Update in Anaesthesia

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SPECIAL EDITION

Guidelines for Management of Emergencies in Anaesthesia

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Update in
Anaesthesia

Editorial

Welcome to Update 25:2, a special edition concentrating on the management of emergencies in anaesthesia. This compilation of the most practical and useful evidence-based algorithms and drills will guide management of emergencies in clinical care. Although prevention of life-threatening events is always preferable to treatment, critical events occur daily in anaesthesia and rely on anaesthetists being prepared to put in place effective clinical management.

Managing emergencies is often influenced by what we have learnt from prior experience - something that anaesthetists and theatre staff do on a daily basis, either as individuals or as a team. This ability to learn from our own and others' past performance, with a view to improving future performance, should now be formalised as a priority in every clinician's daily practice. Critical incident reporting and forums such as morbidity and mortality meetings contribute enormously to improving patient safety. These should be conducted in a supportive and blameless environment. We all work within financial constraints, but these safety-focused practices incur only minor, if any, cost. Detailed discussion of the factors that contribute to adverse events within healthcare is found in the first reference,[1] but the contents of this editorial, and the following articles, focuses primarily on treatment of critical events when they do occur.

Much of our time training and practising as anaesthetists is spent predicting and avoiding serious untoward events in theatre. Some adverse events occur because of errors in the clinical systems within which we work. The Safe Surgery Saves Lives campaign by the World Health Organisation aims to address some of these systemic factors using strategies such as the 'Time out' preoperative checklist.[2] Other potentially life-threatening events are unpredictable and cannot be prevented, for example anaphylaxis complicating induction of anaesthesia. We are trained to recognise these events, react rapidly and initiate treatment. However, well-documented incidents show that despite training, experience and a conscientious approach, the outcome for a patient suffering such an unpredicted event may still be poor.[3]

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An explanation for this is that these scenarios are a major challenge for an individual or group of individuals and they expose our inherent human limitations.[3] The clinical presentation may be non-specific, making diagnosis difficult – for example life-threatening hypoxia has numerous causes. In addition a wide variety of factors contribute to cause such crises. Factors include the patient's pathophysiology, staffing levels and skills, theatre and hospital infrastructure and the equipment that is available. As the crisis deepens the complexity of the situation may increase and the diagnosis may change, for example a case of post-extubation laryngospasm may be further complicated by negative pressure pulmonary oedema. Unfortunately the nature of this type of event, when it occurs during anaesthesia, means that recognition and intervention need to be undertaken rapidly if rapid deterioration is to be avoided. It is well recognised that an individual managing an emergency on their own, and in stressful circumstances, may lose awareness of time passing, and lose sight of the correct diagnosis and appropriate management. Focussing on a task such as intubation may take such concentration that options such as waking the patient up may be missed. Whilst recognising that rare or previously unreported events are harder to identify and therefore potentially harmful,[4] we also know that 'common things are common' and the diagnosis and management of the majority of crises is achievable using own prior experience or that of our colleagues .

Research performed using high-fidelity simulators demonstrates that experience does not mitigate against failure to deal with a simulated anaesthetic emergency.[1] If we rely on our ability to solve a previously un-encountered problem from first principles, progress may be slow, particularly under the stress of an evolving emergency situation. There is a proven advantage to asking for senior help in order to gain a fresh 'unstressed' perspective on the situation. This may also counter the phenomenon of 'confirmation bias' where an individual, mislead by earlier events within an evolving crisis, develops a 'strong but wrong' impression of an evolving situation.

Further information useful to guide management of these emergencies is generated by incident reporting and morbidity and mortality meetings. These systems are essential at a local level, and national data may be compiled to good effect as shown by the 'Crisis management during anaesthesia' resources produced by the Australian Patient Safety Foundation.[3]

A major component of the solution to managing emergencies is to encourage pre-designed responses using written algorithms, drills or protocols. These guide a healthcare worker through their responses at a time when their ability to think rapidly and rationally is compromised by the stress and possible consequences of the event. An algorithm guiding management of an emergency event must be structured, clear, quick and easy to follow, and cover all contingencies.[3] Clearly

there is a balance to be struck between achieving inclusiveness of all possible circumstances within an event and maintaining clarity and practical usefulness.

In compiling this edition we sought compact and clear diagrammatic guidelines that would serve as visual tools to guide management when placed on the walls of anaesthetic rooms and operating theatres in a wide range of healthcare settings. Each guideline is accompanied by a commentary which aims to expand upon the guideline, emphasising the logic behind a sequence of actions, and to describe in more detail practical procedures using unfamiliar equipment.

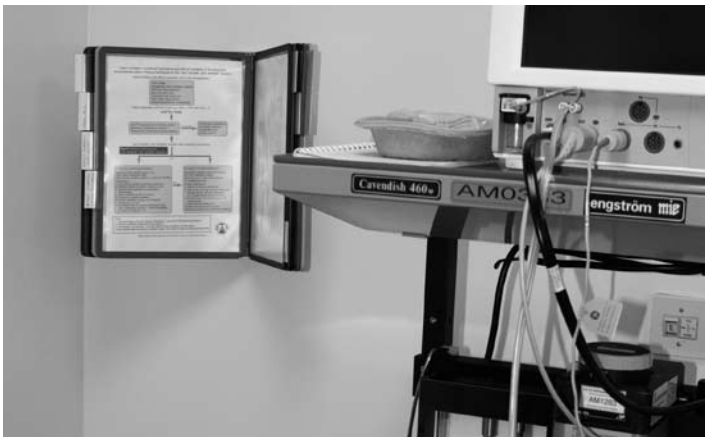
In some areas of practice robust published guidance is available from expert bodies or committees. However, in some areas it has proven particularly difficult to identify high quality guidance, for example for failed intubation in the obstetric setting. For all topics, where guidance is available, but not in a suitable format, we have endeavoured to create a clear single sheet diagrammatic guideline.

Some areas, such as management of major haemorrhage are complex and it is beyond our remit to try to construct a new guideline. Existing guidance for this topic is becoming increasingly outdated as evidence gathered from conflict zones around the world increasingly supports the use of 'massive transfusion packs' at the outset of management of severely injured patients, with the rationale that it is better to prevent coagulopathy than to attempt to treat it once it has occurred. These packs generally consist of four units of red cells that are used in conjunction with fresh frozen plasma and sometimes platelets. Several guidelines from the UK and US are available and these are currently being used by a working party of the Association of Anaesthetists of Great Britain and Ireland to draw up a guideline which should be freely available by the end of 2010. The European Resuscitation Council's updated algorithms for adult basic and advanced life will be published later this year and will be included in a later edition of *Update in Anaesthesia*.

Clearly production and distribution of a protocol is not sufficient on its own – management of life-threatening emergencies within anaesthesia requires training and practice, not just for the anaesthetist, but for the multi-disciplinary theatre team. Take-up of training is limited and it has been documented that most anaesthetists view themselves as 'better than average', a situation that is not mathematically possible and this may explain why uptake of these training sessions is still poor!

In summary, there is growing support for use of algorithms to manage emergency situations within anaesthesia and critical care. In this edition we feature the best guidelines we could find and hope that this compilation of emergency algorithms contributes to improving patient safety within your own practice, your team and your hospital. Display the algorithms in an appropriate place where they can easily be seen for immediate reference (Figure 1). For each one, familiarise yourself with its sequence and contents, in the context of the commentary that follows each algorithm. This process may be aided by discussion at tutorials or departmental meetings - present cases where the algorithm has been used and identify its merits and limitations.

Each algorithm may need to be adapted to the setting in which you work. Remember that practice is fundamental to success. Simulation does not have to be high-fidelity, but can be equally usefully conducted around a set of theatre scrubs laid out on a desk, with enthusiasm from the participants involved. Whilst we are all aware of our own past involvement in management of crises, much can be learnt by implementation of an open and blameless system for discussion of critical incidents within your department as a whole.



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Editor-in-chief

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Update Team

News from the WFSA

The Obstetric Anaesthesia Committee

The past year for the Obstetric Committee has been one of forging links with sister organizations and attempting to build the bridges by which we may collaborate on future multidisciplinary projects.

Late in 2009, following an earlier introductory meeting in London, the Chairman, Dr Paul Howell, travelled to Cape Town as guests of FIGO (the International Federation of Gynecology and Obstetrics) to participate in their World Congress. There he joined round-table panel discussions devoted to the FIGO initiative on Maternal and Newborn Health supported by the Bill and Melinda Gates Foundation. He made clear to parties who habitually forget to include anaesthetic involvement in the planning of maternity projects (e.g. obstetricians, midwives, health planners) how pivotal we are in improving obstetric surgical outcome in resource poor areas.

The WFSA has now become a Partner in the World Health Organization Partnership for Maternal, Newborn and Child Health (PMNCH), a multidisciplinary alliance of interested parties who are working to improve the health of mothers and children worldwide. This will hopefully improve our international profile and our ability to liaise with like-minded organizations on joint future projects – all too pressing since it's now clear that Millennium Development Goals 4 and 5 are far from being met.

Links with the Obstetric Anaesthetists' Association (OAA) and Association of Anaesthetists of Great Britain and Ireland (AAGBI) continue to grow, and the WFSA has joined forces with them on several interesting ventures. Thanks to a generous grant from Baxter, and collaboration with the OAA and Elsevier, publishers of the *International Journal of Obstetric Anesthesia* (IJOA), a two CD set of useful obstetric anaesthetic resource material is being produced for distribution in resource-poor countries. This set, which comprises a variety of different tools including a webcast of the 2008 OAA Three Day Course with slides and abstract book, video of how spinals work, and back copies of *IJOA*, *Update in Anaesthesia* and *Tutorial of the Week*, is almost ready for circulation through usual WFSA routes.

In addition, in collaboration with the Publications Committee, the OAA and the AAGBI, an exciting new handbook of obstetric anaesthesia, specifically targeted at anaesthetic providers in resource-poor area, has just been completed, and is ready for shipping. Already in hardcopy, it is hoped to make it available in electronic format at some point in the future.

As and when these two new educational tools are received, please do feed back to us with your comments, including what is useful, what is not, and what else would you like included (for the next editions)!

Around the world, individual members of the Obstetric Anaesthesia Committee continue to make significant contributions to the practice of obstetric anaesthesia and analgesia in their own regions, and beyond. Everyone plays their part, but special mention should perhaps be made of Dr Medge Owen who heads Kybele, an organisation that takes multidisciplinary teams into transitional level countries and shows how obstetric (anaesthetic) care can be improved through a combination of formal lectures and hands-on practical tuition. Recent publications show that this approach can make a lasting impact, with sustained changes in practice – an excellent example to us all![1,2]

Finally, in the not too far distant future, our next World Congress in Argentina in 2012 approaches. There will, of course, be an obstetric anaesthetic component to the meeting – always popular sessions – so put the dates in your diary, and see you there!

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WFSA

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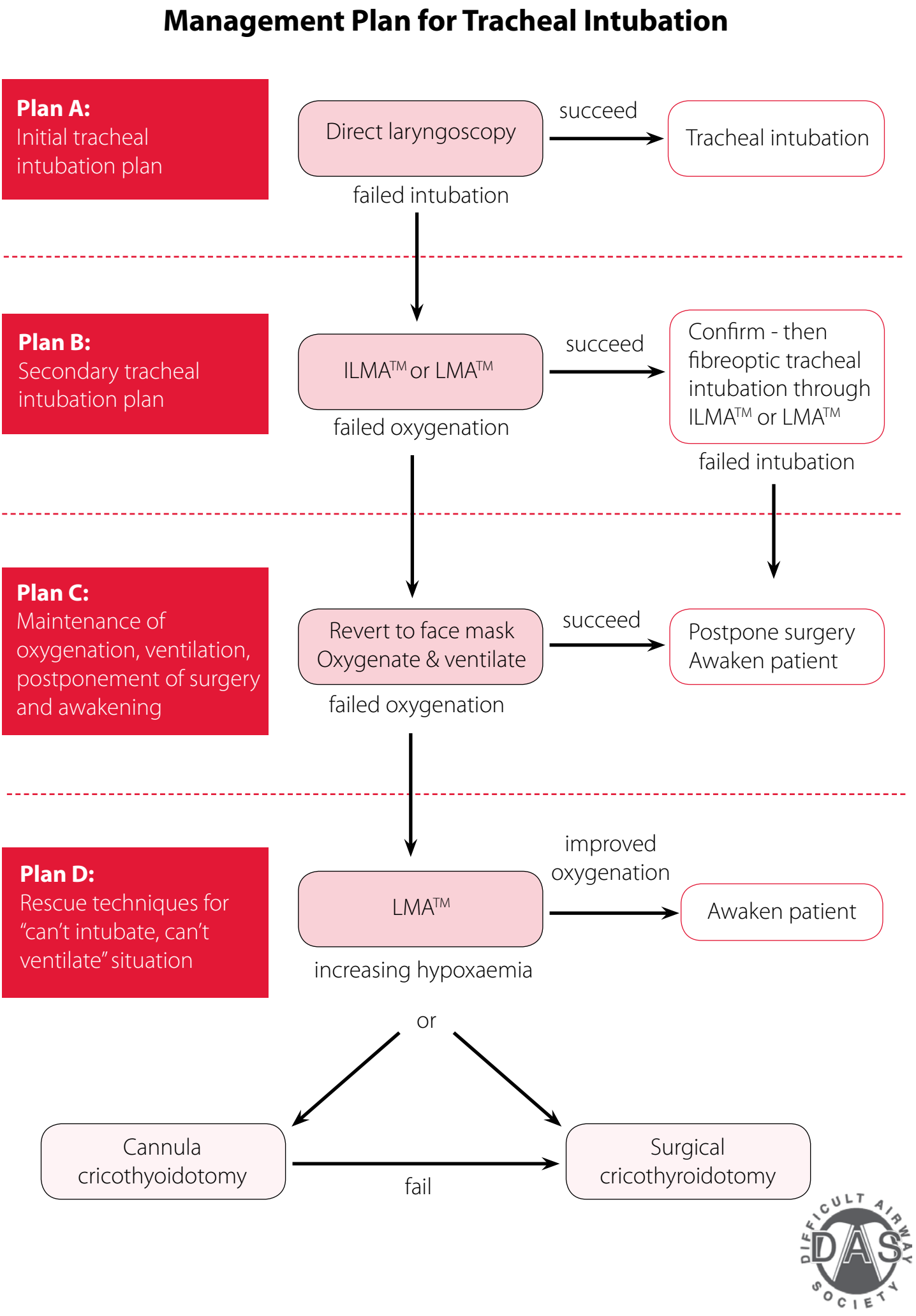

Figure 1. *Reproduced by kind permission of the Difficult Airway Society (UK) and available for download at*: www.das.uk.com/files/simple-Jul04-A4.pdf

Management plan for tracheal intubation

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Summary

Oxygenation takes priority over everything else.

Avoid trauma by minimizing attempts.

Call for help early.

Plan carefully, for every case.

INTRODUCTION

Tracheal intubation is not the main objective of airway management – maintenance of adequate oxygenation is paramount and this can usually be achieved without intubation. The second objective of airway management is to achieve adequate ventilation, i.e. adequate oxygenation plus adequate CO_2 removal. The third objective is to secure the airway from aspiration.

For many years direct laryngoscopy and tracheal intubation has been the mainstay of airway management. While it is true that an endotracheal tube will achieve all three objectives, other techniques such as the various supra-glottic devices (Table 1) or a cricothyroid puncture can at least provide for adequate oxygenation and maybe more, and should therefore be important components of the airway management algorithm. Recently a number of different airway management techniques have been introduced. The use of fiberoptic bronchoscopy has become widespread and supra-glottic devices and video-laryngoscopy have resulted in very significant changes to clinical practice (Figure 2).

Various societies and national organizations have produced guidelines on the management of the difficult airway and intubation. One of the first, and probably the best known, is the American Society of Anesthesiologists (ASA) algorithm which was published in 1993 and revised in 2003.[1] The Canadian, Italian and French anesthesiology societies, amongst others, have also introduced algorithms. In 2004 the Difficult Airway Society (DAS) published their guidelines,[2] which are the subject of this overview and will be analysed in subsequent articles in this issue of *Update in Anaesthesia*.

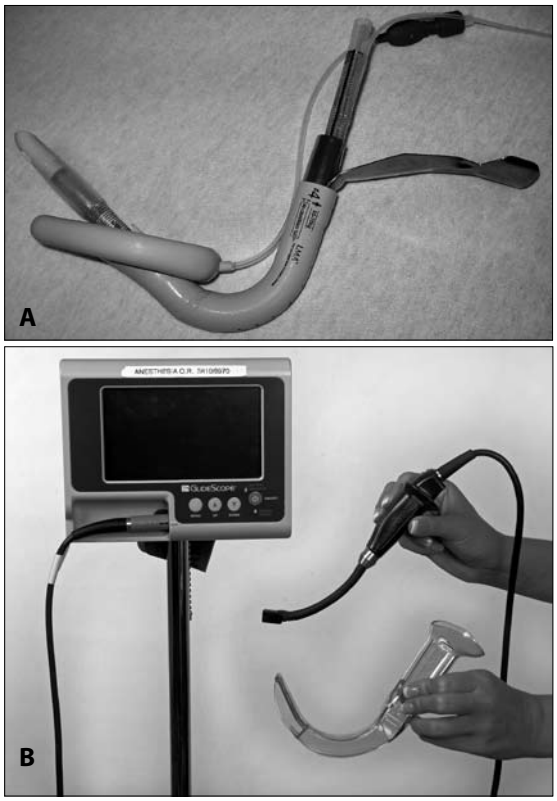

Figure 2. *Examples of airway devices – (A) the intubating laryngeal mask airway (ILMA) and (B) the GlideScope*

TYPES OF ALGORITHMS

An algorithm takes the clinician through a series of decisions and actions from a start point to a final

Table 1. *Airway devices*

Supraglottic airway devices	
	Laryngeal mask airway, ProSeal, Supreme, Air-Q, Slipa, Cobra, I-Gel, Intubating Laryngeal Mask Airway, CombiTube, EZ Tube
Indirect visual laryngoscopy	
Rigid:	GlideScope, McGrath, Pentax AWS, Storz, Bullard, Wu, C-Trach
Stylet:	Shikani, Levitan, Bonfils

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outcome. Anyone designing an algorithm must first decide what the start point should be, and what will constitute an acceptable end state. In the case of the DAS algorithm the start point is when an attempt is made to intubate a patient who is not expected to have a difficult intubation. The construction of the ideal algorithm is difficult because some of the characteristics of the ideal algorithm are contradictory (see Table 2)

Table 2: *Characteristics of the ideal algorithm*

1. Feasible
2. Short (not too much detail)
3. Simple to memorize
4. Covers all possibilities
5. Effective
6. Provides choice
7. Specific (limited or no choice at each point)
8. Deals with anticipated and unanticipated difficult intubations.
9. Evidenced-based

The ASA algorithm is very thorough and offers many choices to the clinician, but this means that it is also complex which limits its clinical usefulness.[1] Studies have shown that many anesthesiologists do not, and perhaps cannot, memorize the algorithm. The DAS algorithm on the other hand offers a binary outcome at each point with no, or very limited choice, with the intention that the definitive and simple flow-charts will be easier to use.

COMMENTARY ON ALGORITHM

The DAS algorithm specifically deals with the ***unanticipated*** difficult tracheal intubation, and consists of a series of plans - Plan A, Plan B, Plan C and Plan D. The structure of the basic algorithm is shown in Figure 1. The basic algorithm is modified depending on the clinical scenario, either routine induction or rapid sequence induction (with an increased risk of aspiration).

The DAS algorithm has been deliberately designed to provide limited choices at each decision point, in order to make it more memorable and easier to follow under stressful circumstances. The DAS algorithm assumes that optimal attempts have been made at direct laryngoscopy including patient positioning, the use of external laryngeal manipulation, an appropriate choice of tube and the use of a gum elastic bougie (Eschmann tracheal tube introducer).

Plan A

Plan A of the DAS algorithm emphasizes that it is necessary to "limit the number and duration of attempts at laryngoscopy in order to prevent trauma and development of a 'can't ventilate, can't intubate' situation." It is difficult to justify use of the same direct laryngoscope more than twice and the maximum number of laryngoscope insertions should be limited to four. However, tracheal intubation may be successful when it is performed by a more experienced anesthetists and one such additional attempt is worthwhile.

Plan B

Plan B requires a change to an alternative technique - there is no point in continually attempting the same technique and expecting a different outcome! The DAS algorithm specifically recommends the use of a Laryngeal Mask Airway (LMA) or Intubating LMA. This recommendation may be changed depending on the specific local conditions. Other supra-glottic devices are acceptable, as would other intubating techniques such as video-laryngoscopy, with the proviso that adequate expertise and equipment are available.

Plan C

Plan C emphasizes the importance of allowing the patient to wake and postponing surgery. This may not always be possible, but it is definitely the safest course of action. When the patient is awake and able to maintain and protect their own airway, then further management can be planned. At this stage the patient is known to have an anticipated difficult airway, which is briefly discussed below.

Plan D

Plan D describes the management of the 'cannot ventilate, cannot intubate' situation. This is a life-threatening situation which is rare, but must be dealt with immediately, and is discussed in more detail in a subsequent article.

Limitations of the DAS algorithm

The DAS algorithm does not cover the anticipated difficult airway, nor does it address prior recognition of a challenging airway. The algorithm also does not deal with the obstetric patient or the paediatric patient.

Anticipating the difficult airway

It is very difficult to predict all difficult intubations. Some clinical situations, such as severe facial trauma and large intra-oral tumors, may be clear-cut, but identification of more borderline cases is a challenge. Tests such as the Mallampati classification, the thyromental distance, the mandibular protrusion and many others have been proposed but none can accurately predict difficult laryngoscopy. Even combinations of these tests cannot provide high levels of sensitivity (predicting cases that will be difficult) or specificity (predicting cases that are not difficult).[3] Part of the problem is that the tests only examine patient factors and do not account for the skill of the intubator. It is therefore appropriate to have a clear plan for the difficult intubation for every case undergoing anesthesia.

All patients must be examined prior to induction of anesthesia. The airway examination must consider the following questions:

Management of an anticipated difficult airway

If difficult intubation is anticipated, always consider whether the proposed surgery can be done under regional anesthesia. Regional techniques are advantageous because the patient can remain awake and can protect and maintain their own airway. However, even in cases when regional anesthesia will be used, a thorough airway examination and appropriate planning must still be done. Patients receiving local anesthesia may develop an anaphylactic reaction, may have a high neuraxial block, or may require conversion to a general anesthetic technique during the procedure. Regional techniques reduce, but do not eliminate, the risk of loss of airway control.

If the answer to Question 1 (Table 3 - will it be possible to bag mask ventilate (BMV) this patient?) is 'No', do not induce general anesthesia, and be cautious about administering sedation. If a regional

Table 3. *Preoperative airway assessment*

1. ***Will it be possible to bag-mask ventilate (BMV) this patient?***
   Indicators of difficulty include a beard, facial trauma, no teeth, history of snoring and sleep apnoea.
2. ***Will it be possible to insert a supra-glottic device?***
   Indicators of difficulty include a small mouth opening, large pharyngeal masses, grossly distorted anatomy.
3. ***Will it be possible to intubate?***
   Indicators of difficulty include small mouth opening, large tongue, severe bleeding, abnormal dentition, inability to move the mandible or neck, and abnormal findings using the various tests mentioned above.
4. ***Will it be possible to perform a cricothyroid puncture?***
   Indicators of difficulty include a very short neck, radiation fibrosis, severe obesity, and a large thyroid goitre.

technique is not possible and the patient requires intubation, then consider awake fiberoptic intubation where the facilities are available. If you do not have the equipment or the necessary skill to perform an awake fiberoptic intubation, then other awake techniques can be done. For example, with adequate topical anesthesia an awake LMA or Intubating LMA can be inserted.[4]

If the answer to Question 1 is 'Yes', then general anesthesia can be induced, provided that adequate preparation has been made for the subsequent airway management. Especially important is to pre-oxygenate the patient thoroughly. A gas induction using halothane has been well-described. Gas induction using sevoflurane should only be performed by experienced anaesthetists since the rapid onset of anaesthesia and the limited metabolism of this agent may result in deep anaesthesia, apnoea and/or airway obstruction. It is far better to use an induction agent such as propofol or etomidate which is rapidly redistributed and allows the patient to wake up fast. Avoid the use of muscle relaxants until it can be shown that airway patency can be maintained after induction. If muscle relaxants are used, use suxamethonium (succinylcholine) in preference to long-acting neuromuscular blocking agents.

The contra-indications to the use of a supra-glottic device are still not clear. For example, in patients with GORD (gastro-oesophageal reflux disease) some people would use a supra-glottic device with a gastric reflux relief tube, such as the ProSeal LMA, while others would not. The same applies to the use of a supra-glottic device in the lateral and prone positions. In the absence of evidence, the choice depends on the operator's familiarity and level of comfort with the device.

A supra-glottic device can also be used as a conduit to place an endotracheal tube, as discussed in a subsequent article. Various other techniques exist which are beyond the scope of this article. For a detailed discussion of this topic, reference 3 is useful.[3]

Extubation algorithm

Any difficult intubation requires careful planning for the extubation. Recently various extubation algorithms have been suggested.[5] First the patient must meet the normal extubation criteria, such as demonstrating adequate tidal volumes and appropriate muscle strength. The cuff leak test (deflating the endotracheal tube cuff after suctioning the pharynx) checks that there is not excessive airway edema. As part of the extubation strategy following difficult intubation, most authors suggest the insertion of a tube exchanger (such as the Cook airway exchange catheter) through the endotracheal tube before the patient is extubated. The endotracheal tube is then removed, leaving the tube exchanger in place. Oxygen can be insufflated via the tube exchanger, and in case of respiratory decompensation a jet ventilator can be attached to the tube exchanger and used to oxygenate and ventilate the patient. Do not use a jet ventilator unless you have been specifically trained in its use. Jet ventilation can be associated with significant morbidity and even mortality. Re-intubation over a tube exchanger is associated with a high success rate, even in cases of a difficult initial intubation. It is recommended that an endotracheal tube one or two sizes smaller should be used. If the patient tolerates extubation well, then the tube exchanger can be removed. A further discussion of aspects of extubation is available in the previous edition of *Update in Anaesthesia*.[6]

CONCLUSION

All patients who have had a difficult intubation should receive a letter describing the difficulty with the airway, which techniques were used, and recommendations for future anesthesia. One copy should be placed in the patient's file, and the other copy given to the patient.

Airway emergencies can occur unexpectedly so familiarity with the management algorithms is essential. Appropriate training must be provided to everyone providing airway management, and the hospital management must ensure adequate functioning equipment.

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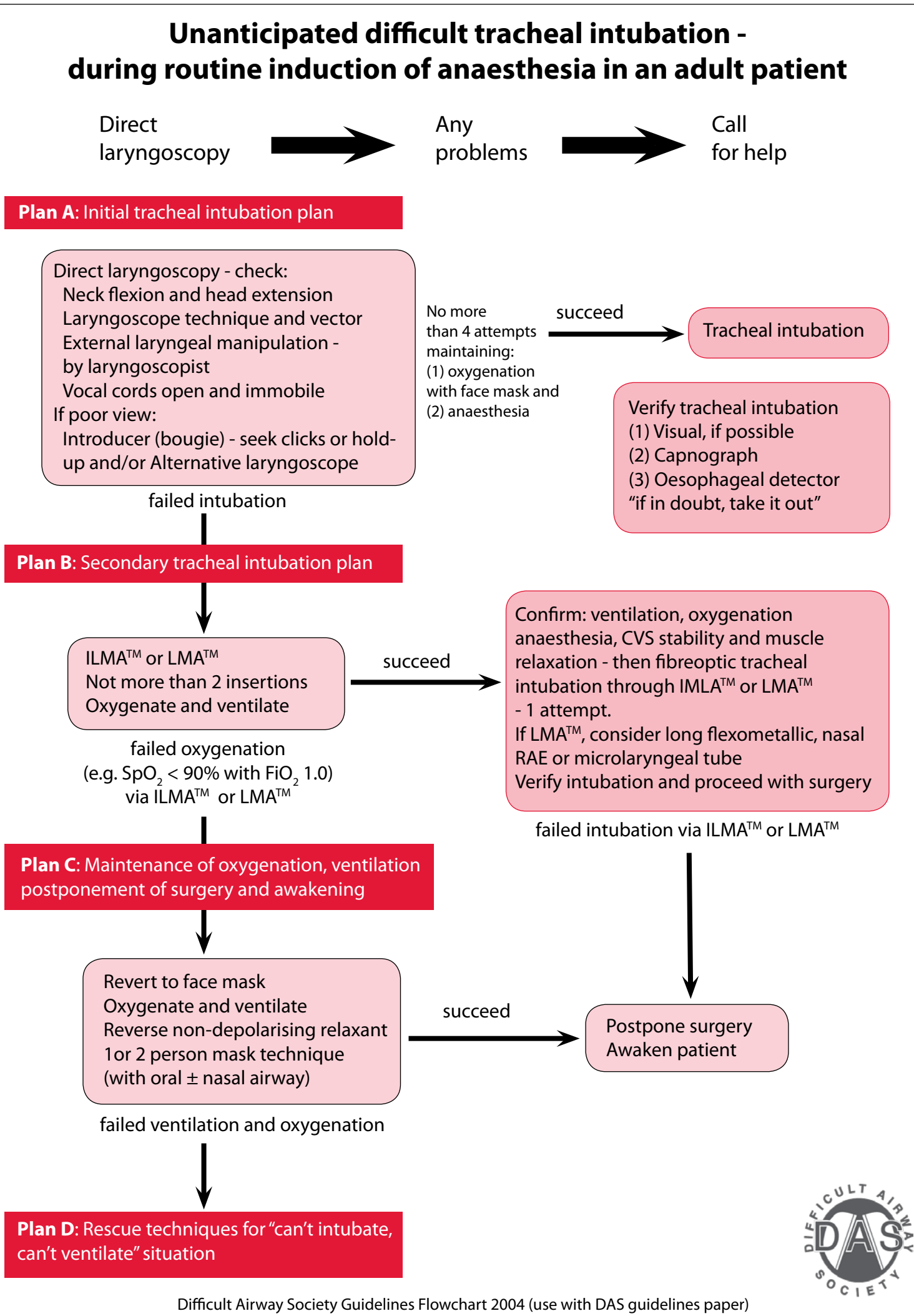

Figure 1. *Reproduced by kind permission of the Difficult Airway Society (UK) and available for download at*: www.das.uk.com/files/ddl-Jul04-A4.pdf

Update in
Anaesthesia

Management of unanticipated difficult tracheal intubation: routine induction and rapid sequence induction of the non-obstetric patient

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Summary

Call for help early.

Always have a backup plan.

No more than 4 attempts at the initial intubation plan.

Be familiar with the various types of laryngeal mask airways.

No more than 2 attempts at the use of a laryngeal mask airway.

Do not traumatize the airway – maintain oxygenation and postpone surgery.

If necessary, do not delay cricothyroidotomy.

