

# *Emergencies in Anaesthesia Course*

*Yangon,  
Myanmar*

# EMERGENCIES IN ANAESTHESIA

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# PRINCIPLES OF CRISIS MANAGEMENT

Hella Deifuss

## 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)

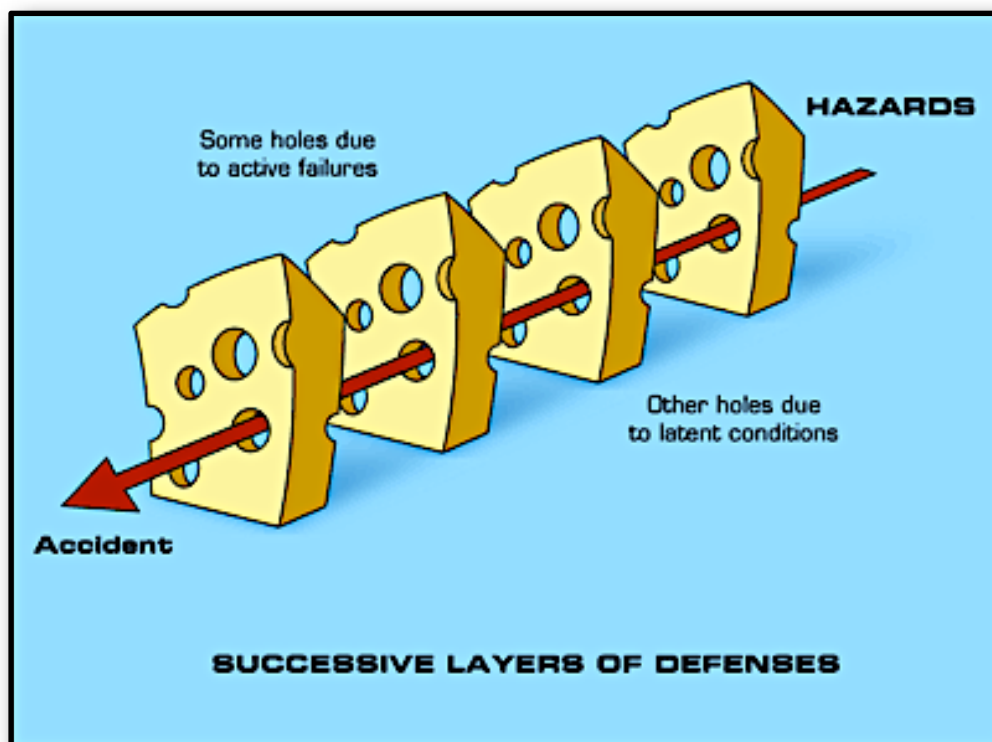
A crisis requires an active response to prevent injury or harm to the patient and it is unlikely to resolve on its own.

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:

- 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.
- 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.
- 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.
- 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!
- 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!**

- 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.

- 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.

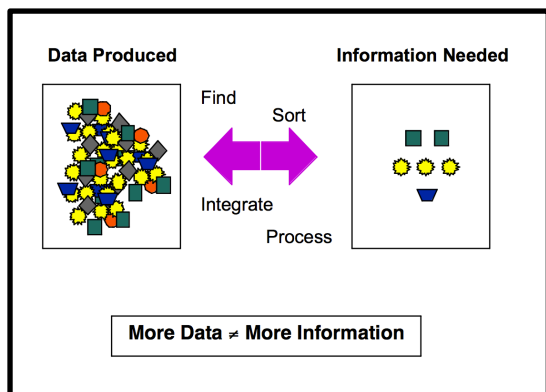
### *Everything but this*

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 anaesthetist 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 judgment, 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:

1. “Don't tell me what to do!” .....Anti-authority
2. “Do something quickly- anything!” .....Impulsivity
3. “It won't happen to me – it's just a routine situation!” .....Invulnerability
4. “I'll show I can do it. I can deal with anything!” .....Macho
5. “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.

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# DIFFICULT INTUBATION

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David Pescod & Stanley Tay

Emergency airway management in the unprepared and unfamiliar patient is often challenging. Although many predictors of difficult intubation have been developed, they all have low positive predictive values, and thus the unanticipated difficult intubation will continue to occur.

Securing and monitoring the airway are among the key requirements of anaesthesia. Failures to secure the airways will drastically increase morbidity and mortality of patients within a very short time.

Endotracheal intubation is often called the 'gold standard' for airway management in an emergency, but multiple failed intubation attempts do not result in maintaining oxygenation; instead, they endanger the patient by prolonging hypoxia and causing additional trauma to the upper airways.

Proficiency in alternative techniques for establishing airway access is of crucial importance when routine measures fail. Intensive training in these situations, as well as protocols based on standardized guidelines and algorithms, allow recognition of common problems and institution of appropriate therapeutic measures without delay.

Thus, knowledge and availability of alternative procedures are essential in every anaesthetic. Given the great variety of techniques available, it is important to establish a well-planned, methodical protocol within the framework of an algorithm. This not only facilitates the preparation of equipment and the training of personnel, it also ensures efficient decision-making under time pressure.

The algorithm for emergency airway management describing the sequence of various procedures has to be adapted to internal standards and to techniques that are available.

## Techniques of Airway Management.

### *Oxygen delivery and clearing the airway*

In all patients who are still spontaneously breathing, sufficient oxygenation should be maintained by oxygen delivery in an adequate concentration. Additionally, in cases of partial or complete airway obstruction with fluids or solid foreign bodies in unconscious patients, the airway has to be cleared by suctioning, or foreign body extraction with Magill forceps under visual control during laryngoscopy.

### *Bag Mask Ventilation*

Bag-valve-mask (BVM) ventilation is a fundamental skill of routine and emergency airway management and must receive a high priority in training. Trainees and anaesthetists must maintain the skill of bag mask ventilation.

A ventilation bag with attached mask can be used to provide both assisted and controlled ventilation of the patient. Generally, a high-flow oxygen source (10 L/minutes) allows better compensation of facemask leaks and generation of sufficient positive pressure to overcome respiratory system resistance to gas flow. Jaw thrust and neck extension is usually necessary to provide a patent airway.

The mask should be sized to cover the nose at the level of the nose bridge and the mouth just above the chin.

In obese patients, the combination of redundant oropharyngeal soft tissue, a bulky tongue, and a thick chin and neck pad may interfere with the ability to ventilate. Several methods may be used to overcome this resistance. Lifting the chin pad while applying a jaw thrust can straighten the soft tissues of the anterior wall in the hypopharynx and facilitate ventilation. Early insertion of an oral airway or tilting the head laterally while ventilating may reduce the risk of the tongue falling backward against the soft palate.

*Finally*, two-person mask ventilation may be more effective and should be attempted. The anaesthetist maintains the mask seal with two hands while the assistant squeezes the bag.

### *Endotracheal intubation*

Endotracheal intubation is considered the gold standard of airway management however this skill is difficult to learn and requires practice to maintain the skills to intubate.

Endotracheal tube intubation protects against aspiration, allows ventilation with peak pressures of 50 cm H<sub>2</sub>O and the administration of endobronchial drugs.

There are no contraindications for tracheal intubation in an emergency. In trauma patients with suspected cervical spine injury, however, all manoeuvres should be carried out with an assistant stabilizing the neck in neutral position.

Performing tracheal intubation, especially in an emergency, requires excellent skills and experience with this relatively complex technology and, additionally, it is necessary to monitor and reliably confirm the placement of the tube tip in the trachea. Undetected oesophageal intubation and inadvertent, unnoticed extubation of the trachea are the most serious incidents in airway management, as they can result in severe hypoxic injury or even death. This underscores the importance of confirming correct endotracheal tube placement.

### *Fiber-optic intubation*

Flexible intubation laryngoscopes (bronchoscope) are the most effective solution in all cases of *anticipated* difficult intubation in a spontaneously breathing patient. They are much less useful in an emergency intubation.

Of course, the use of flexible intubation laryngoscopes requires extended skills with continuous training and appropriate logistics, especially with regard to cleaning and disinfecting the scopes after each use.

The rigid fiber-optic scope allows for indirect laryngoscopy and simplifies orotracheal intubation of patients with various problems that may prohibit successful direct laryngoscopy, such as restricted mouth opening, an immobilized cervical spine, a large tongue, or mandibular retrognathia. However this intubation technique by indirect laryngoscopy is not only more complex than standard laryngoscopy but also requires clinical experience and continuous practice.

### *Supraglottic devices*

Supraglottic devices allow for the blind establishment of an airway that allows oxygenation and ventilation, gives some protection against aspiration and may provide another route for intubation.

### *Combitube.*

The oesophageal/tracheal Combitube (ETC) is primarily used as an emergency tube for ventilating patients during resuscitation. It provides a complete seal of the upper airway and can therefore be used in patients with a risk of regurgitation and aspiration of gastric contents. It has two tubes, one of which resembles a conventional endotracheal tube while the other seals off the oesophagus with an oropharyngeal balloon. The ETC can be inserted blindly through the mouth and is more likely to pass into the oesophagus (95%) than into the trachea (5%). It can safely be inserted in patients with cervical spine injuries because flexion of the neck is not required.

### *Laryngeal mask.*

The cuff around the elliptical body of the laryngeal mask (LMA) seals the larynx posteriorly and enables ventilation of the patient without intubating the trachea. The LMA is available in all sizes from infant to adult and allows, with some experience, rapid manual positioning without additional aids in anaesthetized or unconscious patients.

Numerous reports document the successful use of the LMA in emergencies under difficult conditions, during cardio-pulmonary resuscitation CPR, and in trauma patients even by non-physicians.

### *Intubating laryngeal mask.*

The intubating laryngeal mask airway (ILMA) is an advanced version of the LMA allowing a special endotracheal tube to be passed through the ILMA into the trachea. Its use is recommended especially in cases of difficult intubation, after failed intubation attempts, and for rescuers inexperienced in tracheal intubation.

This device follows a two-step concept: (1) it may be used as a rescue airway when tracheal intubation has failed and in 'cannot intubate, cannot ventilate' situations, allowing rapid oxygenation and ventilation; and (2) it serves for securing the airway as a conduit for tracheal intubation through the ILMA (blind or under vision).

In the 2010 ILCOR (International Liaison Committee on Resuscitation) guidelines, endotracheal intubation is cited as the optimum technique for airway management, but supraglottic devices are explicitly mentioned as alternatives. These alternative devices are described as suitable for use by providers with only limited experience in endotracheal intubation, but also for use in case of failed initial attempts.

According to evidence-based criteria the ETC and LMA are evaluated and classified as follows:

- ETC and LMA are easier to place compared to endotracheal intubation;
- Ventilation with both devices is comparable to that obtained with an endotracheal tube and is definitely superior to BMV ventilation;
- Complication rates are comparable to those of endotracheal intubation;
- ETC and LMA are effective in cases of failed endotracheal intubation.
- Both the ETC and the LMA are therefore recommended as acceptable, safe, and helpful alternatives.
- There is no question that at least one of the supraglottic airway devices described above should be immediately available in an emergency when endotracheal intubation fails.

## Surgical airway

The 'surgical airway' is strictly a means of last resort. It is indicated when the airway cannot be secured by endotracheal intubation or by an alternative techniques and BVM ventilation is not possible.

## ALGORITHM

An algorithm provides stepwise procedures or decision trees to guide the anaesthetist through the management of a particular problem.

The management of the difficult airway is the most important patient safety issue in the practice of anaesthesia. Many national societies have developed algorithms and guidelines for management of the difficult airway. These algorithms differ.

There are two opposite approaches to producing algorithms: one may choose to include a wide choice of techniques at each stage within the algorithm, allowing individual choice of the best technique for each situation, or alternatively one may produce simple and definite flow charts with few items of equipment.

Use of a simple and definitive algorithm with few items of equipment and yes or no responses facilitates familiarity and practice in its use especially as a failed airway algorithm will be used at times of high anxiety.

All algorithms stress the absolute importance of having a series of back-up plans for airway management. Generally, plan B is a secondary airway, plan C emphasizes oxygenation and ventilation while plan D describes rescue techniques for the cannot intubate and cannot ventilate disaster.

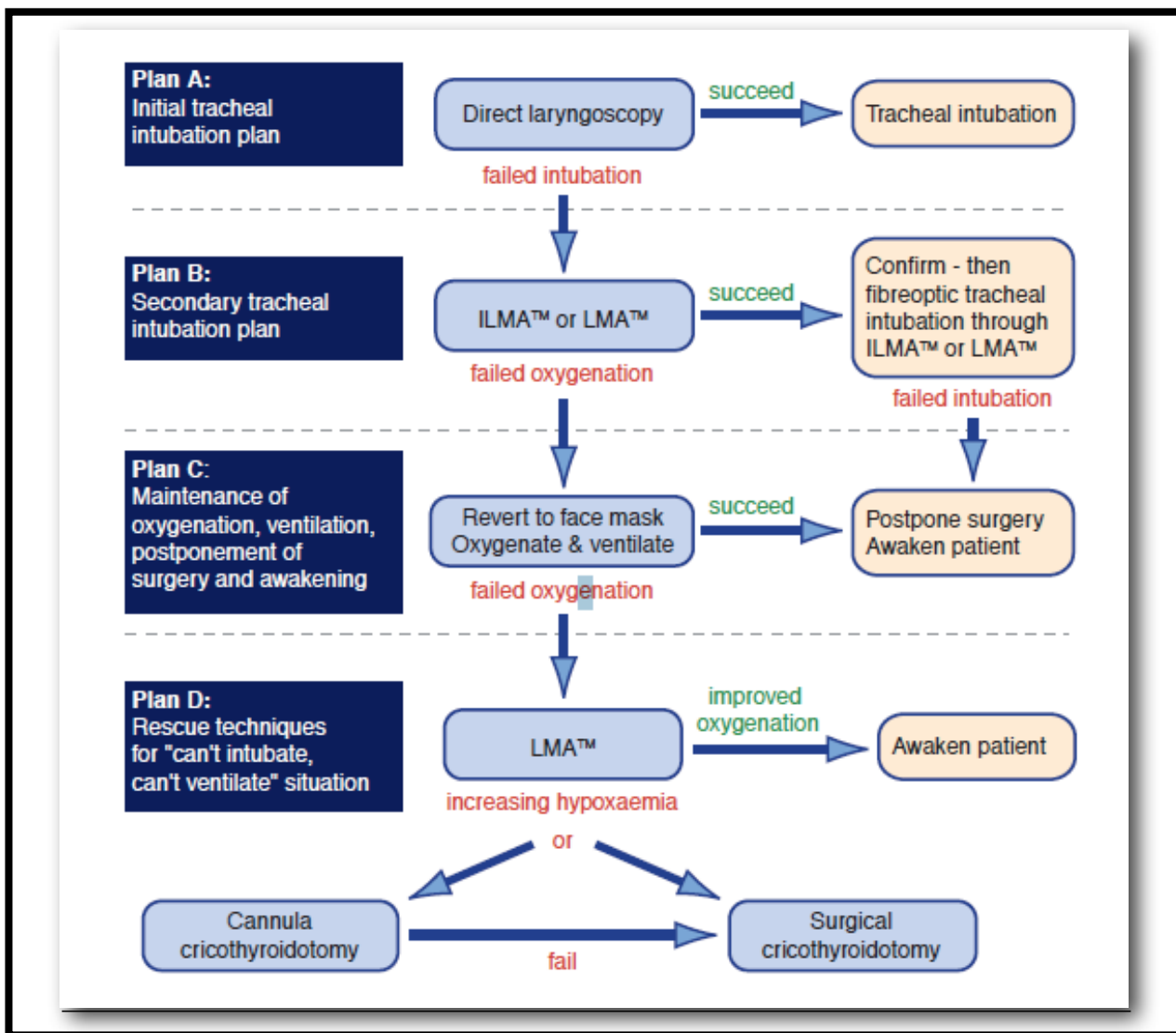
The essential features of back up plans are that they must be decided on before performing the primary plan and the anaesthetist is strongly advised to adhere to the plans.

It is not possible to cover all clinical scenarios with a single flow-chart that remains simple and clear. Flow-charts should be developed and practiced for at least the three scenarios of:

- Unanticipated difficult intubation in routine induction;
- Unanticipated difficult intubation in rapid sequence induction;
- Failed intubation, increasing hypoxaemia and difficult ventilation in the paralysed, anaesthetized patient.

