal and raised intracranial pressure. Look for risk factors for hypertension such as renal disease, hyperthyroidism, intraoperative drugs, pregnancy induced hypertension. Important conditions to exclude are thyroid storm, phaeochromocytoma, malignant hyperthermia, pre-eclampsia and autonomic dysreflexia.

3. Check for the presence of end organ damage including cardiac ischaemia, failure and bleeding from the site of surgery. Perform ECG and blood tests as appropriate.

4. Initiate therapy with short acting antihypertensive agents such as a beta-blocker, nitroglycerin, calcium channel blocker, vasodilator (hydralazine) and alpha blockers such as clonidine. Care must be taken to avoid a large drop in blood pressure.

5. Resume usual oral medications as soon as practical to avoid a recurrence of hypertension

Hypotension:

Hypotension is defined as a drop in blood pressure of 20% or more from baseline, a systolic blood pressure of less than 80 mmHg systolic or 50 mmHg diastolic. There may be evidence of shock and low perfusion to vital organs. Hypotension may be the cause of reduced consciousness, nausea, confusion, low urine output, cardiac ischaemia and cardiac arrhythmias.

Hypotension can be a result of low cardiac output secondary to pump failure (cardiac ischaemia, arrhythmia, low contractility), low circulating blood volume (blood loss, extravasation of fluid from the vasculature) or low vascular tone (vasodilatation from anaphylaxis, secondary to anaesthetic agents or peripheral vasodilator medications). The commonest causes of hypotension in the recovery room are low circulating blood volume secondary to blood loss and residual effects of anaesthetic agents.

The initial treatment of hypotension includes a rapid assessment of the airway, breathing and pulse check. If the patient is unresponsive and cardiac arrest has been confirmed, cardio-pulmonary resuscitation should be commenced. If the patient has not yet arrested, apply monitoring, check the blood pressure and establish intravenous access. The cardiac rhythm is checked and a bolus of intravenous fluids (typically 10-20 ml/kg of crystalloid) is infused. A rapid assessment of the 4 Hs and 4 Ts is performed. (The 4 Hs include: hypoxia, hypovolaemia, hypothermia and hypo/hyperkalaemia. The 4 Ts include: toxins (drugs, poisons), tension pneumothorax, cardiac tamponade and thrombosis -cardiac and pulmonary.)

The management of the hypotension will depend on the cause. It is prudent to give an intravenous bolus of fluid (10-20 ml/kg) if hypovolaemia is suspected. If there is a suspicion of cardiac failure, there may not be any response to intravenous fluid. Vasopressors and ionotropes are then considered. The choice of vasopressor will depend on the need for vasoconstriction, positive chronotropy and ionotropy. An assessment of the cardiac rhythm is made and if necessary, appropriate treatment of tachyarrhythmia or bradyarrhythmia is commenced.

Most anaesthetic agents cause vasodilatation, so hypotension should respond to a modest fluid bolus and vasoconstrictor medication. If anaphylaxis is suspected, the appropriate treatment is administration of oxygen, securing the airway, large volumes of intravenous fluid and adrenaline (0.3-0.5mg **intramuscularly** if the patient has hypotension and severe airway symptoms and has not suffered a cardiac arrest).

Cardiac Failure:

Acute heart failure may present with cardiogenic shock (low blood pressure, low organ perfusion, decreased conscious state, reduced skin blood flow, reduced urine output), acute pulmonary oedema, acute right heart failure (raised jugular venous pressure, increased liver size, peripheral oedema) and high output heart failure (warm

peripheries, vasodilatation, tachycardia and pulmonary congestion with a low to normal blood pressure).

The management consists of a rapid assessment of airway, breathing and circulation (ABC), calling for help and commencement of cardio-pulmonary resuscitation if appropriate (unresponsive, pulseless, abnormal breathing). The patient needs to be monitored with continous ECG, saturations, and non-invasive blood pressure monitoring. High flow oxygen is administered and the patient may benefit from continuous positive airways pressure or intubation. Sitting upright can be helpful if the patient is able to tolerate it. Initial investigations include 12 lead ECG, electrolyte and troponin measurement, chest x-ray and if available, echocardiography.

Acute arrhythmias need to be treated with either electrical cardioversion or medication depending on the rhythm. For tachyarrthmias, amiodarone 300mg over one hour can be infused intravenously. Bradycardias may require atropine, pacing or infusions of adrenaline, isoprenaline or dopamine.