INTRODUCTION

Difficult intubation of the trachea is rare, but it can be encountered after routine induction of general anesthesia even if the airway examination did not provide any suspicion of a difficult airway. A difficult intubation may be defined as not being able to visualize any portion of the vocal cords after multiple attempts of conventional laryngoscopy by an experienced person. However it might be difficult to intubate the trachea even if the larynx can be visualized. Such a situation may be associated with the feared 'can't ventilate, can't intubate' scenario, which may result in brain damage or death if not managed appropriately. In order to avoid this catastrophic outcome, several societies have developed algorithms to provide a decision tree for managing the unanticipated difficult tracheal intubation. The British Difficult Airway Society (DAS) has developed very simple algorithms that break the management strategy into four plans (A to D) using only a few airway devices which are available in many settings, mainly the classic laryngeal mask airway (cLMA) and the intubating LMA (ILMA). There is an algorithm for standard anaesthesia induction and a second algorithm dealing with rapid sequence induction.

COMMENTARY ON ALGORITHMS (Figures 1 and 2)

PLAN A

The initial intubation plan has to be carried out under optimal conditions, including:

1. Optimal head and neck position

The head should be in the sniffing position, where the external auditory meatus is at the level of the sternal notch.

2. Sufficient muscle relaxation

Use either a non-depolarizing agent or suxamethonium, depending on the clinical scenario.

3. Optimal external laryngeal manipulation

Use your right hand to apply backwards, upwards, rightwards pressure on the larynx (BURP manoeuvre).

If these manoeuvres do not improve the laryngeal view to better than a Cormack and Lehane grade III or IV (i.e. some part of the vocal cords are visable), alternative intubation techniques should be used. Different laryngoscopes are useful, such as straight blades (Miller) or flexible tips (McCoy). An introducer ('gum elastic bougie', 'Eschman') is inexpensive and should be readily available. It is easy to use and has a great success rate. The 'gum elastic bougie' can be inserted blindly under the epiglottis in case of a Cormack and Lehane grade III or IV view. The laryngoscope should be kept in place to optimize the view and success rate. Bougie insertion is a blind technique and indications of correct placement of the bougie are:

1. Tracheal clicks

Clicks can be felt when the flexed tip of the bougie is advanced along the tracheal rings.

2. Resistance after approximately 45cm

If there is no resistance felt after about 45cm, the bougie is likely in the esophagus and not in the bronchial tree.

3. Coughing

The sensitive tracheal mucosa will often make the patient cough if there is a foreign body (bougie) in the trachea. If muscle relaxation has been used this sign will be absent.

Once the bougie is in the trachea, an endotracheal tube is railroaded over it. A laryngoscope and approximately 90 degrees of anti-clockwise rotation of the tube will facilitate the passage of the tube past the vocal cords.

Plan A should be abandoned after a total of four attempts (with no more than 2 different techniques) to intubate the trachea, otherwise the risk of trauma and swelling of the upper airway with the potential to result in a 'can't ventilate, can't intubate' situation, becomes too high.

In case of a routine induction of general anesthesia (Figure 1), the next step is Plan B. In case of a rapid sequence induction (Figure 2), Plan B should be skipped and Plan C instituted. Plan B consists of

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Unanticipated difficult tracheal intubation - during rapid sequence induction of anaesthesia in non-obstetric adult patient

Direct laryngoscopy → Any problems → Call for help

Plan A: Initial tracheal intubation plan

Pre-oxygenate
Cricoid force: 10N awake → 30N anaesthetised
Direct laryngoscopy - check:
- Neck flexion and head extension
- Laryngoscopy technique and vector
- External laryngeal manipulation - by laryngoscopist
- Vocal cords open and immobile

If poor view:
- Reduce cricoid force
- Introducer (bougie) - seek clicks or hold-up and/or Alternative laryngoscope

succeed → Tracheal intubation

No more than 3 attempts maintaining:
(1) oxygenation with face mask
(2) cricoid pressure and
(3) anaesthesia

Verify tracheal intubation
(1) Visual, if possible
(2) Capnograph
(3) Oesophageal detector
"if in doubt, take it out"

failed intubation

Plan C: Maintenance of oxygenation, ventilation postponement of surgery and awakening

Maintain 30N cricoid force

Plan B not appropriate for this scenario

Use face mask, oxygenate and ventilate
1 or 2 person mask technique
(with oral ± nasal airway)
Consider reducing cricoid force if ventilation difficult

succeed → Postpone surgery and awaken patient if possible or continue anaesthesia with LMA™ or Proseal LMA™ if condition immediately life-threatening

failed oxygentation
(e.g. SpO_2 < 90% with FiO_2 1.0) via face mask

LMA™
Reduce cricoid force during insertion
Oxygenate and ventilate

succeed → Postpone surgery and awaken patient if possible or continue anaesthesia with LMA™ or Proseal LMA™ if condition immediately life-threatening

failed ventilation and oxygenation

Plan D: Rescue techniques for "can't intubate, can't ventilate" situation

Difficult Airway Society Guidelines Flowchart 2004 (use with DAS guidelines paper)

Figure 2. *Reproduced by kind permission of the Difficult Airway Society (UK) and available for download at*: www.das.uk.com/files/rsi-Jul04-A4.pdf

further intubation attempts with an unprotected airway, which is considered not appropriate in a patient with a full stomach because of a higher risk for aspiration.

PLAN B

Plan B consists of a secondary intubation plan using either the classic LMA (cLMA) or the intubating LMA (ILMA). The primary goal is to establish and confirm ventilation and oxygenation. If oxygenation fails (SaO_2 < 90% with FiO2 1.0) after no more than two attempts at inserting the device, the next step is moving on to Plan C.

If the patient can be ventilated and oxygenated, remains hemodynamically stable and is anesthetized and paralyzed, one attempt at intubating the trachea through either the cLMA or the ILMA should be performed. Ideally this would be performed with the guidance of a fiberoptic bronchoscope, but blind intubations through either the cLMA or the ILMA have high success rates.

Fiberoptic technique for intubation via a laryngeal mask airway:

1. Chose an endotracheal tube which will fit into the stem of the LMA.
2. Ensure that the scope and the endotracheal tube are well lubricated.
3. Pass the fiberoptic scope through the endotracheal tube.
4. Insert the fiberoptic scope into the stem of the LMA, and advance the scope to visualize the aperature bars.
5. Pass between the bars, visualize the vocal cords and pass the scope between the cords.
6. Slide the endotracheal tube over the scope and into the trachea.
7. It is advisable to use a longer than normal endotracheal tube to intubate through a cLMA, because of the longer tube of a cLMA compared to the ILMA.
8. If available, an Aintree tube exchange catheter makes the removal of the LMA possible.

The intubating laryngeal mask airway (ILMA)

The ILMA consists of a metal tube that is shaped to match the curvature of the upper airway. It has a cuff similar to the classic LMA. An epiglottis elevation bar is situated at the distal end of the ILMA stem. It is raised by the inserted endotracheal tube to move the epiglottis out of the way of the tube.

Patient weight	ILMA size
< 50 kg	Size 3
50 - 70 kg	Size 4
> 70 kg	Size 5

The correct size of ILMA must be selected. The manufacturer's recommendations are:

Before starting the procedure it is important to check that the equipment is complete and functional, in particular that there is no cuff leak. The use of an intubating LMA requires 6 pieces of equipment, shown in Figure 2.

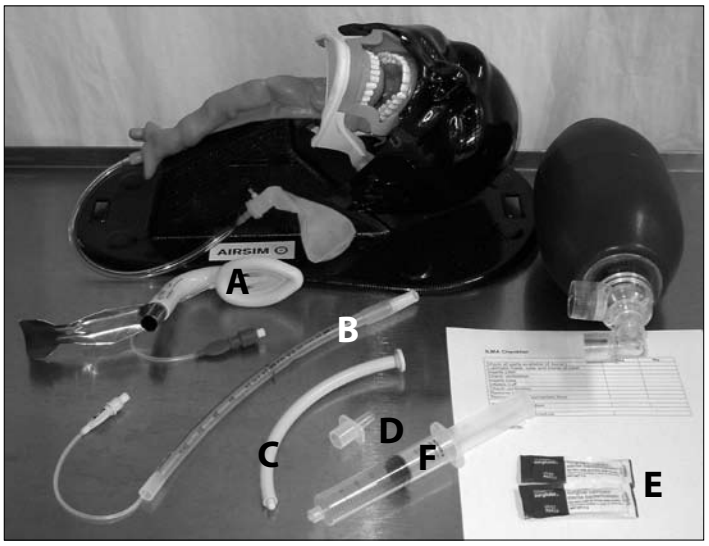

Figure 2. *The components of the ILMA; A – the ILMA itself, B – a flexible wire - reinforced endotracheal tube, C – bougie to secure endotracheal tube position as ILMA is removed from mouth, D – 15mm tube connector and syringe for cuff, E – lubricating gel, F - a syringe to inflate the cuffs of both the LMA and the tube*

The inside of the ILMA stem needs to be lubricated and the ETT should be pushed through the ILMA stem repeatedly until it slides without resistance.

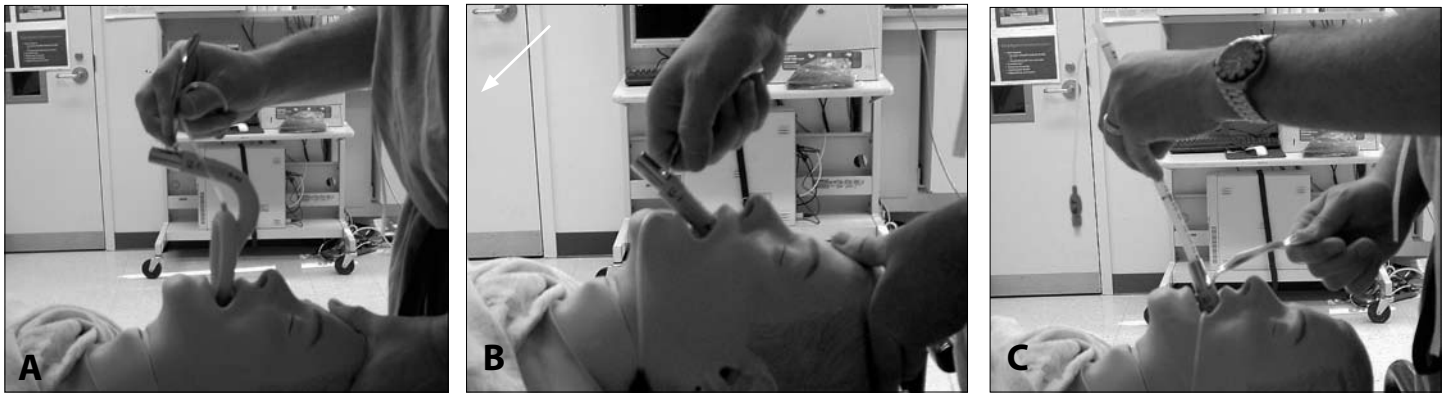

Figure 3. *Intubation using the ILMA*

Insertion of the ILMA

After insertion of the ILMA following its curvature and inflation of the cuff, the ventilation device can be connected to the ILMA. Easy manual ventilation should be confirmed. If ventilation is appropriate one can proceed to intubation. If ventilation is inappropriate, several maneuvers can be performed and repeated, if necessary:

1. Lift ILMA towards anterior neck of the patient to get a better seal using the handle.
2. Rotate ILMA left or right.
3. Remove the ILMA 6cm (markings on tube) out of its position keeping the cuff inflated to allow for a down folded epiglottis to lift up (Up/down or Chandy manoeuvre).

Intubation

Once ventilation is established, push the ETT through the ILMA. The ETT has two black lines, one long longitudinal line and one short vertical line. The longitudinal line should face cephalad. In this position the bevel of the ETT tip will pass between the vocal cords in a sagital position, allowing for an easier passage. The vertical line will enter the ILMA stem when the tip of the ETT exits the ILMA. At this stage a small resistance will be felt. With further advancement of the ETT there should be no more resistance, otherwise the ETT has likely entered the esophagus. After placement of the ETT and cuff inflation, ventilation should again be confirmed. If there are problems with ETT placement or ventilation, the above maneuvers should be performed.

Removal of the ILMA

After confirmation of ventilation the ILMA can be removed. The ETT connector has to be removed and is best left on the ventilation device to prevent it from being lost. If it does get lost, a connector from any regular tube should be readily available. The rod is used to hold the ETT in place while the ILMA is being gently removed. The ETT cuffs should stay inflated while the ILMA cuff can be deflated. As soon as the ETT can be grabbed in the mouth of the patient the rod has to be removed to allow the ETT cuff to fit through the ILMA tube. Now the ETT can be reconnected to the ventilation device and ETT depth can be readjusted, guided by auscultation.

PLAN C

There are two slightly different versions of Plan C, depending on whether the induction is a routine induction of anesthesia or a rapid sequence induction.

Routine induction

If the initial intubation plan as well as the backup plan using a laryngeal airway fails, Plan C involves reverting to facemask ventilation. The goal is to awaken the patient and to postpone surgery after reversal of muscle relaxation. Any further attempts at intubating the trachea can traumatize the airway and may lead to airway obstruction very rapidly.

If face mask ventilation turns out to be difficult every effort should be made to optimize the patency of the upper airway. Successful maneuvers can be:

1. Maximal jaw thrust and chin lift
2. Two handed mask technique
3. Oral airway
4. Nasal airway.

Rapid sequence induction

In the case of a rapid sequence induction, there is a higher risk for regurgitation and vomiting with the subsequent risk for pulmonary aspiration and pneumonitis. Therefore the use of cricoid pressure is still recommended, however its use is controversal. Since the airway may be further compromised by cricoid pressure, one should consider reducing its force or releasing it when ventilation proves to be difficult. If this does not improve ventilation and oxygenation, the use of a laryngeal mask airway is recommended. The patient should be awoken unless surgery is emergent and the patient's condition is immediately life threatening. In this case one should consider performing the surgery with the use of a LMA.

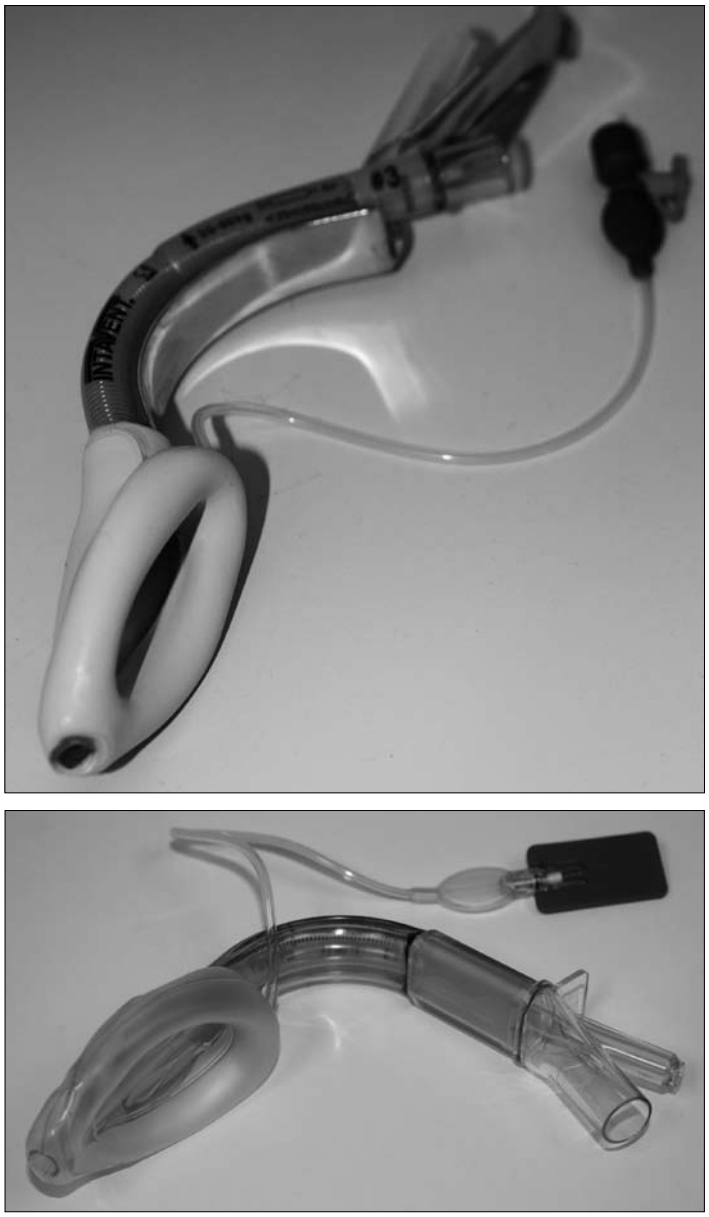

Figure 4. *The proseal LMA and LMA supreme*

In this situation, the Proseal LMA or the LMA Supreme, where available, may be safer alternatives to the classic LMA because of an additional channel to drain the stomach. Placement of both devices is slightly more complex than using a cLMA. Different test should be performed to confirm their correct positioning, i.e separation of the alimentary track and the airway.

1. The "Bubble Test"

The gastric channel is sealed with lubricant. With positive pressure ventilation, there must be no bubbles appearing at the gastric tube opening. Bubbles would indicate an incomplete separation of the airway from the esophagus with the risk of gastric inflation. With the tip of Proseal or Supreme LMA sitting in the upper esophagus, an unobstructed gastric tube would include an air column. Pressing on the sternal notch over the esophagus would move this air column and produce bubbles at the opening of the gastric tube.

2. Gastric tube

A gastric suction tube should be inserted through the gastric tube of the LMA. The tube should pass without problems and the stomach should be suctioned. After suctioning of the stomach the gastric suction tube should be removed. If left in place, the distal opening of the LMAs gastric tube would be occluded and the gastric suction tube could worsen reflux of gastric contents, leading to a higher aspiration risk.

For either of the pathways, routine induction or rapid sequence induction, it is of utmost importance to recognize inadequate oxygenation of the patient. Any delay in moving on to Plan D may result in severe hypoxia with potentially catastrophic consequences, i.e. brain damage or death.

PLAN D

Plan D is the rescue technique for the feared 'cannot intubate, cannot ventilate' scenario. Its endpoint consists of a cricothyroidotomy, using either a large bore cannula or a scalpel. A thorough discussion of Plan D has been published elsewhere in this issue.

FURTHER READING

Henderson J, Popat M, Latto I, Pearce A. Difficult Airway Society guidelines for management of the unanticipated difficult intubation. *Anaesthesia* 2004; **59**: 675-94.

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Bogetz, MS. Using the laryngeal mask airway to manage the difficult airway. *Anesthesiology Clinics of North America* 2002; **20**: 863-70.

Failed intubation, increasing hypoxaemia and difficult ventilation in the paralysed anaesthetised patient. Rescue techniques for the "can't intubate, can't ventilate" situation

failed intubation and difficult ventilation (other than laryngospasm)

Face mask
Oxygenate and Ventilate patient
Maximum head extension
Maximum jaw thrust
Assistance with mask seal
Oral +/- 6mm nasal airway
Reduce cricoid force - if necessary

1

failed oxygenation with face mask (e.g. SpO_2 <90% with FO_2 1.0)

Call for help

2

LMA™ Oxygenate and ventilate patient
Maximum 2 attempts at insertion
Reduce any cricoid force during insertion

succeed

3

Oxygenation satisfactory and stable. Maintain oxygenation and awaken patient

"can't intubate, can't ventilate" situation with increasing hypoxaemia

Plan D: Rescue techniques for "can't intubate, can't ventilate" situation

or

4

Cannula cricothyroidotomy

Equipment: kink-resistant cannula, e.g. Patil (Cook) or Ravussin (VBM)
High pressure ventilation system, e.g. Manujet III (VBM)

Technique

1. Insert cannula through cricothyroid membrane
2. Maintain position of cannula - assistant's hand
3. Confirm tracheal position by air aspiration - 20ml syringe
4. Attach ventilation system to cannula
5. Commence cautious ventilation
6. Confirm ventilation of lungs and exhalation through upper airway
7. If ventilation fails, or surgical emphysema or any other complication develops - convert immediately to surgical cricothyroidotomy

fail

Surgical cricothyroidotomy

Equipment: Scapel - short and rounded (no. 20 or Minitech scalpel)
Small (e.g. 6 or 7mm) cuffed tracheal or tracheostomy tube

4-step technique:

1. Identify cricothyroid membrane
2. Stab incision through skin and membrane
   Enlarge incision with blunt dissection (e.g. scalpel handle, forceps or dilator)
3. Caudal traction on cricoid cartilage with tracheal hook
4. Insert tube and inflate cuff

Ventilate with low pressure source
Verify tube position and pulmonary ventilation

Notes:
1. These techniques can have serious complications - use only in life-threatening situations
2. Convert to definitive airway as soon as possible
3. Post-operative management - see other difficult airway guidelines and flow-charts
4. 4mm cannula with low-pressure ventilation may be successful in patient breathing spontaneously

5

Difficult Airway Society Guidelines Flowchart 2004 (use with DAS guidelines paper)

Figure 1. *Reproduced by kind permission of the Difficult Airway Society (UK) and available for download at*: www.das.uk.com/files/cvci-Jul04-A4.pdf

Update in
Anaesthesia

Management of the 'can't intubate, can't ventilate' situation

Leonard Pott
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Summary

Choose a plan in advance, and practice it thoroughly.

Ensure that everyone is familiar with the plan.

Do not repeatedly try to intubate - allow three good attempts and try an alternative method.

Start cricothyroidotomy BEFORE the patient has hypoxic damage.

If the facilities are not available for a cannula cricothyroidotomy (high-pressure oxygen, kink-resistant cannulas), use the surgical approach.

INTRODUCTION

Even when a patient proves to be unexpectedly difficult to intubate, it is usually not a problem to adequately oxygenate and ventilate the patient using bag-mask ventilation (BMV). Occasionally, and fortunately very rarely, we encounter a patient who is impossible to intubate AND who also cannot be adequately oxygenated. This is the feared 'can't intubate, can't ventilate' situation. The incidence of 'can't intubate, can't ventilate' in patients who are not expected to be difficult intubations is probably around 1 in 10 000 anesthetics. This condition obviously has life-threatening implications and must be resolved within minutes, if not seconds, to avoid hypoxic brain damage or death. The Difficult Airway Society (DAS) has produced a management algorithm, shown in Figure 1.[1]

COMMENTARY ON ALGORITHM

Although the algorithm is intended to be self-explanatory, there are some important points to bear in mind.

BOX 1 – *'failed intubation and difficult ventilation'*

1. Do not persist with intubation attempts. Repeated attempts at intubation will result in bleeding and swelling of the airway structures, which has been shown to increase the risk of complications, even if the patient is eventually intubated. In addition, time is passing which means that the patient will become progressively more hypoxic. Far better to limit intubation attempts to three good attempts, at least one of which should be by the most competent person in the room, and then go onto the next step. Remember, "If at first intubation attempt you do not succeed, repetitive attempts may make the airway bleed."[2]

2. Jaw thrust is very important because it helps lift the base of the tongue from the pharyngeal wall, which is a common site of upper airway obstruction. An oropharyngeal airway will help to achieve this.

3. Likewise, inserting a short nasal cannula (nasopharyngeal airway) bypasses obstruction at the level of the soft palate. This cannula should be long enough to pass beyond the uvula but not long enough to simulate the glottis or even worse, enter the esophagus. The cannula should be passed through the nose and parallel to the hard palate (not directed upwards). The cannula should have a diameter sufficient to provide unrestricted gas flow.

4. Using two hands to hold the mask and an assistant to squeeze the bag may make a significant difference. By using two hands the seal can be optimized and the mandible can be pulled in an anterior direction. This technique is particularly useful in patients with a beard.

5. Cricoid pressure, if not correctly applied, can distort the larynx and make intubation impossible. Release the cricoid pressure on the second intubation attempt. If the intubation proves impossible, reapply cricoid pressure and start bag-mask-ventilation, but if this is also difficult then release the cricoid pressure and check whether there is an improvement.

6. CALL FOR HELP. This is very important! In an emergency situation such as this, you will almost certainly need a second pair of hands to help fetch equipment, or to hold something while you are performing another task. Do not attempt to resolve every problem unaided. In addition, another person will review the situation and may easily spot something that you have overlooked.

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BOX 2 – *'failed oxygenation with face mask'*

1. Attempt the insertion of the laryngeal mask airway (LMA) early. Do not wait until the patient is severely hypoxic and has a significant amount of airway trauma.

2. Always make sure that you have an appropriately sized LMA available before starting any anesthetic induction. You cannot always predict when you will run into difficulties and the equipment for your alternative management strategy should be immediately available.

3. As far as local practice allows, you should ideally be completely comfortable using an LMA. Use an LMA on many of your routine cases who do not necessarily require an endotracheal tube in order to become completely familiar with the intubation technique. The 'can't intubate, can't ventilate' situation is not the time to learn how to insert an LMA!

BOX 3 – *'successful oxygenation'*

1. It is probably best to wake your patient and postpone surgery, because the patient has had a period of desaturation and there is likely to be some degree of airway trauma.

2. Occasionally it will be necessary to proceed with surgery. In this case you need to decide:

   a. Does this patient require general anesthesia or can we awaken the patient and use a regional technique?

   b. Does this patient really need an endotracheal tube for safe anaesthesia? You must consider the risk of aspiration, for example in a woman due to undergo caesarian section.

If you do decide that you must proceed, and that the patient needs an endotracheal tube, then you may consider swapping the LMA for an intubating LMA (Figure 2). Again, you should be familiar with the technique before you even consider it. In some circumstances, for example life-threatening peripartum haemorrhage, analysis of the risks and benefits may lead you to proceed with an LMA.

BOX 4 – *'cannula or surgical cricothyroidotomy'*

Cricothyroidotomy (access to the airway through the cricothyroid membrane) provides access to the trachea BELOW the vocal cords, and is indicated for a supra-glottic obstruction. It is no use for tracheal or bronchial obstruction by foreign bodies or malignant tumours below the upper trachea.

The choice between a cannula cricothyroidotomy and a surgical cricothyroidotomy is very significant for logistic planning and training, and is also rather controversial. This procedure is potentially life-saving and so a more detailed discussion of the alternative techniques is warranted. There are advantages and disadvantages associated with both alternatives, however equipment issues and the unavailability of high-flow or pressurized oxygen make surgical cricothyroidotomy the preferred option in poorly resourced settings.

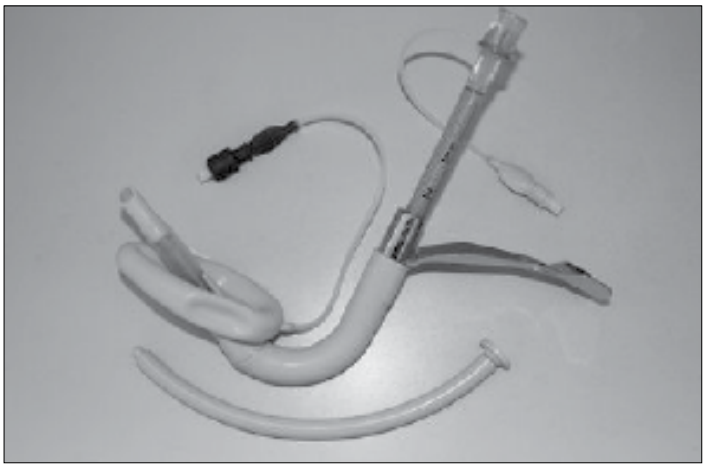

Figure 2. *The intubating laryngeal mask airway*

Cannula cricothyroidotomy

Advantages

1. This technique is considered less invasive and may be associated with less bleeding.

2. Some cannula techniques are performed with a Seldinger technique, with which anesthesia personnel are very comfortable.

Disadvantages

1. Cannula cricothyroidotomy is a temporary measure that allows oxygenation of the patient, but is inadequate for ventilation and carbon dioxide removal. A tube of 4mm internal diameter or greater is necessary to achieve ventilation. The cannula should be replaced by a definite airway, when the appropriate staff and equipment are avialble - ideally this should be within 10 to 15 minutes.

2. The use of a cannula for the cricothyroidotomy requires specialized equipment which may not be readily available at all locations. It is important that the cannula be kink-resistant, and various cannula are available. Examples are the Ravassin, Patil, Quicktrach and others.

3. Because of the cost of the equipment, the expense of providing stock for multiple locations and sufficient stock for training may prove to be inhibitory.

4. An ordinary intravenous catheter has been used successfully, but must be of a large size (14G) and can very easily kink. It is not recommended except in the most severe emergencies.

5. Because the time for inspiration is limited, adequate tidal volumes can only be achieved with oxygen flow rates of around 20 litres per minute. This may be impossible to achieve where in settings where oxygen is provided by oxygen concentrators. A cannula requires a high-pressure source of oxygen in order to provide adequate flow-rates.

6. High-pressure oxygen, typically 50psi (3 to 4 atmospheres), is potentially dangerous because it can result in barotrauma to the lung, pneumothorax, or subcutaneous emphysema. These conditions can rapidly cause death.

7. To minimize some of the risks of high-pressure oxygen, some form of injector which can limit the pressure, is used (Figure 3).

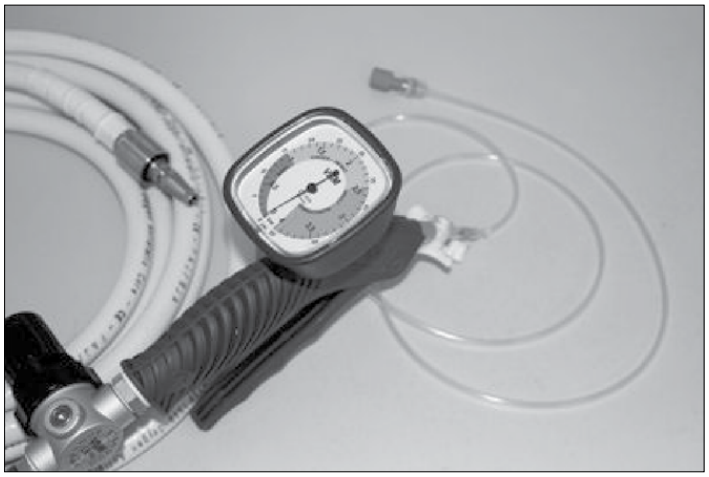

Figure 3. *A Manujet (VBM Medizintechnik GmbH, Sulz, Germany) high-pressure oxygen injector*

8. This specialized equipment must also be readily available at all locations where a 'can't intubate, can't ventilate' situation may arise. A high-pressure oxygen outlet must also be available, and the two systems must be able to be connected to each other. Staff must be able to prepare the equipment within a few minutes and many studies have shown that this cannot be easily achieved.

9. If a specialized injector is not available, wall oxygen can be used via a flowmeter, using a circuit made from readily available components (Figure 4). A three-way tap is essential to decompress the system and control inflation. By using your finger to occlude the tap opening, the oxygen is directed through the cannula. When the tap opening is not occluded, the oxygen takes the low resistance path and is vented to the environment. Use tubing which does not offer significant resistance, and ensure that all connections fit. This equipment should be pre-assembled and immediately available.