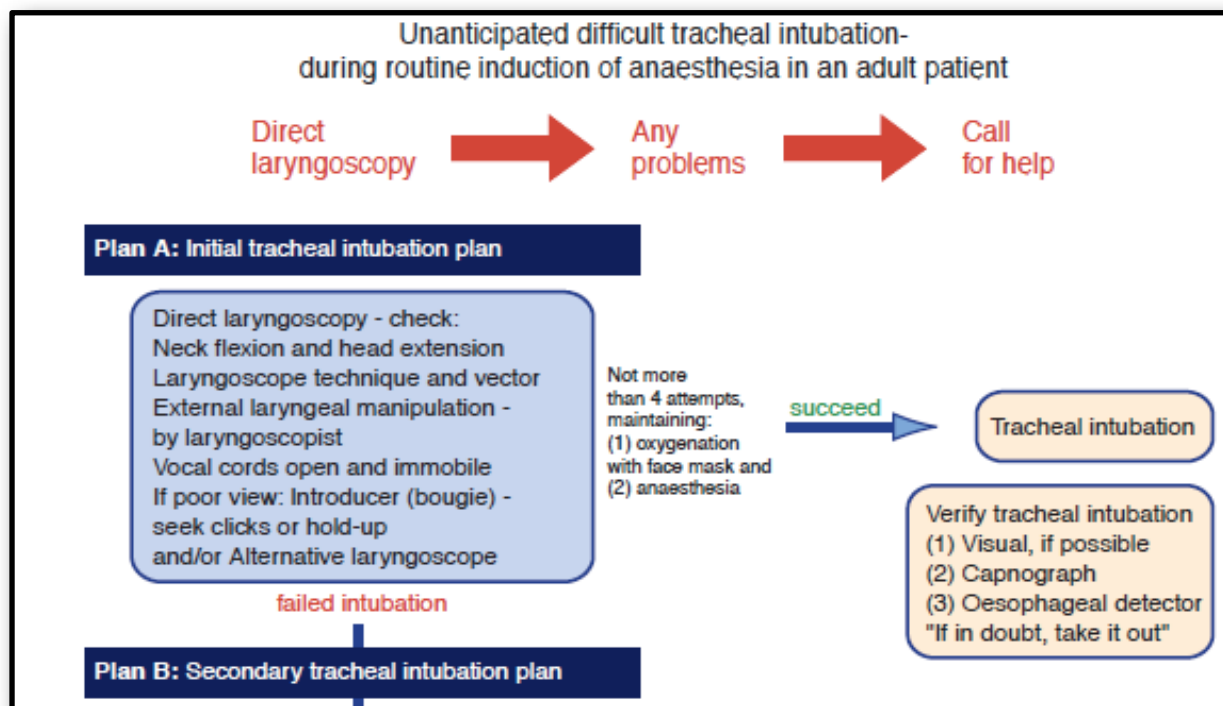
Anaesthetists also need a plan for anticipated difficult airways. This plan should emphasise awake intubation techniques or at least anaesthetic techniques that maintain spontaneous ventilation.

**Unanticipated difficult intubation in routine induction, no risk of aspiration (non-obstetric).**



*Difficult Airway Society*

## Plan A



Intubation is attempted by optimal direct laryngoscopy and this has 5 components:

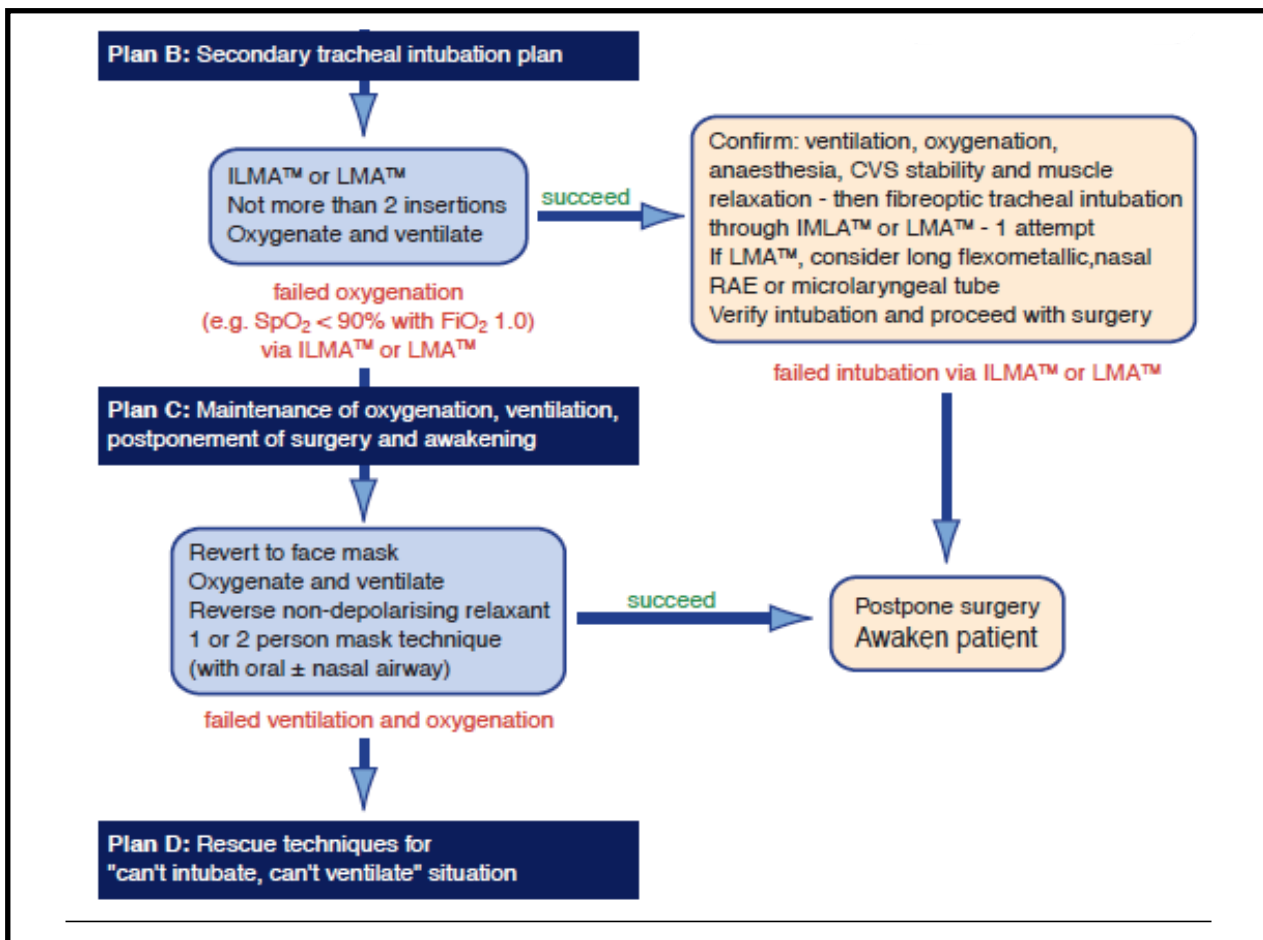
- Optimal head and neck positioning
- Optimal muscle relaxation
- Optimal laryngoscope blade
- Optimal external laryngeal manipulation and
- Optimal use of the bougie.

A number of intubation attempts (3-4) may be undertaken (to change the blade, use a bougie, apply external laryngeal manipulation) but after 3-4 attempts, it is likely that the anaesthetist is repeating fruitless attempts and no further attempts should be made.

The anaesthetist must recognise that intubation has failed and proceed with plan B.

- Start face-mask ventilation,
- ask for a LMA.
- Ensure oxygenation.

## Plan B



Plan B involves the use of a secondary intubation plan.

This is initiated if there is a need to intubate the patient and there is no risk of aspiration. It involves the insertion of an Intubating Laryngeal Mask or other Laryngeal Mask Airway through which an endotracheal tube can be inserted.

The importance of the laryngeal mask is that it usually allows:

- Good ventilation/oxygenation.
- Attachment of a breathing system
- Provides a route for intubation

Both the classic and intubating laryngeal masks are suitable.

Once the patient has been stabilized and oxygenated, the secondary intubation attempt is made through the LMA or intubating LMA. Although blind intubation through the intubating LMA has a high success rate, success can be improved with the use of a fiberoptic scope.

If intubation is successful, the position of the tube in the trachea is confirmed and surgery continues.

If intubation fails or ventilation by LMA is not possible – go to plan C.

If the LMA or intubating LMA has been tried unsuccessfully for intubation but is successful for ventilation, leave it in, and allow the patient to wake up. Surgery should be postponed. Awake intubation should be considered.

## Plan C

If at the initiation of plan B, ventilation by LMA has not been successful then plan B is bypassed to plan C.

Remove the LMA if there is unsatisfactory ventilation through it and return to facemask ventilation with a large oral airway. Consider using two-hand bag mask ventilation. If this fails try 4-hand bag mask ventilation with one person applying the face mask, another applying jaw thrust and neck extension and another squeezing the bag.

If it is not possible to maintain oxygen saturations above 90% with 100% oxygen, this is a situation of failed ventilation and failed intubation. Immediately proceed to plan D.

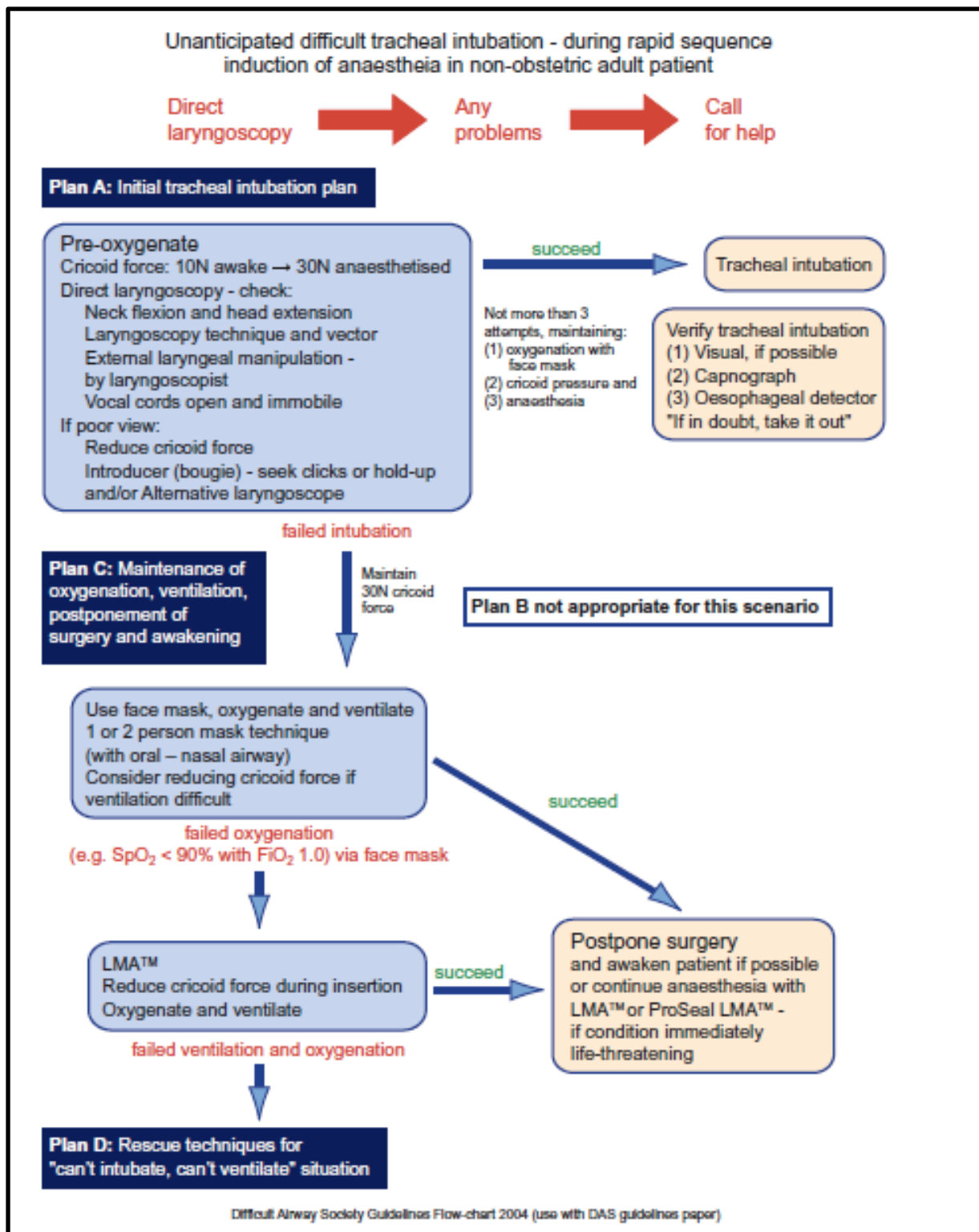
## Plan D

Can't intubate, can't ventilate situation with increasing hypoxaemia!

- Don't waste time trying to intubate.
- Recognise the crisis.
- Oxygenation is the priority.
- Proceed to an emergency surgical airway.



## Unanticipated difficult intubation in rapid sequence induction



The anaesthetic technique includes optimal pre-oxygenation, the use of an induction agent and suxamethonium, with the application of 30 Newton of cricoid pressure at the onset of unconsciousness.

There is no intubation plan B because the patient is going to be woken up without any other attempts at intubation. The only task is to move to plan C – the maintenance of

oxygenation whilst the induction agent and suxamethonium wears off and the patient awakens.

- Call for assistance and keep your first responder present in the room.
- Continue cricoid pressure at 30 N.
- Insert an oral airway and attempt facemask ventilation with 100% oxygen.
- If necessary use two hands to hold facemask and ask responder to squeeze bag.
- If facemask inflation is not possible, maintain 30 N cricoid pressure and insert a laryngeal mask. Release the amount of cricoid force as the laryngeal mask is inserted and reapply it when it is inserted.
- Attempt to ventilate with 100% oxygen via laryngeal mask with cricoid pressure continuing.
- If that fails, release cricoid pressure and try ventilation again through the laryngeal mask.
- If ventilation is still not possible, and the patient is not awakening or is struggling to breathe and the saturations have fallen below 85% go on to Plan D (failed intubation and failed ventilation).

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## REFERENCE:

- Difficult Airway Society



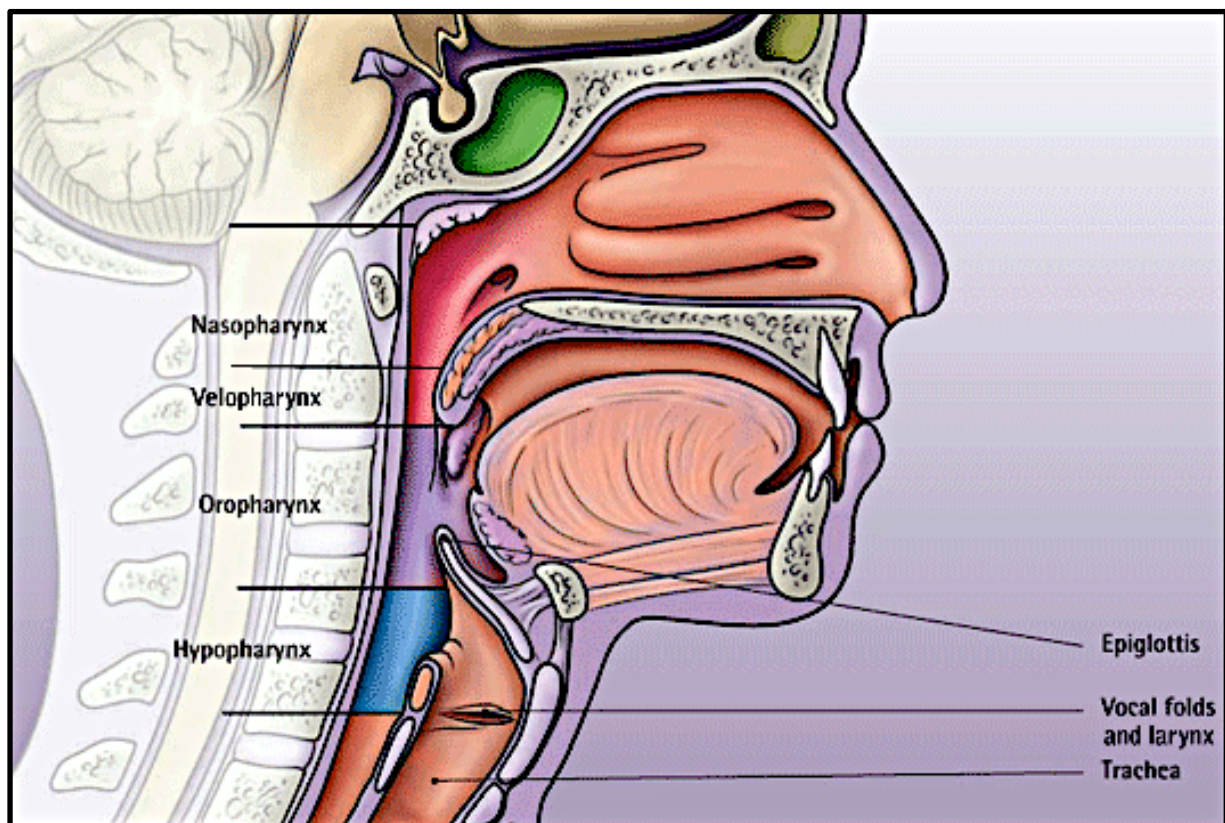
# AIRWAY OBSTRUCTION

Michelle Chan

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 nasopharynx, 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 (nose, trachea, teeth, maxilla, mandible), 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.

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.

Causes of pathological airway obstruction are listed in the tables below.

### Infra-glottic Airway Obstruction:

<ul style="list-style-type: none"> <li>• Compression from neck structures</li> </ul>	Thyroid gland - goitre Haematoma Neck tumour
<ul style="list-style-type: none"> <li>• Foreign body</li> </ul>	Small objects, food, teeth
<ul style="list-style-type: none"> <li>• Trauma</li> </ul>	
<ul style="list-style-type: none"> <li>• Tracheal stenosis</li> </ul>	
<ul style="list-style-type: none"> <li>• Tracheomalacia</li> </ul>	

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.