Digoxin 500 micrograms can be used as a positive ionotrope. For the patient with hypertension, glyceryltrinitrate (GTN) is used to reduce preload in conjuntion with frusemide (20 mg -80 mg). In the hypotensive patient, ionotrope and vasoconstrictor infusion (dobutamine, adrenaline, dopamine and noradrenaline) will help increase afterload to maintain vital organ perfusion. (4)

Myocardial Ischaemia:

Ischaemia may present with acute chest pain or shortness of breath. Occasionally it is silent –no patient symptoms, but ECG and biochemical changes or cardiac arrhythmias only.

The initial treatment consists of assessment of ABC (as above), administration of oxygen to maintain oxygen saturations above 94%, sublingual nitrates (for pain and to reduce preload - contraindicated if the patient is hypotensive), aspirin 150-300 mg oral for its antiplatelet activity and intravenous morphine in small boluses to control pain. Monitoring with ECG (for arrhythmia detection), non-invasive blood pressure monitoring and oximetry is desirable. Investigations include a 12 lead ECG, serum cardiac enzyme measurement (troponin levels may not rise for 6 - 10 hours after an infarction). If it is clear that there is a new ST segment elevation, reperfusion with thrombolytic medication or per cutaneous intervention (angioplasty and coronary stent insertion) is indicated. Thrombolysis is contraindicated after surgery so is only appropriate if there is no risk of bleeding. Other management includes the treatment of hypertension with GTN infusion, controlling the heart rate (between 60-80 beats per minute) with beta blockers, correcting electrolyte imbalance and blood glucose, and treating arrhythmias.

Cardiac arrhythmia:

Sinus bradycardia, sinus tachycardia, junctional rhythms and atrial fibrillation are the most common arrhythmias seen after surgery. They usually represent problems with the residual effects of anaesthetic medication (bradycardia), reduced venous return or pain/anxiety (tachycardia), but they may indicate more serious problems. The initial management involves looking for and treating obvious causes and checking the other vital signs (blood pressure, temperature, respiratory rate, urine output, the surgical wound for bleeding).

Bradycardia is defined as a heart rate of less than 50 beats per minute (or a drop in more than 20% of the resting heart rate) and tachycardia is defined as a heart rate of over 100 beats per minute or increase of more than 20% of the resting heart rate.

If there is clinical instability (shock, syncope, cardiac ischaemia and heart failure) with a tachyarrhythmia, synchronised cardiac defibrillation under sedation is indicated, otherwise, is the patient is stable, a more formal diagnosis can be made and chemical cardioversion or medications can be administered to slow the heart rate depending on the type of arrhythmia (narrow or broad complex).

If there is clinical instability (shock, syncope, cardiac ischaemia and heart failure) or a risk of asystole with a bradycardia, treatment with atropine (500 mcg boluses up to 3 mg total dose) or pacing is indicated (with an infusion of isoprenaline, adrenaline or electrical pacing). If the patient is stable, the patient is observed.

Neurologic Emergencies

The most common "neurologic" problem in recovery is emergence delirium or prolonged emergence. Intracerebral catastrophes and neurologic injury are less common but more serious.

Emergence delirium and Agitation:

Postoperative agitation is common, particularly in pre-school age children who have received short-acting anaesthetic agents. Agitation is usually self-limiting, but some important causes of agitation need to be excluded, including pain, hypoxia, hypotension and metabolic disturbances like low blood sugar. In children, it is useful to try to reorient the patient and reunite the child with his parents. It is very frightening for the child to wake up in a foreign environment with strangers around him.

Occasionally, medication to sedate the child may be required, provided that hypoxia, pain and other physiologic disturbances have been excluded.

Delirium is an acute state of confusion where the patient has loss of contact with his surroundings and carers. It can be as state of agitation, silence or a mixture of both. It is reported to occur in up to 40% of patients and can be potentially life threatening. (5) It occurs with increased frequency in elderly patients and is associated with persisting cognitive decline and increasing physical dependence. Delirium may sometimes go undetected by health professionals and may only be recognised by the family. The risk factors for delirium include advanced age, anaemia, diabetes, infection, duration of surgery, alcohol abuse, intraoperative opioids and benzodiazepines, anticholinergic drugs, as well as higher postoperative pain scores. Other patient factors that predispose to delirium include major depression and dementia. Being in a foreign environment (such as the hospital) and acute medical illness increases the chance of developing delirium. If a patient is disturbed after anaesthesia, it is important to exclude any physical or medical cause such as pain, hypoxia, full urinary bladder, hypothermia, ischaemia and acute infection.