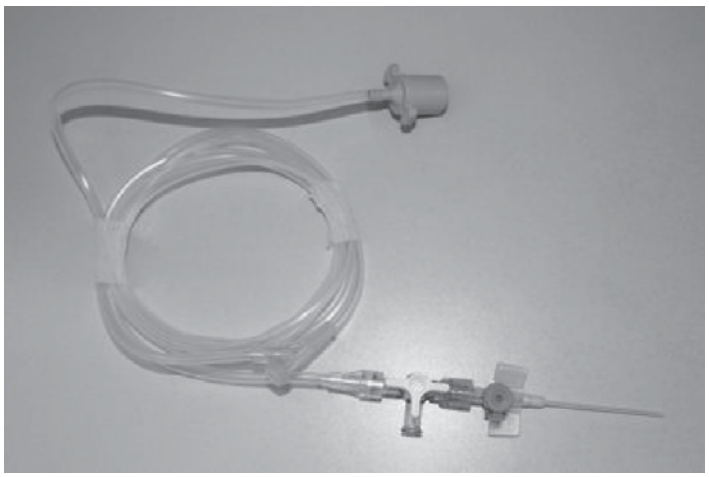

Figure 4. *A. example of an injector constructed using the 15mm connector from a size 5.0 endotracheal tube, an intravenous giving set with the drip chamber cut off, a three-way Luer tap and a large bore (14G) cannula. Note the position of the three-way tap*

10. Adequacy of ventilation is monitored by watching the chest rise and fall, and by the pulse oximetry response. Minute ventilation may be insufficient to avoid hypercapnoea.

11. The oxygen introduced into the patient's lungs must also be exhaled. This cannot be done through the cannula and the intra-thoracic pressure must therefore be released through the mouth. Jaw thrust, or insertion of an oropharyngeal or laryngeal mask airway may be necessary to facilitate expiration. In some cases of 'can't intubate, can't ventilate' the supra-glottic obstruction is complete and then the expired gas cannot be release creating a situation of high intrathoracic pressure and profound haemodynamic compromise.

12. No suction capacity is available.

13. No cuff is available to seal and protect the lower airway from contamination.

Surgical cricothyroidotomy

Advantages

1. Equipment is easily available; a scalpel and a small endotracheal tube are essential. Depending on the technique used, a tracheal dilator, a tracheal hook, and a gum elastic (Eschman) bougie are desirable.

2. Surgeons are usually comfortable with the procedure.

3. Both inspiration and expiration are possible through the endotracheal tube.

Disadvantages

1. Severe bleeding may result from cutting large veins or the thyroid gland.

BOX 5 – *'notes on cricothyroidotomy'*

These techniques are associated with significant complications, and are best avoided by adequate planning and preparation. Complications include severe bleeding, failure to oxygenate, tracheal stenosis, vocal cord damage. If however, the patient is becoming hypoxic and adequate ventilation is not possible, then the diagnosis of 'can't intubate, can't ventilate' should be made early and a cricothyroidotomy performed. Do not allow the patient to be hypoxic for too long.

Although the Difficult Airway Society algorithm only lists two alternative approaches for the percutaneous approach, there are many more available. Examples include the Melker, the Minitrach II, and the Quicktrach. Each has its own particular advantages and disadvantages. See Appendix A for a description of a common technique for surgical tracheostomy.

Table 1. *Factors to consider when choosing between needle and surgical cricothyroidotomy*

	Cannula cricothyroidotomy	**Surgical cricothyroidotomy**
Equipment	Specialized, relatively expensive. Constructed injectors must be pre-assembled	Commonly available
High-pressure oxygen	Required	Not required
Aspiration risk	High	Low
Suctioning	Not possible	Possible
Bleeding risk	Low	High
Barotrauma risk	High	Low

2. It may be difficult to identify the cricothyroid membrane. An incision through the upper tracheal rings, while not ideal, is better than permanent hypoxic brain damage or death.
3. Care must be taken not to lose the tracheal opening before the endotracheal tube is inserted. Use either a tracheal hook, or the handle of the scalpel.

PRE-EVENT PLANNING

1. Chose a particular approach for your institution and prepare adequately. The author's suggestion is the surgical cricothyroidotomy.
2. Make sure the necessary equipment is available at all locations where a 'can't intubate, can't ventilate' situation may arise.
3. Train your personnel. The anesthesia providers, the surgical staff, and all the nursing staff must know where the equipment is kept.

Practise the technique. The incidence of 'can't intubate, can't ventilate' is low, probably around 1 – 2 per 10 000 cases, so experience is difficult to attain, which makes simulation training indispensible. If possible, try to attend an airway management course which demonstrates the technique under the supervision of an expert. Alternatively, and in addition, read a very clear description of the technique in textbooks such as Hagberg,[3] Hung,[4] or Walls[5]. Various alternatives for practice include cadavers, plastic simulation models and animals.

FURTHER POINTS *(Adapted from Reference 2)*

- Repeated attempts at intubation will cause airway bleeding.
- Remember that an elective case can be scheduled for another day.
- Recognize you limitations when dealing with unfamiliar equipment.
- Remember and allow for the risks of aspiration.
- Record all events and timings in detail in the patient's notes.
- Provide a warning for your colleagues embarking on future anaesthetics - fix an ALERT sticker or label to the front of the patient's notes.
- If in doubt – carry the guideline about.

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Appendix A. *Techniques for surgical tracheostomy*

A brief outline of the surgical technique for cricothyroidotomy (based on Reference 6).

1. Identify the cricothyroid membrane. Find the thyroid cartilage, also known as the 'Adam's apple'. Ensure that you are in the midline by moving the thyroid cartilage from side to side, and by identifying the notch in the superior margin of the cartilage.
2. Run your finger down the thyroid cartilage and palpate the inferior margin. At that point you will feel a depression, which is the location of the cricothyroid membrane. This membrane attaches the thyroid cartilage to the cricoid ring below.
3. Depending on time pressure, clean the neck.
4. Make a vertical incision over the cricothyroid membrane, approximately 3 – 4 cm long. The incision is vertical to avoid damage to underlying vessels and other structures.
5. Cut down to the cartilage, using your finger intermittently to dissect the tissues. There will probably be significant bleeding so palpation of the cartilages is important. Stay in the midline!
6. When the cricothyroid membrane has been identified, make a horizontal 5 mm incision through the lower part of the membrane. Watch the depth of incision to avoid damage to the posterior tracheal wall and the underlying oesophagus.
7. Keep the incision open using your scalpel blade and place a gum elastic bougie into the opening, directing it down towards the chest. Advance the bougie until resistance is felt.
8. Pass the endotracheal tube over the bougie. A size 5.0 tube is usually appropriate. Ensure that the bevel of the endotracheal tube is lined up with the horizontal incision of the cricothyroid membrane before advancing further.
9. Apply gentle pressure and advance the endotracheal tube into the trachea. Gentle rotation of the tube may facilitate entry.
10. When the tube is in the trachea, remove the bougie and inflate the cuff.
11. Ventilate the patient in the normal fashion.

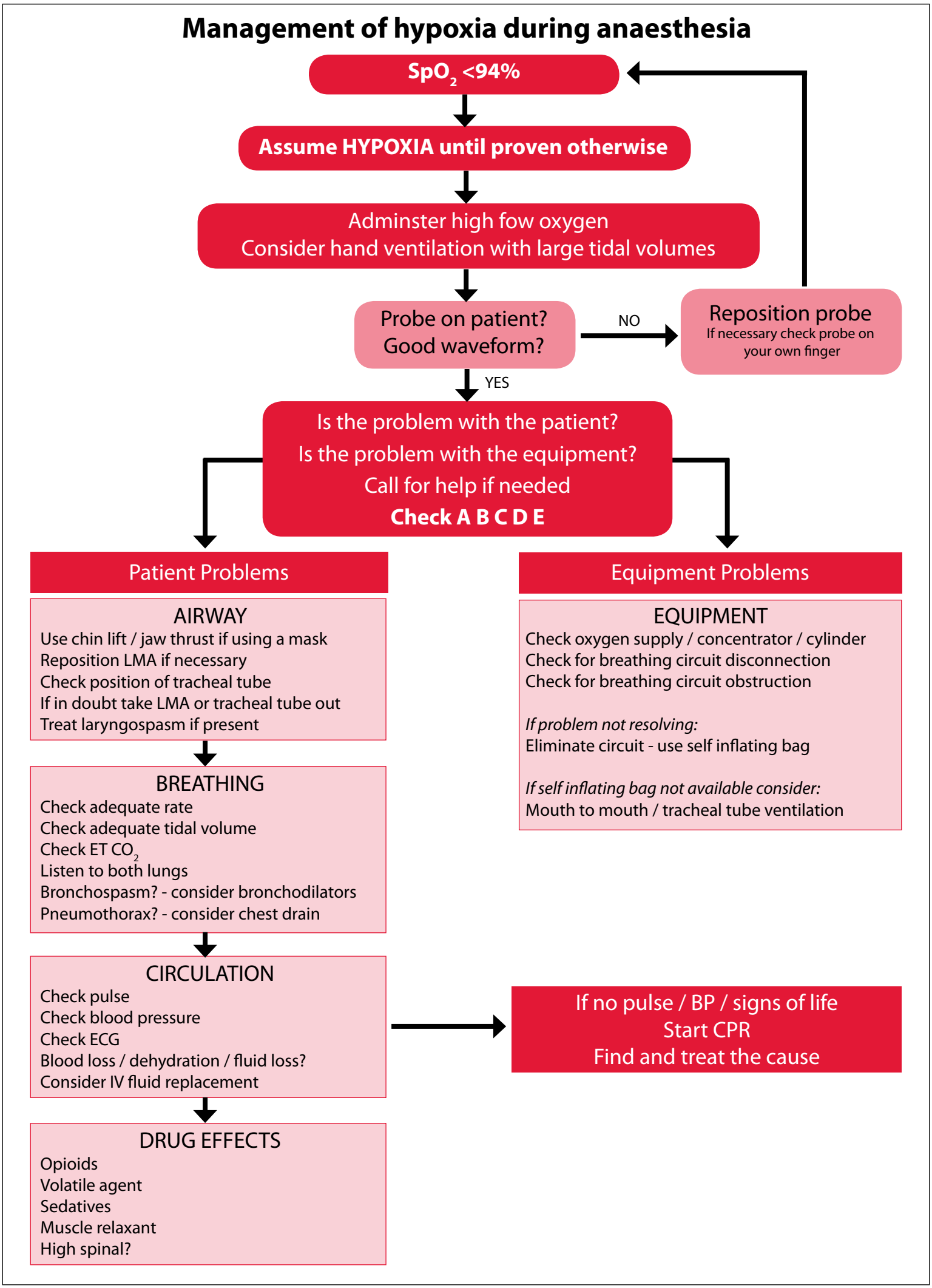

Figure 1. *Available for download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

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Hypoxia

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Summary

During anaesthesia low oxygen saturations must be treated immediately.

Administer 100% oxygen.

Ventilate by hand, call for help and consider 'ABCDE'.

Check for likely causes in a logical sequence.

Treat causes as you identify them.

INTRODUCTION

Hypoxia during anaesthesia is common and is easily detected by a pulse oximeter. This article will describe how to respond to falling SpO_2.

COMMENTARY ON ALGORITHM

Causes of hypoxia during anaesthesia

The causes of hypoxia during anaesthesia are summarised in Table 1. Airway obstruction is the most common cause of hypoxia.

What should be done when the saturation falls?

During anaesthesia, low oxygen saturations must be treated immediately and appropriately. The patient may become hypoxic at any time during induction, maintenance or emergence from anaesthesia. The appropriate response is to administer 100% oxygen, make sure that ventilation is adequate by using hand ventilation and then correct the factor that is causing the patient to become hypoxic. For example, if the patient has an obstructed airway and is unable to breathe oxygen into the lungs, the problem will only be cured when the airway is cleared.

Learning point:

When hypoxia occurs, it is essential to decide whether the problem is with the patient or the equipment. After a quick check of the common patient problems, make sure the equipment is working.

Table 1. *Causes of hypoxia in theatre – consider 'ABCDE'*

Source of problem	Common problem
A. AIRWAY	• An obstructed airway prevents oxygen reaching the lungs • The tracheal tube can be misplaced e.g. in the oesophagus • Aspirated vomit can block the airway
B. BREATHING	• Inadequate breathing prevents enough oxygen reaching the alveoli • Severe bronchospasm may not allow enough oxygen to reach the lungs nor carbon dioxide to be removed from the lungs • A pneumothorax may cause the affected lung to collapse • High spinal anaesthesia may cause inadequate breathing
C. CIRCULATION	• Circulatory failure prevents oxygen from being transported to the tissues • Common causes include hypovolemia, abnormal heart rhythm or cardiac failure
D. DRUGS	• Deep anaesthesia may depress breathing and circulation • Many anaesthetic drugs cause a drop in blood pressure • Muscle relaxants paralyse the muscles of respiration • Anaphylaxis can cause bronchospasm and low cardiac output
E. EQUIPMENT	• Problems with the anaesthetic equipment include disconnection or obstruction of the breathing circuit • Problems with oxygen supply include an empty cylinder or oxygen concentrator not working • Problems with the monitoring equipment include battery failure in the oximeter or a faulty probe

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Whenever the patient has low saturations, administer high flow oxygen and consider 'ABCDE':

A - airway clear?

B - breathing adequately?

C - circulation working normally?

D - drugs causing a problem?

E - equipment working properly?

You must respond to hypoxia immediately by giving more oxygen, ensuring adequate ventilation by hand, calling for help, and running through the 'ABCDE' sequence. Treat problems detected in each element of the sequence as you check it. After you have been through all the checks for the first time, go back and recheck them until you are satisfied that the patient's condition has improved. The aim of the WHO's algorithm or chart (Figure 1) is to help you remember what to look for in a logical sequence. In an emergency, there may not be time to read what to do. You should ask a colleague to read through the chart for you to make sure that you have not forgotten anything.

Learning point:
If SpO_2 is 94% or below, give 100% oxygen, hand ventilate, consider ABCDE

ACTIONS TO TAKE IF THE OXYGEN SATURATION IS 94% OR BELOW

If the oxygen saturation is 94% or below, you should administer 100% oxygen, ventilate by hand, consider whether the problem is with the patient or the equipment, then move through the algorithm 'ABCDE', assessing each factor and correcting it immediately as you go.

Oxygen

Administer high flow oxygen if SpO_2 is ≤94%

A – Is the airway clear?

- Is the patient breathing quietly without signs of obstruction?
- Are there signs of laryngospasm? (mild laryngospasm – high pitched inspiratory noise; severe laryngospasm – silent, no gas passes between the vocal cords)
- Is there any vomit or blood in the airway?
- Is the tracheal tube in the right place?

Action

- Ensure that there is no obstruction. If breathing via a facemask - chin lift, jaw thrust,
- Consider an oropharyngeal or nasopharyngeal airway,
- Check for laryngospasm and treat if necessary.
- Check the tracheal tube/LMA - if any doubt about the position, remove and use a facemask.
- Suction the airway to clear secretions.
- Consider waking the patient up if you have difficulty maintaining the airway immediately after induction of anaesthesia.
- Consider intubation.
- If you 'can't intubate, can't ventilate' an emergency surgical airway may be required (see page 15).

Airway obstruction is the most common cause of hypoxia in theatre. Airway obstruction is a clinical diagnosis and must be acted upon swiftly. Unrecognised inadvertent oesophageal intubation is a major cause of anaesthesia morbidity and mortality. An intubated patient who has been previously well saturated may become hypoxic if the tracheal tube becomes displaced, kinked or obstructed by secretions.

Check the endotracheal tube and - 'If in doubt, take it out'.

B - Is the patient breathing adequately?

Look, listen and feel:

- Are the chest movements and tidal volume adequate?
- Listen to both lungs – is there normal bilateral air entry? Are the breath sounds normal? Any wheeze or added sounds?
- Is the chest movement symmetrical?
- Is anaesthesia causing respiratory depression?
- Is there a high spinal causing respiratory distress?

Bronchospasm, lung consolidation/collapse, lung trauma, pulmonary oedema or pneumothorax may prevent oxygen getting into the alveoli to combine with haemoglobin. Drugs such as opioids, poorly reversed neuromuscular blocking agents or deep volatile anaesthesia may depress breathing. A high spinal anaesthetic may paralyse the muscles of respiration. In an infant, stomach distension from facemask ventilation may splint the diaphragm and interfere with breathing. The treatment should deal with the specific problem.

Action

- Assist ventilation with good tidal volumes to expand both lungs until the problem is diagnosed and treated appropriately.
- If there is sufficient time, consider a chest X-ray to aid diagnosis.

The patient should be ventilated via a facemask, LMA or tracheal tube if the respiration is inadequate. This will rapidly reverse hypoventilation due to drugs or a high spinal and a collapsed lung will re-expand. The lower airway should be suctioned with suction catheters to remove any secretions. A nasogastric tube should be passed to relieve stomach distension.

A pneumothorax may occur following trauma, central line insertion or a supraclavicular brachial plexus block. It may be suspected if there is reduced air entry on the affected side. In thin patients a hollow note on percussion may also be detected. A chest X-ray is diagnostic but you should not delay treatment to wait for this. A chest drain should be inserted as the pneumothorax may worsen. When there is associated hypotension (tension pneumothorax), the pneumothorax should be treated by emergency needle decompression through the

2nd intercostal space in the mid-clavicular line without waiting for an X-ray. A definitive chest drain should be inserted later. Always maintain a high index of suspicion in trauma cases.

C - Is the circulation normal?

- Feel for a pulse and look for signs of life, including active bleeding from the surgical wound.
- Check the blood pressure.
- Check the peripheral perfusion and capillary refill time.
- Observe for signs of excessive blood loss in the suction bottles or wound swabs.
- Is anaesthesia too deep? Is there a high spinal block?
- Is venous return impaired by compression of the vena cava (gravid uterus, surgical compression).
- Is the patient in septic or cardiac shock?

Normally an inadequate circulation is revealed by the pulse oximeter as a loss or reduction of pulsatile waveform or difficulty getting a pulse signal.

Action

- If the blood pressure is low, correct it.
- Check for hypovolaemia.
- Give IV fluids as appropriate (normal saline or blood as indicated).
- Consider head down or leg up position, or in the pregnant mother, left lateral displacement.
- Consider a vasoconstrictor such as ephedrine or phenylephrine.
- If the patient has suffered a cardiac arrest, commence cardiopulmonary resuscitation (CPR) and consider reversible causes (4 H's, 4T's: **H**ypotension, **H**ypovolaemia, **H**ypoxia, **H**ypothermia; **T**ension pneumothorax, **T**amponade (cardiac), **T**oxic effects (deep anaesthesia, sepsis, drugs), **T**hromboemboli (pulmonary embolism).

D – Drug effects

Check that all anaesthesia drugs are being given correctly.

- Excessive halothane (or other volatile agent) causes cardiac depression.
- Muscle relaxants will depress the ability to breathe if not reversed adequately at the end of surgery.
- Opioids and other sedatives may depress breathing.
- Anaphylaxis causes cardiovascular collapse, often with bronchospasm and skin flushing (rash). This may occur if the patient is given a drug, blood or artificial colloid solution that they are allergic to. Some patients are allergic to latex rubber.

Action

- Look for an adverse drug effect.
- In anaphylaxis, stop administering the causative agent, administer 100% oxygen, give intravenous saline starting with a bolus of 10ml/kg, administer adrenaline and consider giving steroids, bronchodilators and an antihistamine.

E - Is the equipment working properly?

- Is there a problem with the oxygen delivery system to the patient?
- Does the oximeter show an adequate pulse signal?

Action

- Check for obstruction or disconnection of the breathing circuit or tracheal tube.
- Check that the oxygen cylinder is not empty.
- Check that the oxygen concentrator is working properly.
- Check that the central hospital oxygen supply is working properly.
- Change the probe to another site; check that it is working properly by trying it on your own finger.

If it is felt that the anaesthesia equipment is faulty, ***use a self-inflating bag to ventilate the patient with air*** while new equipment or oxygen supplies are obtained. If equipment is missing, mouth to tracheal tube, or mouth-to-mouth ventilation, may be lifesaving.

CLINICAL SCENARIOS

Work through problems in each case, deciding why the SpO_2 is low (ABCDE) and what the most appropriate action should be. The first three scenarios are explained in detail. The others should be discussed with your colleagues.

Example 1

A 12-year-old child is scheduled for elective anaesthesia for foot surgery. The patient is ASA 1 and is induced with thiopentone then given halothane in air and oxygen via a face mask. During the induction the patient starts to cough and gets laryngospasm. The SpO_2, which started at 98%, falls to 88% during coughing and then to 74% when laryngospasm occurs. Why has the saturation fallen and what would be the most appropriate actions?

- Give 100% oxygen, assess ABCDE
- A – airway obstruction due to laryngospasm; apply positive pressure to the reservoir bag, deepen anaesthesia. If the situation does not resolve, a small dose of suxamethonium (0.5mg/kg) should be given.
- B – breathing improves after resolution of laryngospasm.
- C – assess pulse rate - bradycardia may occur due to hypoxia or secondary to suxamethonium. Consider atropine after treating hypoxia.
- D – check the halothane has not run out.
- E – check that the anaesthesia equipment is functioning and connected appropriately.

After treating the laryngospasm, the patient improved and the SpO_2 returned to normal.

Example 2

A 56-year-old obese patient is undergoing a laparotomy for bowel obstruction. Preoperatively he is reasonably fit and his SpO_2 is 95%. After rapid sequence induction and intubation, the patient is ventilated and anaesthesia maintained using halothane in air with 30% oxygen. Over the next 10 minutes the patient's SpO_2 falls to 85%. What are the most likely causes and what action would you take?

- Give 100% oxygen, check ABCDE.
- A – check the airway and position of the tracheal tube. Check there is equal air entry to both sides of the chest and that the tube is not kinked. Check that there is no vomit in the mouth to suggest that the patient may have aspirated.
- B – check that there are no added breath sounds to suggest aspiration, lung collapse or bronchospasm. Give large tidal volumes by hand and listed to the chest. Is ventilation easy?
- C – assess whether the circulation is normal.
- D – assess whether the patient is fully relaxed. Check that there are no signs to suggest drug reaction (particularly wheeze + hypotension + rash).
- E – check that the anaesthesia equipment is functioning and connected appropriately.

After ventilating the patient with some large tidal volumes and increasing the inspired oxygen the patient improved. The problem was lung collapse.

Example 3

During a caesarean section under spinal anaesthesia, a fit 23-year-old primigravida complains of tingling in the fingers and difficulty breathing. The SpO_2 falls from 97% to 88%. What are the most likely causes and what action would you take?

- Give 100% oxygen. Check ABCDE
- A – check that the airway is clear
- B - assess breathing. A high spinal may paralyse the muscles of respiration. If breathing is inadequate, ventilate the patient and induce anaesthesia and intubate. Ventilate until the block wears off.
- C – check the blood pressure – hypotension is likely. Treat with left lateral tilt, IV fluids and vasopressors.
- D – check the height of the block. Look for signs of a very high block - difficulty breathing, whispering rather than talking, weak arms and numbness on the shoulders. All indicate the nerves to the diaphragm are becoming blocked. This will make it impossible for the patient to breathe. If the block is not this high, the patient can talk in a normal voice and move their arms normally, but breathing still feels difficult due to the intercostal paralysis, normally the patient can breathe safely using their diaphragm.
- E – always ensure that equipment is ready in case this complication occurs.

After giving oxygen, the anaesthetist determined the block was not too high and the patient settled with reassurance. The SpO_2 improved with oxygen. Any hypoxia in a pregnant patient is dangerous.

THE ASSOCIATION OF ANAESTHETISTS
of Great Britain & Ireland

Management of a Patient with Suspected Anaphylaxis During Anaesthesia

SAFETY DRILL

(Revised 2009)

Immediate management (A)

- Use the ABC approach (Airway, Breathing, and Circulation). Teamworking enables several tasks to be accomplished simultaneously.
- Remove all potential causative agents and maintain anaesthesia, if necessary, with an inhalational agent.
- CALL FOR HELP and note the time.
- Maintain the airway and administer oxygen 100%. Intubate the trachea if necessary and ventilate the lungs with oxygen.
- Elevate the patient's legs if there is hypotension.
- If appropriate, start cardiopulmonary resuscitation immediately according to Advanced Life Support Guidelines.
- Give adrenaline (epinephrine) i.v.
  - Adult dose: 50 mcg (0.5ml of 1:10 000 solution)
  - Child dose: 1.0 $mcg.kg^{-1}$ (0.1$ml.kg^{-1}$ 1:100 000 solution)
- Several doses may be required if there is severe hypotension or bronchospasm. If several doses of adrenaline are required, consider starting an intravenous infusion of adrenaline.
- Give saline 0.9% or lactated Ringer's solution at a high rate via an intravenous cannula of an appropriate gauge (large volumes may be required).
  - Adult: 500 - 1 000 ml
  - Child: 20 $ml.kg^{-1}$
- Plan transfer of the patient to an appropriate Critical Care area.

CONTINUED OVERLEAF

Figure 1A. *Reproduced by kind permission of the Association of Anaesthetists of Great Britain and Ireland and available for download at:* www.aagbi.org/publications/guidelines/docs/ana_laminate_2009.pdf

Secondary management B

• Give chlorphenamine i.v. (chlorpheniramine)	Adult:	10 mg
	Child 6 - 12 years:	5 mg
	Child 6 months - 6 years:	2.5 mg
	Child <6 months:	250 mc.kg^{-1}
• Give hydrocortisone i.v.	Adult:	200 mg
	Child 6 - 12 years:	100 mg
	Child 6 months - 6 years:	50 mg
	Child <6 months:	25 mg

- If the blood pressure does not recover despite an adrenaline infusion, consider the administration of an alternative i.v. vasopressor according to the training and experience of the anaesthetist, e.g. metaraminol
- Treat persistent bronchospasm with an i.v. infusion of salbutamol. If a suitable breathing system connector is available, a metered-dose inhaler may be appropriate. Consider giving i.v. aminophylline or magnesium sulphate.

Investigation C

- Take blood samples (5 - 10 ml blood) for **mast cell tryptase**:
  - Initial sample as soon as feasible after resuscitation has started - do not delay resuscitation to take the sample.
  - Second sample 1 - 2 h after the start of symptoms.
  - Third sample either at 24 h or in convalescence (for example in a follow-up allergy clinic). This is a measure of baseline tryptase levels as some individuals have a higher baseline level.
- Ensure that the samples are labelled with the time and date.
- Liaise with the hospital laboratory about analysis of samples.

Later investigations to identify the causative agent D

The anaesthetist who gave the anaesthetic or the supervising consultant anaesthetist is responsible for ensuring that the reaction is investigated. The patient should be referred to a specialist Allergy or Immunology Centre (see www.aagbi.org for details). The patient, surgeon and general practitioner should be informed. Reactions should be notified to the AAGBI National Anaphylaxis Database (see www.aagbi.org).

Figure 1B. *Reproduced by kind permission of the Association of Anaesthetists of Great Britain and Ireland and available for download at:* www.aagbi.org/publications/guidelines/docs/ana_laminate_2009.pdf

Management of a patient with suspected anaphylaxis during anaesthesia

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Summary

If anaphylaxis during anaesthesia is recognized promptly and managed optimally, severe adverse reactions are avoidable.

Follow basic life support with the ABC approach (Airway, Breathing, and Circulation), epinephrine (adrenaline) is the most effective drug in anaphylaxis during anaesthesia and should be given as early as possible.

Serum mast cell tryptase levels may help the retrospective diagnosis of anaphylaxis, though it does not differentiate IgE-mediated reactions from non-IgE-mediated reactions.

Follow-up investigation is essential to avoid life-threatening re-exposure of the patient to the offending drug or substance and to tailor a safe alternative.

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INTRODUCTION

The exact prevalence of anaphylaxis during anaesthesia is very difficult to estimate and has been calculated to range from 1 in 3,500 to 1 in 13,000 cases according to research in France.[1,2] A report from Australia estimated the incidence to be between 1 in 10,000 and 1 in 20,000,[3] whereas another report from Norway estimated the incidence to be 1 in 6,000.[4] If anaphylaxis during anaesthesia is recognized promptly and managed optimally, severe adverse reactions may be avoidable. However, in most cases, multiple drugs were administered and correct identification of the causative factor is not always straightforward. The Association of Anaesthetists of Great Britain & Ireland (AAGBI) has produced a management guideline (shown in Figure 1).

COMMENTARY ON ALGORITHM

Profound understanding of the consensus and guidelines is crucial for timely and effect diagnosis and management of suspected anaphylaxis during anesthesia. For further detail the guideline is broken into four sections, A to D.

Section A - Immediate management

1. Anaphylaxis during anaesthesia may present in a variety of symptoms. In most cases, the clinical features, associated or not, include severe respiratory manifestations, cardiovascular symptoms, and cutaneous signs.

Cardiorespiratory	Cutaneous
hypotension	cutaneous flushing
tachycardia or bradycardia	rash
cardiovascular collapse	urticaria
bronchospasm	angioedema
hypoxia	

Multisystem involvement is most common, but not always the case. The absence of cutaneous signs does not exclude the diagnosis of anaphylaxis.[5]

2. The diagnosis of anaphylaxis during anaesthesia is usually problematic because clinical features such as hypotension and bronchospasm more commonly have a different cause. Most common anesthetics may cause vasodilation, hypotension, and cardiopulmonary dysfunction as a result of direct and indirect effects on sympathoadrenergic responses. Bronchospasm and wheeze may be provoked by histamine releaseing drugs (such as suxamethonium) and may also develop after endotracheal intubation in smokers or asthmatics. In addition, cutaneous symptoms may be missed as patients are usually hidden by surgical drapes.

3. Up to 90% cases of anaphylaxis during anaesthesia occur within minutes of induction,[6] and are linked mainly to agents administered intravenously.[7] If an adverse event such as hypotension or bronchospasm occurs after drug administration or blood transfusion during anaesthesia, it is appropriate to suspect anaphylaxis unless there exists a significantly more likely cause, such as hypovolaemia, light/deep anaesthesia or extensive regional blockade. Rare but potentially disastrous events should be excluded, for example a misplaced tracheal tube or equipment failure.

4. Recommendations for the treatment of anaphylaxis during anaesthesia, which has a wide variety of presentations, must not be established on a rigid scheme. Treatment should depend on the clinical picture, however, there are general measures used in all cases:

   a Immediately *stop administration of the agent* that you suspect to be the causative factor, interrupting the effects of the preformed mediators that were released in response to the antigen, and preventing more mediator release.

   b *Rapidly control the airway and administer 100% oxygen*. Airway support with 100% oxygen will increase oxygen delivery and compensate for the increased oxygen consumption.

c *Call for help*. In an emergency situation such as this, team-working enables several tasks to be accomplished simultaneously. Do not attempt to resolve every problem unaided. In addition, another person will review the situation and may easily spot something that you have overlooked. Get information from the surgical team as soon as possible which may be helpful to make a decision to cancel, accelerate or stop surgery.

d *Make detailed notes*. For diagnostic purposes and further investigation after a severe adverse reaction during anaesthesia, it is important to document a detailed description of the reaction (e.g. symptoms, severity, time course) and its treatment. All drugs and/or other substances to which the patient was exposed during anaesthesia, as well as their time onset in relation to the reaction, must be recorded.

5. Epinephrine (adrenaline) is the most effective drug in most cases of anaphylaxis and should be given as early as possible. Failure to treat anaphylaxis promptly with epinephrine may result in biphasic or protracted anaphylaxis or in a fatal outcome.[8,9] The α-agonist activity of epinephrine is able to reverse vasodilatation and oedema. In addition, epinephrine is a valuable α-agonist which has a positive inotropic action, dilates bronchial smooth muscle, and reduces the release of inflammatory mediators, such as leukotrienes and histamine.[10] In patients taking β-adrenergic blocking agents, it may be necessary to increase the dose of epinephrine rapidly: for example a first bolus of 100mcg, followed when necessary by 1mg or even 5mg, at 1 to 2 minute intervals.[11] Continuous infusion of epinephrine is advantageous in patients who need repeated doses of epinephrine.[12] It is important to note, however, that epinephrine should be titrated carefully to response, especially when administered intravenously, which have been implicated in a few cases of excessive doses of epinephrine.[13]

6. Fluid therapy is important to counteract the large fluid shifts associated with vasodilatation and capillary leakage. Rapidly restore the vascular volume with isotonic crystalloid and consider use of a colloid when the volume of crystalloid exceeds 30ml.kg^{-1}.[11] Avoid administering the substances that are suspected to be the cause of the reaction.