## Glottic Airway Obstruction:

• Laryngospasm	Recurrent laryngeal nerve palsy
• Vocal cord palsy	
• Tumours	Laryngeal
• Infection	Croup (laryngotracheitis)
• Angioedema	Allergic, hereditary, drug-induced
• Burns	

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.

## Supraglottic Airway Obstruction

<ul style="list-style-type: none"> <li>• Obstructive sleep apnoea</li> </ul>	Obesity Tonsillar and adenoidal hypertrophy Nasal obstruction Craniofacial abnormalities: Down's syndrome Pierre-Robin Treacher-Collins Acromegaly Neuromuscular disorders
<ul style="list-style-type: none"> <li>• Infections</li> </ul>	Epiglottitis Parapharyngeal abscess Retropharyngeal abscess Ludwig's angina
<ul style="list-style-type: none"> <li>• Tumours</li> </ul>	Pharyngeal wall Tongue Tonsillar Mandibular
<ul style="list-style-type: none"> <li>• Foreign bodies</li> </ul>	Loose teeth, dentures Throat pack
<ul style="list-style-type: none"> <li>• Neck haematoma</li> </ul>	Post-surgical: Carotid endarterectomy Thyroidectomy Cervical Spine Post-procedure: CVC insertion
<ul style="list-style-type: none"> <li>• Angioedema</li> </ul>	Allergic Hereditary Drug induced: ACE inhibitors
<ul style="list-style-type: none"> <li>• Burns</li> </ul>	
<ul style="list-style-type: none"> <li>• Trauma</li> </ul>	
<ul style="list-style-type: none"> <li>• Blood/vomit/mucous/secretions</li> </ul>	

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 immune-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.

## Obstruction with/of an Endotracheal Tube in situ

<ul style="list-style-type: none"> <li>• Problem with anaesthetic circuit/filter/ventilator</li> </ul>	Obesity Tonsillar and adenoidal hypertrophy Nasal obstruction Craniofacial abnormalities:      Down's syndrome Pierre-Robin Treacher-Collins  Acromegaly Neuromuscular disorders
<ul style="list-style-type: none"> <li>• Problem with tube</li> </ul>	Tube position :                    oesophageal endobronchial  Obstructed tube:                kinked patient biting on tube cuff herniation foreign matter
<ul style="list-style-type: none"> <li>• Problem with patient</li> </ul>	Inadequate muscle relaxation Bronchospasm Chest wall rigidity from opioids Pneumothorax  Pulmonary oedema Foreign body aspiration

*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 the patient is anaesthetised, by taking a careful history, performing a thorough 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 about 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 due to infection, 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 years old, has a sudden onset, with high temperature >38 degrees. The patient looks anxious, distressed and toxæmic, swallowing is difficult with drooling, cough is muffled and guttural, and the child 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 features of particular syndromes or craniofacial abnormalities.

Size and position of abscesses/tumours/masses should be noted. For thyroid goitres assess for size and for retrosternal extension by palpation and 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 can be quantified. X-rays of the neck can show tracheal deviation or compression. Fiber-optic naso-endoscopy allows awake visualization of the airway, and may be useful to determine the exact location and size of a laryngeal tumour, or assess 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:

- Awake surgical airway
- Awake intubation
- 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, the underlying 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 4%. Sedation should be avoided. Awake intubation methods include fiberoptic 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 uncooperative 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

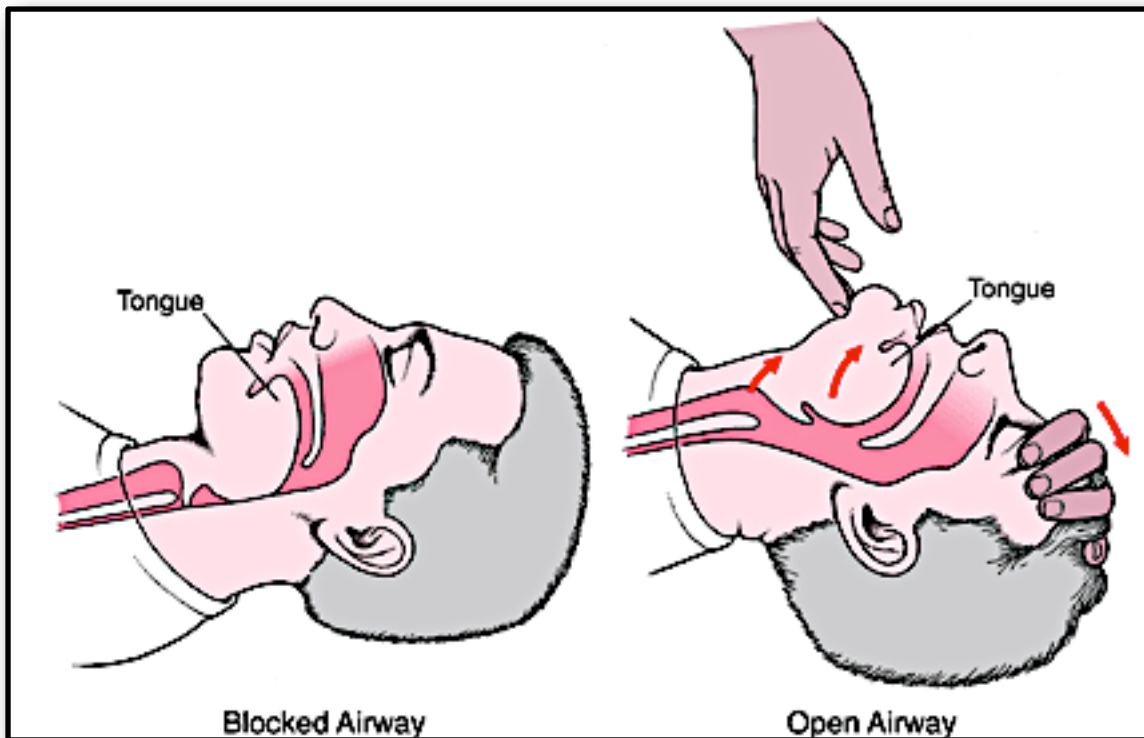
If airway obstruction is impending, whilst deciding how to secure the airway, the patient should be sat up and kept calm. If available, a helium-oxygen mixture (helium 60-80%) can be used to decrease the resistance to breathing by reducing turbulent flow. This however will give a lowered inspired oxygen 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 airway obstruction in the unconscious/anaesthetised patient

The signs of airway obstruction in the unconscious patient will depend on whether the patient is spontaneously breathing or being bag-mask 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 when attempting bag-mask ventilation will present as poor or complete inability to ventilate. There will be loss of the end tidal carbon dioxide (ETCO<sub>2</sub>) trace.

Airway obstruction is managed by firstly visualizing and suctioning of the pharynx to clear it of any secretions, blood, gastric content or foreign bodies. 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. High flow oxygen should be applied.



Source [http://www.merckmanuals.com/home/injuries\\_and\\_poisoning/first\\_aid/cardiac\\_arrest.html](http://www.merckmanuals.com/home/injuries_and_poisoning/first_aid/cardiac_arrest.html)

Immediate assistance should be requested if the airway is still obstructed. 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 to ventilate. 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 and operator skills. 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/angle piece – Blockage, compression, kinking or incorrect connections may cause ventilation problems. The circuit may have a leak, or the ventilator not functioning properly or incorrectly set.
- 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, decreased lung compliance from pneumothorax or pulmonary oedema, or increased airway resistance from bronchospasm, anaphylaxis, aspiration or a foreign body.

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.

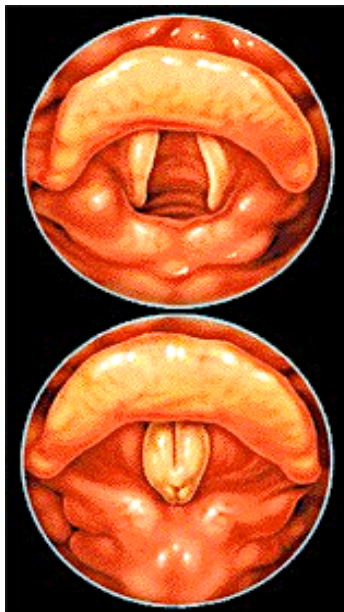
Next the ETT is inspected for any obvious kinking or the patient biting on the tube. Tube placement through the larynx, and not into the oesophagus, should be checked with laryngoscopy. A suction catheter should be passed down the tube to identify any blockages. The cuff should be deflated to exclude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

If the ETT is not the problem, 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.

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



Precipitating factors include:

1. blood or secretions in the airway
2. regurgitation and aspiration
3. excessive surgical stimulation
4. “light” anaesthesia
5. irritant volatile anaesthetic agents
6. airway stimulation or extubation during the excitation phase of anaesthesia
7. highly irritable airway (as with a recent upper respiratory tract infection or smoking)

Top: Normal laryngeal opening

Bottom: Laryngospasm

Source:

<http://anesthesiologia.fullblog.com.ar/laringoespasm-811220930995.html>

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 (IPPV) 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...

Phillipa Hore & Kim Fuller

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.

## OXYGENATION IS THE KEY TO PREVENTING HYPOXIA, BRAIN DAMAGE & DEATH

Approximately 5% of patients are difficult to ventilate and 2% are difficult to intubate. This rises to 5% in the obstetric or obese 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 FOR  
A “CAN’T INTUBATE, CAN’T OXYGENATE”  
(CICO) SITUATION.**

This means that every anaesthetic department must have:

**1. CICO equipment**

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

**2. 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.

**3. 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.

**CICO EQUIPMENT MUST BE READILY ACCESSIBLE  
CICO ALGORITHM MUST BE SIMPLE TO FOLLOW**

**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 **THREE** attempts at intubation as there is a significant risk of airway trauma. If the patient has not been able to be ventilated, the oxygen saturation will be falling.

*Thus there should be recognition of a CICO situation if:*

- 4. There have been 2 or more attempts at intubation**  
and
- 5. There is a failure to oxygenate with a supra-glottic device**  
and
- 6. Oxygenation persistently below <85%**

Importantly, because 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 facemask 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.

**IT IS IMPORTANT TO RECOGNISE A CICO SITUATION  
AND IMPLEMENT THE CICO PLAN IMMEDIATELY**

### **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.



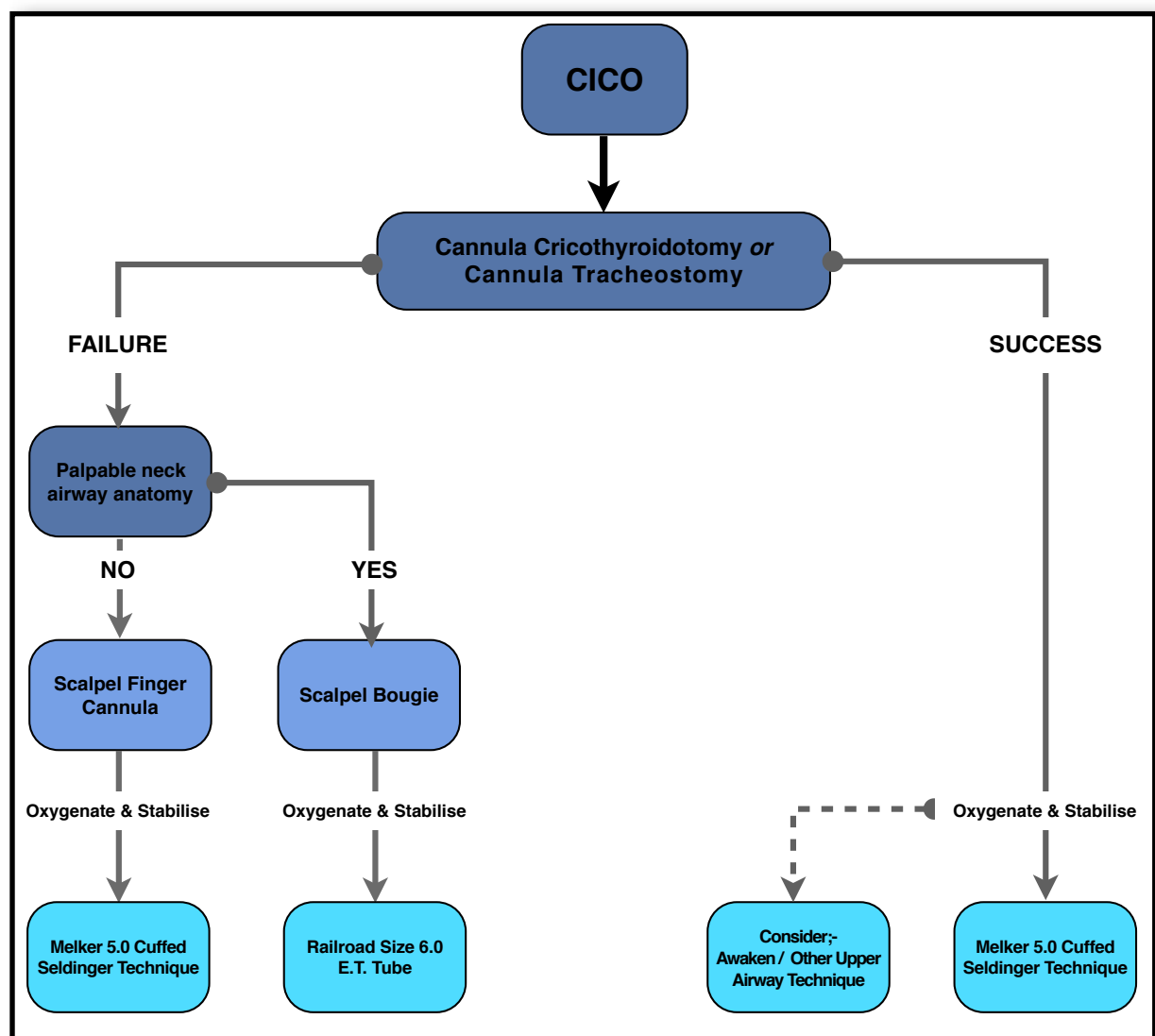
## Cannula versus scalpel cricothyroidotomy

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



**BE TRAINED IN BOTH CANNULA & SCALPEL CRICOTHYROIDOTOMY  
BUT DON'T DELAY DECIDING WHICH METHOD TO USE**

### Cannula Cricothyroidotomy Equipment

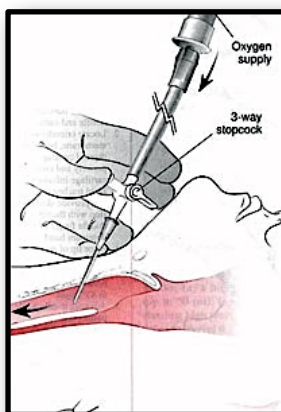
- 14 gauge intravenous cannula
- 5 ml syringe with 1ml saline
- System for oxygen delivery...
  - Jet insufflator if available
  - Oxygen tubing connected to 3-way tap and flow meter

### Method

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

**CANNULA CRICOTHYROIDOTOMY  
ONLY ALLOWS FOR OXYGENATION NOT VENTILATION.  
A DEFINITIVE AIRWAY IS STILL REQUIRED.**

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:

- Attempted intubation via direct laryngoscopy – higher tracheal pressure may open a closed glottis facilitating visualization of glottis structures
- Formal surgical tracheostomy
- Use of a Seldinger technique to secure airway with a cuffed Melker™ tube *or* similar

### **Scalpel + Bougie Technique for Cricothyroidotomy:**

#### *Equipment*

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

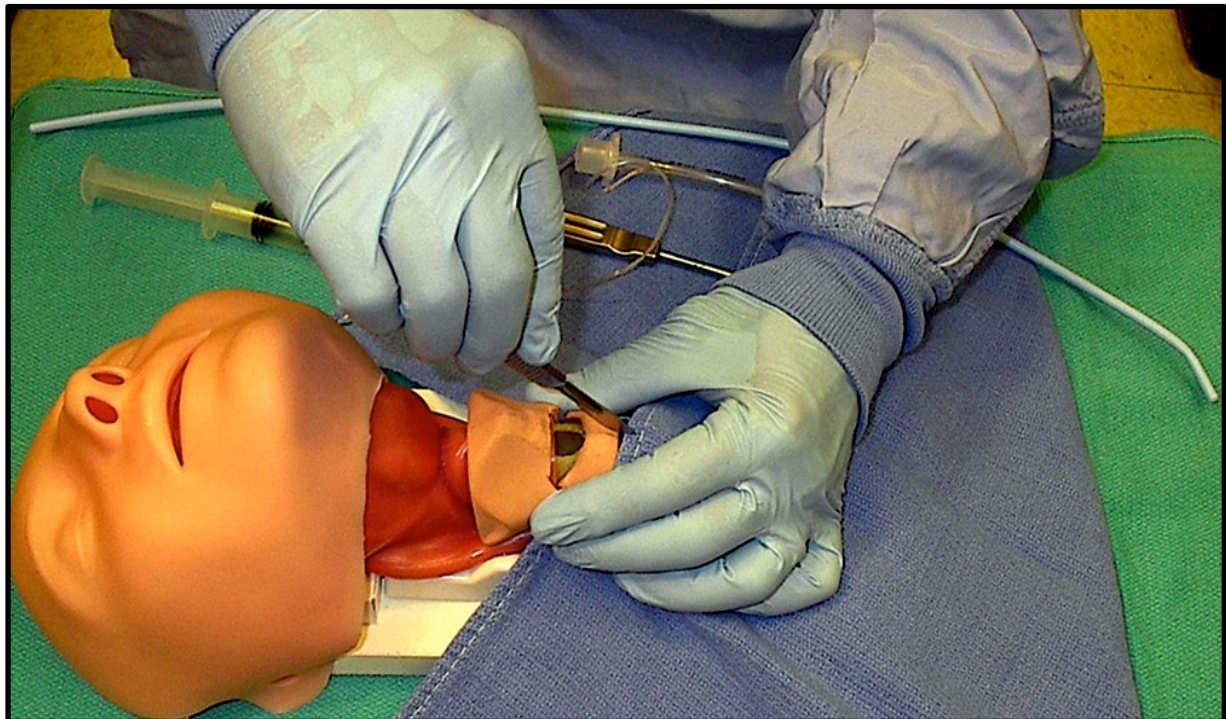
#### *Method*

1. Remove the pillow, extend the neck, identify the cricothyroid membrane and stabilize with the non- dominant hand
2. Make a horizontal stab incision through the cricothyroid membrane with the dominant hand
3. Rotate blade through 90° without removing it so that blade points caudally
4. Pull scalpel towards you so that a triangular space is opened
5. Switch hands so that the non-dominant hand stabilizes scalpel
6. Hold the bougie parallel to floor at right angles to the neck and insert curved tip into hole along scalpel blade