Treatment of delirium includes prophylaxis with geriatric intervention programmes that include early mobilization, nutrition and re-orientation to the patient's surroundings with the help of family members. Acute postoperative delirium may require medication with antipsychotic drugs or sedation. Care must be taken with benzodiazepine use, as it may merely transfer the problem from the recovery room back to the ward.

Delayed emergence:

Delayed emergence can have multiple causes including: hypoxia, hypercarbia, low cardiac output, metabolic or endocrine dysfunction, hypothermia, drugs (opioids, anaesthetic agents, neuromuscular blockers) and primary cerebral events (such as a bleed or ischaemic stroke).

Residual drug effects are a common cause of delayed awakening in recovery. There may have been a relative overdose, or the patient may be particularly sensitive to medication, especially if there is a delay in metabolism (usually hepatic) or excretion (usually renal) of the drugs given. If the procedure is prolonged and the patient is hyperventilating, volatile agents will be excreted more slowly. If the volatile agent is very soluble in blood, they will take longer to excrete than the less soluble agents. The speed of onset and offset of the volatile or gas from fastest to slowest is nitrous oxide, sevoflurane, isoflurane, halothane and ether. The offset of the intravenous agents depends on redistribution initially and then by metabolism and excretion if they are given as an infusion. Both propofol and thiopentone have a rapid initial redistribution phase, but prolonged infusion with thiopentone will have a longer time to awakening than propofol because of the longer elimination half time. (7)

If other drugs are given, such as opioids or benzodiazepines, there will be a potentiation of the central nervous depressant effects and delayed emergence.

Residual neuromuscular blockade is common after intraoperative use of neuromuscular blockers and may be present in up to 64% of patients. (6) The presence of residual neuromuscular blockade will increase the incidence of upper airway obstruction, hypoxaemia and postoperative pulmonary complications including aspiration. The patient who is awake will experience visual disturbances such as double vision, facial weakness or numbness, difficulty speaking and swallowing, fatigue and general weakness and have difficulty breathing and coughing effectively. Some patients are particularly sensitive to neuromuscular blockade, including those with neuromuscular dysfunction such as myasthenia gravis, renal failure and hepatic failure, acidosis or who have received medication that will potentiate neuromuscular blockade such as gentamicin. Hypothermia will also increase the time to recovery from neuromuscular blockade. The intraoperative use of a nerve stimulator will help determine the need for further doses of neuromuscular blocker intra operatively and whether there is any recovery from blockade and the need for reversal with neostigmine (with atropine or glycopyrolate) at the end of the anaesthetic.

The metabolic causes of delayed emergence include hypo or hyperglycaemia and it is recommended that a blood glucose level be checked as both conditions are easily treated with the administration of glucose or insulin respectively. Hyponatremia should be sought if there has been a large amount of hypotonic fluid absorbed, such as with the administration of large volumes of glycine for irrigation during transurethral prostatic resection. Hypothermia with a core temperature of less than 33 C will potentiate the effects of sedative agents and have an anaesthetic effect of its own. (7) Central anticholinergic syndrome is a relatively rare condition that occurs after the use of anticholinergic drugs such as hyoscine or atropine or drugs with some anticholinergic effect such as pethidine and some antidepressants. It is also possible after the administration of ketamine and volatile anaesthetics. It presents as confusion, hallucinations, seizures and coma. The patient may have a dry mouth, tachycardia, blurred vision and dry, flushed skin with dilated pupils. The treatment is physostigmine. (7)

Neurologic complications will result from cerebral hypoxia or ischaemia and raised intracerebral pressure following a bleed or cerebral oedema. It is rare, but should be considered if all other causes of delayed emergence have been excluded.

A systematic approach to the patient with delayed emergence is best. Ensure a patent airway, adequate ventilation, normal heart rate and blood pressure and good cardiac output. The anaesthetic chart should be reviewed to search for potential causes of delayed emergence and basic blood tests performed. The patient needs to be examined from head to toe to look for signs of drug overdose, residual neuromuscular blockade

or cholinergic syndrome and the temperature needs to be measured. If no cause is apparent, consider a brain CT scan or MRI if there is a high suspicion of an intracerebral event.