7. If appropriate, start cardiopulmonary resuscitation immediately, following the usual resuscitation measures for cardiocirculatory insufficiency.

SECTION B - Secondary management

1. Antihistamines and corticosteroids have a place as secondary treatment for anaphylaxis and help to prevent oedema, cutaneous symptoms and relapse of the anaphylactic reaction as seen in biphasic or protracted anaphylaxis.[14] Hydrocortisone is the preferred steroid because it has a fast onset. If intubated, the endotracheal tube should be left in place after successful treatment of a severe anaphylactic reaction, because airway swelling and inflammation may continue for up to 24h.[15]

2. For patients who are refractory to epinephrine, consider other vasoconstrictor drugs such as norepinephrine (starting with 0.1mcg.kg^{-1}.min^{-1})[11] or vasopressin – see below.

3. Vasopressin is an important alternative for vasodilatory shock associated with anaphylaxis for anaesthetists who may not have access to epinephrine to treat anaphylaxis. The pressor response to vasopressin may be mainly due to the ability of vasopressin to block the potassium channels in vascular smooth muscle and interfere with multiple signaling pathways.[17,18] In patients with a reasonable arterial blood pressure consider starting at doses of 1–2 units; in patients with cardiac arrest, 40 units are part of Advanced Cardiac Life Support guidelines.[16] It is reported that the effects of vasopressin are similar to those of epinephrine in the management of ventricular fibrillation and pulseless electrical activity, but vasopressin is superior to epinephrine in patients with asystole.[19] Several case reports and experimental models of severe anaphylactic reactions have also suggested vasopressin's efficacy.[20,22]

4. For patients on β-blocker treatment, if epinephrine is not efficient, glucagons could also be effective (1 to 2mg intravenously, repeated every 5 minutes).[10,11]

5. In cases of bronchospasm without arterial hypotension, a β_2-adrenergic agonist (such as salbutamol, 2.5-5mg) can be administered through an inhalation chamber adapted to the ventilatory circuit. If this treatment is resistant, consider the intravenous route: administer a 100 to 200mcg bolus of salbutamol, followed by continuous perfusion of this drug (5 to 25mcg.min^{-1}).[11]

SECTION C - Investigation

1. Serum tryptase is a mast cell protease that is increased in cases of anaphylaxis, signaling an immune-mediated mechanism. Tryptase concentrations can be measured in serum (or plasma) 30 minutes after onset of symptoms and reach a peak between 15 minutes and one hour.[23] The half-life of tryptase is about 120 minutes and the levels gradually decrease over time.

2. Mast cell tryptase can also be released by pharmacologic drugs that cause direct non-immunologic mast cell activation.[24] Therefore, an increase of serum tryptase does not differentiate IgE-mediated reactions from non-IgE-mediated reactions. There have been a few reports of anaphylaxis with positive tests for IgE antibodies and an absence of serum tryptase.[25] In other words, a negative test for serum tryptase does not exclude an anaphylactic reaction.

3. To enable comparison with baseline levels, a control sample value should be measured either on a pre-operative sample or a minimum of 24 h after the reaction.

4. If the sample tubes cannot be transported to the local laboratory within 2 hours, they must be stored in a refrigerator at +4°C (for not longer than 12 hours). After centrifugation, the serum should be stored at -20°C in several aliquots.

5. In order to make a valid interpretation of serum tryptase values, the timing of blood sampling in relation to the reaction should be recorded.

SECTION D - Later investigations to identify the causative agent

1. Follow-up investigation is necessary in order to identify the drug or substance responsible and the mechanism behind the reaction. This is essential to help a patient avoid potentially life-threatening re-exposure to the offending substance and tailor a safe alternative. If necessary, the anaesthetist should consider referral to an immunologist or allergy specialist for further investigation.
2. The anesthetist is responsible for:
   a Initiating the investigation, in collaboration with a consulting allergy specialist.
   b Informing the patient about the the reaction and giving written and verbal recommendations for subsequent anaesthesia.
   c Reporting the event to the pharmaco-vigilance centre if a drug is suspected to be the cause.
3. Given the present state of knowledge, skin tests (prick and intradermal reaction) remain the gold standard for the detection of IgE-dependent allergies.[11]
4. Currently, radioallergosorbent test (RAST) and fluoroimmunoassay (Pharmacia CAP System) are available in some centres to measure specific IgE antibodies in the blood. However, IgE measurement is only commercially available for a few drugs used during anaesthesia.
5. Other cellular assays based either on the release of sulphidoleukotrienes or on flow cytometry are not sufficiently validated to enter daily clinical practice.

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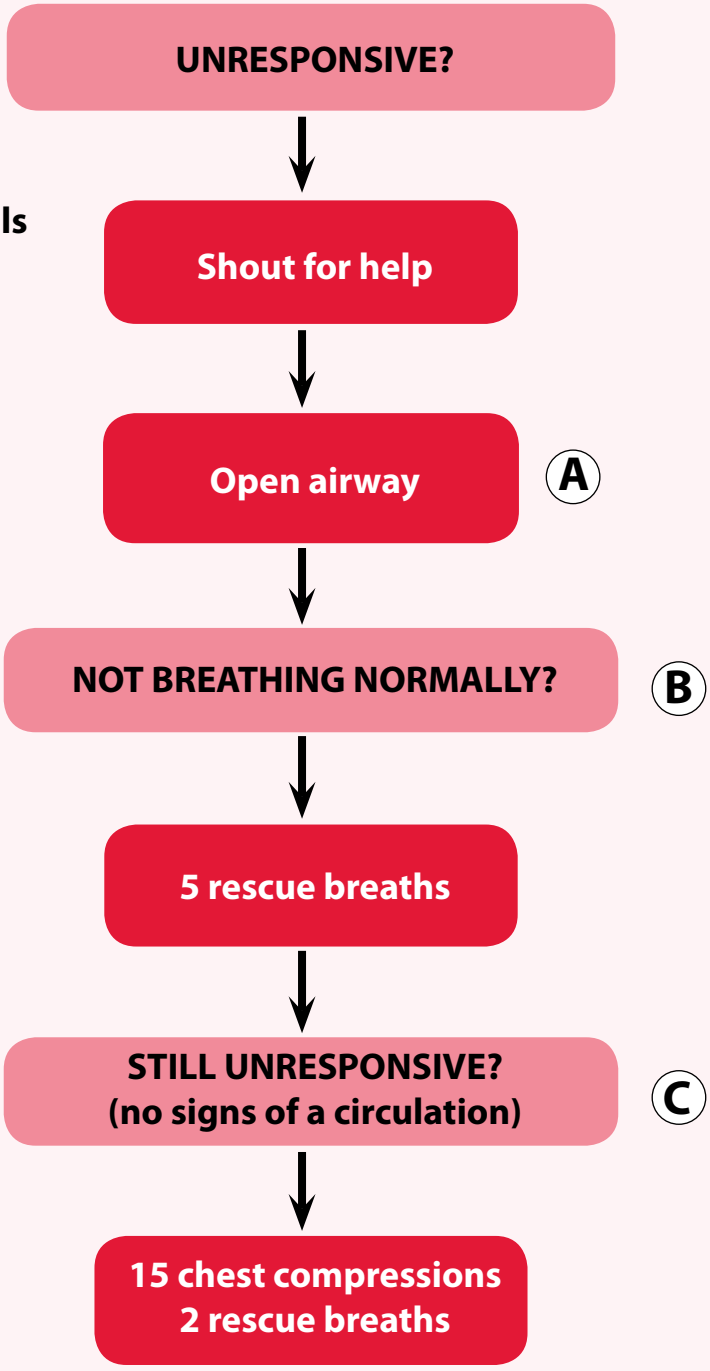
Resuscitation Council (UK)

Paediatric Basic Life Support

(Healthcare professionals with a duty to respond)



After 1 minute call resuscitation team then continue CPR

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Paediatric life support

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Summary

Children usually suffer cardiac arrest secondary to hypoxia or ischaemia due to respiratory or circulatory failure.

Cardiac arrest is commonly reversed by simple interventions.

Early recognition of a child at risk of deterioration is essential.

Avoid interruptions in chest compressions.

There are some differences between resuscitation techniques for children and adults but there are also many similarities. There is no doubt that a child in cardiorespiratory arrest will be harmed more by doing nothing than by using adult resuscitation guidelines.

Children usually suffer from secondary cardiac arrest – the heart stops secondary to hypoxia or ischaemia caused by respiratory or circulatory failure. The main implication of this is that there is potential to recognise the primary cause early on and prevent its progression to full blown arrest. Respiratory or circulatory failure is initially compensated by the body's physiological mechanisms and the signs are fairly subtle.

Signs of compensated respiratory failure

- Tachypnoea or bradypnoea (e.g. in narcotic overdose)
- Increased work of breathing:
  - Anxious appearance
  - Use of accessory muscles of respiration
  - Noises – stridor, grunting or wheeze
  - Nasal flaring.

Signs of compensated circulatory failure

- Tachycardia
- Slow capillary refill
- Cool peripheries
- Thirst
- Lethargy.

In the compensated phase there are good opportunities to prevent deterioration by the administration of general treatment such as oxygen and fluid (in circulatory failure) and specific treatments such as salbutamol in asthma and antibiotics in pneumonia or sepsis. This compensated phase may progress to decompensation, however and, if immediate action is not then taken, to cardiorespiratory arrest.

Signs of decompensation

Diminishing level of consciousness is an important sign of decompensation and imminent arrest

In addition, for decompensating respiratory failure

- Sudden fall in respiratory rate
- Exhaustion
- Very quiet or silent chest.

Decompensating circulatory failure

- Hypotension
- Sudden fall in heart rate.

Fortunately, the actions required to reverse this process are usually simple and follow the familiar ABC format.

COMMENTARY - BASIC LIFE SUPPORT (Figure 1)

A – Airway

Opening a child's airway is similar to opening that of an adult – a head tilt and chin lift. The most important difference is to avoid pressing on the soft tissues underneath the jaw as this pushes the tongue backwards into the oropharynx and can worsen airway obstruction. Infants have a prominent occiput and simply require the head to be placed in a neutral position – overextension is not helpful. If this simple manoeuvre is ineffective a jaw thrust (performed in the same way to that in adults) usually works.

Sometimes an airway adjunct is required and the most useful is an oropharyngeal airway. The size is selected so that tip of the airway is level with the angle of the jaw when the flange is lined up with the lips. It can be inserted in the same way as for an adult airway (i.e. introduced upside down and then rotated 180 degrees into its final position) but care should be taken not to damage the hard palate.

Successful opening of the airway should be assessed by ***looking*** (for chest movement), ***listening*** (for air flow at the nose and mouth) and ***feeling*** (for expired air on your cheek held close to the child's nose and mouth).

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- If there is chest movement and you can hear and feel expired air then the airway is clear and oxygen (if available) should be given.
- If there is chest movement but no expired air then the airway is still obstructed and it should be repositioned.
- If there is no chest movement, positive pressure ventilation is required.

B – Breathing

Positive pressure ventilation (IPPV) may be given with expired air (mouth to mouth) or bag mask ventilation (BMV) with a self-inflating bag/mask system.

Mouth to mouth ventilation requires no equipment but is inefficient as it only delivers expired oxygen concentrations (about 17%). Nevertheless it can be lifesaving.

The most important points are to open the airway effectively (as above) and to achieve a good seal with your lips over the child's mouth (or nose and mouth in a small infant). You should deliver enough breath to make the child's chest rise as if they had taken a normal breath.

The same principles apply to BMV – the airway should be open and there should be a good seal, this time between the mask rim and the child's face. If this is difficult it may help to have 2 people – one to do a jaw thrust and to achieve a seal with the mask using both hands and the other to squeeze the reservoir bag. Again, the aim is to make the chest rise as if the child has taken a normal breath. Five rescue breaths should be delivered in this fashion and then an assessment of the circulation should be made.

C – Circulation

In diminished level of consciousness due to decompensated respiratory or circulatory failure, failure to respond to positive pressure ventilation by moving, coughing or resuming breathing is a sure sign of absence of an effective circulation and external chest compression (ECC) should be immediately commenced. Prolonged searching for a pulse (>10 seconds) is unnecessary may result in error or delay.

ECC is performed by compression of the chest to a depth of 1/3 to 1/2 of the A-P diameter, at a point just (1 finger's breadth) above the xiphisternum. Don't be afraid of pushing too hard. Compressions should be at a rate of 100 per minute and 2 breaths should be given after every 15 compressions. Compressions should be interrupted as little as possible so, if the trachea is intubated, they should be continuous with about 10 breaths delivered every minute. Generally, people ventilate too vigorously during resuscitation and this has been shown to impede venous return and reduce blood flow.

If a monitor or defibrillator is available it should be applied to check whether there is a shockable cardiac rhythm (ventricular fibrillation or ventricular tachycardia) or not. Adrenaline (10mcg.kg^{-1}) should be given every 3-5 minutes during ECC as it increases cerebral and myocardial perfusion.

COMMENTARY - ADVANCED LIFE SUPPORT (Figure 2)

1. Shockable rhythms - ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT)

If a shockable rhythm is present defibrillation with 4J.kg^{-1} should be performed immediately. Chest compression should be restarted immediately even if a rhythm change is seen on the monitor. This is important, as the heart will not be able to support the circulation for a minute or so, even if sinus rhythm resumes. If defibrillation is unsuccessful, CPR should be continued for a further 2 minutes and the defibrillation cycle repeated. If a third shock is necessary, epinephrine (adrenaline) should be given immediately before and an anti-arrhythmic should be used before a fourth shock. Amiodarone (5mg.kg^{-1}), where available, is preferred but lidocaine (1mg.kg^{-1}) is an acceptable alternative.

2. Non-shockable rhythm - asystole or pulseless electrical activity (PEA)

If the rhythm is not shockable, the emphasis is on good quality CPR with minimum interruption in ECC together with adrenaline administration every 3-5 minutes.

3. Reversible causes

In both shockable and non-shockable rhythms treatable causes should be sought and dealt with. Children rarely suffer from primary heart disease, so there is often a precipitating cause and resuscitation will not be successful if this is not removed. Treatable causes can be remembered by the 4Hs and the 4Ts mnemonic.

4**Hs**	4**Ts**
Hypoxia	**T**ension pneumothorax
Hypovolaemia	Cardiac **T**amponade
Hypo/hyperkalaemia	**T**oxicity
Hypothermia	**T**hromboembolism

4. Other points

Drugs

By far the most important treatment in resuscitation is good quality basic life support with continuous chest compression and effective, but not excessive, lung inflation. The next important action is to remove any reversible precipitating causes. Although drugs are commonly administered, there is little evidence to support routine administration of many of them. As the tracheal route of administration is largely ineffective, circulatory access had to be achieved rapidly; this is most effectively performed by intraosseous cannulation unless a peripheral vein can be accessed immediately.

Oxygen

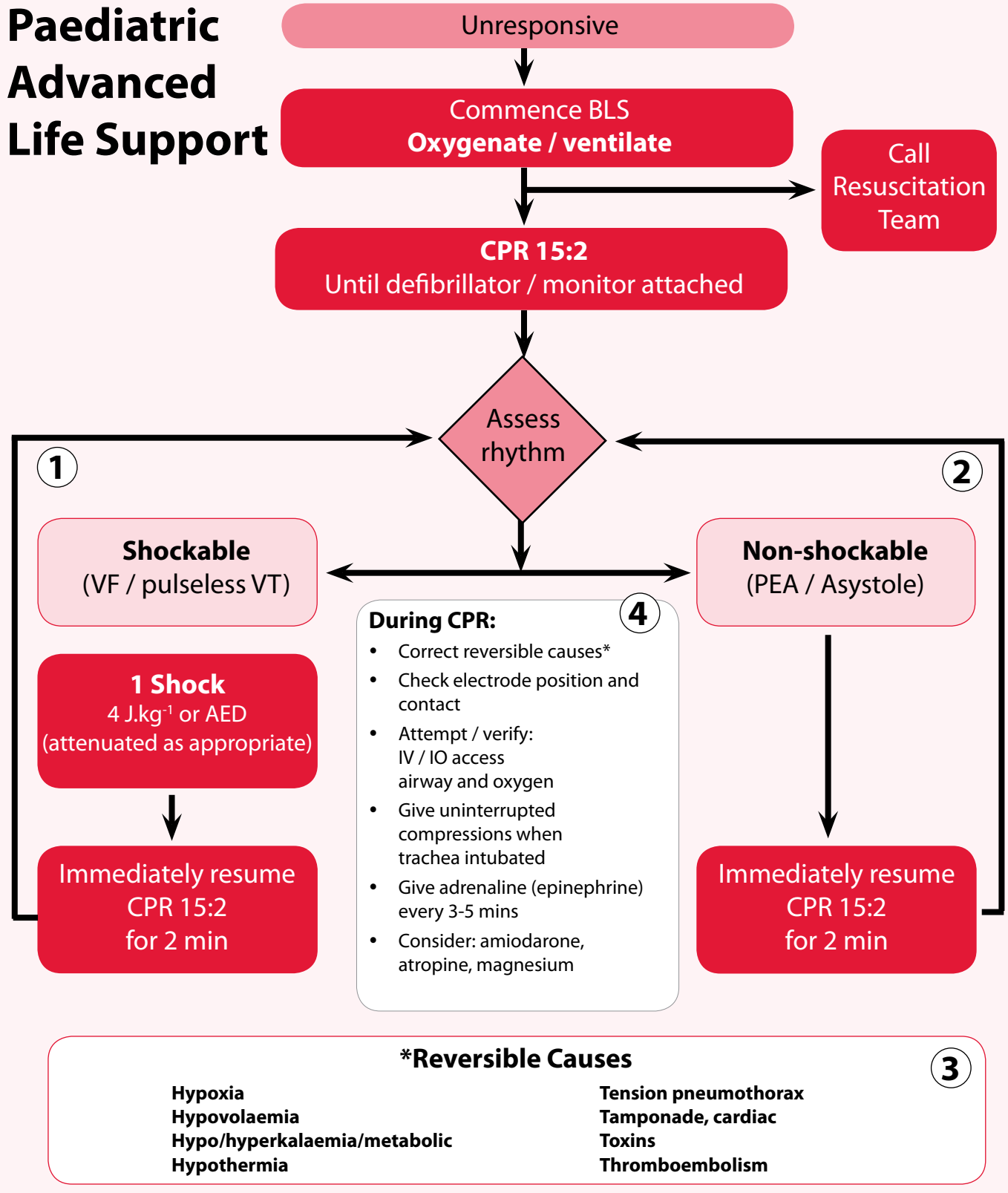
This is the most important drug in paediatric resuscitation as many arrests in children are due to hypoxia. Although high concentrations are often used, effective airway opening and lung inflation are by far the most important steps in achieving adequate oxygenation.

Epinephrine (adrenaline)

Epinephrine been shown to increase the chances of restoring spontaneous circulation and should be administered in a dose of 10mcg.kg^{-1} every 3-5 minutes during resuscitation. Larger doses have not been shown to be effective and should not be used.

Resuscitation Council (UK)

Paediatric Advanced Life Support



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Sodium bicarbonate ($NaHCO_3$)

Bicarbonate neutralises acidosis by releasing carbon dioxide. During resuscitation, this cannot be cleared as there is insufficient pulmonary gas exchange, consequently, it has not been shown to be effective and should not be used routinely. $NaHCO_3$ may be indicated in specific circumstances such as hyperkalaemia or in drug toxicity (e.g. tricyclic antidepressants).

Calcium has not been shown to be effective in resuscitation and it may even be harmful, consequently it should not be used routinely. It may however, be effective in hyperkalaemia, hypocalcaemia and calcium receptor blocker overdose.

Amiodarone ($5mg.kg^{-1}$) has been shown to be the most effective anti-arrhythmic in resistant VF or pVT but lidocaine is an acceptable alternative. Amiodarone is incompatible with saline and should be diluted in 5% glucose.

OUTCOMES

Although it is often thought that children have extremely poor outcome after cardiac arrest, this is not entirely true. Large North American databases have shown that children that have a full cardiac arrest in a hospital have a 27% chance of survival to discharge and that 75% of these will have a good neurological outcome. Out of hospital resuscitation has poorer survival rates but these figures are significantly biased by infants with sudden infant death syndrome (SIDS). Older children and adolescents have survival rates of about 9%.

Children with respiratory arrest only who haven't progressed to full cardiac arrest have an excellent chance of survival with about 70% alive after 1 year.

SUMMARY

The most important intervention in paediatric resuscitation is early recognition of the child at risk of deterioration and the instigation of treatment intended to prevent progression to cardio-respiratory arrest.

Once cardio-respiratory arrest has occurred, early and good quality CPR is the most important step for a favourable outcome. Interruptions in chest compression should be avoided and compressions can be continuous once the trachea is intubated. Reversible causes should be actively sought and treated as many paediatric arrests are secondary to another event.

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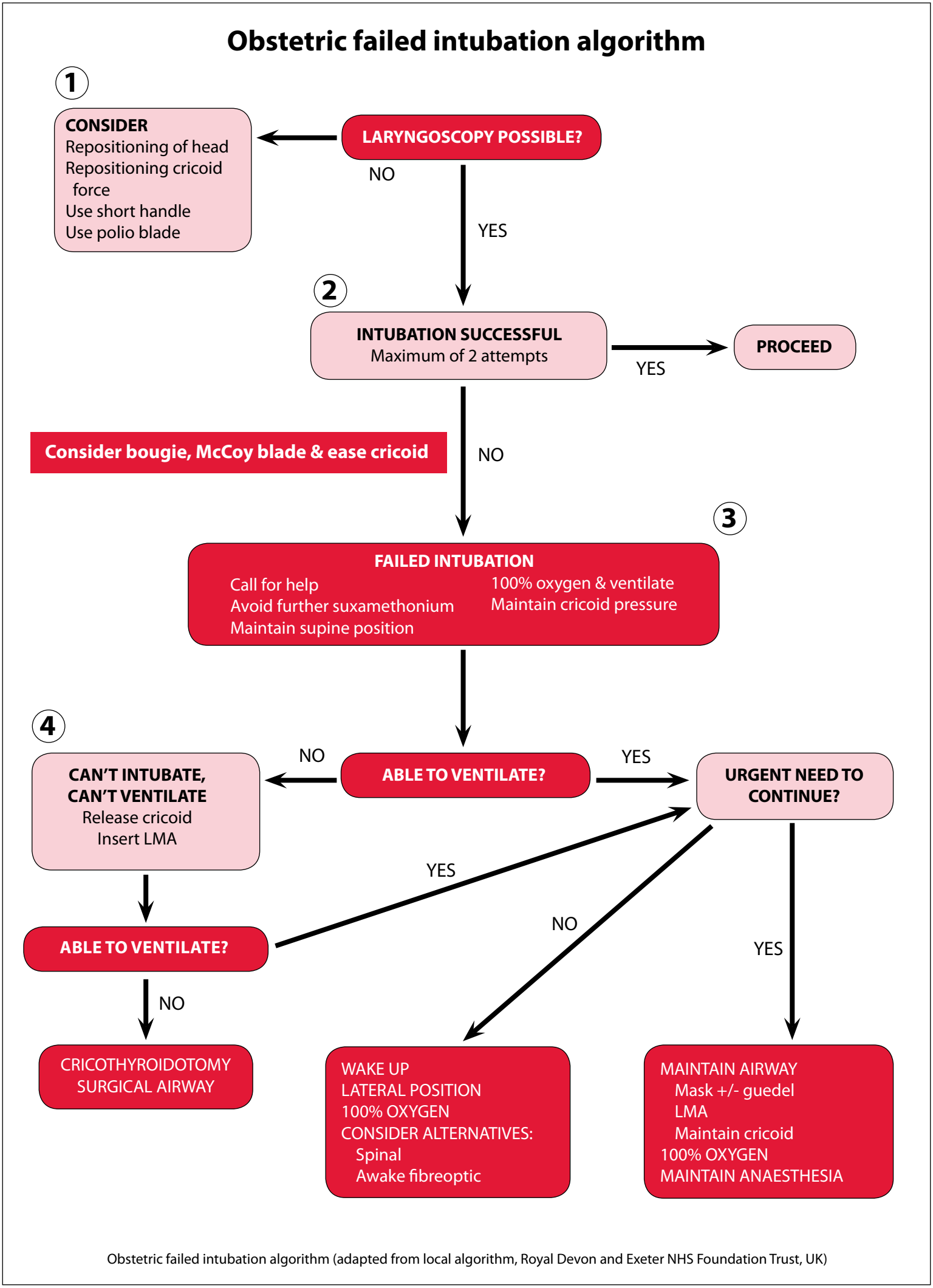

Obstetric failed intubation algorithm (adapted from local algorithm, Royal Devon and Exeter NHS Foundation Trust, UK)

Figure 1. *Available for download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

Management of obstetric failed intubation

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Summary

Assess the airway before induction of anaesthesia.

Check all intubation equipment daily and be familiar with its use.

Position the patient correctly before induction.

Remember that oxygenation is more important than intubation.

Call for help early.

Maternal welfare is paramount and takes priority over foetal considerations.

There is no single definition for failed tracheal intubation. The inability to intubate following a single dose of succinylcholine is an pragmatic definition in the obstetric setting.[1]

Failed intubation is an important factor contributing to both maternal and foetal mortality.[2,3] Ideally we should be able to predict, and plan for, all difficult intubations. However, most airway tests are unreliable so we will inevitably be faced with some unexpectedly difficult or impossible intubations. The next best option is to have a robust plan for the management of such a situation.

The incidence of failed tracheal intubation in the general surgical population is approximately 1:2200, but the incidence in the obstetric population may be as high as 1:250.[1,4] Pharyngeal oedema may explain some of this difference and it has been shown that Mallampati scores worsen throughout pregnancy.[5]

PREOPERATIVE ASSESSMENT

A clinical assessment of the airway and risk of difficult intubation can be performed in a matter of seconds. It should include the following;

1. Mouth opening (should be greater than three of the patient's fingerbreadths).
2. Mallampati view (pharynx should be visible).
3. Identifying large protruding incisors.
4. Jaw slide (should be able to push the lower incisors anterior to the upper incisors).
5. Neck movement (full, unhindered range of at least 90°).
6. Evidence or possibility of laryngeal swelling (severe pre-eclampsia or upper respiratory tract infection).
7. History of previous problems.

If difficulty is anticipated and surgery is not urgent a consultant anaesthetist should be present.

EQUIPMENT

The following should be readily available.

1. Selection of laryngoscopes (long and standard blade, short-handled or polio blade, McCoy).
2. Selection of tracheal tubes (size 5.0mm upwards).
3. Gum elastic bougie - with selected tracheal tube already threaded on.
4. Selection of oropharyngeal airways.
5. Laryngeal mask airway (size 3).
6. Alternative airway devices eg. ILMA/Airtraq optical laryngoscope (only to be used by those with prior experience).
7. Cricothyroidotomy kit (or equipment for transtracheal ventilation and suitable connectors).

Many obstetric centers have produced algorithms to guide clinicians in the failed intubation scenario. The following version is based upon the existing algorithm used within our hospital. It has been revised following an appraisal of a number of alternative local practice algorithms. No appropriate guideline was found that has been published by a national body. It is largely self-explanatory but a commentary is included to highlight the important aspects.

COMMENTARY ON ALGORITHM

Box 1 - *Laryngoscopy not possible*

- Obstruction to the insertion of the laryngoscope by the patient's breasts or the anaesthetic practitioner's hand can be overcome by using the polio blade. Alternatively, insert the ordinary blade into the patient's mouth before attaching the handle.

Box 2 - *Initial attempts at intubation unsuccessful*

- If intubation is difficult but the epiglottis is visible try using a gum elastic bougie and/or a McCoy blade. When railroading the ETT over a bougie rotate the ETT 90° anti-clockwise. This often helps to overcome resistance.
- A smaller ETT is often needed in obstetrics, especially if there is a history of URTI or pre-eclampsia, both of which predispose to laryngeal oedema.
- An obscured laryngeal view is often due to incorrectly applied cricoid pressure. Tilting the patient laterally can exacerbate this problem. Careful readjustment should improve the view.

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- Do not persist with repeated intubation attempts. This will increase bleeding and swelling resulting in a higher rate of complications.

Box 3 - *Failed intubation*

- It is important to ventilate the lungs with 100% oxygen via bag and mask as soon as possible.
- Using a two hand technique to hold the mask may improve the seal for ventilation.
- Avoid a second dose of suxamethonium as the paralyzing effect may last for a significantly longer duration.

Box 4 - *Can't intubate can't ventilate*

- If ventilation of the lungs is impossible with a bag and mask, cricoid pressure should be eased as excessive force can obstruct the airway. If this is ineffective the problem is probably anatomical.
- Insertion of an oropharyngeal airway may help but the laryngeal mask airway (LMA) is generally considered to be the most useful device if ventilation is not achieved. Cricoid pressure should be released to allow correct insertion of the LMA. Once in place it may be possible to reapply cricoid pressure as long as ventilation is not compromised.

Box 5 - *Consider the need to continue surgery*

- As soon as satisfactory ventilation and oxygenation has been established, consideration should be given to the degree of urgency of the procedure. The following grades can be used to aid the anaesthetist in deciding between continuing general anaesthesia (GA) without the protection of an endotracheal tube, and using an alternative technique that will result in some delay.

BOX 6 - *Cricoidotomy / surgical airway*

- Needle cricothyrotomy should be attempted by the anaesthetist if ventilation is still impossible. All anaesthetists should familiarise themselves with the local cricothyroidotomy kit.

Surgical cricothyroidotomy

1. Place the patient supine.
2. Consider extending the neck to improve access. Otherwise, maintain a neutral alignment.
3. Identify the cricothyroid membrane.
4. Prepare the skin and, if the patient is conscious, infiltrate with local anaesthetic.
5. Place your left hand on the neck to stabilise the cricoid and thyroid cartilages, and to protect the lateral vascular structures from injury.
6. Make a small vertical incision in the skin, and press the lateral edges of the incision outwards, to minimise bleeding.
7. Make a transverse incision through the cricothyroid membrane, being careful not to damage the cricoid cartilage.
8. Insert tracheal spreader, or use the handle of the scalpel by inserting it through the incision and twisting it through 90° to open the airway.
9. Insert an appropriately sized endotracheal or tracheostomy tube. It is advisable to use a slightly smaller size than would have been used for an oral or nasal tube.
10. Ventilate the patient and check that this is effective.
11. Secure the tube to prevent dislodgement.
12. If you have performed a cricothyroid puncture you may need to use intravenous agents such as propofol to keep the patient asleep.