7. Rotate bougie to align with trachea, lift and pass it into trachea
8. Oxygenate via bougie if it has hollow core (e.g. Frova)
9. Railroad lubricated 6.0 ETT (15mm connector removed), rotate continuously to help passage
10. Remove bougie
11. Reattach connector and ventilate via circuit
12. 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, oesophagus, 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 Cricothyroidotomy

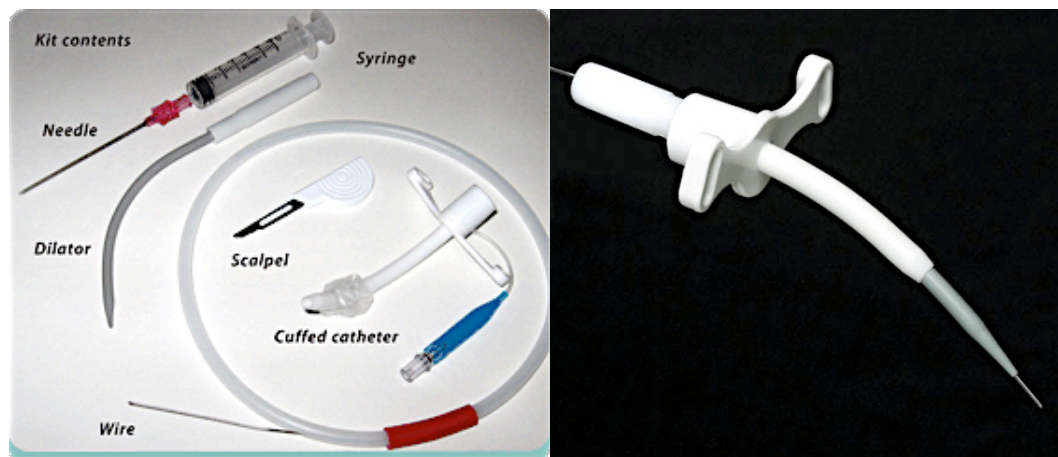
When it is difficult to identify landmarks in the neck, most often due to oedema, surgical emphysema, trauma or gross obesity, it may be necessary to perform a larger vertical midline incision with a scalpel and use blunt finger dissection down to the cricothyroid membrane. This is an invasive procedure 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

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

1. Insert wire through in situ cannula (preferably) or through needle passed into cricothyroid membrane after making a small stab incision.
2. Carefully withdraw cannula over wire ensuring plenty of wire is left in trachea
3. Make a stab incision caudally with scalpel along wire
4. Pass lubricated dilator and tube assembly over wire
5. Ensure dilator is seated completely within airway
6. Grip assembly device firmly preventing backward movement of dilator
7. Advance assembly over wire, through skin and into airway (moderate force required)
8. Remove wire and dilator
9. Inflate cuff
10. 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 likely if:

**ALL STAFF MEMBERS ARE FAMILIAR WITH THE LOCATION AND USE OF CICO EQUIPMENT**

**EQUIPMENT IS SIMPLE TO USE AND EASY TO MAINTAIN**

**ALL STAFF ARE FAMILIAR WITH THE 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 enables oxygen delivery in the shortest possible time.

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# INTRA-OPERATIVE HYPOTENSION & HYPERTENSION

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Sam Kennedy

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 20% 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 50-60mmHg. 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) = Cardiac Output (CO) x Total Peripheral Resistance (TPR)

CO = Heart Rate (HR) x Stroke Volume (SV)

SV = End Diastolic Volume (EDV) - End Systolic Volume (ESV)

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

## Intraoperative Factors Affecting Blood Pressure and Treatment

### Hypotension

#### Decreased Total Peripheral Resistance (Vasodilation)

- Anaesthetic drugs: these drugs have a direct effect 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

- Drugs including beta blockers
- High sympathetic block in the setting of regional anaesthesia
- Vasovagal effect
  - peritoneal stretch at laparoscopy
  - bowel dilatation at colonoscopy

#### Decreased End Diastolic Volume

- Hypovolaemia
- Air embolism/pulmonary embolism (thrombus)/fat embolism
- Tension pneumothorax
- Restrictive cardiomyopathy
- Pericardial disease

#### Increased End Systolic Volume

- Decreased contractility
  - Ischaemia
  - Fluid overload
  - Valvular heart disease (especially aortic stenosis)
  - Sepsis
  - Drugs including local anaesthetic toxicity
  - Tachyarrhythmia
    - Ventricular tachycardia/Ventricular fibrillation
    - Rapid atrial fibrillation
    - Supraventricular tachycardia

### *Treatment of Intraoperative Hypotension*

- Assess Severity
  - Is the degree of hypotension SERIOUS?
  - If YES then validate reading.
    - Check NIBP monitor - repeat cycle
    - Confirm with palpation for pulse
    - Check SpO<sub>2</sub>
    - Check ETCO<sub>2</sub>
  - If SERIOUS proceed to CRITICAL MANAGEMENT.
  - Otherwise proceed as NON-CRITICAL MANAGEMENT.



## CRITICAL MANAGEMENT

1. Increase inspired O<sub>2</sub>.
2. Consider:
  - Surgical causes
  - IVC compression (obstetrics/laparoscopy)
  - Femoral shaft reaming
  - CO<sub>2</sub> insufflation
  - Tourniquet or Vascular clamp release
3. Check ECG
  - Asystole/VF/VT - start Advanced Life Support
  - Tachyarrhythmia - control rate pharmacologically or synchronized cardioversion
  - Bradyarrhythmia - increase rate pharmacologically
4. Provide circulatory support
  - Volume resuscitation
  - Vasopressors
5. Assess CAUSE and provide SPECIFIC treatment
  - Blood loss (surgical)
  - Impaired venous return (surgery/posture/high airway pressures/pneumothorax)
  - Vasodilation (neuroaxial block, anaesthetic agents, anaphylaxis)
  - Embolism (Air, CO<sub>2</sub>, fat, venous thromboembolism)
  - Cardiac dysrhythmia
  - Cardiac dysfunction (Ischaemia/Infarction)
6. Continue to support blood pressure
  - Call for assistance
7. If cause is unknown, *review*:
  - Airway (Pressures, Minute Volume)
  - Breathing (CO<sub>2</sub> exchange, Oxygenation)
  - Circulation (Rhythm, Ischaemia, Volume)
  - Drugs (Doses, Agents)
  - *Consider* RARE CAUSES

## NON-CRITICAL MANAGEMENT

1. Identify and treat cause (decreasing anaesthetic depth, volume, vasopressors)
2. Consider:
  - Relative Hypovolaemia (neuraxial block, inadequate fluid replacement)
  - Excessive relative depth of anaesthesia (too much volatile or IV agent)
  - High airway pressures
  - Surgical (blood loss, venous return compression, release of tourniquet or vascular clamp)
  - Mild rhythm disturbance (sinus bradycardia, slow AF)
3. If cause is unknown, *review*:
  - Airway (Pressures, Minute Volume)
  - Breathing (CO<sub>2</sub> exchange, Oxygenation)
  - Circulation (Rhythm, Ischaemia, Volume)
  - Drugs (Doses,
  - *Consider* RARE CAUSES

## RARE CAUSES

- Anaphylaxis
- Drug Error
- Transfusion Incompatibility
- Acute mitral valve rupture
- Pericardial tamponade
- Septic shock
- Adrenocortical insufficiency

## Hypertension

Increased Sympathetic Response (increase TPR, HR, contractility)

- Inadequate anaesthesia/awareness
- Painful stimulus
- Hypoxaemia/Hypercarbia
- Emergence
- Bladder distension
- Tourniquet
- Cerebral ischaemia: raised ICP, carotid/vertebral occlusion

Increased Total Peripheral Resistance (Afterload)

- 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 doses or if inadvertent intravenous injection takes place.
- Hypothermia
- Surgical: aortic cross clamp, pneumoperitoneum

Increased End-Diastolic Volume

- Volume overload – iatrogenic or Conn's syndrome

Pre-existing Hypertension

- Essential hypertension
- Renovascular
- Pre-eclampsia
- Autonomic dysreflexia
- Metabolic: thyroid storm, pheochromocytoma

### *Treatment of Intraoperative Hypertension*

1. Validate reading.
2. Assess Severity
  - Mean BP > 120mmHg or 30% above baseline
  - ST changes
  - Known ischaemic heart disease
  - Known cerebral vascular disease (aneurysm, bleeding)
3. Assess CAUSE and provide SPECIFIC treatment
  - Inadequate Anaesthetic Depth
  - Inadequate Anaesthetic Delivery
  - Vasoactive drug administration (check correct drug and dose)

- Inadequate Ventilation and Oxygenation
  - Surgical factors
  - Stimulation (increase depth, analgesia)
  - Tourniquet, pneumoperitoneum, cross clamp
4. Treatment for isolated hypertension
- Vasodilators (hydralazine, GTN, Sodium Nitroprusside)
  - Alpha blockers (clonidine, phentolamine) *-useful if heart rate is low to normal.*
  - Beta blockers (metoprolol, esmolol) *useful if there is also tachycardia*
  - **Contraindicated** in patients with bronchospasm,phaeochromocytoma or iatrogenic adrenaline overdose.
5. Treatment for raised intracranial pressure
- Head up, avoid ties around neck, low airway pressures
  - Mannitol
  - Hyperventilation
  - Frusemide
  - Neurosurgical intervention

## PERI-OPERATIVE ARRHYTHMIA

**Sion Davies & Kim Fuller**

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:

- Acute ischaemia
- Sympathetic stimulation
- Drug effects
- Electrolyte imbalance (especially hypokalaemia or hypomagnesaemia)
- Hypoxia, hypercarbia
- 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

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**DEFINITION: HR <40 BEATS PER MINUTE or  
AN INAPPROPRIATELY LOW HR IN THE CONTEXT**

### TREATMENT: REMOVE VAGAL STIMULATION CHEMICAL OR ELECTRICAL MANAGEMENT

- Initial drug therapy is atropine 500-600 mcg intravenously 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.
- If there is no response to Atropine, Adrenaline is the recommended second line medication
- Third line medications may include the other drugs listed below
- Glucagon may be considered if there has been a Beta-blocker or Calcium channel blocker overdose

### CHEMICAL (Drug) MANAGEMENT

- Atropine
- Adrenaline
- Other; aminophylline, isoprenaline, dopamine, glucagon, 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.

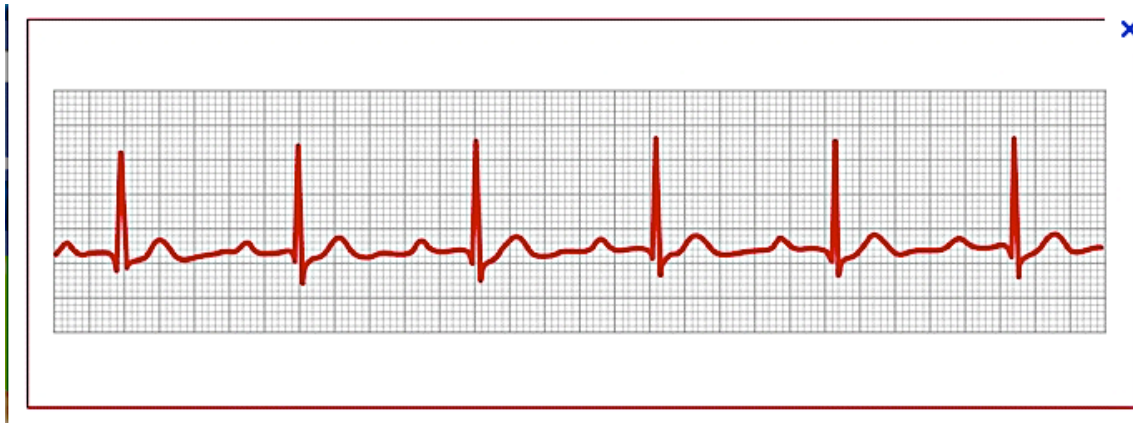
Peri-operative bradyarrhythmias are usually caused by

- vagal stimulation
- medications
- electrolyte disturbances
- hypoxaemia
- schaemia.

Underlying cases should be identified and corrected *pre-operatively if possible*

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,  $\beta$ -blocker or verapamil.
- Second-degree block involves intermittent failure of atrio-ventricular (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.

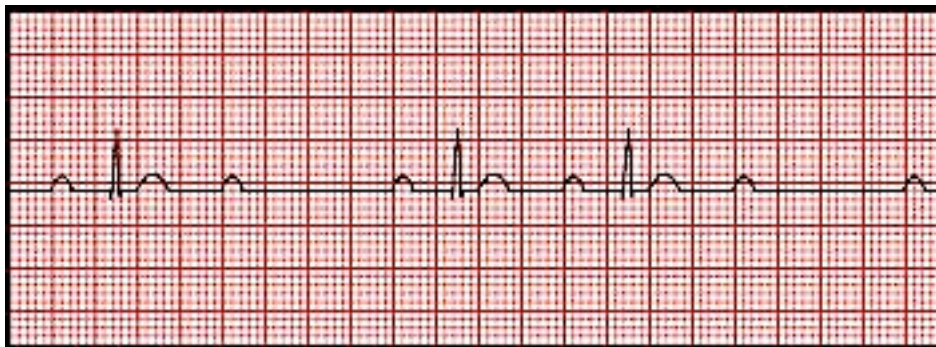


### First-degree heart block



### 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.
- Intermittent failure of AV conduction with loss of QRS complexes
- No progressive increase in delay between P waves and the QRS
- Irregular QRS rhythm
- 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:*

- Total failure of A-V conduction
- 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° heart block*

## Emergency Pacing

The indications for emergency pacing are

- haemodynamically unstable bradycardia (systolic BP <90 mmHg, altered mental state, angina, pulmonary oedema)
- bradycardia unresponsive to drug treatment
- bradycardia with pause dependent ventricular rhythm  risk of VT/VF
- cardiac arrest secondary to drug overdose, acidosis, electrolyte disturbance or other reversible cause
- after cardiac surgery

The relative contraindications for emergency pacing 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 can be used if there is no access to the chest.
- Selecting the demand mode and increasing the ECG gain initiate pacing.
  - 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.