Endocrine and Metabolic Emergencies

There are a large number of potential metabolic and endocrine emergencies that can arise during anaesthesia and the recovery period. The commonest is hypothermia and this is usually related to heat loss before and during surgery. It is usually mild (32-35C) but may delay emergence or cause a sympathetic response and shivering. This is usually not a problem in a previously well patient, but may have important physiologic implications for the elderly or those with systemic disease, particularly cardiac ischaemia. It is usually treated with active re-warming with a forced air-warming blanket. One of the discharge criteria for recovery is a temperature of above 36 degrees, so it may delay return to the ward. Other metabolic problems include electrolyte disturbances (often related to fluid management or blood transfusion), anaphylaxis, hyper- or hypoglycaemia, thyroid storm and hypothyroid coma, and the potentially fatal problem of malignant hyperthermia, which may not manifest until after surgery.

Hypothyroid coma may present as delayed emergence, low core temperature, hypotension and hypoventilation. There is usually a history of thyroid disease. The patient may experience low blood glucose and hyponatremia. The patient needs to be admitted to intensive care for ventilation, cardiovascular support, temperature and glucose control and very cautious thyroid hormone replacement.

Thyrotoxic storm is a life-threatening exacerbation of hyperthyroidism that may present 6-24 hours after surgery. It has a high mortality (20-30%). The clinical manifestations are a high temperature of over 41C, tachycardia, initial hypertension followed by hypotension, cardiac failure, nausea and vomiting, diarrhoea, jaundice, agitation, delirium and coma. Treatment consists of attention to ABC and rehydration with saline and glucose, controlling temperature with exposure, fans and paracetamol, intravenous beta blocker (propranolol) to control the hyperadrenergic response, hydrocortisone for the adrenal insufficiency followed by prophythiouracil and intensive care management.

Conclusion

Recovery from anaesthesia is a time when the patient may be quite unstable until his airway reflexes and respiratory function has returned, the effects of anaesthetic agents have worn off and cardiovascular instability has subsided. Common problems in the recovery period include airway obstruction, hypoxaemia, hypoventilation, hypo and

hypertension, pain, nausea and vomiting and delayed emergence or delirium. Many problems can become life threatening if they are not managed appropriately in a timely manner. Close supervision of the patient is required until they have emerged from anaesthesia, been observed for immediate surgical complications and recovered from the effects of medications administered in the perioperative period.

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MALIGNANT HYPERTHERMIA

Malignant hyperthermia (MH) is a rare but potentially lethal reaction that can be triggered under general anaesthesia. It is a pharmaco-genetic disorder of skeletal muscle that, when triggered, results in hypermetabolism, muscle rigidity and muscle breakdown.

Malignant hyperthermia was first described in Melbourne, Australia in 1960, in a letter published about the symptoms that occurred during anaesthesia of a man from a family where ten family members had died under general anaesthesia. Since then much has evolved about the diagnosis and management of this condition.

Epidemiology

The disorder has a pattern of autosomal dominant inheritance with variable penetrance. It has an incidence of approximately 1:40,000 anaesthetics administered in adults, and 1:15,000 in children. There is a male predominance, and it is common for susceptible patients to have had more than one (and on average three) previous uneventful general anaesthetics. Due to increased vigilance of the condition and improved monitoring and treatments, the mortality of MH in developed nations is now 2-3%, down from 80%.