Table 1. *Urgency of procedure – decision making*[6]

Grade	
1	Mother's life depends on completion of surgery e.g. cardiac arrest, massive haemorrhage.
	No alternative but to continue GA
2	Maternal pathology makes alternative regional techniques impossible e.g. decompensated heart disease or coagulopathy.
	Probably acceptable to continue GA but should consider awake fibreoptic intubation.
3	Sudden and severe fetal distress not recovering between contractions e.g. in placental abruption or prolapsed cord.
	This is the most difficult grade. Abandoning GA may lead to foetal death but it could be argued that maternal well-being is paramount and waking the patient for a regional technique would be appropriate. This decision must be made based on obstetric circumstances and the quality of the maintained airway.
4	Long standing fetal distress of varying severity with good recovery between contractions.
	The patient should be woken and a regional technique performed.
5	Elective procedure or maternal distress.
	Absolutely no indication to continue under GA and the patient should be woken and an alternative technique used.

- Cannula cricothyroidotomy is a temporary measure that allows oxygenation but not ventilation. A tube of 4mm internal diameter or greater is necessary to achieve adequate ventilation. The cannula should be replaced by a definite airway, when appropriate staff and equipment are available.
- Definitive (surgical) cricothyroidotomy should be undertaken by the clinician with the most experience. It is important not to delay this procedure if other attempts at oxygenation have failed.

CONCLUSION

The algorithm serves only as a guide to facilitate decision making and clinical judgement must be exercised. It is important to remember that patients do not die from failure to intubate but failure to oxygenate. Ensuring adequate pre-oxygenation may buy time if a problem is encountered.

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Management of Obstetric Haemorrhage

3 Initial management

- **CALL FOR HELP**
- Assess and treat Airway, Breathing, Circulation
- High flow **OXYGEN** via facemask
- Head down tilt (left lateral if APH)
- **IV ACCESS** - Two14G (orange) cannulae
- Take blood - FBC, coagulation, +match 6 units
- Request cell salvage
- **GIVE WARMED FLUIDS** (level 1 infuser)
  - crystalloid up to 2000ml
  - colloid up to 1500ml
- **CONTACT HAEMATOLOGY**
- 0-ve blood if bleeding uncontrolled
- **Type specific blood** if the time permits (20 minutes)

1 Definitions

MINOR	500 - 1000ml
MODERATE	1000 - 2000ml
SEVERE	>2000ml

Blood loss is frequently underestimated

2 Diagnosis

APH Placenta praevia or placental abruption

If severe consider immediate delivery

PPH **Tone** (atonic uterus - 70%) **Trauma** (20%)

Tissue (retained products - 10%)

Thrombin (coagulapathy)

4 Management of PPH

PHARMACOLOGICAL:

- Syntocinon 2-5IU slowly IV (repeat once)
- Ergometrine 500mcg slowly IV (caution in hypertension)
- Syntocinon IV infusion 30IU in 500mls at 125ml.hr^{-1}
- Carboprost 0.25mg IM every 15min - 8 doses max (caution in asthmatics)
- Misoprostol 1mg PR

SURGICAL

- Examination under anaesthetic
- Uterine massage if atony
- Bimanual uterine compression
- Balloon tamponade (e.g. Rusch balloon)
- B-Lynch suture
- Ligation of uterine/internal iliac arteries
- Hysterectomy

RADIOLOGICAL:

- Contact radiology consultant
- Embolisation / arterial balloon occlusion

5 Blood loss and coagulation

- Team member to coordinate sample delivery and blood product collection
- Contact senior haematologist for advice
- Give packed blood cells (RBC)
- After 4U RBC give 1 unit FFP for each further unit of blood
- If INR >1.5 give **FFP**
- Give **platelets** if <50x10^{9}/l
- **Cyroprecipitate** 1 unit per 5kg, if fibrinogen 1.5gl^{-1}
- If DIC suspected, transfuse platelets and cyroprecipitate early
- Repeat coagulation studies and regular Hemocue®
- Consider **recombinant factor VIIa** 90mcg.kg^{-1} and/or Octaplex if available (D/W haemotologist)
- Consider 10ml 10% calcium gluconate
- Consider **gluconate tranexamic acid** 15mg.kg^{-1} IV

6 Anaesthetic considerations

- Call consultant anesthetist
- Liaise early with ICU
- Avoid hypothermia: early use of warm air blower and warmed fluids
- Weigh swabs to aid accurate estimation of blood loss
- Monitor urine output and temperature
- Consider arterial line early
- Take regular blood samples for FBC, coagulation, ABG and Hemocue®
- Avoid regional anesthesia if cardiovascular instability: **GA and RSI is usually indicated**
- Consider oesophageal Doppler and/or CVP monitoring
- A vasopressor may be required despite fluid resuscitation. Use phenylephrine in first instance; norepinephrine infusion (4mg in 40ml 5% glucose) may be necessary

Figure 1. *Available for download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

Management of obstetric haemorrhage

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Summary

Obstetric haemorrhage is frequently underestimated.

Ensure your Obstetric Unit has a protocol for the management of haemorrhage – and practice it.

Call for senior help.

Successful management requires multidisciplinary input.

Remember to replace both blood and blood components.

INTRODUCTION

Obstetric haemorrhage remains one of the leading causes of preventable maternal morbidity and mortality worldwide. Life-threatening haemorrhage occurs in around 1 in every 1000 deliveries. Prompt recognition and management of obstetric haemorrhage is essential.

The majority of maternal deaths due to haemorrhage in the 2003-2005 UK Confidential Enquiry into Maternal and Child Health report were deemed to have received 'major substandard care' (10 out of 17 fatalities). The implications of suboptimal management of severe obstetric haemorrhage are numerous, threatening not only the well being of the mother, but also that of her neonate and family.

The guideline shown in Figure 1 is an adapted version of the local guideline in use in our centre – it has been adapted with reference to the Royal College of Obstetricians and Gynaecologists (RCOG) Green-top Guideline No.52, that deals specifically with postpartum haemorrhage and is for use by both anaesthetists and obstetric staff.

For the purpose of this review, the points made in the boxes of the guideline in Figure 1 are expanded and discussed in sequential order. However, in a case of major obstetric haemorrhage resuscitation, monitoring, investigation and management should occur simultaneously. Note that the core guideline gives recommendations for management of postpartum haemorrhage, whilst additional points appropriate to antepartum cases are also highlighted.

Some of the facilities and equipment detailed in the guideline will not be available in many healthcare settings. They are included for completeness, in the knowledge that practitioners will be able to use those parts of the guideline which are applicable to the system within which they work.

COMMENTARY ON ALGORITHM

Box 1 – Definitions

Antepartum haemorrhage (APH)

APH is defined as vaginal bleeding after gestation of 22 weeks. Blood loss may be concealed and lead to underestimation of haemorrhage.

Causes include:

- Placenta praevia
- Placental abruption
- Infection
- Trauma

Postpartum haemorrhage (PPH)

Post partum haemorrhage may be primary or secondary.

Primary PPH can be defined as the loss of more than 500ml of blood within 24 hours of delivery. It can be further subdivided into minor (500ml-1000ml) or major (more than 1000ml). Exact numerical definitions of blood loss are unimportant. It is essential to remember that blood loss is frequently underestimated and physiological variables, especially that of systolic blood pressure, may change little until 30 to 40% circulating blood volume has been lost. The clinician must therefore maintain a high index of suspicion for major obstetric haemorrhage.

An estimated blood loss of 1000ml (or less with concurrent clinical signs of haemorrhagic shock such as tachycardia, tachypnoea, prolonged capillary refill, oliguria and, in extremis, hypotension and altered cognitive function) should initiate the protocol for the management of major obstetric haemorrhage. Education of staff in the use of more accurate measurement of blood loss, near patient testing of haemoglobin (Hemacue™) and an obstetric Early Warning Score may facilitate earlier diagnosis and treatment of obstetric haemorrhage (see appendices).

Box 2 - Diagnosis

Causes of primary PPH can be divided into the "Four Ts": (See Table 1. *Risk factors for PPH*)

- **Tone**: Atonic uterus (The most common, accounting for 70% of PPH)
- **Tissue**: Retained products (10%)
- **Trauma**: Genital tract trauma (20%)
- **Thrombin**: Coagulopathy, e.g. DIC (1%)

Secondary PPH is defined as abnormal or excessive bleeding from the birth canal between 24 hours and 12 weeks postnatally. Causes include retained products of conception and sepsis.

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Table 1: *Risk factors for PPH*

Classification	Risk factor
Tone	Multiple pregnancy
	Previous PPH
	Obesity (BMI>35)
	Large baby (>4kg)
	Prolonged Labour (>12hrs) / prolonged 2nd stage
	Advanced maternal age (>40 years, primiparous)
	Retained placenta
	Asian ethnicity
	Placenta Praevia
Tissue	Retained placenta
	Placenta accreta, increta and percreta (high mortality: associated with previous caesarean section)
Trauma	Delivery by caesarean section (emergency>elective)
	Operative vaginal delivery
	Mediolateral episiotomy
	Large baby (>4kg)
Thrombin	APH (placental abruption)
	Pre-eclampsia
	Sepsis
Other	Pre-existing coagulation problems, thrombocytopaenia, women taking anti-coagulannts

Women with pre-existing coagulation problems, thrombocytopaenia or those on anticoagulants are at increased risk of obstetric haemorrhage. The specific management is not described within this article.

Box 3 - Initial management of obstetric haemorrhage, monitoring and effective fluid and transfusion strategies

Call for help

This is essential. An obstetric emergency requires multidisciplinary input. Several pairs of hands are needed to aid resuscitation, to fetch equipment, prepare the operating theatre and transport blood samples and blood products to and from the laboratory. In the author's hospital, switchboard can be asked to put out a Major Obstetric Haemorrhage Call, which summons the appropriate staff including the obstetrician, anaesthetist, haematologist, operating department assistant, midwives, theatre staff and porter.

A, B, C and oxygen

Assess Airway, Breathing and Circulation in accordance with the ALS guidelines. It is important to perform concurrent evaluation and resuscitation. Ensure monitoring is attached (BP, ECG, SaO_2).

Resuscitation should include:

- High flow oxygen via a face mask with a reservoir bag.
- Head down tilt to increase venous return to the heart and help preserve cardiac output.
- Intravenous access with two large bore (14G) short cannulae remembering to take blood for FBC, coagulation, and cross-match (minimum of 6 units).
- Consider invasive arterial blood pressure (IABP) monitoring early, once resuscitation is underway. IABP monitoring provides accurate, continuous blood pressure measurement as well as access to arterial blood for blood gas analysis and blood samples for evaluation of coagulation.

Fluid resuscitation

Commence intravenous fluid resuscitation. Aim to aggressively restore circulating volume using pressure bags and a fluid warmer (or Level 1 infuser if available). It is essential to infuse warmed fluids as large volumes of cold fluids place the patient at risk of hypothermia. Hypothermia may induce shivering and subsequently increase O_2 consumption in a patient with decreased O_2 carrying capacity and decreased O_2 reserves. Hypothermia may also impair coagulation, affect renal and liver function and delay wound healing.

Once 3500ml of warmed crystalloid (2000ml) and/or colloid (1000ml) have been infused, further resuscitation should continue with blood. Give O Rhesus negative blood (immediate) or group specific blood (20 minutes) until crossmatched red blood cells are available (40-60 minutes). This ensures improvement in the O_2 carrying capacity. If the haemorrhage continues, inform the haematology laboratory of the likely need for additional blood and blood products.

Note that use of major haemorrhage packs is now established for management of traumatic blood loss in conflict zones. These packs contain four units of blood with a unit of fresh frozen plasma (and, in some instances, platelets or cryoprecipitate) and are aimed at pre-emptive control of the coagulopathy that complicates major haemorrhage. The most recently constructed algorithms for management of major haemorrhage in trauma recommend this approach, although currently available guidance for the obstetric setting recommends conventional use of packed cells and other blood products guide by laboratory investigations. Guidance from the Association of Anaesthetists of Great Britain and Ireland is anticipated in October 2010 and will be reproduced in a future edition of *Update*.

Specific points for management of APH

Many of the initial resuscitative measures remain identical to those in PPH. In addition, consideration must be given to assessment and optimisation of foetal well being. The patient should be placed in a head down position with left lateral tilt or uterine displacement in order to avoid aorto-caval compression. Foetal monitoring should be applied and an assessment of the foetal gestation and viability made. Severe APH usually mandates urgent surgery and delivery of the baby.

Box 4 - Management of PPH

Measures aimed at minimising blood loss can be divided into pharmacological, surgical, radiological and haematological. Treatment must be tailored to the underlying cause of PPH, for example use drugs to stimulate uterine contraction in cases of uterine atony, or undertake evacuation of the uterus for retained products or surgical repair of genital tract trauma.

Box 4A – Pharmacological management of PPH

The most common cause of PPH is uterine atony. In addition to uterine massage, the following drugs stimulate uterine contraction:

Syntocinon

- Syntocinon is a synthetic analogue of oxytocin and 5IU is given by slow intravenous injection.
- Rapid injection may result in vasodilatation and subsequent hypotension and tachycardia.
- The dose may be repeated once (maximum of 10IU).
- Thereafter, an infusion of 30 to 40IU in 500mls 0.9% Saline may be commenced at a rate of 125ml.hr^{-1}.

Ergometrine

- The dose is 500mcg by slow intravenous or intramuscular injection.
- Adverse effects include nausea, vomiting and vasoconstriction leading to a marked rise in BP. It is therefore best avoided in patients with cardiovascular disease and pre-eclampsia.

Carboprost

- Carboprost is a prostaglandin F2 receptor agonist which stimulates uterine contraction
- 250mcg is given by intramuscular injection. This may be repeated at 15 minute intervals to a maximum of eight doses.
- Adverse effects include bronchospasm, hypoxia, flushing, nausea and vomiting. It should be avoided in patients with asthma.

Misoprostol

- Misoprostol is a prostaglandin E1 analogue which also stimulates uterine contraction. It rarely causes any serious side effects.
- The dose is 1mg rectally.

Box 4B - Surgical treatments for PPH

If pharmacological measures fail to adequately control the haemorrhage, attempt mechanical measures. In the case of uterine atony, uterine massage or bimanual uterine compression may stimulate uterine contractions and aid control of PPH. However, if the preceding measures fail, the following invasive surgical measures should be considered:

- Balloon tamponade
- B-Lynch suture
- Bilateral ligation of uterine or internal iliac arteries
- Hysterectomy

Balloon tamponade has largely superseded uterine packing in the control of PPH secondary to uterine atony. A variety of hydrostatic balloon catheters (including the Foley catheter, Bakri balloon, Rusch balloon and Senstaken Blakemore tube) are suitable for intrauterine placement and subsequent balloon inflation. If PPH is arrested, the catheter should be left in situ for at least 6 hours. Failure to achieve haemorrhagic control after balloon inflation indicates the need for laparotomy.

B-Lynch sutures are a form of haemostatic brace suturing which require hysterotomy for insertion. They are most useful in the control of PPH following caesarean section (as the uterus has already been opened). Modified techniques which do not require hysterotomy have been described. Haemostatic sutures may reduce the need for hysterectomy but should only be used by a skilled surgeon familiar with the technique.

The choice of surgical procedure will depend on the expertise of the staff and availability of equipment. Temporising measures may be undertaken pending the arrival of an experienced clinician skilled in both judging the need for, and in performing, peripartum hysterectomy which is often challenging. Unnecessary delay must be avoided however and the difficult decision to perform a peripartum hysterectomy should be made before the woman is in extremis.

Box 4C - Radiological strategies in PPH

Where facilities for interventional radiology are available, arterial embolisation or balloon occlusion of iliac vessels may obviate the need for hysterectomy. However, few centres have this service immediately available and many patients will be too unstable to tolerate potentially long X-ray procedures which may also require transfer to a radiology department. Transfer for an interventional radiological procedure may be particularly useful in the stable patient who is still bleeding despite the surgical interventions described above.

Box 5 - Blood loss and coagulation management

At term, the blood supply to the uterus may exceed 800ml.min^{-1}. Haemodynamic compromise may occur rapidly or more insidiously (e.g. retained products). The latter may be associated with a delay in recognition of the severity of haemorrhage due to the patient's ability to compensate until life threatening haemorrhage has already occurred.

It is important to remember that haemorrhage results in the loss of not only red blood cells but also blood components and platelets. Once four units of packed red blood cells (RBCs) have been transfused, consideration should be given to the replacement of other blood components. There is recent evidence from military medicine that aggressive replacement of coagulation products may improve outcome. Following transfusion of 4 units RBCs, a 1:1 ratio of FFP to RBC is recommended in major haemorrhage. Transfusion of RBCs alone increases the oxygen carrying capacity of blood but will not correct an underlying coagulopathy.

The endpoint of the coagulation cascade is the conversion of fibrinogen to fibrin. Even with adequate factor levels, fibrinogen is essential for coagulation. During pregnancy, fibrinogen levels increase and women should be considered severely hypofibrinogenaemic and transfused

fibrinogen in the form of cryoprecipitate if their fibrinogen level falls below 1.5g.l^{-1}.

A named team member should coordinate the delivery of blood samples and collection of blood products to and from the haematology laboratory. Delivery of multiple blood samples and collection of blood products may be required.

Alert the oncall haematologist. They are best able to guide transfusion requirements based on regular full blood count and coagulation studies, however in a major haemorrhage, waiting for coagulation results from the laboratory must not delay transfusion of coagulation factors. The following may serve as a guide to the main haematological goals in the management of massive blood loss:

Suspicion of disseminated intravascular coagulation should prompt earlier administration of platelets and cryoprecipitate.

Table 2. *Guide to use of blood products*

	Target	Action
Hb	> 8g.dl^{-1}	If less, transfuse RBCs
INR	< 1.5	If prolonged, transfuse Fresh Frozen Plasma (FFP)
Platelets	> 50 x 10^{9}.l^{-1}	If less, transfuse platelets
Fibrinogen	> 1.5g.l^{-1}	If less, transfuse cryoprecipitate 1unit/5kg

Pharmacological manipulation of coagulation

Recombinant factor VIIa - 90mcg.kg^{-1}

Recombinant activated Factor VII was originally developed for the treatment of haemophilia but has been used in the management of major trauma and obstetric haemorrhage. Evidence of its efficacy is limited. However, as the activation of factor VII by tissue factor is one of the initial steps in the coagulation cascade, it may prove to be useful in the management of life threatening PPH. Ensure adequate levels of fibrinogen and platelets prior to administration.

Human pro-thrombin complex (e.g. Octaplex)

Octaplex is a human pro-thrombin complex which may have a role in the management of persistent non surgical haemorrhage.

Tranexamic acid - 15mg.kg^{-1} IV

Tranexamic acid is an antifibrinolytic agent which inhibits the conversion of plasminogen to plasmin. It may have a role in uncontrolled haemorrhage although there is little supporting evidence for its efficacy.

Near patient testing of coagulation and fibrinolysis by thromboelsatography or thromboelastometry if available, may be very useful in guiding the management of coagulation as a result of massive obstetric haemorrhage.

Box 6 - Anaesthetic considerations

Call a senior anaesthetist

This is essential. Obstetric patients with severe haemorrhage may decompensate rapidly. An experienced pair of hands is vital to aid in resuscitation and decision making. Seek early Intensive Care involvement.

Continue resuscitation with warmed fluids and apply early active patient warming (e.g. forced warm air device) to avoid hypothermia. Consider upgrading monitoring (arterial +/- CVP) if situation allows but **DO NOT DELAY URGENT SURGERY** to facilitate insertion. Haemostasis takes precedence.

If surgery is required, remember to ensure that routine safety precautions are taken including anaesthetic history, airway assessment, antacid prophylaxis and pre-oxygenation. Regional anaesthesia is usually best avoided in the case of major obstetric haemorrhage as it may prevent sympathetic compensation in a patient who is intravascularly deplete, leading to marked hypotension and inadequate perfusion of vital organs. General anaesthesia following a rapid sequence induction with cricoid pressure is therefore usually the technique of choice.

Drug dosages for induction and maintenance of general anaesthesia may need to be modified according to the circulatory status of the patient. Some induction agents (e.g. thiopentone, propofol) may result in significant hypotension in the presence of hypovolaemia. In major haemorrhage, ketamine (1.5mg.kg^{-1} IV) may better preserve cardiovascular stability. Remember that the volatile agents cause uterine relaxation and excessive concentrations should be avoided, especially in the case of uterine atony.

The focus of resuscitation should be preservation of the woman's life rather than preservation of her uterus.

Resuscitation must be guided by taking regular blood samples for FBC (Hemacue™ if available), coagulation and arterial blood gases. Cell salvage may reduce requirements for homologous blood and is increasingly used in obstetric haemorrhage. Other means of assessing cardiovascular status include the use of an oesophageal Doppler and/or a central line. Although the latter may be used to monitor trends in central venous pressure it has perhaps greater benefit in facilitating inotrope administration if required.

Postoperative care

The patient should be managed where she can be watched closely, given oxygen and her vital signs monitored at regular intervals. Ideally this should be in a high dependency area.

SUMMARY

This guideline and accompanying article describe the emergency management of an obstetric patient with major haemorrhage. Haemorrhage in this setting can be sudden, profuse and unexpected. Management is therefore likely to be more efficient and effective if guided by a protocol, which combines the common objectives of anaesthesia, midwifery and obstetric staff. It is important to call for senior anaesthetic and obstetric assistance at an early stage. As blood is transfused it is important to remember to administer blood products - it is likely that future guidelines will advocate use of 'massive transfusion packs' (with blood, fresh frozen plasma and platelets) from the onset of resuscitation.

FURTHER READING

1. Royal College of Obstetricians and Gynaecologists. Prevention and Management of Postpartum Haemorrhage. Green-top Guideline No.52. London RCOG; 2009. Available at: www.rcog.org.uk/womens-health/clinical-guidance/prevention-and-management-postpartum-haemorrhage-green-top-52
2. Royal College of Obstetricians and Gynaecologists. Blood Transfusion in Obstetrics. Green-top Guideline No.47. London RCOG; 2008. Available at: www.rcog.org.uk/files/rcog-corp/uploaded-files/GT47BloodTransfusions1207amended.pdf
3. Confidential Enquiry into Maternal and Child Health. Saving Mothers Lives 2003-2005. Seventh Report on Confidential Enquiries into Maternal Deaths in the United Kingdom. London: CEMACH; 2006. Available at: www.cmace.org.uk/getattachment/927cf18a-735a-47a0-9200-cdea103781c7/Saving-Mothers--Lives-2003-2005_full.aspx
4. Bose P, Regan F, Paterson-Brown S. Improving the accuracy of estimated blood loss at obstetric haemorrhage using clinical reconstructions. *BJOG* 2006; **113**: 919–24.
5. P. Moor, D. Rew, M.J. Midwinter, H. Doughty. Transfusion for trauma: civilian lessons from the battlefield? *Anaesthesia* 2009, **64**, 469-72.

Appendix 1. *Example of an Obstetric Early Warning Scores Chart. Reproduced with permission from the RCOG from Green-Top Guideline No. 52 (2009)*

OBSTETRIC EARLY WARNING CHART. FOR MATERNITY USE ONLY

NHS Forth Valley

NAME: _____ **DOB:** _____

CHI: **WARD:**

CONTACT DOCTOR FOR EARLY INTERVENTION IF PATIENT TRIGGERS ONE RED OR TWO YELLOW SCORES AT ANY ONE TIME

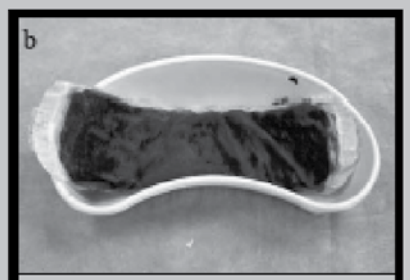
	Date :																		
	Time :																		
RESP (write rate in corresp. box)	>30																		>30
	21-30																		21-30
	11-20																		11-20
	0-10																		0-10
Saturations	90-100%																		90-100%
	<90%																		<90%
O2 Conc.	%																		%
Temp	39																		39
	38																		38
	37																		37
	36																		36
	35																		35
HEART RATE	170																		170
	160																		160
	150																		150
	140																		140
	130																		130
	120																		120
	110																		110
	100																		100
	90																		90
	80																		80
	70																		70
	60																		60
	50																		50
	40																		40
Systolic blood pressure	200																		200
	190																		190
	180																		180
	170																		170
	160																		160
	150																		150
	140																		140
	130																		130
	120																		120
	110																		110
	100																		100
	90																		90
	80																		80
	70																		70
	60																		60
	50																		50
Diastolic blood pressure	130																		130
	120																		120
	110																		110
	100																		100
	90																		90
	80																		80
	70																		70
	60																		60
	50																		50
	40																		40
Passed Urine	Y or N																		Y or N
Lochia	Normal																		Normal
	Heavy / Foul																		Heavy / Foul
Proteinuria	2+																		2+
	> 2+																		>2+
Liquor	Clear / Pink																		Clear/Pink
	Green																		Green
NEURO RESPONSE (√)	Alert																		Alert
	Voice																		Voice
	Pain / Unresponsive																		Pain /Unresponsive
Pain Score (no.)	2-3																		2-3
	0-1																		0-1
Nausea (√)	YES (√)																		YES (√)
	NO (√)																		NO (√)
Looks unwell	YES (√)																		YES (√)
Looks unwell	NO (√)																		NO (√)
Total Yellow Scores																			
Total Red Scores																			

Appendix 2. *Reprinted with permission from Miss Paterson-Brown, Consultant Obstetrician, Queen Charlottes Hospital (colour version available on the British Maternal & Fetal Medicine Society Website at:* http://www.bmfms.org.uk/docs/Guide%20to%20Aid%20Visual%20Estimation%20of%20Blood%20Loss%20at%20Obstetric%20Haemorrhage.pdf)

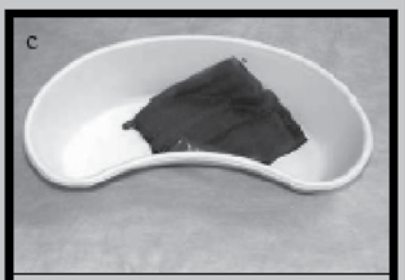
A Pictorial Reference Guide to Aid Visual Estimation of Blood Loss at Obstetric Haemorrhage: Accurate Visual Assessment is Associated with Fewer Blood Transfusions

Dr Patrick Bose, Dr Fiona Regan, Miss Sara-Paterson Brown

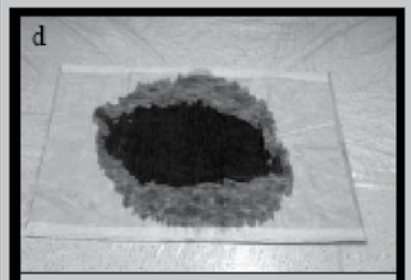
Soiled Sanitary Towel
30ml



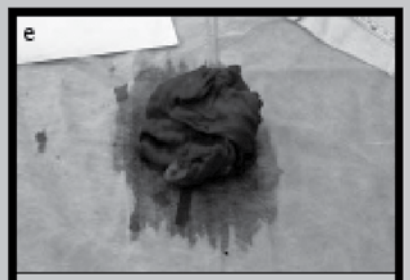
Soaked Sanitary Towel
100ml



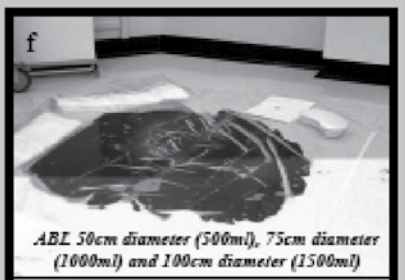
Small Soaked Swab 10x10cm
60ml



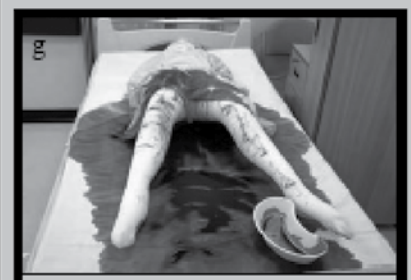
Incontinence Pad
250ml



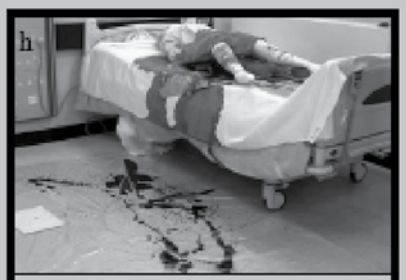
Large Soaked Swab 45x45cm
350ml*



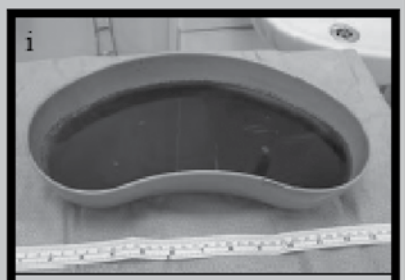
100cm Diameter Floor Spill
1500ml*



PPH on Bed only
1000ml



PPH Spilling to Floor
2000ml



Full Kidney Dish
500ml

***Multidisciplinary observations of estimated blood loss revealed that scenarios (e-f) are grossly underestimated (> 30%)**

For Further Information please contact Miss Sara Paterson-Brown
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Adminstration of magnesium sulphate ($MgSO_4$) for eclampsia

Combined intramuscular (IM) and intravenous (IV) regimen

Loading dose:

- ❑ Add 8ml 50% $MgSO_4$ **(4g)** in 100ml 0.9% saline or 5% glucose

  → ***administer IV over 20min***

OR, if you have syringe-driver pump

- ❑ Add 8ml 50% $MgSO_4$ **(4g)** to 12ml 0.9% saline or 5% glucose and

  → ***infuse IV over 20min at 60ml.h^{-1}***

AND

- ❑ Give **2.5g** $MgSO_4$ IM into each buttock

(Total initial dose 4g IV + 2 x 2.5g IM = 9g)

If the convulsions do not stop:

Administer a further 2g $MgSO_4$

- ❑ Draw 4ml (2g) of 50% $MgSO_4$ into 10ml syringe and add 6ml 0.9% saline or 5% glucose

  → ***inject over 2min (5ml.min^{-1})***

Do not exceed 9g total IV dose of $MgSO_4$ during the first hour

- ❑ If convulsions still continue, consult medical staff and consider diazepam 5mg or lorazepam 1mg (IV or IM)
- ❑ Be aware of risk of respiratory depression

Maintenance

- ❑ 2.5 $MgSO_4$ IM 4 hourly using alternate buttocks if there are no signs of $MgSO_4$ overdose
- ❑ Check reflexes before giving $MgSO_4$
- ❑ Continue for 24 hours after the last convulsion or delivery

Intravenous (IV) regimen

Loading dose:

- ❑ Fill a paediatric infusion burette set with 22ml 5% glucose
- ❑ Add 8ml 50% $MgSO_4$ (4g)

  → ***administer at 60ml.h^{-1} - the total will run over 30min***

If the convulsions do not stop:

As above for combined IV/IM regime

Maintenance

- ❑ Fill a paediatric infusion burette with 112ml 5% glucose
- ❑ Add 8ml 50% $MgSO_4$ (4g)

  → ***administer at 30ml.h^{-1}- the total will run over 4 hours (1g.h^{-1})***

- ❑ Repeat the same management every 4hr for at least 24 hours after the last convulsion or delivery

For recurrent siezures

- ❑ Administer a second loading dose or increase the infusion to 1.5 or 2g.h^{-1}

Adverse effects of $MgSO_4$

- ❑ hypotension, arrhythmias
- ❑ respiratory depression
- ❑ flushing, nausea/vomiting
- ❑ drowsiness, slurred speech, double vision

Figure 1. *Available to download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

Management of severe pre-eclampsia and eclampsia

Juliet Hull* and Matt Rucklidge
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Summary

Magnesium sulphate administration is indicated to treat eclamptic seizures and prevent seizures in women with severe pre-eclampsia.