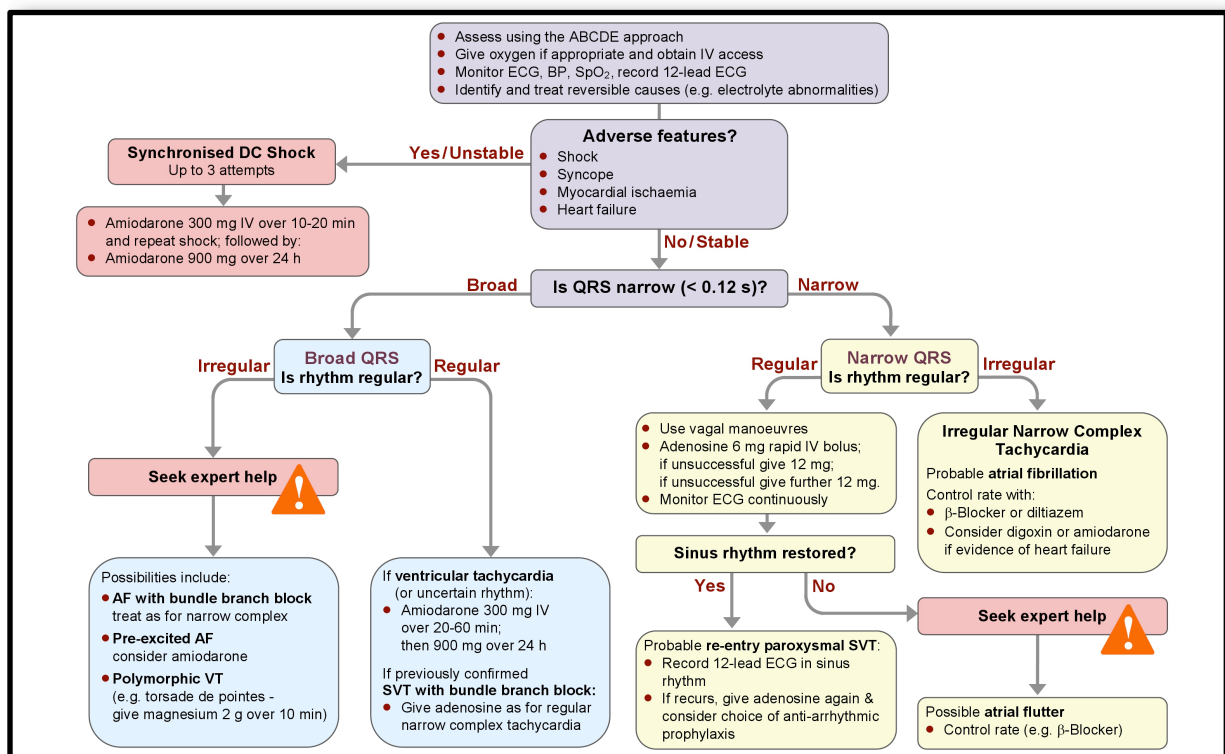




# TACHYARRHYTHMIAS

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.

Haemodynamically 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 & 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<sup>+</sup> current, thus inhibiting re-entry. Amiodarone also blocks sodium channels,  $\alpha$ -receptors and calcium channels.

Vasodilatation ( $\alpha$ -blockade, Ca<sup>++</sup> 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 (over 10-20 minutes) 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 the 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*

8 mmol. 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 bronchoconstriction).

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  $\beta$ -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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# MYOCARDIAL (PERI-OPERATIVE) ISCHAEMIA

Roni Krieser & Kim Fuller

## Why is this important?

- Major cause of post-op morbidity and mortality
- Perioperative ischaemia is associated with adverse cardiac events
- Most perioperative ischaemia is silent
- Early detection may allow therapeutic intervention

## How common is perioperative ischaemia?

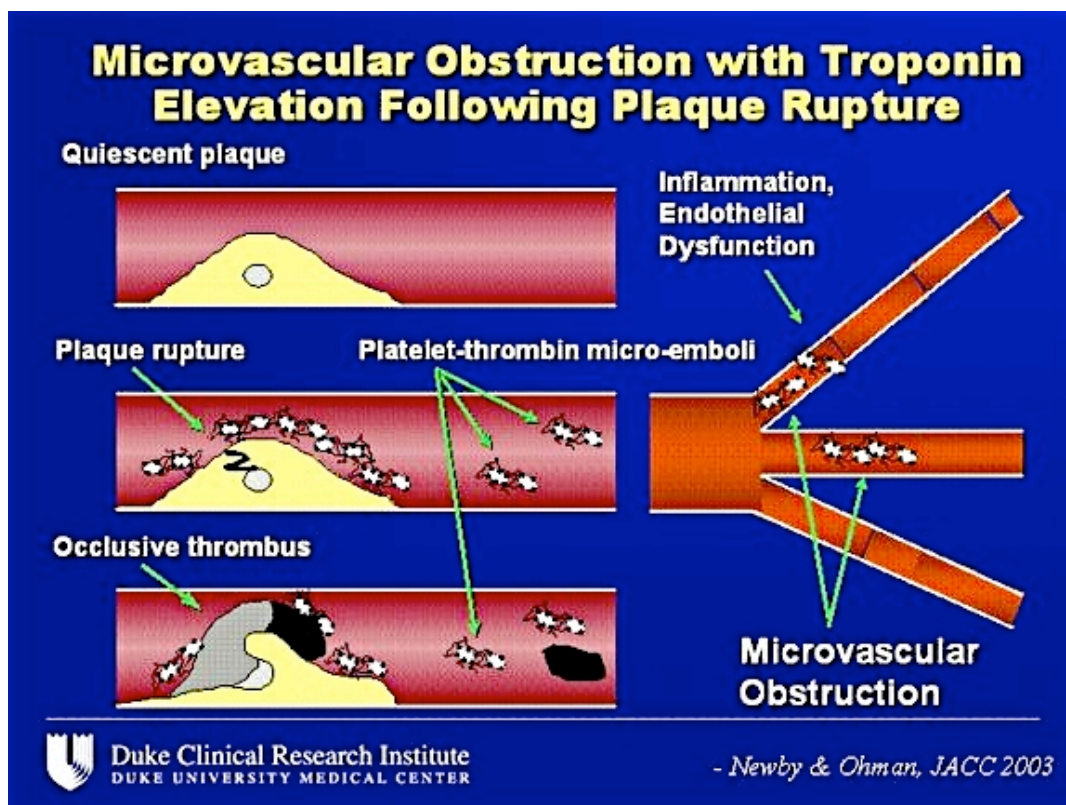
- Incidence is difficult to determine due to the silent nature of ischaemia
- Some studies have shown up to 30%
- A perioperative myocardial infarction (PMI) after non-cardiac surgery carries a 15-25% mortality

## Why does it occur?

There are two main mechanisms:

### 1. Acute Coronary Syndrome

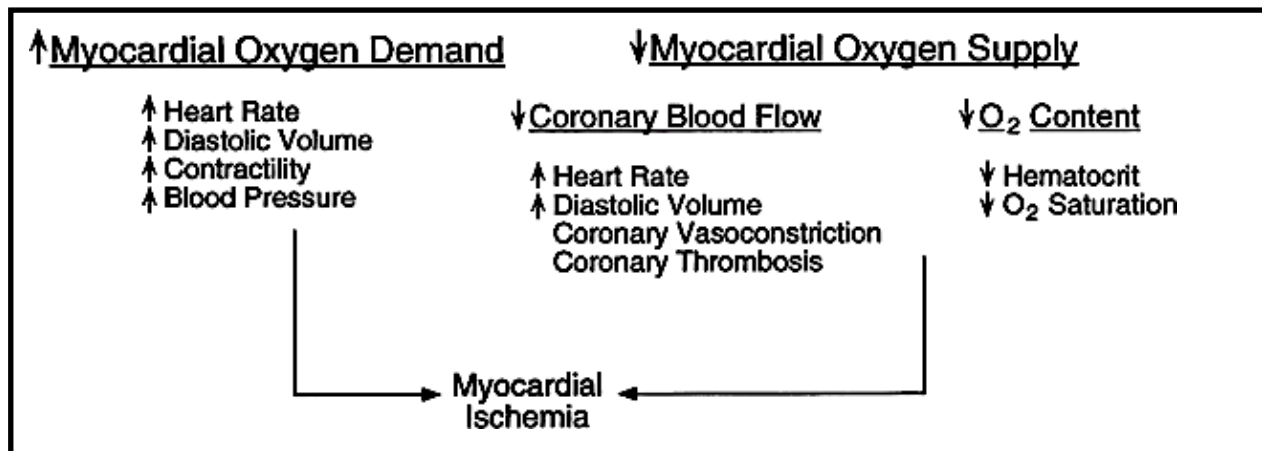
- Plaque rupture, smooth muscle contraction, thrombus formation
- Patients often have multiple cardiac risk factors
- Lesions may be critical or non-critical





## 2. Myocardial Oxygen Supply-Demand Imbalance

- Severe BUT stable stenotic lesion
- Prolonged ST depression especially from tachycardia



### Who is at risk?

- Patients with known coronary artery disease (CAD)
- Patients at increased risk of CAD
- Diabetes, hypertension, smoking, hyperlipidaemia, family history, peripheral or cerebrovascular disease
- Patients at increased risk of cardiovascular complications
- Renal insufficiency, age >65, history of cardiac failure, poor functional capacity (METS <4), abnormal ECG
- Surgical factors
- Major urgent surgery, vascular surgery, surgery with significant fluid shifts or blood loss

### How can we reduce the risk?

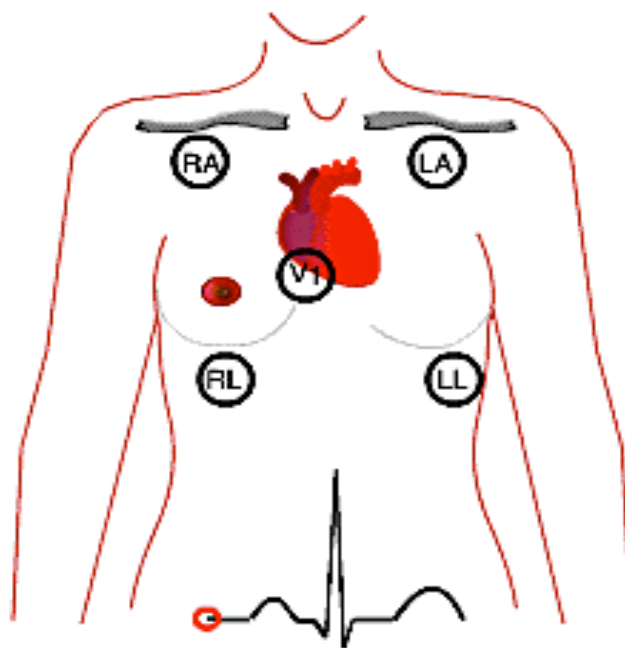
- Sympathetic Modulation
- Beta-blockade (controversial), Alpha-2 agonists (also controversial)
- Anxiolysis (premedication)
- Good analgesia
- Nerve blocks or neuraxial analgesia/anaesthesia
- Maintain normothermia preoperatively
- Haemoglobin >100g/l
- Avoid hypoxia (prolonged supplementation postoperative, 3 days)
- Coagulation Modulation (perioperative hypercoagulable state)
- Aspirin, ketorolac
- Heparin
- Warfarin

## How can we monitor for ischaemia?

- Symptomatic
  - Most often NO symptoms
  - Can have pain, SOB, sweating, N&V, altered mental state
- Clinical Signs
  - Again, often none
  - May have dysrhythmia, hypotension, Congestive Cardiac Failure
- ECG changes
  - ECG is the MOST valuable perioperative monitor (see below)
  - Pulmonary artery catheter and trans-oesophageal echo (not discussed here)

## How can we use the ECG?

Must choose appropriate leads and place them correctly



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**Five Lead Placement**

Electrodes must be applied adequately

Mode, calibration and analysis must be set appropriately

Leads V4 or V5 most accurately detect ischaemia

Lead II is best to detect abnormal rhythms

### What will we see in ishaemia?

ST segment changes

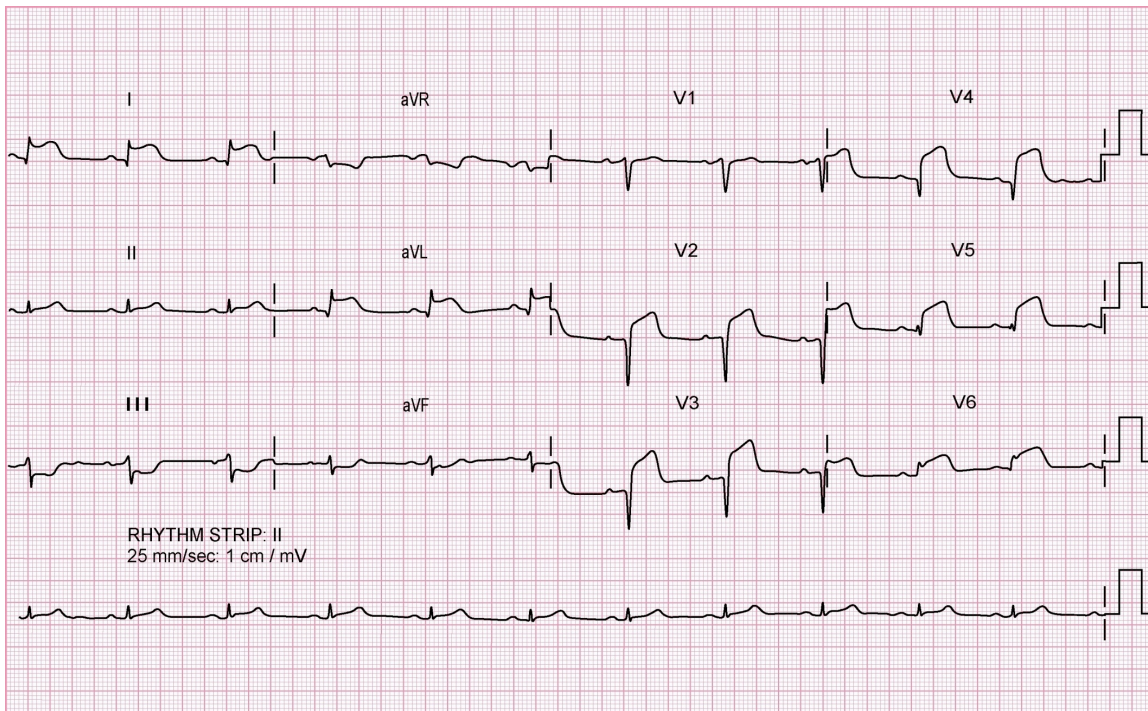
T-wave changes (especially T-wave inversion)

Abnormal rhythm

New conduction abnormalities

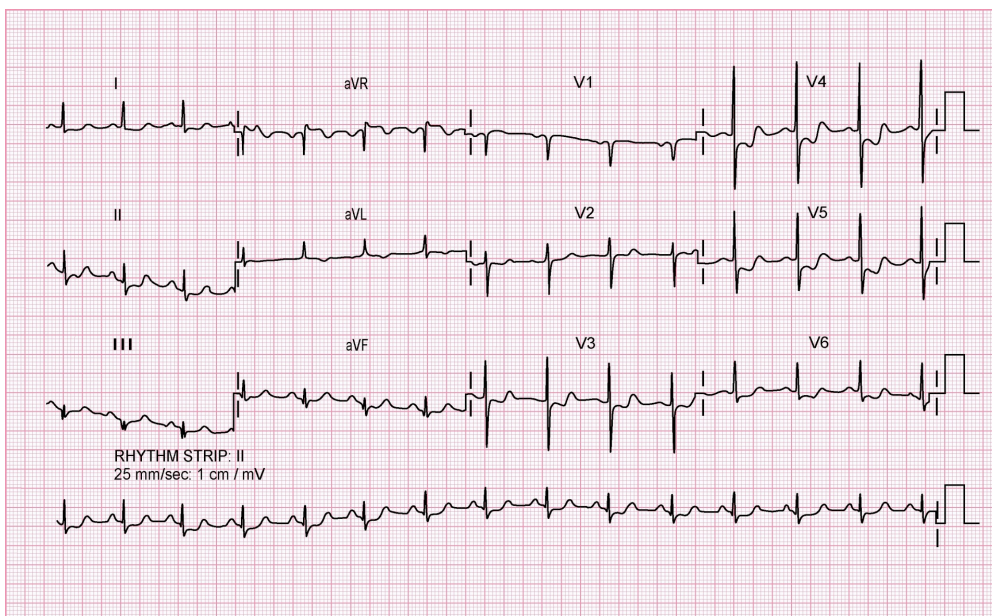
New atrioventricular block

Heart rate changes



## ST ELEVATION MYOCARDIAL INFARCTION

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



## NON-ST WAVE ELEVATION MYOCARDIAL INFARCTION

- ST depression, T wave inversion
- Elevated cardiac enzymes

## What part of the myocardium is involved?

- Anterior ►► ST elevation V2-V5
- Anterolateral ►► ST elevation I, aVL, V5-6
- Inferior ►► ST elevation II, III, aVF

## Are there any other causes for acute ST segment changes?

- Other cardiac causes:  
Conduction disturbances, R wave changes, myocardial contusion
- Other pathology:  
Sub Arachnoid Haemorrhage, acute pancreatitis, spinal injury
- Physiologic changes:  
Hyperventilation, electrolyte abnormalities, hypothermia, etc.,

## How do we manage suspected intra-operative ischaemia?

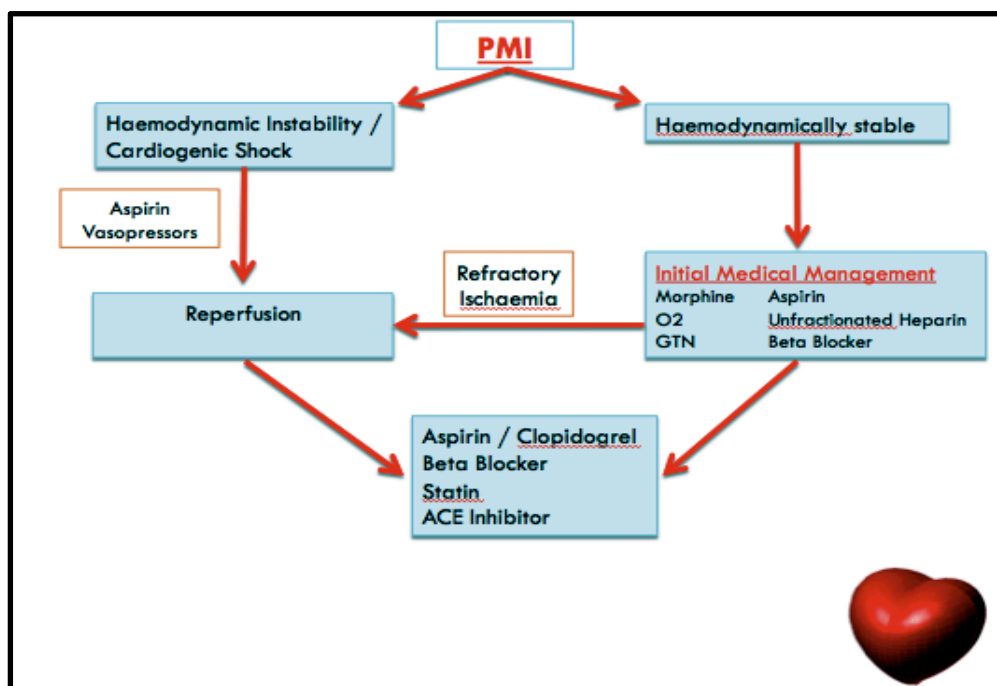
**FIRST** *ensure adequate oxygenation, BP, volume and Hb*