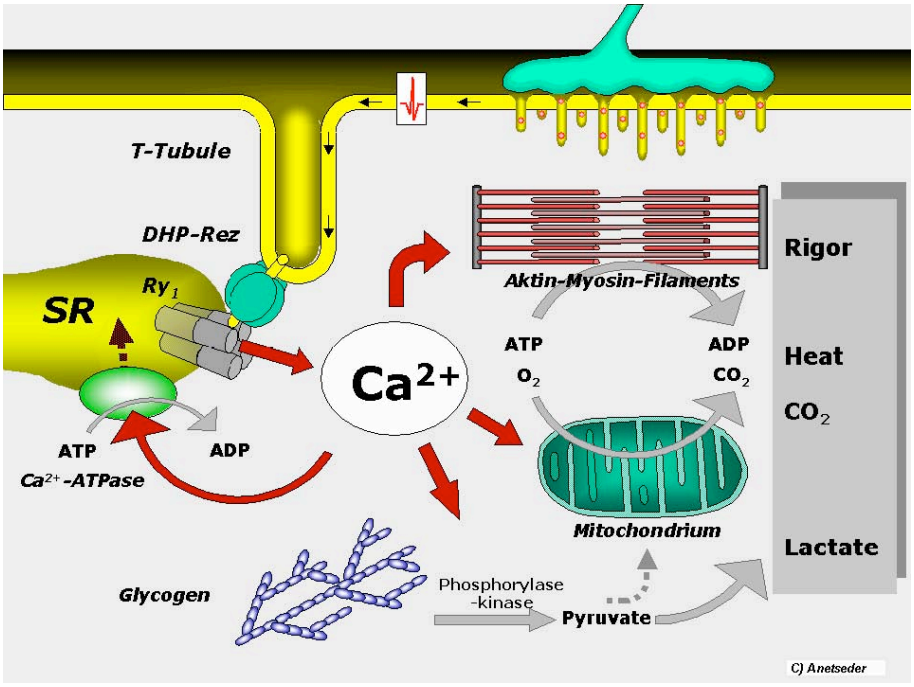
Aetiology

Anaesthetists have a particular interest in malignant hyperthermia because the only known pharmacological triggering agents of this condition are volatile inhalational anaesthetic agents (halothane, enflurane, isoflurane, sevoflurane, desflurane) and the depolarizing muscle relaxant succinylcholine. All other drugs, including nitrous oxide, benzodiazepines, thiopentone, ketamine, non-depolarising neuromuscular blockers, local anaesthetics, opioids and sympathomimetics are considered safe.

Pathophysiology

The primary defect in malignant hyperthermia is a mutation in the RYR1 gene on chromosome 19, which codes for the ryanodine receptor. The ryanodine receptor is a calcium release channel found in the sarcoplasmic reticulum of skeletal muscle. In a normal person, calcium is intermittently released from the sarcoplasmic reticulum into the muscle cell in response to an action potential to allow muscle to contract via actin-myosin interaction. In MH, the mutation of the ryanodine receptor leads to the

continuous release of calcium into the muscle cell, leading to continuous muscle contraction.



source: http://www.humpath.com/IMG/jpg_malignant_hyperthermia.jpg

The consequences of the persistently raised myoplasmic calcium levels are many. There is continuous actin-myosin interaction resulting in continuous muscle contraction and rigidity. Hypermetabolism results from increased demand for ATP from the persistent actin-myosin interaction, as well as from membrane calcium pumps trying to restore calcium homeostasis.

Heat, oxygen consumption, carbon dioxide production, and lactate production result from the use of ATP for energy. Because skeletal muscle constitutes 40% of body mass, the heat produced can lead to a lethal increase in body temperature of over 1 degree celcius every 10 minutes.

Oxygen delivery may be insufficient to meet metabolic demands of the contracting muscle, resulting in tissue hypoxia. Carbon dioxide and lactic acid production leads to respiratory and metabolic acidosis.

Rhabdomyolysis occurs due to excessive muscle contraction, as well as calcium activation of phospholipases resulting in higher turnover of membrane phospholipids.

Rhabdomyolysis leads to extrusion of potassium ions from damaged muscle, and the resulting hyperkalaemia can lead to life-threatening arrhythmias. Rhabdomyolysis also causes release of myoglobin, which can damage renal tubules and cause acute renal failure.

Disseminated intravascular coagulation can occur from release of tissue clotting activators from necrosing muscle as well as from the excessive heat.

Clinical Features

Early recognition of an impending malignant hyperthermia crisis and instituting immediate treatment is vital for patient survival. The clinical signs associated with MH are not specific, so anaesthetists must be able to recognize a pattern of signs in order to make a rapid diagnosis.

It is best to classify signs into early signs, developing signs, then later signs as the condition progresses.

Early signs:

The earliest signs of an impending MH reaction are those of metabolic stimulation (hypercapnoea, tachycardia) and masseter muscle rigidity (if suxamethonium is used).

Hypercapnia resistant to treatment by increasing minute ventilation is the most reliable initial clinical sign of an MH reaction. Excessive carbon dioxide (CO2) production is caused by cellular hypermetabolism. In the mechanically ventilated patient there will be a marked rise in end-tidal CO2 on capnography. In the spontaneously ventilating patient a marked increase in respiratory rate will occur to try to blow off the extra CO2. The large amount of exhaled CO2 will quickly heat and exhaust the CO2 absorbent.

Inappropriate sinus tachycardia is another early sign of MH, a response to the increased metabolic demands of muscle.