Pharmacological strategies for control of blood pressure are described in detail.

Intravenous regimes for magnesium administration are described with alternative intramuscular regimes for settings where infusion pumps are not available.

Multidisciplinary input is essential.

INTRODUCTION

Administration of magnesium sulphate is one of the prime treatment modalities in eclampsia and severe pre-eclampsia. Figure 1 is a guideline for administration of magnesium sulphate to treat severe pre-eclampsia and eclampsia. The guideline has been drawn up with reference to the guidance of the Royal College of Obstetricians and Gynaecologists,[1] with additional guidance for those working in settings where infusion pumps are unavailable and intramuscular administration of magnesium sulphate is more practical.[2] Use of magnesium is described, along with other modes of treatment, in the context of a case history.

DEFINITIONS

Pre-eclampsia is the most likely diagnosis. Pre-eclampsia is a multisystem disorder which occurs after 20 weeks of pregnancy with variable features, severity and rates of progression. There are a number of definitions of hypertension in pregnancy which lack consistency and can be confusing.

In essence, high blood pressure in pregnancy can be:

- Pre-existing hypertension
- Pregnancy induced hypertension
- Pre-eclampsia.

Most definitions of hypertension in pregnancy are based on a diastolic BP >90mmHg on two occasions or diastolic >110mmHg on one occasion. Failure to record and treat systolic hypertension in women with severe pre-eclampsia was highlighted as a common problem in the most recent Confidential Enquiry into Maternal and Child Health (CEMACH). Treatment was recommended if the systolic BP was >160mmHg, on two consecutive readings at least 4 hours apart.

ASSESSMENT

Table 1. *Symptoms and signs of pre-eclampsia*

Symptoms	
• Headache	
• Visual disturbance	
• Epigastric pain / right upper quadrant pain	
• Nausea / vomiting	
• Increasing swelling of legs, fingers, face.	
Signs	
Cardiovascular system	Hypertension, vasoconstriction leading to cool peripheries, peripheral oedema
Respiratory system	Pulmonary oedema, facial and laryngeal oedema, acute respiratory distress syndrome (ARDS)
Renal system	Proteinuria, oliguria, acute renal failure
Central nervous system	Hyperreflexia, clonus, cerebral haemorrhage, convulsions (eclampsia), papilloedema, coma
Others	HELLP (Haemolysis, Elevated Liver Enzymes and Low Platelets), thrombocytopenia, DIC (disseminated intravascular coagulopathy)
Foetal signs	CardioTocoGraphy (CTG) abnormalities, pre-term labour, and intrauterine growth retardation.

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Some definitions of hypertension rely on a rise in BP, rather than an absolute value, e.g. a rise in systolic BP of 30mmHg, or diastolic BP of 25mmHg above the earliest BP taken in pregnancy. It is essential to monitor blood pressure closely throughout pregnancy and to identify other signs and symptoms suggestive of pre-eclampsia. The distinction between pregnancy induced hypertension and pre-eclampsia is important since pre-eclampsia is associated with worse outcomes. Pregnancy induced hypertension, like pre-eclampsia, occurs in the second half of pregnancy, but without proteinuria or other signs of pre-eclampsia.

CASE HISTORY

A 28 year old woman in her first pregnancy is admitted to the labour ward at 38 weeks of gestation. She has no past medical history of significance. Her blood pressure when pregnancy was first confirmed at 8 weeks was 120/70. Today she presents with a mild frontal headache and increasing swelling of her ankles. Blood pressure is 170/120, urine dip stick testing shows 3+ of protein and there is oedema of both ankles to the mid-calf.

- ***What is the most likely reason for these clinical signs?***
- ***What other symptoms and signs should be looked for?***
- ***What investigations should be performed?***

Severe pre-eclampsia[1]

Clinical features of severe pre-eclampsia (in addition to hypertension and proteinuria) are:

- symptoms of severe headache
- visual disturbance
- signs of clonus
- papilloedema
- epigastric pain and/or vomiting
- abnormal liver enzymes (ALT or AST rising to above 70IU.l^{-1})
- liver tenderness
- HELLP syndrome
- platelet count falling to below 100x10^{6}.l^{-1}

INVESTIGATIONS

Proteinuria demonstrated by urine dipstick and 24hr collection of urine:

1+	=	0.3g.l^{-1}
2+	=	1g.l^{-1}
3+	=	3g.l^{-1}

Urine

More recently, urine protein: creatinine ratio (PCR): a PCR >30 is significant. Assessment of urine output is important.

Blood tests

Consider full blood count, urea and electrolytes, clotting, uric acid, liver function tests, magnesium, serum calcium, group and save.

Cardiotocogram

Regular assessment of fetal well-being using CTG, ultrasound scan to assess fetal growth and uterine artery Doppler blood flow to assess placental blood flow.

CASE HISTORY

The aims of management at this stage are:

- ***confirmation of diagnosis,***
- ***control of blood pressure,***
- ***prevention of convulsions and***
- ***a decision regarding timely delivery.***

The patient is reviewed by a junior obstetrician and moved into a room with equipment for cardiovascular monitoring. A 14G intravenous cannula is inserted and the above bloods are sent to the laboratory. Continuous CTG monitoring is commenced. She complains that her headache is worsening and she is seeing flashing lights. She is found to be hyper-reflexic and she is given 200mg oral labetalol. At this stage her BP is being monitored every 15mins and despite the oral antihypertensive her BP remains high at 170/120 two hours later.

This woman has severe pre-eclampsia which is a serious threat to the lives of both mother and foetus.

She is reviewed by a senior obstetrician and anaesthetist who decides to commence an IV antihypertensive agent.

What IV antihypertensive drugs are suitable in this case?

MANAGEMENT OF PRE-ECLAMPSIA

Labetalol

Control of acute hypertension in pre-eclampsia may be achieved by:

- Labetalol bolus – 25mg IV bolus (5ml of 5mg.ml^{-1} neat solution) over at least 1 minute
- Repeat above at 15 minute intervals to a maximum dose of 200mg until blood pressure is controlled and then start infusion:
- Labetalol maintenance infusion – dilute 200mg (40ml of 5mg.ml^{-1} neat labetalol with 10ml 0.9% sodium chloride gives a final concentration of 4mg.ml^{-1})
- Commence infusion at 5ml.hr^{-1} (20mg.hr^{-1})
- Double infusion rate every 30 min to maximum 40ml.hr^{-1} (160mg.hr^{-1})
- Titrate to keep diastolic between 90 – 100mmHg

Labetalol should be avoided in women with asthma

If the woman remains hypertensive on maximum rate or labetalol is contraindicated or causing side effects, add / replace with a hydralazine infusion.

Hydralazine bolus

- Dilute 40mg hydralazine in 40ml 0.9% saline to give a concentration of 1mg.ml^{-1}
- Give 5ml (5mg) slowly (e.g. over 15 minutes using an infusion pump set at 20ml.hr^{-1})
- Check BP. After 20 minutes, if diastolic >100mmHg give further 5ml (5mg) over 15 minutes (pump rate 20ml.hr^{-1})
- When diastolic 90–100mmHg commence maintenance infusion.

Hydralazine infusion

- Using same concentration as above set pump rate to 5ml.hr^{-1} (5mg.hr^{-1})
- Titrate to keep diastolic 90-100mmHg and systolic 140 - 150mmHg
- Usual maintenance dose is 2-3ml.hr^{-1} (2-3mg.hr^{-1})
- Maximum dose 18ml.hr^{-1} (18mg.hr^{-1})
- Reduce if significant side effects (see below) or maternal tachycardia >130bpm.

Hydralazine causes headache, tremor, nausea and tachycardia and may be less well tolerated than labetalol

A fluid bolus of 250ml should be considered before commencing IV antihypertensive therapy as there is some evidence that this may avoid the hypotension observed with initiation of vasodilator therapy.

CASE HISTORY

Given the severity of this woman's symptoms, measures to control BP with an IV agent should have been considered earlier in this case.

A bolus dose of labetalol is given, followed by a continuous infusion of labetalol and her BP starts to stabilise and her diastolic falls to 90mmHg. A litre of Hartmann's solution (Ringers lactate) is commenced at a rate of 85ml/hr, aiming for urine output of >100ml in 4 hours (excessive fluid administration is harmful in severe pre-eclampsia and fluids should be restricted).

Observations of BP, oxygen saturations, heart rate and respiratory rate are performed every 15 minutes along with continuous CTG monitoring. While considering a plan for delivery she has a grand mal fit.

What is the most likely reason for the fit?

How should it be managed?

The fit is most likely to be an eclamptic seizure. Other causes of a fit in this situation include:

- Epilepsy
- Intracranial event (e.g. subarachnoid haemorrhage, cerebrovascular accident)
- Vaso-vagal (may be caused by rapid fall in BP due to treatment)
- Hypoglycaemia.

MANAGEMENT OF AN ECLAMPTIC SEIZURE

- The patient should be turned to the left lateral position
- Call for help
- Assess and support **A**irway, **B**reathing and **C**irculation
- High flow oxygen by face mask
- Obtain IV access
- **Treat with IV magnesium sulphate (see Figure 1)**
- Monitor ECG, BP, respiratory rate and oxygen saturations
- Check blood sugar

It is beneficial for obstetric departments to set up an "eclamptic box" for use in this kind of emergency. Within the box is stored: magnesium sulphate, normal saline, syringes, needles and instructions for the correct dose and administration. This will improve the timely administration of magnesium and reduce errors in dosing and administration.

Would earlier administration of magnesium have reduced the risk of fitting in this particular case?

The drug of choice to manage eclampsia and reduce subsequent fits is magnesium sulphate. The use of magnesium to prevent seizures in women with pre-eclampsia is less clear. Guidelines on the management of pre-eclampsia by the Royal College of Obstetricians and Gynaecologists (RCOG) in 2006, recommended prophylactic magnesium sulphate should be considered for women with severe pre-eclampsia for whom there is concern about the risk of eclampsia.[1] This is based on the Magpie Trial.[3] This study showed that magnesium sulphate given to women with pre-eclampsia reduced the risk of an eclamptic seizure by around 58%. It should be noted however that not all women with pre-eclampsia will progress to eclampsia; only 1–2% of women in the UK with pre-eclampsia will fit in the absence of anticonvulsant treatment. The Magpie trial calculated that the number of pre-eclamptic women needed to be treated with magnesium to prevent one of them from fitting is 91 (i.e. of 91 pre-eclamptics receiving magnesium, 1 would gain benefit by not fitting while 90 will derive no benefit from its administration and may be at risk of its side effects.)

CASE HISTORY

A bolus dose of magnesium is administered and she is commenced on an infusion of magnesium at a rate of 1g.h^{-1}. Her respiratory rate and reflexes are checked regularly. She is nursed in the left lateral position.

The CTG now demonstrates repetitive and severe fetal heart rate decelerations.

She is reviewed by the senior obstetrician and a decision is made for an emergency delivery by Caesarean section (category 1). The anaesthetist attends to make a plan for anaesthesia.

What is the most appropriate anaesthetic technique? GA or regional?

Women with severe pre-eclampsia should be encouraged to have regional anaesthesia for caesarean section.

Regional anaesthesia is not contraindicated after an eclamptic fit if the mother has regained consciousness and treatment for seizures and BP control has been commenced. If an epidural has already been in place for labour, this can be topped up as long as it has been effective. If there is no epidural (as in this case) or it is considered that there is not enough time to top up the epidural, spinal anaesthesia should be provided.

The benefits of spinal anaesthesia include provision of a rapid, dense and predictable block suitable for surgery while avoiding general anaesthesia which has the risk of BP surges due to the pressor response of laryngoscopy, intubation and extubation.

There have been concerns with spinal anaesthesia and severe pre-eclampsia due to the fear of causing a sudden and significant drop in BP. This fear appears unfounded because women with pre-eclampsia have high levels of circulating catecholamines which may protect them against a fall in BP as a result of a spinal induced sympathetic block.[4] If a spinal technique is chosen there should be cautious use of vasopressors as an exaggerated hypertensive response to vasopressors may be observed.

Sometimes a regional block may be contraindicated, for example: maternal refusal, coagulopathy, thrombocytopenia and poorly controlled seizures or there is no time because of severe foetal distress. In these cases general anaesthesia will have to be undertaken.

Caesarean section under general anaesthesia in severe pre-eclampsia is a high risk procedure.

The factors which make general anaesthesia in pre-eclampsia particularly hazardous include:

- the increased risk of difficult airway and intubation
- marked pressor response at laryngoscopy, intubation and extubation resulting in dangerous surges in blood pressure

There is a significant risk of intracranial haemorrhage secondary to uncontrolled severe hypertension at induction of general anaesthesia. Therefore in patients who require general anaesthesia, BP and convulsions should be maximally controlled and ideally, invasive monitoring inserted prior to induction of general anaesthesia. A senior anaesthetist should be present to manage these challenging cases.

CASE HISTORY

The blood tests which were sent earlier are now available and demonstrate a platelet count of $55x10^9.l^{-1}$. She is assessed by the anaesthetist and consented for a general anaesthetic. A thorough assessment of her airway is performed and she denies any history of stridor, voice change or hoarseness. She is transferred to theatre and full monitoring is attached to the patient. An arterial line is inserted prior to induction. She is pre-oxygenated in the left tilted position and a rapid sequence induction is performed with thiopentone 450mg and suxamethonium 100mg. No other drugs are administered prior to laryngoscopy. The trachea is intubated after 45 seconds with a size 7 endotracheal tube at which point her BP surges to 240/140. Anaesthesia is maintained with isoflurane in nitrous oxide and oxygen, she is paralysed with atracurium 40mg and prophylactic antibiotics are given. After delivery of the baby she is given morphine 10mg.

At the end of the caesarean section she is slow to wake up.

How can you obtund the pressor response to laryngoscopy and intubation?

What might be the cause of her slow recovery?

The response to laryngoscopy can be obtunded by the following:

- A short acting opiate bolus (e.g. alfentanil 10-20mcg.kg^{-1} or remifentanil 1mcg.kg^{-1})
- A bolus dose of labetalol 10-20mg IV
- Bolus of magnesium 40mg.kg^{-1} IV
- Bolus of lidocaine 1.5mg.kg^{-1} IV 3-5 min before induction

If opiates are given prior to delivery, inform the neonatal team of the possibility of neonatal respiratory depression.[5]

Laryngoscopy can cause significant surges in blood pressure in severe pre-eclampsia and measures to obtund the pressor response should be undertaken.

There are several reasons why she is slow to wake up and these include:

- Effect of excess anaesthetic agents
- Effect of excess opiates
- Inadequate reversal of neuromuscular block: magnesium potentiates non-depolarising neuromuscular blocking drugs
- Respiratory depression due to magnesium toxicity
- Hypoglycaemia.

The most concerning possibility is that she has experienced an intracranial event due to excessive hypertension during intubation. This should be diagnosed by pupil examination and response and emergency CT scan.

Fortunately, over the next 15 minutes she slowly wakes and makes an uneventful recovery from her general anaesthetic.

What on-going management of her pre-eclampsia should be continued?

After her caesarean section this lady should go to a high dependency area for close observation and blood tests.

It is important she also has:

- Effective postoperative analgesia as this reduces the stress response and consequent hypertension caused by poorly controlled pain; however NSAIDs should be avoided until proteinuria has resolved and there is no evidence of impairment of renal or platelet function.

- Ongoing anti-hypertensive therapy. This should be continued after the delivery as dictated by her blood pressure. Although blood pressure usually falls initially after delivery it may rise again around 24hours postpartum. IV antihypertensive therapy should be converted to oral therapy with further reductions made in a stepwise fashion.
- Close monitoring must continue as she is at risk of further eclamptic seizures. Magnesium therapy should be continued until 24hrs after her delivery (or the last convulsion, whichever is the later).
- Cautious fluid intake. She should continue with a total of 85ml.h^{-1} of fluids (subtracting oral intake from IV prescription). Fluid overload must be avoided and transient rises in plasma urea and creatinine concentrations are acceptable in the short term as a spontaneous diuresis is usual within 1-2 days of delivery.

Most women with severe pre-eclampsia or eclampsia will need inpatient care for 4 days or more following delivery.

SUMMARY

Administration of magnesium sulphate is indicated to treat eclamptic seizures and also in the prevention of seizures in women with severe pre-eclampsia. This treatment should considered in the context of pharmacological control of the woman's blood pressure and provision of safe anaesthesia to allow delivery of the baby.

FURTHER READING

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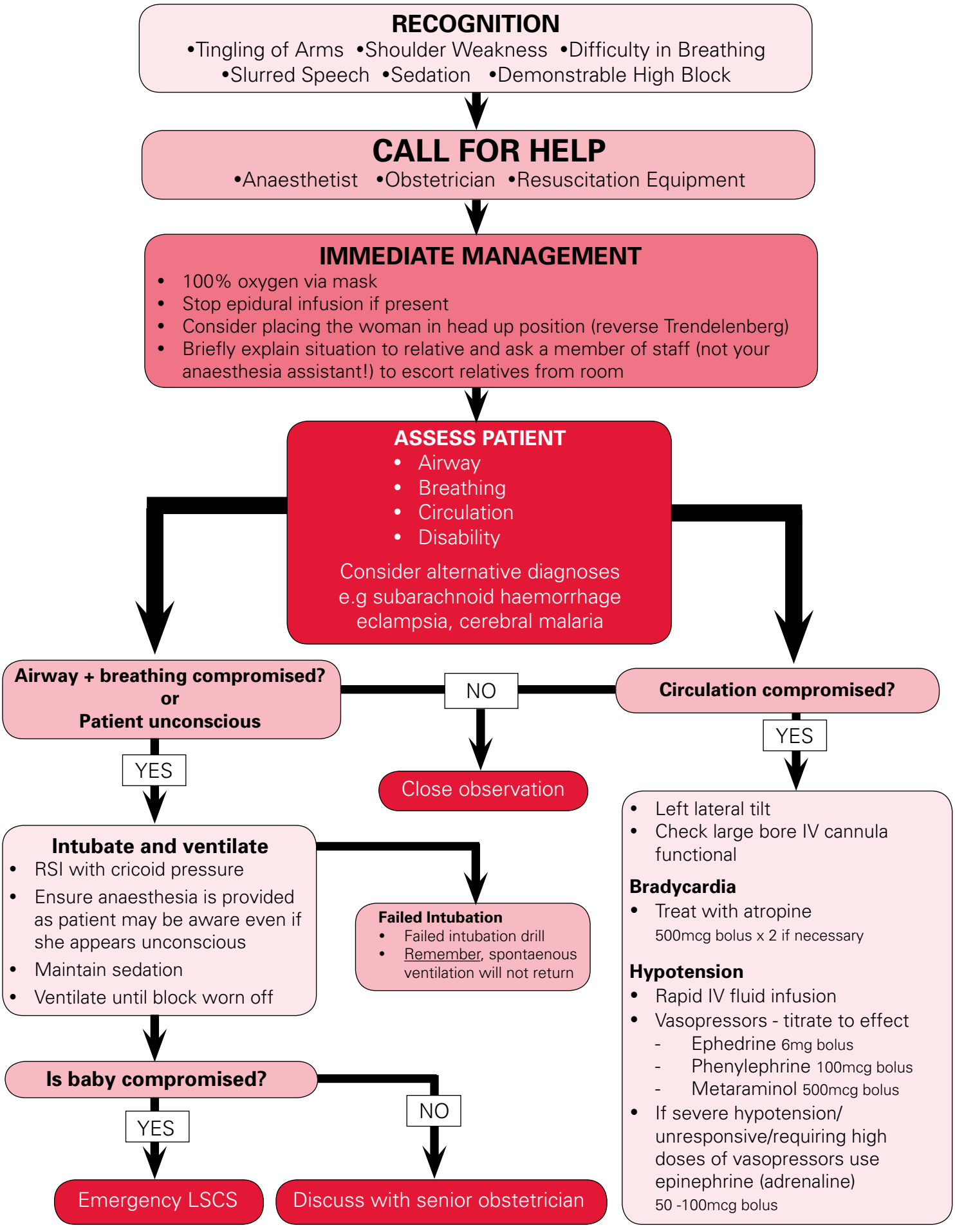
Algorithm for the Management of a High Regional Block in Obstetrics



Adapted from Southampton University Hospital's High Spinal Drill

Figure 1. *Available for download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

Management of high regional block in obstetrics

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Summary

Ensure all staff on the maternity unit are aware of the risk.

Early recognition and treatment will prevent harm to mother and baby.

Communicate with the rest of the team, the patient and partner.

Be prepared – have a plan and practice a drill regularly.

INTRODUCTION

Traditionally, it has been the potentially catastrophic situation of 'can't intubate, can't ventilate' during induction of general anaesthesia that has preoccupied obstetric anaesthetists. The incidence of this was reported to be 1 in 885 in a survey of 60,000 anaesthetics for caesarean section in 1997,[2] and the Confidential Enquiries into Maternal Deaths in the United Kingdom have highlighted mortality due to failed intubation. This has lead to an emphasis on algorithm-based training for this specific scenario. However, in our endeavours to be prepared for failed intubation, have we neglected another potentially catastrophic event – the high regional block?[3]

The reported incidence of high regional block varies considerably, but a large survey in 1997 demonstrated an incidence of 1 in 5334 for elective cases whilst for emergency cases the incidence reported was 1 in 2470 for epidural top ups, and 1 in 3019 for spinal anaesthesia.[1] In 2001, Kar and Jenkins reported an incidence of 1 in 27107 for high regional block following obstetric epidurals.[4] Clearly then, the incidence is much lower than that of failed intubation. However, the majority of obstetric procedures are performed under regional anaesthesia and thus the sheer volume makes it increasingly likely that an anaesthetist will encounter a high regional block. An additional factor is that concerns about achieving adequate anaesthesia (and the litigation consequences of inadequate anaesthesia) have influenced and elevated the planned level of block.

DEFINITIONS

High spinal – spread of local anaesthetic block affecting the spinal nerves above T4. The effects will depend upon the nerves involved.

Total spinal – intracranial spread of local anaesthetic resulting in loss of consciousness.

Both of the above terms refer specifically to blocks from a spinal (subarachnoid / intrathecal) injection of local anaesthetic. However, both of these effects can also be seen from epidural infusions or epidural top ups. For this article the term high regional block will be used, defined as an excessively high block that may require tracheal intubation.

PREVENTION IS BETTER THAN CURE!

So, what should we do? First, endeavour to reduce the incidence of high regional block. All anaesthetists undertaking spinal or epidural anaesthesia should have meticulous pre-procedure preparation to try and prevent a high regional block.

Spinal (subarachnoid) anaesthesia

- Consider the *level required for adequate analgesia/ anaesthesia*. For example the level (and therefore local anaesthetic agent dose) required for removal of a retained placenta in less than that for caesarean section.
- *Local anaesthetic dose* – consider the volume and dose to be used as a number of factors can affect the spread of a block:
- *Patient position* – especially when using hyperbaric or "heavy" solutions of local anaesthetic. If head down position is used to establish a block, it should be reversed as soon as possible.

  Block height can be manipulated with positioning for 20-30mins when using hyperbaric solutions.
- *Patient characteristics* - height, age, weight.
- *Technique* - site of injection, direction of needle, speed of injection, use of barbotage.

Epidural anaesthesia or analgesia

- Use *low concentrations* of local anaesthetic agent for labour analgesia.
- *Assess block* prior to giving a top up.
- Always *aspirate* using a 2ml syringe to check that the catheter is not in the CSF (or a vein). It is important to do this every time you bolus an epidural and not just on insertion.
- *Give a test dose* – ensure the volume you use is adequate to manifest a spinal block if the catheter is intrathecal.
- Consider giving large volumes of local anaesthetic in *divided dose* (weigh up the risk against the benefits, and clinical urgency of establishing a block rapidly)

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Subdural blocks occur when the arachnoid mater is separated from the dura mater (see Figure 2). This can occur with epidural catheters and it is potentially dangerous as a bolus injection may rupture the arachnoid mater converting it into a subarachnoid/intrathecal block. Suspect a subdural catheter if an assumed epidural block spreads high (but slowly), with patchy sensory loss and sacral sparing, and only mild hypotension. The epidural catheter should be removed and a new epidural sited.

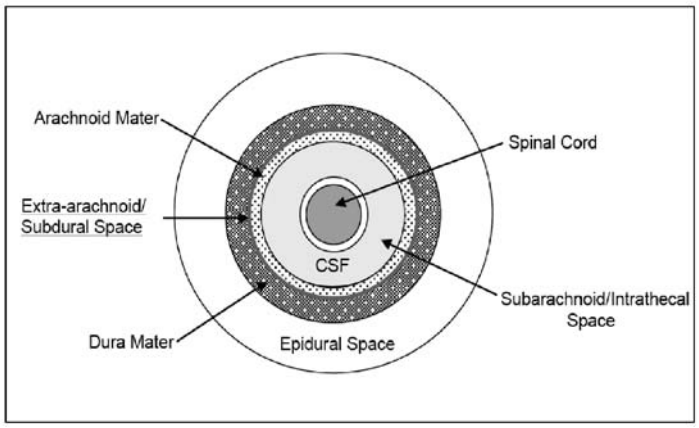

Figure 2. *The anatomy of an extra-arachnoid, subdural block. Adapted from Grady K, Howell C, Cox C. Managing Obstetric Emergencies and Trauma: The MOET Course Manual. 2nd ed. London: RCOG Press. 2007:* **34***; 3225*

EDUCATION

It is important that all staff recognise the potential for a high regional block, since early recognition will allow prompt management of an ascending block and prevent harm to mother and baby. Help should be called immediately, with simple interventions instigated in the meantime.

Anaesthetists should ensure that they are familiar with the recognition and management of a high regional block and should practice a drill regularly. Figure 1 is an algorithm for the diagnosis and management of a high regional block.

COMMENTARY ON ALGORITHM

Recognition and diagnosis

Early recognition is vital and allows appropriate management before either the mother or baby is harmed.

During labour, an epidural bolus, either at initial placement or as a top up that is followed by rapid analgesia that is excessive sensory or motor block and hypotension, should alert to the risk of accidental subarachnoid placement. If the patient complains of shoulder weakness this is a warning sign that diaphragmatic weakness may soon develop.

In theatre, regular checking of the block height and continuous communication with the mother in addition to monitoring of ECG, heart rate and oxygen saturations should help early detection of an ascending block.

The sensory level of a block can be checked by loss of sensation to ice cold (using ice cubes or ethyl chloride spray) or pin-prick. Altered sensation to light touch should also be demonstrated and this is usually one level lower. The clinical effects of spinal or epidural anaesthesia depend upon the spinal roots blocked and knowledge of this allows detection of an ascending block (see Table 1).

A high regional block often develops early and rapidly but it can have a later onset, so remain alert to the possibility. High regional blocks that occur in the postoperative period are particularly hazardous as the staff's attention may be focused elsewhere.

Initial management

With careful monitoring an ascending block can be detected before it has risen to a level causing significant cardiovascular or respiratory compromise. In this situation, placing the patient in the head up/ reverse Trendelenberg position can prevent the block from ascending further.

If a block is rapidly ascending it is important to reassure the partner but ask him to leave - try to give a brief explanation if time and the situation allow. Get a member of staff to escort him but do not send a skilled staff member whose help you will need. It will be a stressful

Table 1. *Clinical effects resulting from ascending neuraxial block*

Root level	System affected	Effects
T1-T4	Cardiac sympathetic fibres blocked	• Bradycardia
		• Can result in severe hypotension as bradycardia compounds hypotension from vasodilation
C6-C8	Hands and arms	• Paraesthesia (tingling) and weakness
		• Accessory muscles of respiration affected
C3-5	Diaphragm and shoulders	• Diaphragmatic innervation – definite respiratory compromise that will require intubation and ventilation
		• Shoulder weakness is a warning sign of impending diaphragmatic compromise
Intracranial spread		• Slurred speech, sedation, loss of consciousness

situation and your concentration needs to be focused on the patient not on reassuring her partner. A full explanation of events can be given at a later time.

Circulatory compromise

A drop in maternal blood pressure of greater than 20% requires immediate action. Severe hypotension will compromise placental blood flow and if not treated will result in maternal cardiac arrest.

Relieve aorto-caval compression with manual uterine displacement or by left lateral tilt – this can be achieved using a wedge or pillow under the right hand side of the patient or by tilting the operating table.

Rapidly infuse one litre of crystalloid, unless otherwise contraindicated, and give vasopressors. Phenylephrine, ephedrine and metaraminol are all acceptable and in very severe or unresponsive maternal hypotension it may be necessary to use epinephrine (adrenaline). There is some evidence that phenyleprine preserves placental blood flow better than other vasopressors in maternal hypotension, but in a crisis use whichever vasopressor you have readily available. Repeated bolus doses should be given and titrated to the blood pressure. If high doses are required an infusion may be needed. See Table 2 for practical information on using vasopressors.

If cardiac arrest occurs follow the advanced life support guidelines discussed on page 62 in this edition of *Update in Anaesthesia*.

Intubation and ventilation

If breathing or conscious level is affected tracheal intubation and ventilation will be necessary. As soon as you identify an ascending block ensure that drugs, an assistant and equipment for a rapid sequence induction (RSI) are prepared and close to hand. Ensure that your assistant knows how to apply cricoid pressure. It is important to administer drugs to provide anaesthesia for RSI, as the patient may be fully aware even if apparently unconscious. Assume that the mother is aware until drugs have been administered. Talk to her calmly and explain what you are doing even if she appears unconscious.

If there is a failed intubation remember that spontaneous ventilation will not return. Proceed to a failed intubation drill for a paralysed patient. Management of a failed intubation in a pregnant woman is described on page 38 of this edition of *Update*.

The patient must be ventilated, by hand if necessary, until the block wears off – usually 1-2 hours for a spinal. This may be much longer if the high regional block is a consequence of intended epidural analgesia/anaesthesia due to the larger doses used. Sedation must be provided and the woman's spontaneous ventilation carefully evaluated to ensure it is adequate prior to extubation.

Consider the baby

Stabilising the mother is in the best interests of the baby but once this is achieved consideration needs to given to whether or not to deliver. If there is foetal compromise then urgent delivery via lower segment caesarean section (LSCS) is appropriate. If there is no foetal distress then a vaginal delivery may still be possible once the block has receded. Ensure a senior obstetrician is involved.

FURTHER POINTS

Debrief

Ensure that all staff members involved have a chance to discuss the case and raise any issues or areas for concern. Consider if the drill worked well or if improvements could be made.

Ensure accurate documentation of events

It may not have been possible to document events as they happened so ensure that you record an accurate reflection of the incident with maternal observations and times of all drugs and interventions undertaken.