**SECOND** *confirm the change*

- Optimise haemodynamics ►► HR and BP
- Ensure adequate analgesia, depth of anaesthesia if hypertension ►► Morphine
- Check PEEP, surgical manipulation, volume status if hypotensive ►► vasopressors (Metaraminol, Phenylephrine)
- Manage tachycardia ►► beta-blockers, Ca-channel blockers or alpha-2 agonists

**THIRD**

- Increase FiO<sub>2</sub>
- Consider GTN
- Increase monitoring
- Inform the surgeon
- Plan for the postop period



## What do I do if haemodynamics have been optimized and ischaemia persists?

Start GTN infusion and continue to increase it (may need vasopressor for hypotension)  
Consider Trans Oesophageal Echo, Pulmonary Capillary Wedge Pressure, Central Venous Pressure monitoring

- Aspirin or Ketorolac
- Heparin (*if surgery permits*), 5000U bolus then 1000U/hr
- Continue beta-blockade
- Discuss with cardiology
  - urgent reperfusion
  - Intra-aortic balloon pump

## What about my postoperative management?

ALL patients will need

- Serial 12-lead ECGs
- Serial cardiac enzymes
- Analgesia
- Supplemental oxygen
- Adequate monitoring (consider Intensive Care if available)
- Referral to cardiology
- Risk factor management
  - Aspirin, statin, beta-blockade, Angiotensin Converting Enzyme inhibitors depending on current evidence and local guidelines

## What is the current opinion for using fibrinolytic agents in the perioperative period?

- There are NO universal guidelines
- The current recommendations are that it must be a case-specific decision
- There is NO data available that shows a beneficial effect from any one particular therapeutic regimen
- The risk of bleeding seems to depend more on the duration of thrombolysis than on the dosage of the drug
- The cases where it has been used successfully include peri-operative cardiopulmonary resuscitation, massive pulmonary embolus and peri-operative stroke
- The incidence of intracranial haemorrhage following thrombolytic therapy for acute myocardial infarction is about 1.5% with a mortality ranging from 50-80%

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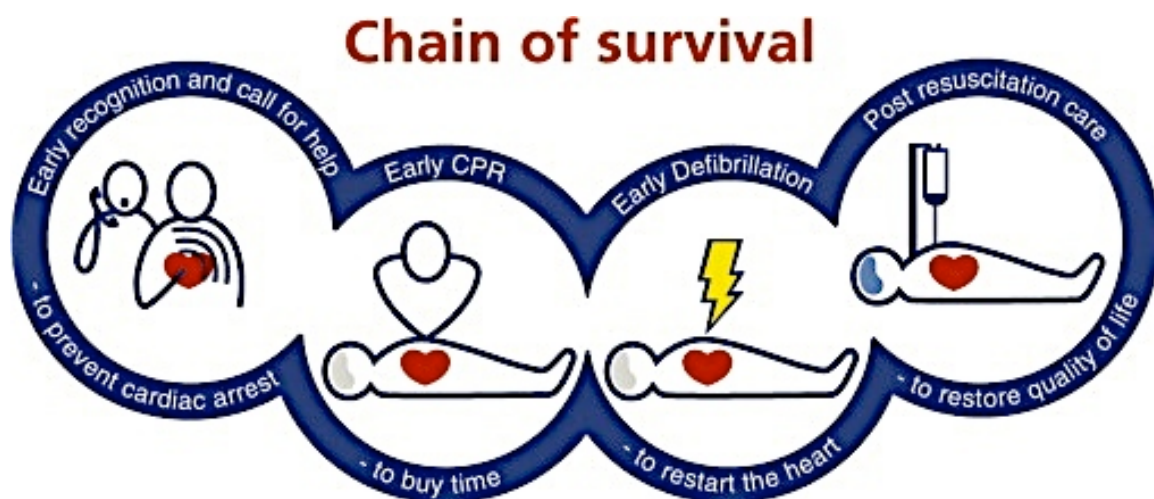
# BASIC LIFE SUPPORT (BLS) & ADVANCED LIFE SUPPORT (ALS) TREATMENT ALGORITHMS

Brendan Munzel & Michelle Chan

## The Chain of Survival

The actions that link the victim of sudden cardiac arrest with survival are called the Chain of Survival.

- Recognising those at risk of cardiac arrest and calling for help
- Provision of *immediate* Cardio Pulmonary Resuscitation (CPR).
  - Immediate CPR can double or triple survival from out of hospital ventricular fibrillation (VF) cardiac arrest (1)
- Give *early* Defibrillation.
  - Following VF cardiac arrest, CPR plus defibrillation within 3-5 minutes of collapse can produce survival rates as high as 49-75%. (2) Every minute of delay before defibrillation reduces the probability of survival to discharge by 10-12%. (3)
- Effective post-resuscitative care.



*Reproduced from ERC Guidelines*

## Adult Basic Life Support

Basic Life Support describes an initial assessment for and management of cardio-respiratory arrest. Immediate CPR may double or triple survival from VF arrest. (4) It can safely be performed by anyone. It can be performed as a one person, or two-rescuer technique.

The safety of patient, resuscitator and bystanders should be checked before proceeding. Check the collapsed victim for a response. This can be done by gently shaking the patient's shoulders and shouting, "are you alright?" If the patient is unconscious, then call for help. The patient should be gently placed upon their back. The airway should be opened using head tilt and chin lift, or jaw thrust.

Keeping the airway open, assess for the presence and quality of breathing. The patient airway is assessed by 'looking, listening and feeling'. Holding an ear over the patients' mouth

and nose, listening for breath sounds, feeling for exhalation on the cheek, and observing any rise/fall of the chest. Gasping is an early sign of cardiac arrest, and should be recognised.

If breathing normally, the patient should be placed in the recovery position (left lateral), monitored, and help should be summoned. If breathing is abnormal or absent, help should be summoned. If there are two people present, the first should leave to call for help, whilst the second continues with the BLS algorithm. A single resuscitator should quickly call for help. The patients' side should only be left if there is no other option. Checking for pulses is an inaccurate method of confirming the presence or absence of circulation, both for laypersons and professionals.

High quality chest compressions are essential. This should be performed over the lower half of the sternum at a rate of 100 compressions per minute and at a ratio of 30:2 (compression to respiration). Chest compressions should be performed in a comfortable position kneeling beside the patient. The heel of the hand is placed over the lower half of the sternum. The other hand is placed on top, and fingers interlaced. Elbows are extended, and power generated from flexing the hips.

Chest compressions are combined with rescue breaths. After 30 compressions, the airway is opened with head-tilt and chin-lift. Pinch the soft part of the nose closed. Take a normal breath and place your lips around the patients' mouth with a good seal. Blow steadily until the chest rises for 1 second. This is an effective rescue breath. Take your mouth away, and watch the chest fall as air comes out. This should be repeated a second time to achieve two effective rescue breaths. Chest compressions and rescue breaths should be conducted simultaneously in two-person resuscitation.

Chest compressions should be un-interrupted during connection of the defibrillator, or during other advanced life support manoeuvres (including laryngoscopy). There should be only a brief pause in the cycle during ALS for rhythm assessment, defibrillator discharge and the passage of an endotracheal tube (if appropriate). It is a good idea to change over the person doing chest compression every 2 minute cycle to avoid fatigue.

The use of an AED (automated external defibrillator), which is a biphasic defibrillating device, is included in European Resuscitation Council (ERC) and Australian Resuscitation Council (ARC) basic life support protocols. However, this is subject to device availability, and has been omitted from this discussion.

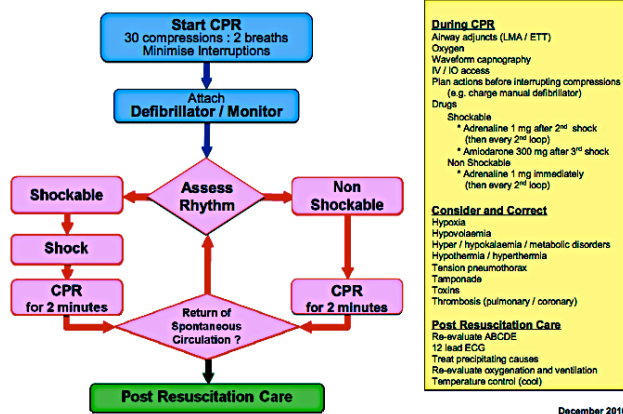
## Advanced Life Support

*(Summarised from ERS and ARC resuscitation guidelines.)*

Advanced life support is the algorithm for assessment and early intervention of cardio-respiratory arrest. Advanced life support is a continuum from adult basic life support. The recommended sequence will depend on the location, staffing experience, and available equipment. Out-of-hospital cardiac arrest is usually the result of a primary cardiac event (coronary artery disease, ischaemic cardiomyopathy, valvular heart disease). In contrast, inpatient cardiac arrest is usually preceded by slow physiological deterioration (hypoxia, hypotension), rather than a primary cardiac event. Once cardiac arrest occurs, fewer than 20% of patients survive to go home. (5)

Some in-hospital cardiac arrests may be preventable by identifying and treating the critically ill or deteriorating patient. Such patients can be alerted by clinical 'track-and-trigger' systems; such as the response criteria for a medical emergency team call (MET criteria) or aggregated weighted scoring systems (such as early warning scores EWS). Early assessment and intervention of patients with respiratory, cardiac or circulatory failure follow the ABCDE algorithm.

## Advanced Life Support for Adults



The collapsed patient is approached, and help called for, as described in the BLS algorithm. The rescuer must ensure the safety of patients, bystanders and themselves. After attempting to elicit a response, the patient is placed on their back and the airway is assessed as in BLS. The airway is opened by head-tilt and chin-lift manoeuvres. The mouth is inspected for foreign body and debris removed with a gloved finger, suction or Magill forceps.

A blind finger sweep is not recommended. Breathing is assessed by *'look, listen and feel'*. Those with clinical experience can inspect the patient for signs of circulation. Pulse can be assessed at the carotid while other signs of life are observed for no more than 10s.

If there is a pulse or signs of life, further urgent medical assessment is required. For those not confident in palpation of a carotid pulse, or if there are no signs of life, proceed immediately to CPR. In the hospital environment, appropriate staff or the emergency response team should be called depending on local policy.

In Advanced Life Support, the importance of good quality chest compressions with minimal interruption is again stressed. Whilst waiting for the clinical response team, CPR should be administered as described in the BLS algorithm. Positive pressure ventilation, with high flow oxygen, should be performed with the most appropriate equipment at hand. This may be bag-valve-mask or supraglottic airway depending on availability. Ventilation can be aided by airway adjuncts such as an oropharyngeal or nasopharyngeal airway. Tracheal intubation should be attempted only by those who are trained, competent and experienced in this skill. No tracheal intubation attempt should interrupt chest compressions for more than 10 seconds. Waveform capnography should be available for confirming tube placement. If the trachea has been intubated, cardiac compression can proceed uninterrupted, and the patient ventilated at a rate of 10 breaths per minute. Each breath should be sufficient to allow the chest to rise for 1 second. Avoid over-ventilation.

Appropriate monitoring should be attached to the patient, including a defibrillator (pads or paddles) to expedite rhythm assessment. This should occur without interruption to CPR. Once attached, CPR should be paused briefly for rhythm assessment.

A patient found to be in ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) is said to have a shockable rhythm. The defibrillator is charged whilst CPR continues, to 360J if using a monophasic defibrillator, or 200J if using a biphasic defibrillator. Once the defibrillator is charged, pause chest compression, ensure that all rescuers are clear of the patient and that high flow oxygen is removed. After a rapid, thorough safety check, a shock can be given. Defibrillation is delivered as a single shock. The precordial thump in this setting is no longer recommended. It is no longer recommended that a fixed period of CPR precede defibrillation of the shockable rhythm.

After defibrillation, CPR is immediately restarted without rhythm assessment or pulse check. Ideally, the pause between stopping and restarting chest compressions should be less than 5 seconds. Once resuscitation has resumed, prepare intravenous cannulae and drugs likely to be used by the resuscitation team. Circulatory access should be obtained. Large calibre peripheral cannulae are preferable for drug and fluid boluses. If peripheral cannulation proves difficult, the intraosseous route should be considered. Central venous cannulation is a good alternative, if it is able to be performed quickly by experienced operators. Once



access is established, blood is taken for further investigation and a fluid bolus can be administered. After two minutes of resuscitation, CPR is paused, and the rhythm assessed. If the patient is still in VT/VF, give a second shock at 360J. Without reassessing the rhythm or feeling for a pulse, resume CPR at a ratio 30:2.

This cycle is continued. After the second shock has been delivered, and CPR recommenced, Adrenaline can be administered at a dose of 1mg intravenously or via the intraosseous route. This can be repeated every 3-5 minutes (alternate cycles) until return of spontaneous circulation. After the third shock, Amiodarone should be administered at a dose of 300mg. The algorithm continues until return of spontaneous circulation. If a narrow complex regular rhythm is present, try to palpate a pulse. If return of spontaneous circulation has been achieved, begin post-resuscitation care.

The patient found to be in asystole or pulseless electrical activity, is said to have a non-shockable rhythm. CPR should continue with delivery of high concentrations of inspired oxygen, with 2 minutely brief interruption for rhythm re-assessment. Adrenaline should be administered immediately at a dose of 1mg IV or IO. Subsequent doses are given at 3-5 minutely intervals. An intravenous fluid bolus of 20ml kg<sup>-1</sup> (up to 1L) should be given. The underlying pathology must be identified and treated. The 4Hs and 4Ts, should be aggressively sought and treated. These include hypoxia, hypovolemia, disorders of metabolism (hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia) and hypothermia, tension pneumothorax, cardiac tamponade, toxins (or medications), and thromboembolism (pulmonary embolism, myocardial infarction).

During advanced life support, a team leader should be identified. This person is responsible for monitoring progress through the ALS algorithm, the quality of chest compression, alternating CPR providers and communicating with members of the team.

There are a number of important therapeutic goals for patients who have a return to spontaneous circulation following resuscitation. This post-resuscitative phase aims to make the diagnosis and provide definitive treatment of ischaemic heart disease, reduced cardiac complications, and reduce neurological sequelae. Appropriate level monitoring, arrhythmia prevention, fluid and electrolyte therapies, and glycaemic control are other important objectives. Some patients will require ventilatory support and sedation. Other post-resuscitation treatment considerations include therapeutic hypothermia and seizure prevention.

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# COMMON INTRA-OPERATIVE PROBLEMS

Amanda Baric

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 checklist may be modified to suit local requirements.

Surgical Safety Checklist			World Health Organization	Patient Safety A World Alliance for Safer Health Care
<b>Before induction of anaesthesia</b>	<b>Before skin incision</b>	<b>Before patient leaves operating room</b>		
(with at least nurse and anaesthetist)	(with nurse, anaesthetist and surgeon)	(with nurse, anaesthetist and surgeon)		
<p><b>Has the patient confirmed his/her identity, site, procedure, and consent?</b></p> <input type="checkbox"/> Yes	<p><input type="checkbox"/> <b>Confirm all team members have introduced themselves by name and role.</b></p> <p><input type="checkbox"/> <b>Confirm the patient's name, procedure, and where the incision will be made.</b></p> <p><b>Has antibiotic prophylaxis been given within the last 60 minutes?</b></p> <input type="checkbox"/> Yes <input type="checkbox"/> Not applicable	<p><b>Nurse Verbally Confirms:</b></p> <input type="checkbox"/> The name of the procedure <input type="checkbox"/> Completion of instrument, sponge and needle counts <input type="checkbox"/> Specimen labelling (read specimen labels aloud, including patient name) <input type="checkbox"/> Whether there are any equipment problems to be addressed		
<p><b>Is the site marked?</b></p> <input type="checkbox"/> Yes <input type="checkbox"/> Not applicable	<p><b>Anticipated Critical Events</b></p> <p><b>To Surgeon:</b></p> <input type="checkbox"/> What are the critical or non-routine steps? <input type="checkbox"/> How long will the case take? <input type="checkbox"/> What is the anticipated blood loss? <p><b>To Anaesthetist:</b></p> <input type="checkbox"/> Are there any patient-specific concerns? <p><b>To Nursing Team:</b></p> <input type="checkbox"/> Has sterility (including indicator results) been confirmed? <input type="checkbox"/> Are there equipment issues or any concerns?	<p><b>To Surgeon, Anaesthetist and Nurse:</b></p> <input type="checkbox"/> What are the key concerns for recovery and management of this patient?		
<p><b>Is the anaesthesia machine and medication check complete?</b></p> <input type="checkbox"/> Yes	<p><b>Is essential imaging displayed?</b></p> <input type="checkbox"/> Yes <input type="checkbox"/> Not applicable			
<p><b>Is the pulse oximeter on the patient and functioning?</b></p> <input type="checkbox"/> Yes				
<p><b>Does the patient have a:</b></p> <p><b>Known allergy?</b></p> <input type="checkbox"/> No <input type="checkbox"/> Yes				
<p><b>Difficult airway or aspiration risk?</b></p> <input type="checkbox"/> No <input type="checkbox"/> Yes, and equipment/assistance available				
<p><b>Risk of &gt;500ml blood loss (7ml/kg in children)?</b></p> <input type="checkbox"/> No <input type="checkbox"/> Yes, and two IVs/central access and fluids planned				

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. The ABCDE approach that is part of the advanced life support approach. 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.