Masseter muscle rigidity (MMR) is the inability to open a patient's mouth 2 minutes after the administration of succinylcholine due to rigidity of the masseter muscles. Masseter muscle tension may increase in normal patients after the administration of succinylcholine but typically only lasts a few seconds. When it persists, there is a 25-50% chance that the patient is MH susceptible. The sign by itself is not specific enough to make a definitive diagnosis of MH in the absence of the other signs of

hypermetabolism. However if MMR is observed, any triggering agents should be stopped and the patient observed for other signs of MH.

Developing signs:

After these earliest signs of MH, other clinical signs of a developing MH reaction are an increase in oxygen (O2) consumption, with mottling of the skin and a decline in oxygen saturation (SaO2). The patient may become haemodynamically unstable, and arrythmias may be observed from hyperkalemia (ventricular ectopics, bigeminy, ventricular tachycardia or fibrillation).

Hyperthermia can happen minutes to hours following the initial onset of symptoms. It may be marked, with a rise in temperature of 1 degree every 10 minutes, and profuse sweating may be seen. Generalised rigidity may develop. Blood gases will reveal a mixed respiratory and metabolic acidosis, as well as hyperkalemia from muscle breakdown.

Late signs:

Blood myoglobin levels and creatine kinase levels often peak at 14 to 24 hours after an acute MH episode. Dark coloured urine occurs from myoglobinuria.

Disseminated intravascular coagulation (DIC) from severe hyperthermia is a poor prognostic indicator and often, terminal event.

Differential diagnosis

Because the signs of MH are not specific, there are many differential diagnoses one must consider. These include:

Causes of raised ETCO2:

- Inadequate mechanical ventilation
- Patient who have been given opioids
- Laparoscopic surgery (CO2 gas insufflation)
- Expired soda lime (high inspired CO2)

Causes of tachycardia:

- Inadequate anaesthesia
- Inadequate analgesia
- Infection
- Septicaemia
- Anaphylaxis
- Phaeochromocytoma
- Thyroid crisis
- Drug reactions with or during anaesthesia (neuroleptic malignant syndrome, mono-amine oxidase inhibitors, selective serotonin reuptake inhibitors)
- Recreational drugs (ecstasy, cocaine, ketamine)

Treatment

Once malignant hyperthermia is suspected, treatment is of utmost urgency. An emergency must be declared and help must be obtained. Surgery should be completed or abandoned as soon as possible. Then there are a multitude of tasks to be done simultaneously. These include stopping the cause, definitive treatment, monitoring, and symptomatic treatment.

Stop the cause:

The volatile agent should be turned off and the vaporizer disconnected. Do not waste time changing the anaesthetic machine or circuit. Hyperventilate the patient with 100% O2 at a high fresh gas flow to eliminate the volatile agent (>15L/min).

Change to intravenous (non-trigger) anaesthesia such as a propofol infusion at 50 ml/hr.

Definitive treatment:

The only known antidote for MH is Dantrolene, so it needs to be administered as soon as possible. It binds to the ryanodine (RYR1) receptors and directly inhibits sarcoplasmic reticulum calcium release, thereby reversing skeletal muscle hypermetabolism.

One bottle contains dantrolene 20mg and mannitol 3g. It is an orange powder that needs to be mixed with 60ml of sterile water. The first dose of dantrolene is 2.5mg/kg intravenously, and repeated doses of 1mg/kg should be administered until the tachycardia, hypercapnoea and pyrexia start to subside. Up to 10mg/kg or more may be required (for example in muscular males) but the average dose is about 3mg/kg. In most cases, dantrolene reverses the acute hypermetabolic process within minutes. Further doses may be required in the next 48 hours if the reaction recurs.

Monitoring and lines:

There needs to be constant monitoring of end tidal CO2, SaO2, electrocardiogram, blood pressure, and temperature.

Arterial blood gases should be repeated frequently for potassium levels, acid-base status, arterial CO2, arterial O2 levels, glucose and lactate levels.

Large bore intravenous (IV) access needs to be established, and an arterial line inserted. A central line may be useful for multiple infusions that may be required.

A urinary catheter is required to monitor renal function and urine colour.

Symptomatic treatment:

Active cooling measures should be undertaken to treat hyperthermia. Blankets and drapes should be removed, refrigerated IV fluid should be administered, wet cold sheets can be placed on the patient with fans blowing, ice can be applied to the axillae and groin. Active cooling should be stopped when core temperature decreases to below 38.5 degrees.