Don't forget to explain what happened to both the patient (when she has regained consciousness) and her partner. This will probably

Table 2. *Preparation and use of vasopressors*

Vasopressor	Standard neat concentration (check preparation)	How to dilute	Final concentration	Bolus dose (initial dose, titrate to effect)
Phenylephrine	10mg.ml^{-1}	Take 1 ml and dilute with 0.9% sodium chloride to a total volume of 100mls	100mcg.ml^{-1}	100mcg (1ml)
Ephedrine	30mg.ml^{-1}	Take 1 ml and dilute with 0.9% sodium chloride to a total volume of 10mls	3mg.ml^{-1}	6 – 9mg (2 – 3mls)
Metaraminol	10mg.ml^{-1}	Take 1ml and dilute with 0.9% sodium chloride to a total volume of 20mls	500mcg.ml^{-1}	500mcg (1ml)
Epinephrine 1 in 1 000	1mg.ml^{-1}	Take 1ml and dilute to a total volume of 10mls	100mcg.ml^{-1} (1:10,000 solution)	**UNDILUTED SOLUTION MUST NOT BE GIVEN INTRAVENOUSLY** **DILUTE TO 1:10,000 AND USE AS BELOW**
Epinephrine 1 in 10 000	100mcg.ml^{-1}	Use neat	100mcg.ml^{-1}	50mcg - 100mcg (0.5 -1ml)

have been a distressing and frightening event for them and consider that she may have recall of some events when she appeared to be unconscious.

FINAL COMMENTS

High regional blocks requiring intubation are rare but the volume of obstetric procedures performed under regional anaesthesia is high. It is increasingly likely that it may happen to a patient in your care. Prompt recognition and treatment should prevent mother and baby coming to any harm - have a plan, practice the drill and make sure that all staff are prepared.

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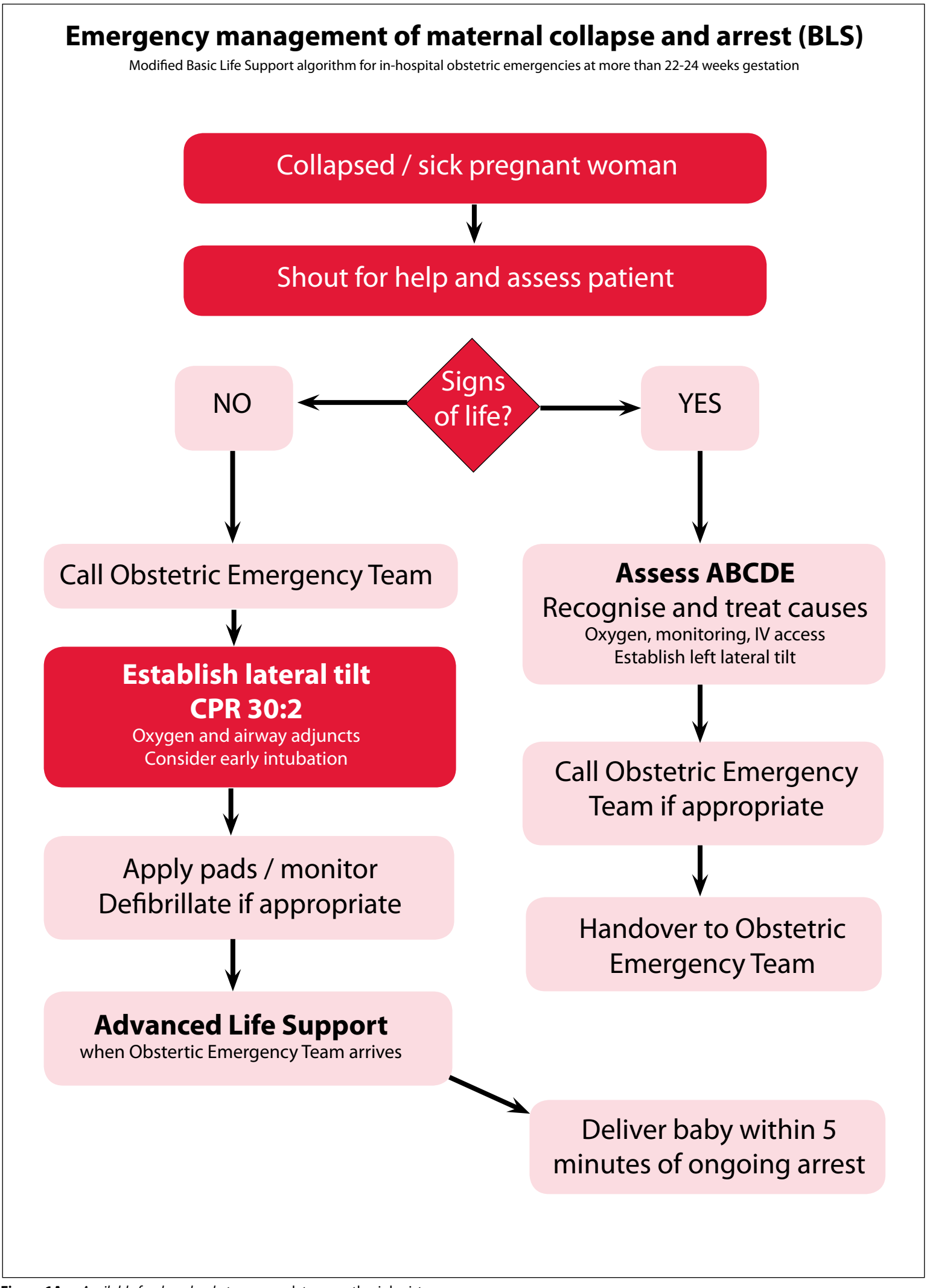

Figure 1A. *Available for download at*: www.update.anaesthesiologists.org

Emergency management of maternal collapse and arrest (ALS)

Modified Advanced Life Support algorithm for in-hospital obstetric emergencies at more than 22-24 weeks gestation

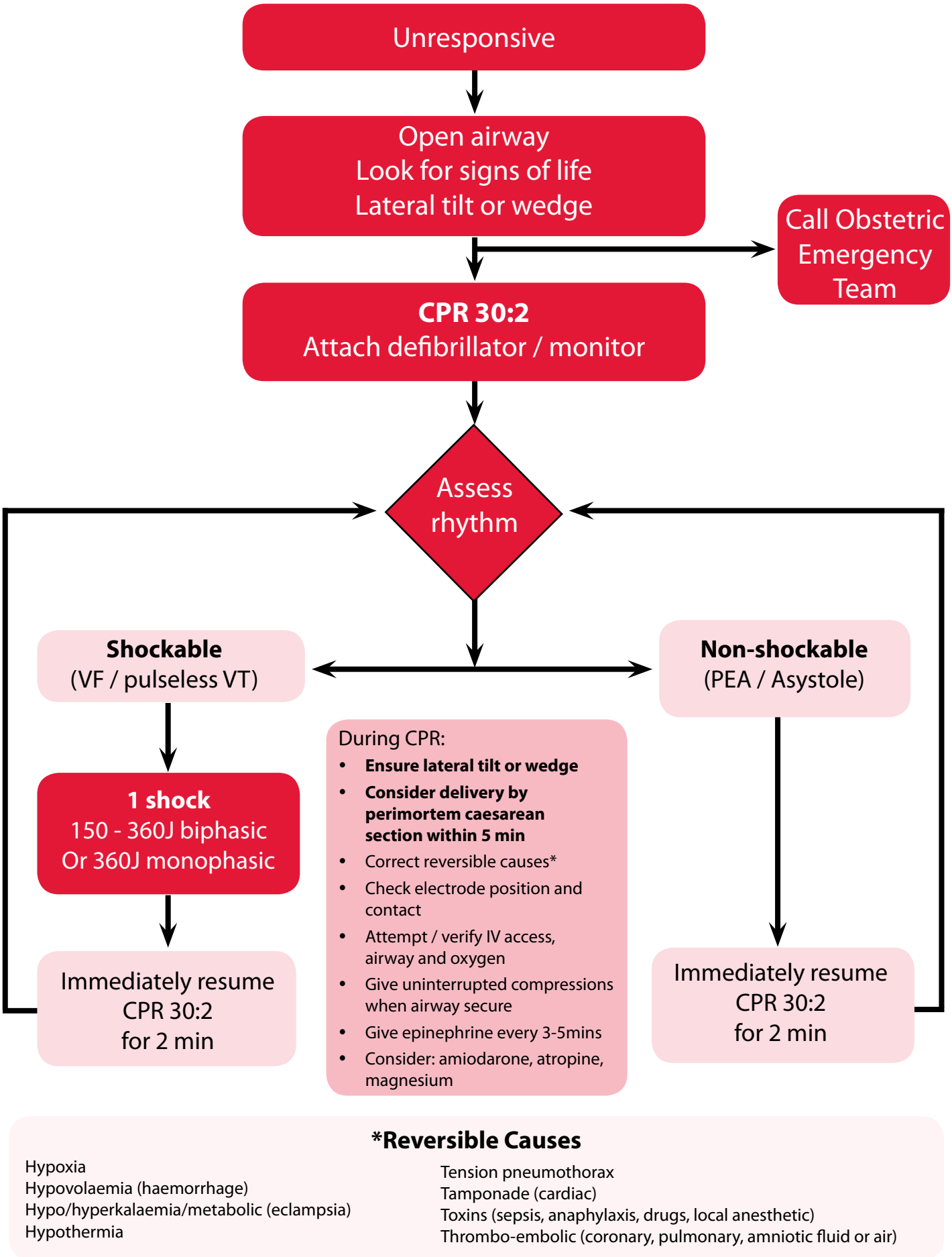

Figure 1B. *Available for download at*: www.update.anaesthesiologists.org

Update in
Anaesthesia

Emergency management of maternal collapse and arrest

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Summary

The cause of maternal collapse and arrest is not always immediately apparent.

A generic approach based on Basic and Advanced Adult Life Support is recommended.

Key modifications to these algorithms are required in pregnancy. These include early intubation and the use of lateral tilt or uterine displacement.

If cardiopulmonary resuscitation is unsuccessful, delivery of the baby by perimortem caesarean section should be accomplished within 5 minutes.

Senior multidisciplinary help should be summoned immediately by defined emergency pathways.

INTRODUCTION

Maternal collapse is a spectrum of clinical presentations from an uncomplicated faint to sudden unexpected cardiac arrest in a term mother.

Around two-thirds of pregnancy-related deaths occur during childbirth or in the immediate postpartum period.[1] The commonest causes of worldwide maternal mortality are shown in Table 1, although it should be noted that there is widespread regional variation.

Less common causes include pulmonary or amniotic fluid embolism, cardiovascular disease, trauma and problems related to anaesthesia. Importantly the reason for the collapse may not initially be obvious, therefore a generic approach to resuscitation may be helpful, and this can be augmented by specific treatments as the diagnosis becomes apparent.

RESUSCITATION DURING PREGNANCY

Prior to 22-24 weeks gestation, resuscitation of a collapsed pregnant woman follows the European Resuscitation Council Basic and Advanced Life Support algorithms (BLS and ALS, see *Update* 22, 2007). After this gestation, resuscitation is complicated by the progressively significant maternal anatomical and physiological changes discussed in this article.

Whilst the algorithms and the ABC (airway, breathing, circulation) approach remain the basis of cardiopulmonary resuscitation, modifications are required in this group. Sample obstetric arrest algorithms are shown in Figures 1 and 2.

COMMENTARY ON ALGORITHMS

A - Airway

Prompt and effective airway management is critical to successful resuscitation. Efforts are directed at early intubation of the trachea, as it protects from aspiration of stomach contents and facilitates effective ventilation of the mother. Tracheal intubation should be considered early in resuscitation, although attempts must not be at the expense of oxygen delivery. In the face of respiratory arrest, simple airway manoeuvres and positive pressure mask ventilation with cricoid pressure should be started until intubation can be achieved. Repeated attempts at intubation may lead to trauma and hypoxia, worsening an already grave situation.

The increased rate of difficult or failed intubation in obstetric patients is multi-factorial. Proposed factors include a reduction in training and expertise due to the increasing use of regional techniques and situational stress. The presence of large breasts, obesity and oedema of the soft tissues and airway may further complicate airway management.

Difficult airway equipment, in a well-organised trolley, should be available in clinical areas and staff should be trained in its use. Gum elastic bougies, alternative laryngoscopes such as the 'polio blade' (Figure 3), intubating laryngeal mask airways (ILMAs) and advanced fibre-optic devices may improve success, but should not delay ventilation by other means. In a 'cannot intubate, cannot ventilate' situation, emergency cricothyroidotomy may be required (see page 39).

Table 1. *Leading global causes of direct maternal death (2000)*[1]

Cause	Number of maternal deaths	% of all direct deaths
Haemorrhage	132 000	28
Infection	79 000	16
Unsafe abortion	69 000	15
Eclampsia / HELLP syndrome	63 000	13
Obstructed labour	42 000	9

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Table 2. *Factors affecting airway management*[1,2]

High reported difficult intubation rate (1:250)
Worsened by obesity and oedema (including larynx)
Increased aspiration risk
Increased intragastric pressure, reduced oesophageal tone and gastric motility delayed

Figure 3. *Polio laryngoscope blade and short laryngoscope handle*

B - Breathing

Table 3. *Factors affecting breathing (ventilatory) management*[3]

Higher ventilatory requirements
Decreased functional residual capacity (FRC) by 10-15%
Increase in basal oxygen requirements by 20-30%
Decreased chest compliance due to raised abdominal pressure

A combination of increased oxygen requirements and reduced ventilatory capacity results in rapid hypoxia once normal breathing ceases. The diaphragm is displaced upwards by the gravid uterus and exacerbates the difficulties in achieving effective positive pressure ventilation. Whilst an endotracheal tube allows high positive pressures to be employed, this may have a further deleterious effect on the cardiac output from chest compressions. This is improved following perimortem caesarean section (see below).

Ventilation should follow Adult Life Support guidelines, with 100% oxygen if available, and become uninterrupted following intubation.

C - Circulation

Both blood volume and basal cardiac output increase dramatically from the first trimester, with around 25% of cardiac output supplying the utero-placental circulation at term. During cardiac arrest, in non-pregnant subjects, closed chest compressions provide up to 30% of normal cardiac output.[1] In pregnancy, the effect of aorto-caval compression by the bulky uterus in the supine position is likely to worsen this considerably. For this reason, it is imperative to mechanically displace the uterus leftwards from the midline to reduce this effect. The ideal full left lateral position is not compatible with cardiopulmonary resuscitation and so a compromise must be reached. Many authorities advocate a tilt of up to 30° by rolling the patient or placing a wedge under the right side. Beyond this, chest compressions are not effective.[1] Alternatively, manual leftwards displacement of the uterus using external pressure can be employed.

Table 4. *Factors affecting circulatory management*[3]

Anatomical
Mediastinum displaced upwards in chest
Aortocaval compression by gravid uterus when supine
Physiological
Increased cardiac output at rest (around 40-50%)
Increased blood volume (up to 60%)

Circulatory life support should generally follow standard guidelines, with large bore IV access, use of epinephrine, atropine, defibrillation as appropriate and identification and treatment of the underlying cause. Exclusion of the four 'H's and the four 'T's in the ALS algorithm may help (Figure 2).

Perimortem caesarean section

It has become clear that cardio-pulmonary resuscitation remains significantly impaired by the gravid uterus after 22-24 weeks gestation, despite the above management. Accordingly, surgical evacuation of the uterus has preceded many successful resuscitation attempts. Therefore immediate delivery by perimortem caesarean section is strongly recommended to begin within 4 minutes of cardiac arrest if no spontaneous circulation has been restored, aiming for delivery within 60 seconds. The indications for this are shown in Table 5. The logistics of this are challenging, although arguably it is unjustifiable to move the patient to an operating theatre prior to the procedure. A simple kit of gloves, scalpel and swabs is potentially life saving and should form part of a readily accessible emergency obstetric trolley.

If unknown, estimation of gestational age should be made clinically by observation and palpation. Intervention should not be delayed for formal uterine or foetal assessment.

Table 5. *Indications for perimortem caesarean section*

- No spontaneous maternal circulation at 4 minutes despite ongoing cardiopulmonary resuscitation
- Estimated gestational age > 22 weeks
- Skilled person available to perform procedure
- Resources to allow post-operative care of mother (and ideally child, although of secondary importance)

Whilst primarily a life-saving procedure for the mother, infants appear to have the best chance of survival when delivered within 5 minutes of maternal arrest (although some reports show survival up to 30 minutes[1] and perimortem caesarean section should still be considered after prolonged resuscitative efforts). The recommendation for performing perimortem caesarean section within 4 minutes of arrest was made by the American Heart Association in 1986. Following this, a review of cases to 2004 suggests that early delivery of the infant in cardiac arrest is associated with much improved outcomes for both mother and child (including neurologically), and certainly does not worsen the situation.[1]

Multidisciplinary team involvement

Effective management of obstetric emergencies relies heavily on the skills and support of several individuals and services (Table 6).

Table 6. *Services involved in effective obstetric emergency plan*

- Obstetricians
- Midwives
- Anaesthetist
- Critical care
- Haematology
- Ancillary (theatre staff, porters etc.)

Adequate planning, preparation and rehearsal of emergency drills are crucial to this process. Many hospitals will have protocols and activation pathways to ensure that these services are rapidly engaged in the event of an emergency. Daily tasks involve checking of equipment, drugs and communication systems. Long term tasks involve training, audit, service development, case review and risk management.

Thorough records should be kept throughout and following the resuscitation, noting times of drugs, decisions, interventions and transfers.

Post resuscitation care

Following successful resuscitation, meticulous attention must be paid to ongoing support and treatment of the mother, ideally in a high dependency or intensive care environment. Less immediate complications of obstetric emergencies, such as myocardial damage from post-partum haemorrhage,[1] renal failure and pulmonary thrombo-embolic disease,[2] may be underestimated contributors to mortality and morbidity.

It is good practice that senior staff members take responsibility for informing the family of key progress and outcomes throughout. Additionally, a team debrief should be carried out whether the resuscitation is successful or not.

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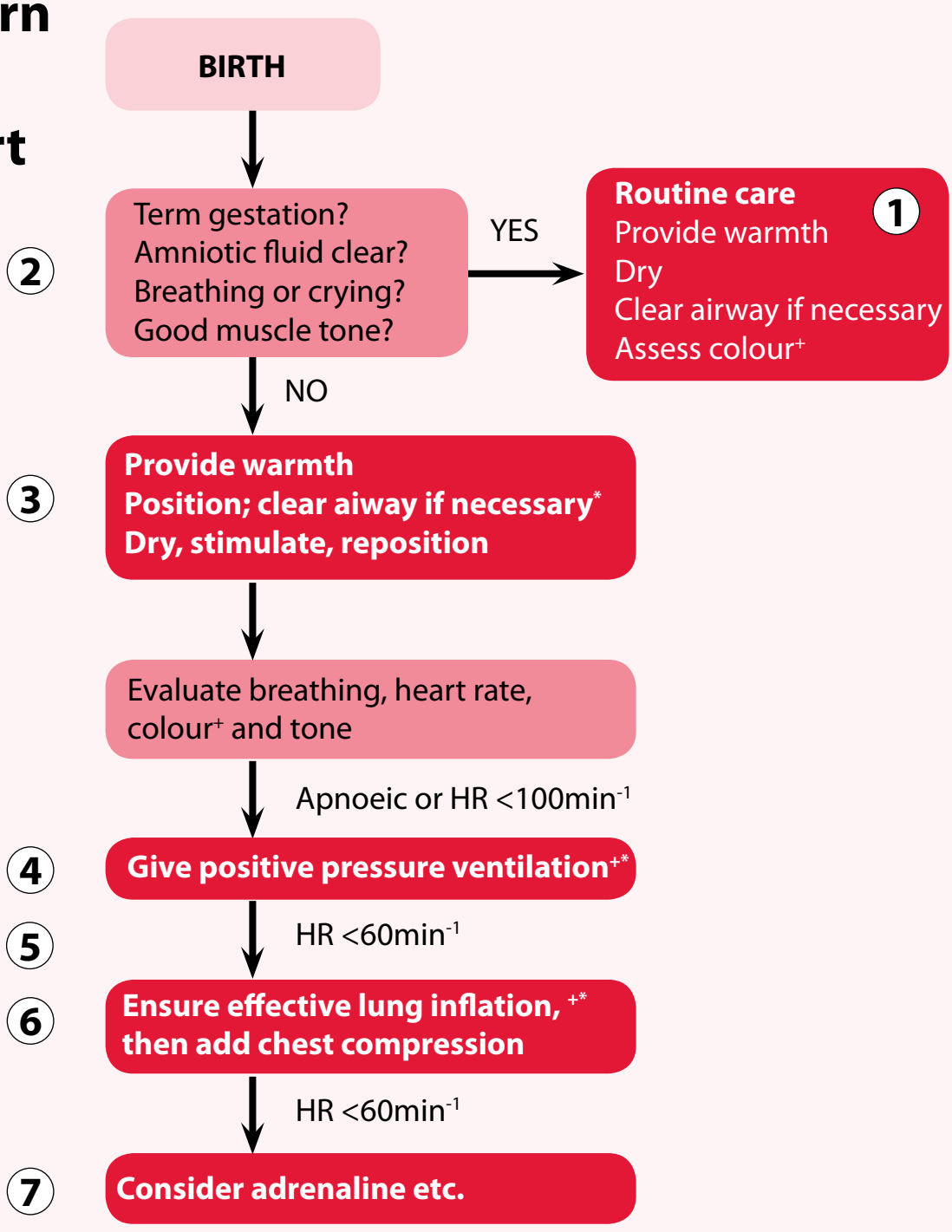
Resuscitation Council (UK)

Newborn Life Support



* Tracheal intubation may be considered at several steps

+ Consider supplemental oxygen at any stage if cyanosis persists

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Figure 1. *Reproduced by kind permission of the European Resuscitation Council and available at*: www.resus.org.uk/pages/nlspost.pdf

Update in
Anaesthesia

Resuscitation at birth

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Summary

A floppy baby is unconscious - a baby with good tone is not.

Good airway management and effective rescue breaths are key to achieving oxygenation of fluid-filled lungs.

Chest compressions and drug administration are rarely needed.

INTRODUCTION

Resuscitation of a newborn infant at birth is straightforward and much more likely to be successful than resuscitation of a collapsed adult. The principles underlying the approach are simple and the issue is not complicated by a need to interpret ECGs or manage arrhythmias. Babies are well adapted to withstand the periods of intermittent hypoxia which are a feature of normal labour and delivery. At term their hearts are packed with glycogen and, by switching to anaerobic respiration, can – in extremis - maintain some circulation for up to about twenty minutes in the face of anoxia. Of those few who get into difficulties, the vast majority will recover rapidly once their lungs have been successfully inflated. However, it is necessary to be aware of some important differences between babies at birth and adults. It is equally necessary to maintain a logical approach, evaluating and completing each step before proceeding to the next.

NEONATES COMPARED TO OLDER CHILDREN

One obvious difference between babies and older children or adults is that babies are small and have a large surface area to weight ratio. They are also always born wet which means they are particularly prone to rapid evaporative heat loss. The initiating insult will virtually always be an interference with placental respiration but the condition that a baby is born in can vary from healthy to extremely sick and all shades between. However, perhaps the most important difference to remember is that a baby at birth is in transition from placental to pulmonary respiration. It will therefore have fluid-filled lungs that have never yet been inflated with gas.

COMMENTARY ON ALGORITHM

Let us now approach the algorithm shown in Figure 1.[1] This algorithm deals primarily with term infants and to some extent this approach can be extended to preterm infants in similar difficulty. The management of transition in significantly preterm infants is beyond the scope of this article even though this process is also often referred to as "resuscitation".

1. Heat loss

The first item addresses the issue of minimising heat loss. The baby should be received into warm towels, rapidly dried, the now wet towels removed, and the baby then covered in warm dry towels and, ideally, placed on a flat surface under a radiant heater. This will take 20 to 30 seconds during which time one can also begin to assess the condition of the baby.

2. Assessment

The baby then needs to be rapidly assessed. A healthy baby will adopt a flexed posture with good tone, will have a normal heart rate which rapidly rises to above 100 beats per minute (bpm), will cry and breathe normally within about 30 seconds of delivery. Although born blue, he will rapidly become pink even though the extremities will remain somewhat cyanosed. An asphyxiated baby will be very floppy with a slow or even absent heart rate, will make no attempt to breathe or may give only a shuddering gasp. He will remain blue or maybe appear very pale due to restriction of blood flow to the skin in an attempt to maintain central circulation. You will certainly need help if the baby is like this.

Of these four attributes the most indicative of a serious problem is tone.

A floppy baby is in serious difficulty, a baby with good tone is not.

A floppy baby with a low heart rate is in serious difficulty whereas a baby with a slow heart rate but good tone is probably OK.

The next most important attribute is heart rate. In a baby in difficulty the heart rate will almost instantly respond as soon as oxygenated blood reaches the heart. This will therefore give you the first sign that your resuscitative efforts are having a positive effect. You therefore need to know what the heart rate is at the start so as to be able to judge whether it has later improved.

A B C D

From here on the algorithm follows a familiar pattern – Airway, Breathing, Circulation and Drugs. However, it is vital that these items are dealt with in sequence.

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Whereas in adult collapse 'compression only' CPR may be effective, the reason for this is that in adults one is usually dealing with a cardiac problem. In babies the problem is a respiratory one and performing chest compressions before inflating the lungs merely attempts to circulate blood through fluid filled lungs where it has no hope of acquiring oxygen. This is a time-consuming distraction.

3. Airway

An unconscious baby placed on its back will tend to obstruct its airway due to loss of tone in the oropharynx and jaw, resulting in the tongue falling back to obstruct the oropharynx. This tendency is exacerbated by the relatively large occiput of the newborn baby which will tend to flex the neck. In order to open the airway of a baby the head is best held in the neutral position with the face supported parallel to surface on which the baby is lying. Over-extension of the neck is likely to obstruct the airway, as is flexion.

Supporting the jaw and, in very floppy babies, providing formal jaw thrust, is sometimes necessary. Given the relatively large size of the newborn baby's tongue compared to size of the mouth an oropharyngeal airway may also be helpful.

Special case - meconium aspiration

Some babies who get into difficulties before delivery may pass meconium in utero. If insulted further, they may inhale this meconium into the oropharynx or airways during episodes of anoxic gasping before birth. Therefore, if a baby is born through heavily meconium stained liquor and if the baby is unresponsive at delivery – and only if unresponsive[2,3] - it is worth inspecting the oropharynx and removing any thick particulate meconium by means of a large bore suction device. If the infant is unresponsive and the operator has the appropriate skill then intubating the larynx and 'hoovering out' the upper trachea by applying suction to the tracheal tube during withdrawal may remove a potential blockage. Attempting to remove meconium or other endotracheal blockages by passing a suction catheter down through the tube itself is unlikely to be successful as the bore of the catheter will be too small for the purpose.

4. Breathing

If the baby has not yet responded then the next step is to ventilate the lungs. Remember the lungs will be fluid filled if the baby has made no attempts to breathe. Apply a well fitting mask to the mouth and nose and then attempt to inflate the lungs with air at a pressure of around 30 cm of water aiming for an inspiratory time of 2 to 3 seconds. Five such 'inflation breaths' will usually be successful in aerating the lung to an extent that will allow any circulation to bring some oxygenated blood back to the heart producing a rapid increase in heart rate.

5. Circulation - re-evaluate heart rate

Having given five inflation breaths you should then assess whether the heart rate has increased. If it has, then this is a firm indication that you have aerated the lung and it also tells you that all that is necessary is for you to gently ventilate the baby until it starts to breathe normally. A rate of 30 or so 'ventilation breaths' per minute each with an inspiratory time of around one second will usually be sufficient to maintain the baby's heart rate above 100 bpm during this period.

However, if the heart rate has not improved you still need to know whether this is because your attempts at lung aeration have not been successful – which is the most likely reason – or have you actually succeeded in aerating the lungs but the circulation has deteriorated to such an extent that this alone is not going to be sufficient. The only way to judge this is to see if you can detect passive chest movement in response to attempts at lung inflation. Is the chest moving when you try to inflate it?

Initial chest movement is likely to be subtle and you may have to stoop down and look carefully from the side during further attempts at inflation to be sure on this point. The commonest error is to assume successful chest inflation when it is not present. It is, however, absolutely crucial that this question is answered correctly. If you assume that you have inflated the lungs when you have not, then proceeding to chest compressions will not have any hope of success and you are merely wasting time. Equally, if you assume you haven't inflated the chest when you have, then you will fail to initiate chest compressions when they are necessary and will also waste precious time. If you have inflated the chest but not recognised this, then the rapidly improving chest compliance will make chest movement easier to see with subsequent breaths, so chest movement should eventually become obvious.

If chest movement is not seen, then the airway is the problem and this must be addressed before going any further. Unless and until the lung is successfully inflated nothing else will have any chance of success. Apart from checking for obvious problems such as failing to switch on the oxygen supply, or a big leak from the mask, check the following issues:

Consider:

- Is the baby's head truly being supported in the neutral position?
- Is jaw thrust necessary?
- Would use of an oropharyngeal airway be helpful?
- Might you achieve better airway control if two people were employed controlling the airway?
- Are you actually delivering an appropriately long inspiratory time?
- Might there be a blockage in the oropharynx or trachea?

Though the presence of meconium on a collapsed baby may give a clue to a blocked airway it is well known that other less obviously visible substances such as blood clots, lumps of vernix or thick mucus plugs can equally be inhaled and block the airway in exactly the same way.[4]

Once chest movement has been achieved – and only then – consider chest compressions if the heart rate remains slow or absent.

6. Chest compressions

If the heart rate has not responded to lung inflation alone, then a brief period of chest compressions may be all that is necessary to bring a little oxygenated blood from the lungs back to the coronary arteries which will then produce a rapid cardiac response. The most effective way to perform chest compressions is with both hands encircling the chest. Place the thumbs together centrally over the lower sternum with the fingers overlying the spine at the back and briskly compress

the chest between fingers and thumbs at a rate of about 120 beats per minute. Current advice is that one should intersperse breaths at a rate of one breath for every three beats during this manoeuvre, though there is no clear evidence as to the most appropriate compression: inflation ratio.

The need to proceed as far as this is relatively rare – probably around 1 in 1000 births. The length of time compressions are needed is also relatively short – a few minutes at most.[5]

Having given 30 to 60 seconds of chest compressions one should look for a response. Once again you are looking for an increase in heart rate indicating successful delivery of oxygenated blood to the heart. Virtually all babies will have responded by this stage. Because this is the expectation it is important to check once again that lung inflation has definitely been successful and that chest compressions are being delivered as expected before deciding that further intervention is needed. However, if the heart remains slow – less than about 60 per minute - or is absent, then further intervention should be considered.

7. Drugs

What else is available? There is very little published evidence to support any of the drugs which have been suggested for use at this stage. Epinephrine (adrenaline) is traditional in these situations and, if given centrally – ideally via an umbilical venous cannula – does improve coronary artery perfusion pressure in animal experiments. Early animal studies also appear to show a possible place for the use of alkalinising agents such as bicarbonate (with dextrose) - again given centrally - in boosting a failing circulation at this point. Intuitively one might also expect that babies who are seriously hypovolaemic, perhaps from blood loss, would respond to appropriate fluid expansion.

If any of these manoeuvres are to be employed then it is necessary to rapidly establish central venous access. This is easily done by inserting a catheter into the umbilical vein.

It must be said, however, that babies who appear to require this degree of help are at very high risk of permanent and severe neurological damage, if they survive. Those with the least risk will be those who have undergone a severe but sudden and recent insult rather than those whose insult has been intermittent and chronic.

SUMMARY

Resuscitation of babies at birth boils down to good airway management and effective lung inflation with the need to add chest compressions on very rare occasions. Air is almost certainly all that is necessary for lung inflation[6] and drugs have a very limited place.