### **A B C D E** / *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.

## 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 PaO<sub>2</sub> 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 (up to 100% if required) 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 depends 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 up-sloping 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 is to exclude 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, pheochromocytoma 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, labetalol). (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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# ANAPHYLAXIS

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Elmo Mariampillai

## 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, IgG or complement activation and non-allergic/non-immune-mediated anaphylaxis. The term anaphylactoid 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. In the operating room, the most common causative agents are muscle relaxants. Other common allergens include:

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

The risk factors for anaphylaxis include a history of allergy, intravenous administration of the allergen, 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%	
Angioedema	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 simultaneous treatment.

- 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 a biphasic reaction, protracted anaphylaxis or in a fatal outcome.

The dose of adrenaline is – 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 has suffered a cardiac arrest (no pulse or blood pressure), then the standard 1mg of IV adrenaline should be given according to the advanced life support algorithm.

### *Other management:*

2. IV fluids
3. Antihistamine
4. Steroids – hydrocortisone 100mg or Prednisolone 50mg
5. 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 because 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 tryptase. 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.

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# MANAGEMENT OF MASSIVE HAEMORRHAGE

Sion Davies

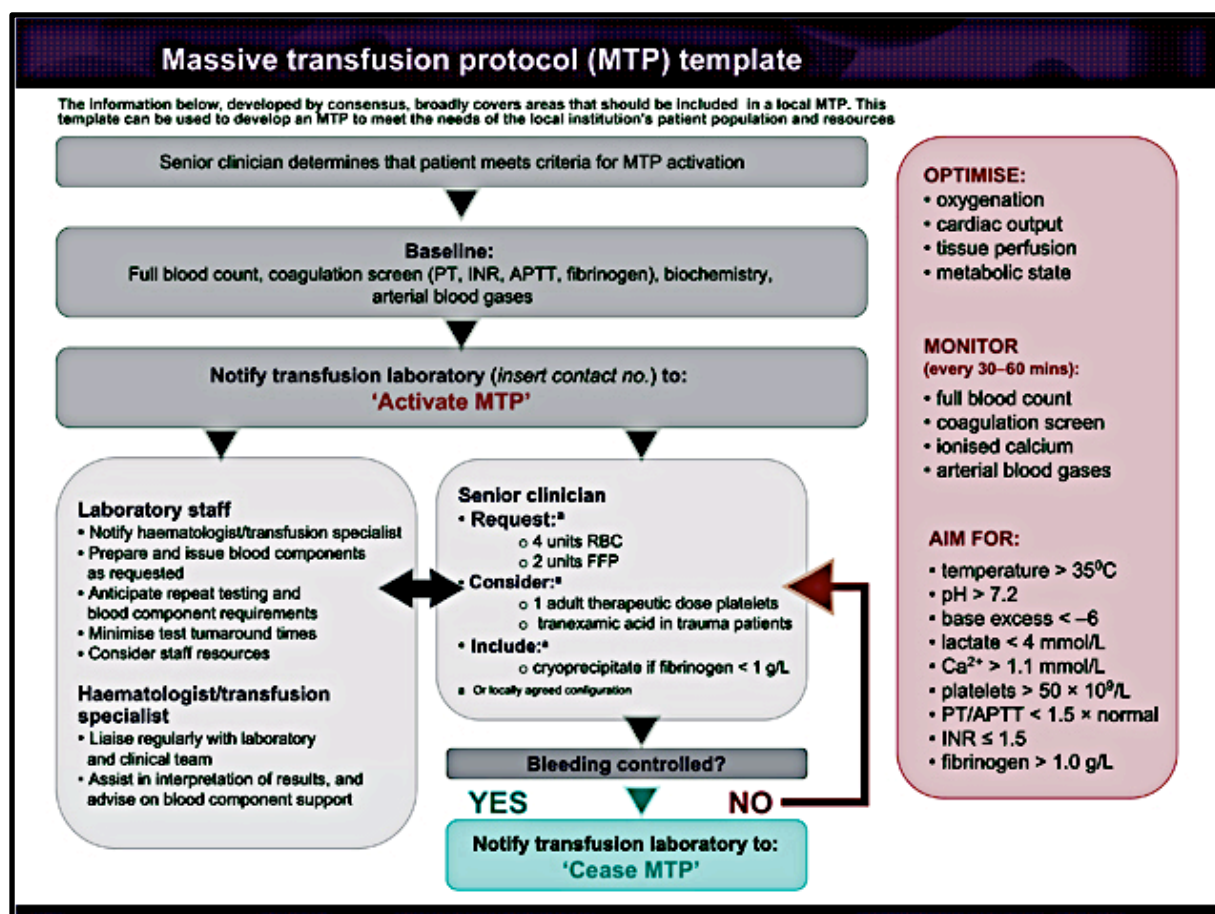
Trauma is the leading cause of death in all ages from 1 to 44 years in the UK. Haemorrhagic shock accounts for 80% of deaths in the operating theatre and 50% of deaths in the first 24 hours after injury. (1)

## Massive transfusion may be defined:

- 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) templates are reproduced here (below).



### Suggested criteria for activation of MTP

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

<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Initial management of bleeding</h4> <ul style="list-style-type: none"> <li>• Identify cause</li> <li>• Initial measures:             <ul style="list-style-type: none"> <li>- compression</li> <li>- tourniquet</li> <li>- packing</li> </ul> </li> <li>• Surgical assessment:             <ul style="list-style-type: none"> <li>- early surgery or angiography to stop bleeding</li> </ul> </li> </ul>	<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Resuscitation</h4> <ul style="list-style-type: none"> <li>• Avoid hypothermia, institute active warming</li> <li>• Avoid excessive crystalloid</li> <li>• Tolerate permissive hypotension (BP 80–100 mmHg systolic) until active bleeding controlled</li> <li>• Do not use haemoglobin alone as a transfusion trigger</li> </ul>								
<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Specific surgical considerations</h4> <ul style="list-style-type: none"> <li>• If significant physiological derangement, consider damage control surgery or angiography</li> </ul>	<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Special clinical situations</h4> <ul style="list-style-type: none"> <li>• Warfarin:             <ul style="list-style-type: none"> <li>• add vitamin K, prothrombinex/FFP</li> </ul> </li> <li>• Obstetric haemorrhage:             <ul style="list-style-type: none"> <li>• early DIC often present; consider cryoprecipitate</li> </ul> </li> <li>• Head injury:             <ul style="list-style-type: none"> <li>• aim for platelet count &gt; 100 × 10<sup>9</sup>/L</li> <li>• permissive hypotension contraindicated</li> </ul> </li> </ul>								
<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Cell salvage</h4> <ul style="list-style-type: none"> <li>• Consider use of cell salvage where appropriate</li> </ul>	<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Considerations for use of rFVIIa<sup>b</sup></h4> <p>The routine use of rFVIIa in trauma patients is not recommended due to its lack of effect on mortality (Grade B) and variable effect on morbidity (Grade C). Institutions may choose to develop a process for the use of rFVIIa where there is:</p> <ul style="list-style-type: none"> <li>• uncontrolled haemorrhage in salvageable patient, and</li> <li>• failed surgical or radiological measures to control bleeding, and</li> <li>• adequate blood component replacement, and</li> <li>• pH &gt; 7.2, temperature &gt; 34°C.</li> </ul> <p>Discuss dose with haematologist/transfusion specialist</p> <p><sup>b</sup> rFVIIa is not licensed for use in this situation; all use must be part of practice review.</p>								
<h4 style="text-align: center; background-color: #4b4b8b; color: white; padding: 2px;">Dosage</h4> <table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 50%;">Platelet count &lt; 50 × 10<sup>9</sup>/L</td> <td style="width: 50%;">1 adult therapeutic dose</td> </tr> <tr> <td>INR &gt; 1.5</td> <td>FFP 15 mL/kg<sup>a</sup></td> </tr> <tr> <td>Fibrinogen &lt; 1.0 g/L</td> <td>cryoprecipitate 3–4 g<sup>a</sup></td> </tr> <tr> <td>Tranexamic acid</td> <td>loading dose 1 g over 10 min, then infusion of 1 g over 8 hrs</td> </tr> </table> <p><small>a Local transfusion laboratory to advise on number of units needed to provide this dose</small></p>	Platelet count < 50 × 10 <sup>9</sup> /L	1 adult therapeutic dose	INR > 1.5	FFP 15 mL/kg <sup>a</sup>	Fibrinogen < 1.0 g/L	cryoprecipitate 3–4 g <sup>a</sup>	Tranexamic acid	loading dose 1 g over 10 min, then infusion of 1 g over 8 hrs	
Platelet count < 50 × 10 <sup>9</sup> /L	1 adult therapeutic dose								
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Fibrinogen < 1.0 g/L	cryoprecipitate 3–4 g <sup>a</sup>								
Tranexamic acid	loading dose 1 g over 10 min, then infusion of 1 g over 8 hrs								

ABG	arterial blood gas	FFP	fresh frozen plasma	APTT	activated partial thromboplastin time
INR	international normalised ratio	BP	blood pressure	MTP	massive transfusion protocol
DIC	disseminated intravascular coagulation	PT	prothrombin time	FBC	full blood count
RBC	red blood cell	rFVIIa	activated recombinant factor VII		

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

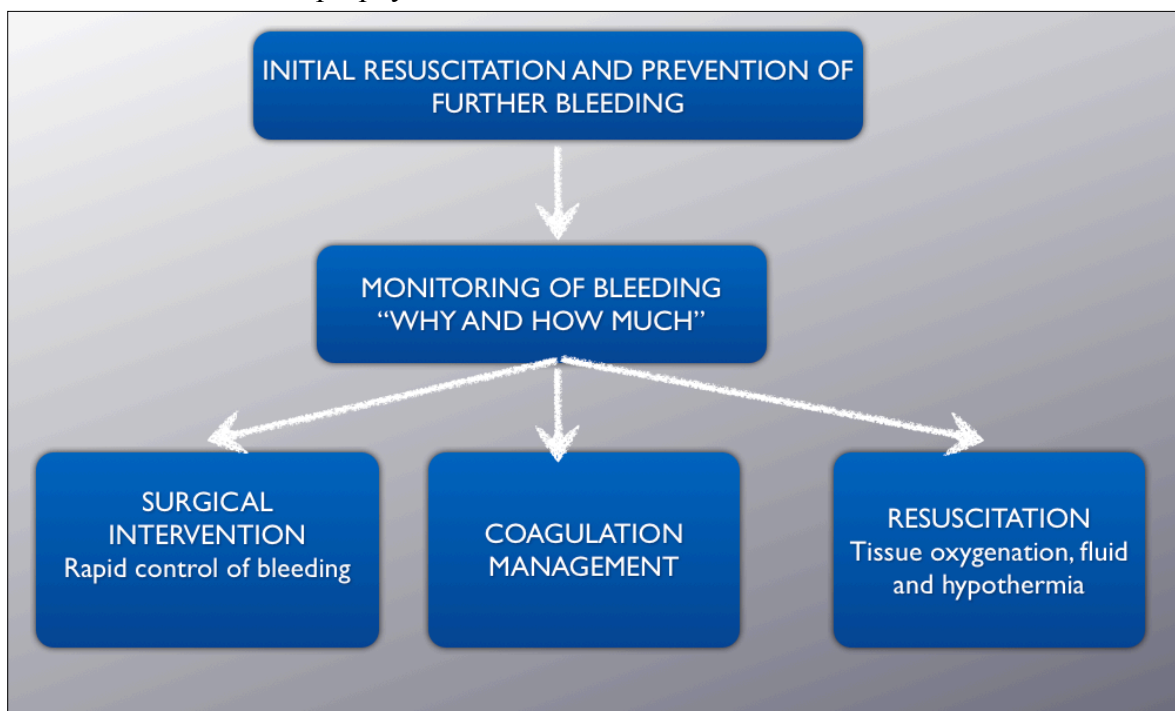
### Management of massive haemorrhage

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.

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 dates 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 are 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 surgical 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

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)<sup>19</sup>  
 Reproduced with permission from ACS  
 Note: Values are estimated for a 70 kg male

## 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 significant.

The aim of management is to try and prevent the development of a coagulopathy. This is achieved by the administration of fresh frozen plasma (FFP), platelets, and cryoprecipitate or fibrinogen with blood as soon as the MTP is activated.

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.

Coagulopathy in massive haemorrhage is 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°C
- pH > 7.2
- base excess < - 6
- lactate < 4 mmol/L
- Ca<sup>2+</sup> > 1.1 mmol/L
- Platelets > 50 x 10<sup>9</sup>/L (some authorities recommend maintaining platelet count at 75 x 10<sup>9</sup>/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.

## 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](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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# PRINCIPLES OF TRAUMA MANAGEMENT

Simon Hendel & Kim Fuller

## UNDERSTANDING HOW AND WHEN PEOPLE DIE ALLOWS US TO MAKE TIMELY ASSESSMENT, DECISIONS AND ACTIONS

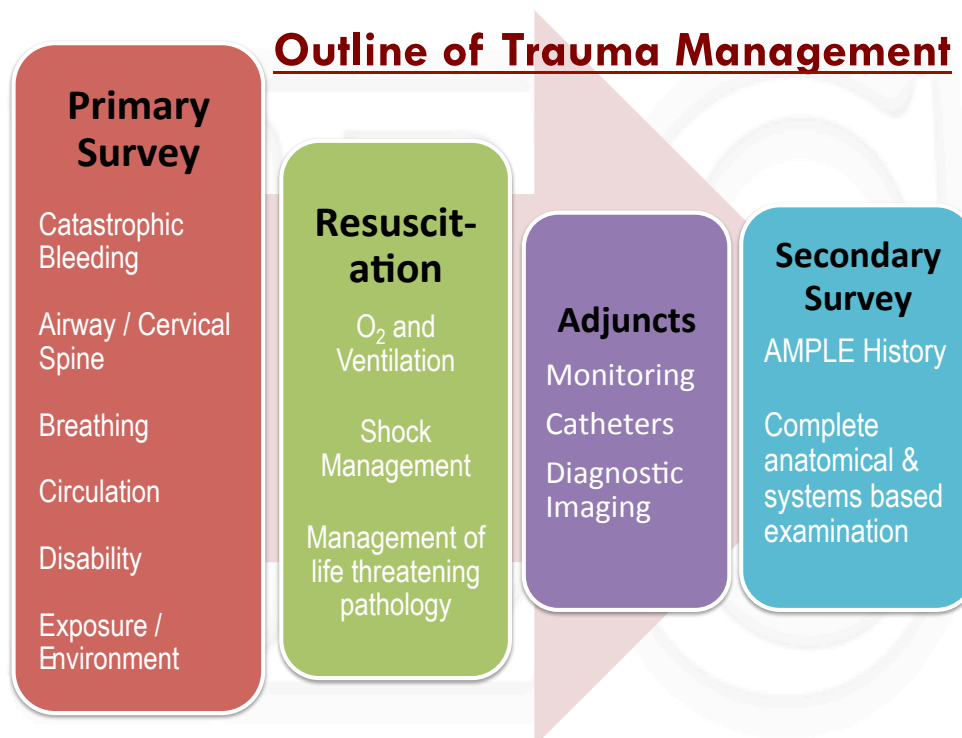
Injury from trauma is a major economic burden

Trauma is the most common cause of death in those <45 yrs of age

Deaths occur in three peaks

- Early deaths
  - Occur at the scene
  - Account for 50% of total mortality
  - Usually the result of brain or spinal cord injury, heart and great vessel damage
  - Often from rapid deceleration
- Intermediate deaths
  - Occur within 1-2 hours
  - Account for 35%
  - Head, chest, abdominal injuries and fractures associated with large blood loss
- Late deaths
  - Occurs days to weeks after initial trauma
  - Sepsis and organ failure

Where there is NO specifically organised trauma care system in place it is estimated up to 30% of deaths are preventable