Hyperkalemia should be treated with insulin and dextrose (10 International Units of insulin with 50ml of 50% dextrose in an adult). Calcium chloride should be given for cardiac protection against arrhythmias 0.1 mmol/kg IV. That is, 7 mmol=10ml of calcium chloride for a 70kg adult.

Treatment of acidosis includes hyperventilation to return to normocapnoea. Sodium bicarbonate 0.5mmol/kg IV should be considered if the pH<7.2.

Arrhythmias should be treated with lignocaine 1-2mg/kg or amiodarone 2-3mg/kg over 15 minutes. Beta-blockers can be given if tachycardia persists. Calcium channel blockers should NOT be given as in combination with dantrolene can cause marked cardiac depression.

Blood should be taken 14-24 hours after the crisis to measure the peak plasma creatinine kinase (CK) and myoglobin levels. Urine output needs to be maintained >2ml/kg/hr in order to limit renal tubular damage by myoglobin. IV fluid should be given, and diuretics as required (such as, frusemide 0.5mg/kg). The patient's muscle compartments should be carefully monitored for compartment syndrome from rhabdomyolysis.

Coagulation studies should be done to look for abnormal paramenters. If disseminated intravascular coagulation occurs coagulation factors should be administered (fresh frozen plasma, platelets, cryoprecipitate).

Ongoing care:

After the event, the patient should be transferred to an intensive care or high dependency unit for ongoing monitoring. Recrudescence occurs in up to 25% of patients after initial treatment, and further doses of dantrolene may be required for 48 hours.

The mhANZ (malignant hyperthermia Australia and New Zealand group) have divided the various tasks during an MH crisis onto 8 MH task cards, which are delegated to 8 different people during a crisis, to aid with the effective management of the patient. The link to their resource kit, which includes the task cards is:

http://www.anaesthesia.mh.org.au/mh-resource-kit/w1/i1002692/

Testing for malignant hyperthermia

After the successful treatment of a suspected MH reaction, the patient should be referred to an MH centre for confirmation of the clinical diagnosis.

Patients and family members should be counseled about the condition and the implications under anaesthesia.

Confirmation of the diagnosis is by in vitro contracture testing (IVCT), performed at specialist centres. A fresh muscle biopsy from the vastus muscle is required, done under a non-triggering anaesthetic. The tension generated by the muscle in response to separate exposures of halothane and caffeine is increased in individuals with MH susceptibility.

If MH is confirmed by the IVCT, DNA testing (a blood test) can be done on the patient to look for a mutation of the RYR1 gene. If a mutation is found, family members may be screened for MH by looking for the same mutation. If the same mutation is found, they are deemed MH susceptible without needing a muscle biopsy to confirm diagnosis. However if no mutation is found, a biopsy is still required for diagnosis as it is not safe to reject the diagnosis on DNA testing alone.

Anaesthesia in susceptible patients

The following patients should be treated as MH susceptible:

1. Previous malignant hyperthermia reaction

2. Positive in vitro contracture test (IVCT) on muscle biopsy

3. Positive DNA test for MH

4. The patient has a relative with MH, and the patient has not been proven to be negative for MH by IVCT

Anaesthesia should not be denied to these patients. Triggering drugs (succinylcholine and all volatile anaesthetics) should be avoided, and regional anaesthetic techniqes are appropriate where feasible.

If a general anaesthetic is to be administered in an MH susceptible patient, the anaesthetic machine must first be prepared. Vaporisers must be removed to avoid accidental administration. Soda lime must be fresh, and new airway equipment, breathing circuit and bag should be used. The machine and ventilator must then be flushed with 10 L/min oxygen or medical air for at least 20 minutes (and at least 30 minutes if isoflurane has been used recently in the anaesthetic machine).

MH susceptible patients should preferably be placed first on the operating list. They should be kept asleep with total intravenous anaesthetic agents (such as propofol infusion). If muscle relaxation is required a non-deopolarising neuromuscular blocker can safely be given. High flows of gas should be used throughout the anaesthetic to avoid accumulation of small quantities of volatile agent. Monitoring of ETCO2, temperature, ECG and SaO2 is mandatory. If having a day procedure patients should stay for 4 hours in the post-anaesthetic care unit before being sent home. Ensure that dantrolene is available, but if precautions are taken to avoid the triggers, the use of prophylactic dantrolene is not indicated

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