**FOR EVERY PATIENT WHO DIES, 10 MORE ARE ADMITTED TO HOSPITAL AND 2 SUFFER LONG-TERM DISABILITY**

There are three main causes of preventable death in major trauma

- Exsanguination
- Tension pneumothorax
- Airway obstruction due to maxillofacial injuries

Assessment and management should be dynamic and continuous

- Divided into *Pre-hospital* and *Hospital Care*
- Handover between the two phases and before/after procedures must be smooth and avoid interruption of patient care
- Goals of assessment and resuscitation
  - Minimize time from injury to definitive care
  - Don't allow the obvious injury to distract you from diagnosing other, less obvious, injuries
  - NO patient should leave the resuscitation area without a clear management plan
  - There should be no need for further clinical guesswork after 2 hours from the arrival of the patient



*A fatal pelvic fracture was missed with this severe arm injury*

**AIMS**

**SAVE LIVES**

**PREVENT MAJOR DISABILITY**

**DIAGNOSE & MANAGE ALL INJURIES**

**AVOID UNNECESSARY INTERVENTIONS**

**AVOID UNNECESSARY INVESTIGATIONS**

## THE PRIMARY SURVEY

### RESUSCITATION OCCURS CONCURRENTLY WITH THE PRIMARY SURVEY AS PROBLEMS ARE ENCOUNTERED

#### CATASTROPHIC HAEMORRHAGE

- Extremity or major body cavity
- More common with penetrating or blast trauma
- Needs attention IMMEDIATELY
- Direct pressure, elevation, tourniquets, pressure points
- Haemostatic agents

#### *AIRWAY & CERVICAL SPINE (A)*

If the patient is talking

- The airway is patent (for now)
- They are breathing
- There is enough circulation to perfuse the brain
- ➡ Protect the Cervical-spine

If the patient is not talking

- Basic airway maneuvers (care of C-spine)
- Airway adjuncts
- +/- Intubation (manual in-line stabilization)
- +/- Surgical Airway

#### *BREATHING & VENTILATION (B)*

- The causes of fatal chest trauma are... **BL-ATOM-FC**
  - **B**last lung
  - **A**irway obstruction
  - **T**ension pneumothorax
  - **O**pen pneumothorax
  - **M**assive haemothorax
  - **F**lail chest
  - **C**ardiac tamponade
- Assessment of breathing
  - Look, listen and feel for adequacy of ventilation
  - Look at patient ➡ colour, bruising, abrasions
  - Measure respiratory rate
  - Look at chest movement ➡ *is there a flail segment?*
  - Listen to breath sounds
  - Feel for tracheal position, subcutaneous emphysema



**IF THERE IS A TENSION  
PNEUMOTHORAX TREAT IT  
IMMEDIATELY WITH  
NEEDLE DECOMPRESSION**

### *CIRCULATION & HAEMORRHAGE CONTROL (C)*

- Look for signs of external bleeding
- Look for signs of shock
- Rapid pulse
- Low blood pressure
- Poor capillary return
- Colour of skin
- Adequacy of organ perfusion
  - Mentation
  - Urine output
- Main causes of hypovolaemia are
  - Chest or abdominal injury
  - Pelvic or femur fracture
  - Penetrating injuries to arteries or veins
  - External haemorrhage
- Insert 2 large bore cannula
- Send blood for x-match
- Give IV fluids and consider O-ve blood
- Establish monitoring (BP, ECG, HR, O2 sats)
- Titrate response to radial pulse and conscious state

**IN THE ABSENCE OF HAEMORRHAGE CONTROL  
AIM FOR A SYSTOLIC BLOOD PRESSURE OF 90 mmHG**

### *DISABILITY*

- Look at the pupils with a torch
- Check size, symmetry, reactivity
- Assess level of consciousness (AVPU)
  - A Awake
  - V Responds to Voice
  - P Responds to Pain
  - U Unresponsive
- Alteration in conscious state indicates brain injury until proven otherwise
- NEVER assume an altered level of conscious is due to alcohol or drugs

**A RESPONSE TO PAIN ONLY = GCS 8 & MEANS THEY ARE  
LIKELY TO NEED INTUBATION**

*ENVIRONMENT & EXPOSURE (E)*

- There is no place for modesty in trauma ➡ if you don't look for injury you will NOT find it
- Hypothermia increases oxygen consumption, decreases coagulation, and leads to further cardiovascular instability

**EXPOSING THE PATIENT IS VITAL & ENSURES SERIOUS  
INJURY IS NOT MISSED**

**HYPOTHERMIA IS VERY BAD FOR INJURED PATIENTS**

*BASIC IMAGING*

- Trauma Series
  - Chest x-ray, cervical spine x-rays, pelvic x-ray
- Focused Abdominal Sonography in Trauma (FAST) Scan
- CT scans if available
  - C-spine and brain *especially*

**SECONDARY SURVEY**

1. Head to toe, thorough examination
2. “*Hands & tubes in every orifice*”
3. Log roll to examine back
4. Take an ‘**AMPLE**’ history
  - a . **A**llergies
  - b . **M**edications
  - c . **P**ast history
  - d . **L**ast meal
  - e . **E**vents surrounding trauma

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# SKILL STATION: INTERCOSTAL CHEST TUBE INSERTION

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

## Indications

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

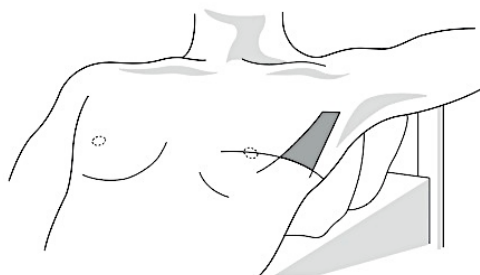
- Pneumothorax (open, simple, tension)
- Haemothorax
- Significant chest injuries in a patient who is undergoing general anaesthesia or being transferred by aircraft.
- Chest Trauma
- Empyema and parapneumonic pleural effusion
- Malignant pleural effusion
- Post operative for thoracotomy and oesophagectomy

## Equipment needed:

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

## Technique:

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



**Figure 3** Diagram to illustrate the "safe triangle".

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

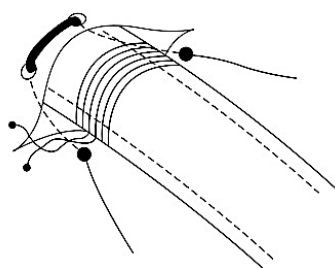
The patient is positioned for the procedure. Preferably the patient is sitting up slightly, with monitoring including

ECG, pulse oximetry and oxygen by a facemask.



A vasovagal reaction may occur due to intense vagal stimulation, so caution must be exercised in the sitting position. The patient's hand is placed behind the head to expose the axilla. The site of insertion is usually the 4<sup>th</sup> or 5<sup>th</sup> intercostal space (approximately at the nipple line), just anterior to the mid-axillary line on the affected side. On expiration, the diaphragm rises to the 5<sup>th</sup> rib at the level of the nipple, and thus chest drains should be placed above this level. Rib spaces are counted down from the 2<sup>nd</sup> rib at the sterno-manubrial joint.

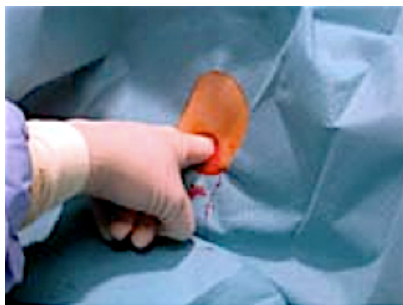
There is a safe triangle for insertion of the ICC. Its boundaries are the anterior border of latissimus dorsi and the lateral border of the pectoralis major muscle; an anterior line superior to the horizontal level of the nipple and the apex of the triangle is below the axilla.



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

Infiltrate the skin and deeper tissues including the pleura with local anaesthetic.

The needle is then directed perpendicular to the skin and local anaesthetic infiltrated through the layers of the chest wall down onto the rib below the actual intercostal space.



Here, local anaesthetic is injected around the periosteum of the rib. The needle is then angled above the rib and advanced slowly until air is aspirated.

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

Blunt dissection is performed using a curved clamp along the track. The clamp is inserted into muscle tissue and is spread to split the fibres. Insert a gloved finger to confirm the track, and that you are in the chest cavity and other organs are not present.



A large-bore (32-36 F) chest tube is mounted on the clamp and passed along the track into the pleural cavity. It is directed posteriorly and basally to a desired length (15cm in adults).

The tube is connected to an underwater seal.

Confirm correct placement by observing bubbling in the drain

bottle, as well as swinging of the fluid in the tubing with inspiration.

- Suture the tube in place and apply a sterile dressing.
- A chest X-ray is taken to confirm placement & position.





## Complications

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

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

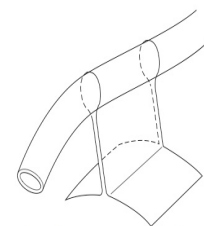


Figure 5 Clement tag to support the tube while allowing it to lie a little away from the chest wall.

### Chest drain (underwater seal)



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

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

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

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

### Key points regarding chest drains:

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

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## NEEDLE THORACOSTOMY

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The patient with a suspected tension pneumothorax needs immediate decompression, preferably with a thoracostomy tube using a standard technique. As an alternative, a large bore intravenous catheter can be used in hemodynamically unstable patients for whom a suspicion of tension pneumothorax is high.

A 14 to 16 gauge intravenous needle/catheter attached to a 5 or 10 mL syringe is inserted along the superior margin of the second or third rib in the midclavicular line.

The intravenous needle/catheter is advanced until air is aspirated into the syringe. The needle is withdrawn and the catheter is left open to air. An immediate rush of air out of the chest indicates the presence of a tension pneumothorax, which has thus been converted to a simple pneumothorax.

Because these catheters are small bore thin-walled catheters, they are prone to kinking and they may not completely relieve a tension pneumothorax. Dislodgement can also occur and lead to re-accumulation of air and recurrent tension pneumothorax. Thus, immediately following needle decompression, a standard thoracostomy tube should be performed, the size of which depends upon the expected pathology.

Other complications of needle thoracostomy include lung laceration and air embolism.  
(Source UpToDate)

## SKILL STATION: VASCULAR ACCESS

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### Intravenous access:

- Required for drug administration, fluid administration, blood sampling and transvenous pacing
- Circulation time varies from peripheral (5 mins) to central (30 sec)
  - Best to achieve most central position for vascular access AND to flush any drugs given with 20 mL saline

### *Cannula Sizing*

- Standard wire gauge (G)
  - Diameter of cannula increases with a DECREASE in number
- French gauge (F)
  - Diameter of cannula increases with INCREASE in number
- A large diameter, short cannula is best for rapid infusions

### Peripheral Access

- Inserted using either
  - A introducer in needle technique (most common practice)
  - A Seldinger technique (small cannula, wire, dilator over wire)
- Common sites for cannulation during circulatory collapse are
  - External jugular
  - Femoral vein
  - Long saphenous vein

### Central Cannulation

- Inserted using Seldinger technique
- Internal jugular vein and subclavian vein
- Complications
  - Arterial puncture
  - Haematoma
  - Haemothorax/pneumothorax
  - Venous air embolism
  - Cardiac arrhythmias
  - Damage to nerves (Brachial Plexus)
  - Sepsis

## Intra-osseous access

Utilizes direct access into the intramedullary cavity. Technique uses a rigid cannula directly into a long bone. There are dedicated intra-osseous devices but a 16 or 18 G needle with stylet can also be used.

### *Effective Route for*

- Fluid resuscitation
- Drug delivery
- Laboratory evaluation

## Indications

- In decompensated shock if intravenous access cannot be attained in <90 secs
- Cardiac arrest
- Acceptable method for all ages except newborn where umbilical venous access is still the preferred method.

## Contra-indications

- Fractures, burns or trauma at, or proximal to, the insertion site
- Previous sternotomy (for sternal IO site)
- Prosthetic joints near site of insertion
- Bone abnormalities (osteoporosis, osteogenesis)
- Inability to accurately identify insertion site (morbid obesity)

## Complications

- Failure to enter bone marrow → extravasation, subperiosteal infusion
- Through and through penetration of bone
- Epiphyseal plate injury
- Rare complications like infection, compartment syndrome fat/bone emboli

## Sites for intra-osseous access

- Proximal tibia (2cm medial, 1 cm superior from tibial tuberosity)
- Distal tibia (proximal to medial malleolus)
- Distal femur (midline, 2-3 cm above external condyle)
- Proximal humerus (1cm proximal to surgical neck)
- Upper sternum

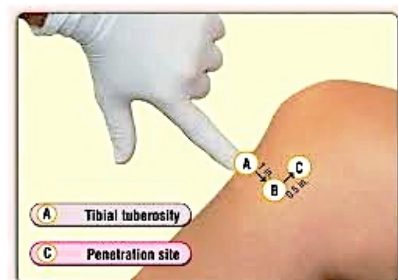


Photo courtesy of Waismed, Ltd. Houston, Texas.

**INTRA-OSSEOUS CANNULATION IS  
A TEMPORARY FORM OF ACCESS < 24 HOURS**

*Technique for insertion*

- Universal precautions
- Find the insertion point
- Clean the skin over the insertion site
- Local anaesthetic may be required in the conscious patient
- Immobilize the limb and place the tip of the needle over your insertion site
- Insert the needle at 90 degrees to the skin using a rotational motion (or drill if Ezi-IO)
- Advance needle until 'give' is felt
- Remove stylet
- Aspirate marrow if possible
- Inject 10 ml normal saline to confirm position (there should be no resistance or extravasation)
- Start infusion
- Up to 125 ml/min but pressure bag (up to 300 mmHg) may be required

*Technique for removal of intra-osseous cannula*

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

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# RECOVERY ROOM PROBLEMS

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Amanda Baric

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 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 once 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-Hydroxytryptamine-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 facemask 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

Cardiovascular 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)

- Confirmation of the severity of hypertension, check the pre-operative blood pressure and determine whether the patient is already being treated for hypertension.
- 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, pheochromocytoma, malignant hyperthermia, pre-eclampsia and autonomic dysreflexia.
- 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.
- 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.
- 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 inotropes 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 continuous 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 tachyarrhythmias, 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 inotrope. For the patient with hypertension, glyceryltrinitrate (GTN) is used to reduce preload in conjunction with frusemide (20 mg -80 mg). In the hypotensive patient, inotrope 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, if 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 re-orient 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 care-givers. It can present as a 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, myocardial 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 propylthiouracil 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

Michelle Chan

Malignant hyperthermia (MH) is a rare but potentially lethal reaction that can be triggered under general anaesthesia. It is a pharmacogenetic 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

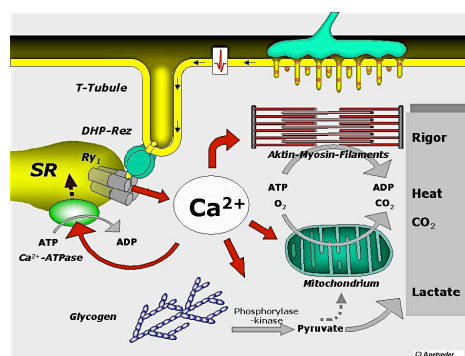
The disorder has a pattern of autosomal dominant inheritance with variable penetrance. It has an incidence of approximately 1:40,000 anaesthetics administered in adults, and 1:15,000 in children. There is a male predominance, and it is common for susceptible patients to have had more than one (and on 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. 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.



Source: [http://www.humphath.com/IMG/jpg\\_malignant\\_hyperthermia.jpg](http://www.humphath.com/IMG/jpg_malignant_hyperthermia.jpg)



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 (hypercapnia, 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 (CO<sub>2</sub>) production is caused by cellular hyper-metabolism. In the mechanically ventilated patient there will be a marked rise in end-tidal CO<sub>2</sub> on capnography. In the spontaneously ventilating patient a marked increase in respiratory rate will occur to try to blow off the extra CO<sub>2</sub>. The large amount of exhaled CO<sub>2</sub> will quickly heat and exhaust the CO<sub>2</sub> 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 (O<sub>2</sub>) consumption, with mottling of the skin and a decline in oxygen saturation (SaO<sub>2</sub>). The patient may become haemodynamically unstable, and arrhythmias 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 hyperkalaemia 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 ETCO<sub>2</sub>:

- Inadequate mechanical ventilation
- Patient who have been given opioids
- Laparoscopic surgery (CO<sub>2</sub> gas insufflation)
- Expired soda lime (high inspired CO<sub>2</sub>)

Causes of tachycardia:

- Inadequate anaesthesia
- Inadequate analgesia
- Infection
- Septicaemia
- Anaphylaxis
- Pheochromocytoma
- Thyroid crisis
- Drug reactions with or during anaesthesia (neuroleptic malignant syndrome, monoamine 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% O<sub>2</sub> 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, hypercapnia

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 hyper-metabolic 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 CO<sub>2</sub>, SaO<sub>2</sub>, electrocardiogram, blood pressure, and temperature.

Arterial blood gases should be repeated frequently for potassium levels, acid-base status, arterial CO<sub>2</sub>, arterial O<sub>2</sub> 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 normocapnia. 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 parameters. 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 techniques 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 anaesthetized with total intravenous anaesthetic agents (such as propofol infusion). If muscle relaxation is required a non-depolarising 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 ET<sub>CO</sub><sub>2</sub>, temperature, ECG and SaO<sub>2</sub> 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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