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THE ASSOCIATION OF ANAESTHETISTS
of Great Britain & Ireland

Guidelines for the management of a Malignant Hyperthermia Crisis

Successful treatment of a Malignant Hyperthermia (MH) crisis depends on early diagnosis and aggressive treatment. The onset of a reaction can be within minutes of induction or may be more insidious. Previous uneventful anaesthesia does not exclude MH. The steps below are intended as an *aide memoire*. Presentation may vary and treatment should be modified accordingly. Know where the dantrolene is stored in your theatre. Treatment can be optimised by teamwork.

Call for help

Diagnosis - consider MH if: 1

1. Unexplained, unexpected increase in end-tidal CO_2 together with
2. Unexplained, unexpected tachycardia together with
3. Unexplained, unexpected increased in oxygen consumption

Masseter muscle spasm, and especially more generalised muscle rigidity after suxamethonium, indicate a high risk of MH susceptibility but are usually self-limiting.

Take measures to halt the MH process: 2

1. Remove trigger drugs, turn off vaporisers, use high fresh gas flows (oxygen), use a new, clean non-rebreathing circuit, hyperventilate. Maintain anaesthesia with intravenous agents such as propofol until surgery completed.
2. Dantrolene; give 2-3 $mg.kg^{-1}$ i.v. initially and then 1 $mg.kg^{-1}$ PRN.
3. Use active body cooling but avoid vasoconstriction. Convert active warming devices to active cooling, give cold 3 intravenous infusions, cold peritoneal lavage, extracorporeal heat exchange.

Monitor: 4

ECG, SpO_2, end-tidal CO_2, invasive arterial BP, CVP, core and peripheral temperature, urine output and pH, arterial blood gases, potassium, haematocrit, platelets, clotting indices, creatine kinase (peaks at 12-24h).

Treat the effects of MH: 5

1. Hypoxaemia and acidosis: 100% O_2, hyperventilate, sodium bicarbonate.
2. Hyperkalaemia: sodium bicarbonate, glucose & insulin, i.v. calcium chloride (if *in extremis*).
3. Myoglobinaemia: forced alkaline diuresis (aim for urine output >3 $ml.kg^{-1}.h^{-1}$, urine pH >7.0).
4. Disseminated intravascular coagulation: fresh frozen plasma, cryoprecipitate, platelets.
5. Cardiac arrhythmias: procainamide, magnesium, amiodarone (avoid calcium channel blockers - interaction with dantrolene).

ICU management: 6

1. Continue monitoring and symptomatic treatment.
2. Assess for renal failure and compartment syndrome.
3. Give further dantrolene as necessary (recrudescene can occur for up to 24h).
4. Consider other diagnoses, e.g. sepsis, phaeochromocytoma, myopathy.

Late management: 7

1. Counsel patient and/or family regarding implications of MH.
2. Refer patient to MH Unit.

This poster is produced by the AAGBI and is endorsed by the British MH Association.

Figure 1. *Reproduced by kind permission of the Association of Anaesthetists of Great Britain and Ireland and available for download at:* www.aagbi.org/publications/guidelines/docs/malignanthyp07amended.pdf

Guidelines for management of a malignant hyperthermia (MH) crisis

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Summary

Malignant hyperthermia (also termed as malignant hyperpyrexia) is a life threatening emergency.

More appropriately it is a "malignant hypermetabolic" disorder.

Increase in temperature is a hallmark of MH but it may be a late sign.

The operating room area and recovery area should be equipped with appropriate resuscitative measures and equipment.

Practice drills and simulation training are strongly recommended due to the rarity of this condition.

The importance of teamwork and communication is paramount to successful management of an MH crisis.

INTRODUCTION

Malignant hyperthermia (MH) is a rare pharmacogenetic autosomal dominant disease. This is generally unmasked when a susceptible individual is exposed to general anaesthesia and it can present during or after delivery of anaesthesia. The common precipitants are volatile anaesthetic agents and succinylcholine (suxamethonium). In these individuals there is increased skeletal muscle oxidative metabolism leading to increased oxygen consumption, increased production of carbon dioxide and increased body temperature. Circulatory collapse and death frequently follow if the condition is not recognised and treated promptly.

EPIDEMIOLOGY

The incidence of MH is 1 in 4500 to 1 in 60000 under general anaesthesia. It occurs worldwide and in all racial groups.

PATHOGENESIS

Sixty to seventy per cent of cases are due to a mutation in ryanodine receptor (RYR1) in the sarcoplasmic reticulum (SR), the site of calcium storage in skeletal muscle cells. In health RYR1 receptors mediate release of calcium from the SR into the muscle cell cytoplasm, causing muscle contration. Defective RYR1 receptors allow exaggerated calcium release and also have a higher threshold for deactivation and muscle relaxation. Other mutations have also been recognised to cause MH.

Various musculoskeletal abnormalities like scoliosis, hernias or strabismus have been stated to be associated with MH susceptibility, but an analysis of over 2500 patients has not supported this. Based on a recent review, the association of MH in patients with dystrophies (Duchenne muscular dystrophy and Becker dystrophy) has been found to be very weak.[1] There is also a very weak link to disorders such multiple sclerosis, myasthenia gravis, and other neuromuscular disorders and enzymopathies.[2]

COMMENTARY ON ALGORITHMS

1 – Clinical features of MH

The clinical features of MH are not specific. Prompt diagnosis depends on knowledge of features and recognising those in a pattern consistent with an evolving MH reaction and exclusion of other differential causes. Increasing end-tidal CO_2 is usually the first sign of MH. Tachycardia, mixed respiratory and metabolic acidosis are present due to the hypermetabolic state.[3] There is an accompanied increase in oxygen consumption. Total body or truncal rigidity could be an isolated presentation. Masseter spasm may be an isolated feature after succinylcholine. Increased temperature is usually a delayed sign.[3]

Masseter spasm

In the absence of a positive family history, susceptibility to MH may be suspected by exaggerated increase in the tension of the jaw muscles. Jaw stiffness after succinylcholine may be present in most individuals and is often more pronounced in children. When the jaw stiffness is prolonged and severe the condition is termed masseter spasm. There are reports showing a relationship between masseter spasm and MH susceptibility.[4] When presented with this situation one should avoid the trigger agents mentioned below and follow the MH treatment guidelines. If the surgical procedure is non-urgent it should be aborted. The patient should be referred for testing and the family should be counselled.

2 - Trigger agents for MH

Table 1 - *Trigger agents unsafe in patients with MH*[7]

Inhaled agents
• Desflurane
• Enflurane
• Halothane
• Isoflurane
• Sevoflurane
• Ether
Depolarising muscle relaxants
• Succinylcholine

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Table 2. *Safe anaesthetic agents for MH patients*[7]

Intravenous anaesthetics
- Etomidate
- Ketamine
- Methohexital
- Pentobarbital
- Propofol
- Thiopental

Benzodiazepines
- Diazepam
- Midazolam
- Lorazepam

Inhaled non-volatile agents
- Nitrous oxide

Narcotics (opioids)
- Alfentanil
- Codeine
- Diamorphine
- Fentanyl
- Hydromorphone
- Meperidine
- Methodone
- Morphine
- Naloxone
- Oxycodone
- Remifentanil
- Sufentanil

Other
- Neostigmine
- Atropine
- Glycopyrrolate
- Ephedrine

Muscle relaxants
- Atracurium
- Cisatracurium
- Mivacurium
- Vecuronium
- Pancuronium
- Rocuronium

Local anaesthetic agents
- Amethocaine
- Bupicavaine
- Lignocaine
- Levobupivacaine
- Ropivacaine
- Prilocaine
- Etidocaine
- Articaine

The breathing circuit should be changed and fresh gas flows increased. Hyperventilate with 100% oxygen at flows of 10l.min^{-1} or more. Consideration should be given to stopping the procedure unless it is an emergency.

3A - Dantrolene

This is the only available specific and effective treatment for MH.

Mechanism of action

Dantrolene is a skeletal muscle relaxant and has been shown, via action on the ryanodine receptor, to inhibit the calcium release channel of the skeletal muscle sarcoplasmic reticulum. This prevents the increase in intracellular calcium concentration. The molecular mechanism of action is unclear.

Presentation and reconstitution

Pharmacologically it is a hydantoin derivative, highly lipophilic and poorly soluble in water – it is therefore advisable to dedicate one to two members of staff with the specific role of preparing the dantrolene. It is available as 20mg lyophilized dantrolene sodium added to 3grams of mannitol to improve water solubility. The contents of the vials have to be dissolved in 60ml of water. Prewarming the water (<39°C) may improve the solubility of dantrolene. This gives a final concentration of 0.33 mg.ml^{-1} with a pH of 9.5. The prepared solution should be protected from light and stored at 15-25°C. Once reconstituted it should be used with 6 hours.

Mode of administration

The alkaline nature of the solution makes it irritant to veins and it should be injected through a large vein or a fast flowing infusion. Before administering the solution should be clear and devoid of any particles. Consider central venous access once the initial crisis is under control. A lot of help is needed to reconstitute dantrolene in a crisis. Call for help as soon as possible.

Pharmacokinetics and pharmacodynamics

Dantrolene is metabolized by liver microsomes to an active metabolite and excreted via urine and bile. The mannitol present in the formulation causes an osmotic diuresis and fluid shifts. A urinary catheter is usually necessary and helps monitoring of output and fluid balance. Watch carefully for rhabdomyolysis and renal failure.

Other indications for dantrolene

Dantrolene is also used in the management of neuroleptic malignant syndrome (NMS), spasticity, and ecstasy intoxication.

In patients with high risk for MH, prophylaxis with dantrolene is no longer recommended because oral therapy does not guarantee reliable plasma concentrations.

Side effects

These include muscle weakness, phlebitis, respiratory failure and gastrointestinal discomfort. It may prolong the duration of neuromuscular blockade and ventilatory support post-crisis is frequently needed.

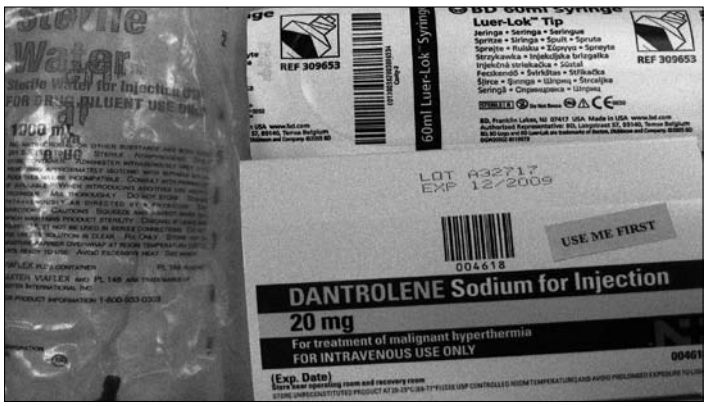

Figure 2. *Dantrolene*

3B - Active cooling

Commence active cooling of the patient if their core temperature is greater than 39°C. Consider cold intravenous fluids and lavage of the stomach, bladder or rectum, and body cavities that are open for surgery. Stop cooling once the core temperature is below 38°C to prevent a fall to less than 36°C.

4 - Monitoring

Monitoring during and after the MH crisis is very important. Core temperature, ECG (electrocardiogram), blood pressure (continuous and invasive if available), capnography, urine output, and observation of urine colour (for myoglobinuria) are essential. Blood gas analysis is useful, where available, to identify acidosis and initiate treatment with bicarbonate and hyperventilation to compensate for hypercapnia. Where available monitor the creatine kinase (CK) level during the crisis and every 6 hours for 36 hours.

5 – Treat the effects of MH

CK levels of more than 10,000IU.l^{-1} indicate significant rhabdomyolysis (skeletal muscle breakdown) and myoglobinuria and you can predict that acute kidney injury (AKI) is likely. AKI may be prevented or limited by aggressive hydration aiming to maintain a urine output of greater than 3ml.kg^{-1}.h^{-1}. Alkalinization of the urine with a bicarbonate infusion may improve the solubility of myoglobin and is advocated in the algorithm, aiming to achieve a urine pH of greater 7.0. Watch for hyperkalaemia.

Management of hyperkalaemia

Treat hyperkalaemia if the blood concentration is >5.5mmol.l^{-1} or there are ECG changes.

Treatment of hyperkalaemia involves:

- Infusion of glucose and insulin - 10 units of short-acting insulin (e.g. Actrapid) in 50ml 50% dextrose given over 30 minutes (monitor blood sugar),
- Calcium chloride or calcium gluconate 10ml of the 10% solution injected intravenously over 10 minutes,
- 1-2mEq.kg^{-1}sodium bicarbonate IV – (an 8.4% solution of sodium bicarbonate contains one mEq per ml),
- A β-agonist such as nebulised salbutamol (2.5-5mg).

Avoid calcium channel blockers, which may increase potassium levels or cause cardiac arrest in the presence of dantrolene.

6 – ICU and late management

After the initial management of the crisis, continuing intensive care is very important. Watch for signs for disseminated intravascular coagulation (DIC) and compartment syndrome. Supportive and symptomatic treatment is essential. Dantrolene treatment may be required for up to 24 hours post crisis (1mg.kg^{-1} every 4-6 hours). Consider differential diagnoses.

Neuroleptic malignant syndrome (NMS)

MH may been confused with NMS. The clinical features include the triad of pyrexia, rigidity and raised CK levels (indicating rhabdomyolysis). The presentation is usually over 24-72 hours and is the result of the central antidopaminergic effects of major tranquillizer drugs. The most important clue to diagnosis is a careful patient history with particular emphasis on the medications being taken around the time of presentation. Most often it follows administration of neuroleptic drugs. It is postulated that dopamine D_2 receptor antagonism by the neuroleptic drugs either block the heat loss pathway or produce heat due to extrapyramidal rigidity.

Table 3. *Differential diagnosis of suspected MH*[3]

Neuroleptic malignant syndrome
Inadequate anaesthesia and analgesia
Inappropriate breathing circuit, fresh gas flow or ventilation
Infection or sepsis
Tourniquet ischaemia
Anaphylaxis
Phaeochromocytoma
Thyroid storm
Heat stroke
Other muscle disease

Treatment involves stopping the neuroleptic drug and is predominantly supportive and intensive care therapy. Dantrolene and dopamine agonists such as bromocriptine and amantidine, may be beneficial. Dantrolene is used intravenously in the same dose used for MH.[3]

7 - Follow up and MH testing

An alert bracelet or information regarding this condition should always be carried by the susceptible patients. The patient and their family should be referred to an MH unit for testing and biopsy.

ANAESTHESIA FOR MH SUSCEPTIBLE PATIENTS

- Consider alternatives to general anaesthesia.
- Schedule these patients as the first case on the operating list.
- Vaporizers should be removed from the anaesthesia machines.
- Breathing circuits should be new.
- The anaesthesia machine should be flushed at 10l.min^{-1} oxygen for at least 20 minutes. In addition the circuit may be used to ventilate a bag until there is no volatile agent is detected in the circuit.
- Avoid potential trigger agents for MH.
- MH crisis resuscitation drugs should be readily available in the vicinity of the operating room area.

MANAGEMENT OF MH SUSCEPTIBLE PARTURIENT OR A POTENTIAL MH SUSCEPTIBLE FETUS[6]

- Review history of the both parents to ascertain the risk for MH.
- **The mother should be treated as MH susceptible until delivery of the fetus.**
- The anaesthesia provider should be notified immediately when the patient arrives in the delivery unit.

- Epidural/spinal anaesthesia is strongly recommended if caesarean section or operative intervention is needed.
- If regional anaesthesia is contraindicated or general anaesthesia is indicated, non-trigger anaesthesia should be administered.
- Suxamethonium should be avoided and alternative muscle relaxants should be considered. Careful airway assessment should be performed. With the potential risk for aspiration, modified rapid sequence induction with non-depolarizing relaxant should be considered. Rocuronium in dose of $1mg.kg^{-1}$ can provide good intubating conditions within 60 seconds. If the facilities for awake intubation are available, it should be considered if difficult airway management is anticipated.
- Nitrous oxide in the form of Entonox may be used for labour analgesia.

POSTOPERATIVE MONITORING OF MH SUSCEPTIBLE PATIENTS

The presentation of MH varies in onset and course. It can be evident within ten minutes of administration of a trigger agent but may present up to several hours later. Care should be taken for monitoring these patients carefully in the recovery area. Studies advise that between three to six hours is safe.[5] MH susceptible patients can be safely managed as day case patients.

MH TROLLEY

A dedicated MH trolley or box is worth considering and this must be restocked on a regular basis. A careful log of stocking and restocking should be maintained. There should be a dedicated person or group of people responsible for maintaining the contents of the trolley. This should contain adequate dantrolene, sterile water for mixing, sodium bicarbonate, glucose, insulin, calcium chloride, mannitol and temperature probes.

A flow chart diagram in laminated printed format should be available in the operating room environment for easy access and reference to follow treatment guidelines.

SUMMARY

- Adequate knowledge and a guideline-based approach is key to successful outcomes following unanticipated MH crisis.
- Management of known MH susceptible patient involves avoidance of trigger agents and adequate preparation. Consideration should be given to regional/local anaesthesia if feasible.
- MH susceptible patients should be observed in the recovery area for three hours.[5]
- The operating room and recovery area should have dantrolene readily available and this must be restocked on a regular basis.
- Physicians and nurses should know where it is stored.

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AAGBI Safety Guidelines

Management of Severe Local Anaesthetic Toxicity

1 Recognition

Signs of severe toxicity:

- Sudden alteration in mental state, severe agitation or loss of consciousness, with or without tonic-clonic convulsions
- Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias may occur
- Local anaesthetic (LA) toxicity may occur some time after an initial injection

2 Immediate management

- Stop injecting the LA
- Call for help
- Maintain the airway and, if necessary, secure it with a tracheal tube
- Give 100% oxygen and ensure adequate lung ventilation (hyperventilation may help by increasing plasma pH in the presence of metabolic acidosis)
- Confirm or establish intravenous access
- Control seizures: give a benzodiazepine, thiopental or propofol in small incremental doses
- Consider drawing blood for analysis, but do not delay definitive treatment to do this

3 Treatment

IN CIRCULATORY ARREST

- Start cardiopulmonary resuscitation (CPR) using standard protocols
- Manage arrhythmias using the same protocols, recognising that arrhythmias may be very refractory to treatment
- Consider the use of cardiopulmonary bypass if available

GIVE INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Continue CPR throughout treatment with lipid emulsion
- Recovery from LA-induced cardiac arrest may take >1h
- Propofol is not a suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

WITHOUT CIRCULATORY ARREST
Use conventional therapist to treat:

- hypotension,
- bradycardia,
- tachyarrhythmia

CONSIDER INTRAVENOUS LIPID EMULSION
(following the regimen overleaf)

- Propofol is not suitable substitute for lipid emulsion
- Lidocaine should not be used as an anti-arrhythmic therapy

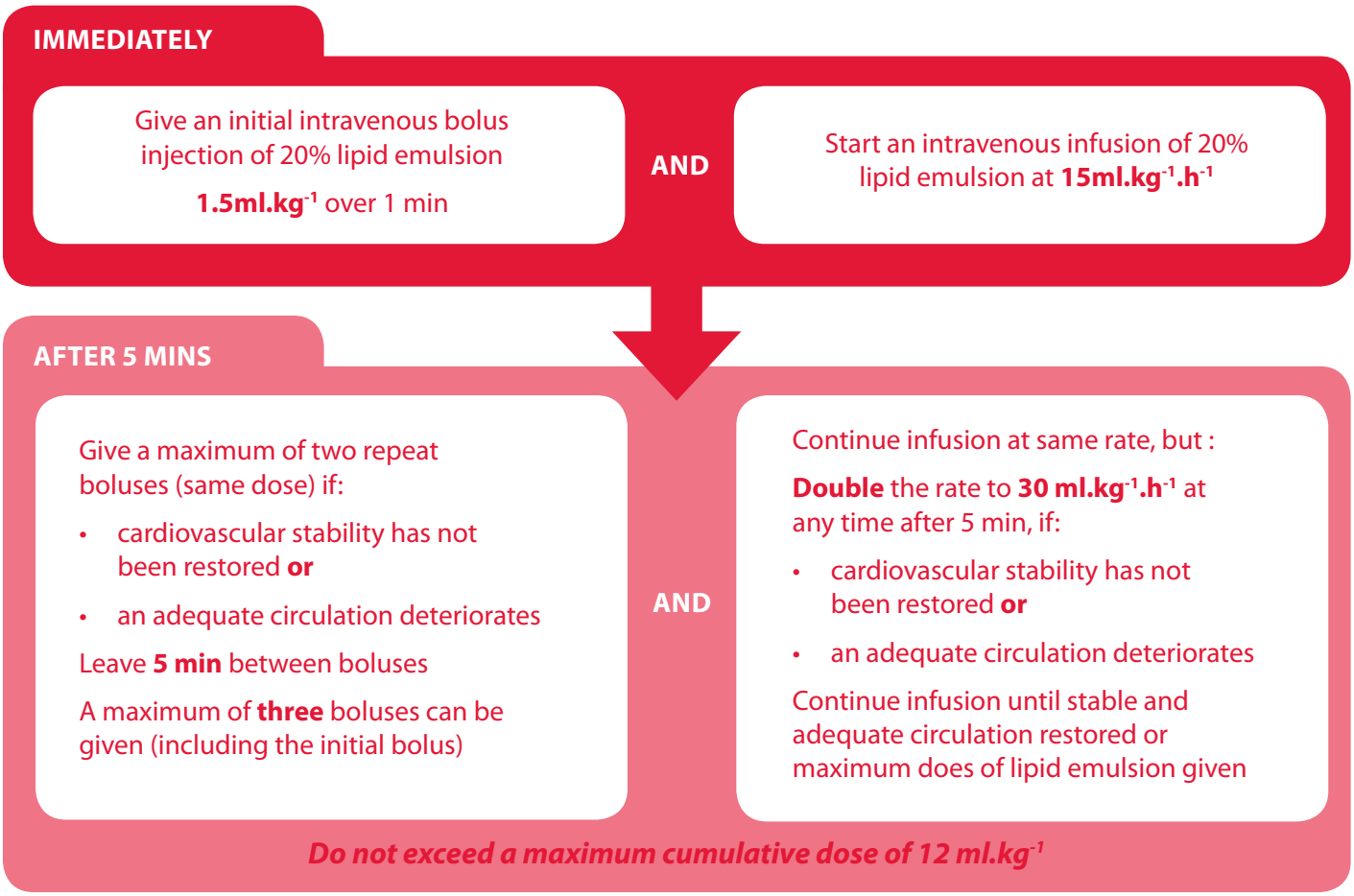
4 Follow-up

- Arrange safe transfer to a clinical area with appropriate equipment and suitable staff until sustained recovery is achieved
- Exclude pancreatitis by regular clinical review, including daily amylase or lipase assays for two days
- Report cases as follows:
  in the United Kingdom to the National Patient Safety Agency (via **www.npsa.nhs.uk**).
  in the republic of Ireland to the Irish Medicines Biard (via **www.imb.ie**).
- If Lipid has been given, please also report its use to the international registry at **www.lipidregistry.org**. Details may also be posted at **www.lipidrescue.org**

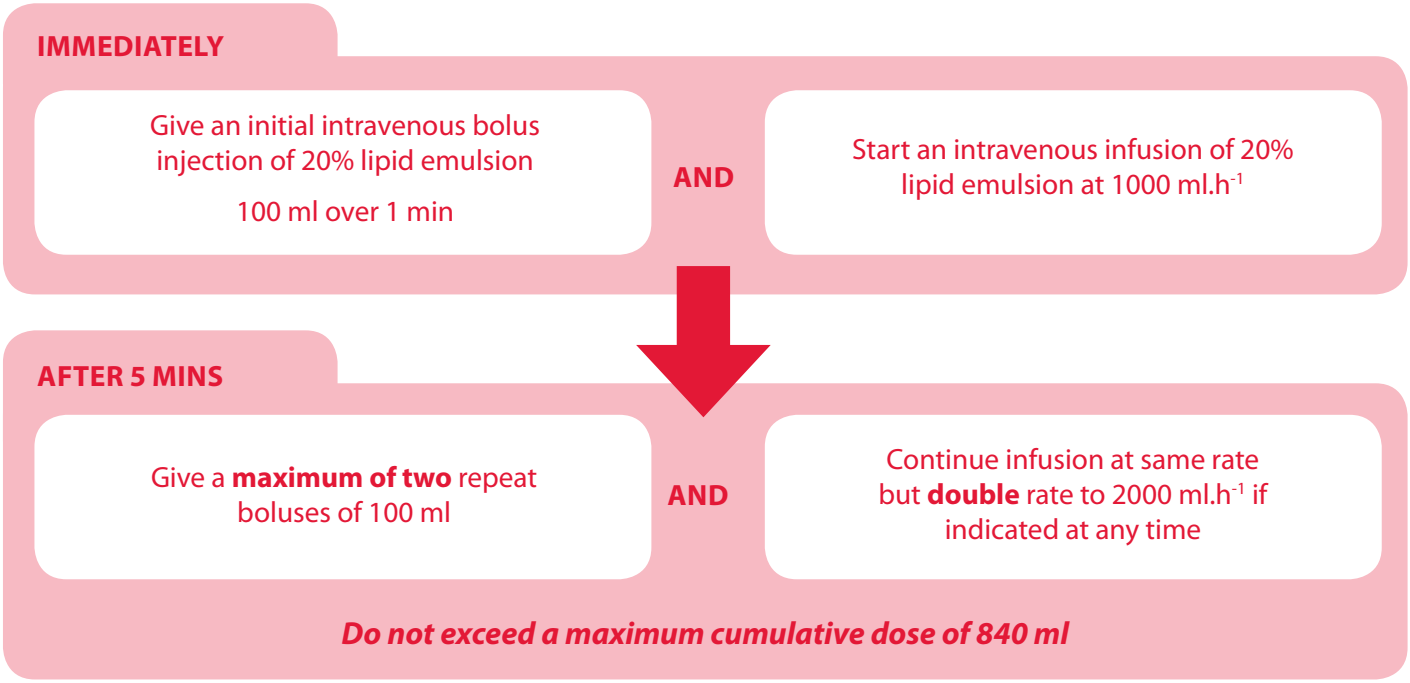
Your nearest bag of Lipid Emulsion is kept .....

This guideline is not a standard of medical care. The ultimate judgement with regard to a particular clinical procedure or treatment plan must be made by the clinician in light of the clinical data presented and the diagnoses and treatment options available.

Figure 1A. *Reproduced by kind permission of the Association of Anaesthetists of Great Britain and Ireland and available for download at:* www.aagbi.org/publications/guidelines/docs/la_toxicity_2010.pdf



An approximate dose regimen for a 70kg patient would be as follows:



This AAGBI Safety Guideline was produced by a Working Party that comprised:

Grant Cave, Will Harrop-Griffiths (Chair), Martyn Harvey, Tim Meek, John Picard, Tim Short and Guy Weinberg.

This Safety Guideline is endorsed by the Australian and New Zealand College of Anaesthetists (ANZA).

Figure 1B. *Reproduced by kind permission of the Association of Anaesthetists of Great Britain and Ireland and available for download at*: www.aagbi.org/publications/guidelines/docs/la_toxicity_2010.pdf

Update in
Anaesthesia

Management of severe local anesthetic toxicity

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Summary

An ounce of prevention is worth a pound of cure: Follow the guidelines for prevention of Local Anesthetic toxicity.

Cardiac toxicity from local anesthetics is often irreversible.

Prolonged circulatory arrest can cause brain damage even if resuscitation is successful.

Some authors recommend the use of Intralipid infusion at the first sign of cardiovascular toxicity rather than waiting for cardiac arrest.

Selection of local anesthetic type, concentration and dose must be made on an individual basis after considering patient factors.

Constant vigilance, early detection and treatment are key.

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INTRODUCTION

Local anesthetic (LA) agents are widely used, not just by anesthesiologists but by medical staff from all specialties. It is important to be aware of their toxic potential so that any toxic reactions can be detected and treated early. Whilst it is important to be able to treat LA toxicity effectively, it is clearly desirable to avoid LA toxicity whenever possible. For this reason the first section in this article outlines strategies for minimizing the risk of LA toxicity. Knowledge of the properties of local anesthetics as they relate to toxicity will enable the clinician to choose the appropriate technique and local anesthetic for each case.

PROPERTIES OF LOCAL ANESTHETIC AGENTS

Local anaesthetic agents can be classified on the basis of their chemical structure (amides or esters) or their physicochemical properties (short, intermediate or long acting).

Toxic plasma levels of LA can occur following either direct intravascular injection or absorption from the site of injection, resulting in peak plasma levels associated with neurological or cardiovascular symptoms. These plasma level values have been determined for an "average" patient. They need to be individualized for patients with comorbidites or extremes of age.

The amount of systemic absorption depends on the local anesthetic (physicochemical and intrinsic vasoactive properties), site of injection, dose of local anesthetic, addition of vasoconstrictors, and the patient's clinical condition.

Physicochemical properties

Systemic absorption of the more lipid soluble longer acting agents is generally slower. This has implications during continuous administration techniques. The longer acting agents have greater local accumulation while the shorter acting agents have greater systemic absorption.

Intrinsic vasoactive properties

Ropivacaine and levobupivacaine have intrinsic vasoconstrictor properties which may contribute to their longer duration of action and slower systemic absorption. This contributes to a higher safety profile than racemic bupivacaine, which has an intrinsic vasodilator action.

Site of injection

Independent of the local anesthetic used, systemic absorption increases in the following order:

Sciatic and femoral block < brachial plexus block < epidural < caudal < intercostal block

Since intercostal blocks are associated with the greatest systemic absorption and hence potentially toxic plasma levels of local anesthetics, it is prudent to use an agent with a good safety profile and consider adding a vasoconstrictor (such as epinephrine). Avoid continuous intercostal blocks unless the patient can be closely monitored.

Dose (concentration and volume) of LA

Increasing the local anesthetic concentration can prolong the duration of the nerve block. However, beyond a ceiling level there is a disproportionate increase in systemic absorption, possibly from saturation of local binding sites and the greater vasodilator effects of more concentrated solutions. This should be kept in mind while selecting the drug concentration. Higher concentrations of local anesthetics do not necessarily translate into longer duration blocks and have a greater potential for systemic toxicity. The recommended maximum single doses for different local anesthetics can be obtained from manufacturer's guidelines (Table 1). This recommendation is not applicable to all patients. As described above, the peak plasma level of a local anesthetic depends on multiple factors. The dose recommendations are only guidelines and should be individualized based on patient factors, type of local anesthetic used and type of block performed.

Addition of vasoconstrictors

When added to the local anesthetic solution, vasoconstrictor agents such as epinephrine could slow systemic absorption and prolong the intensity and duration of action of the nerve block. The extent to which this happens depends on the type and concentration of local anesthetic, and the site of injection. It is more pronounced with the short acting

Table 1. *Local anesthetic agents and the recommended maximum dose for infiltration and peripheral nerve blocks, based on a 70kg adult*

Local anaesthetic	Recommended maximum single dose
Lidocaine	300mg
Lidocaine with epinephrine	500mg
Prilocaine	600mg
Mepivacaine	400mg
Mepivacaine with epinephrine	500mg
Bupivacaine	225mg
Procaine	1000mg
Chloroprocaine	1000mg

amides (which tend to have a greater systemic absorption), and after intercostal blocks.

The intrinsic vasoactivity of the local anesthetic also modifies the effect of adding epinephrine. Therefore higher concentrations of local anesthetics, which tend to produce vasodilation, benefit more from the addition of epinephrine. However epinephrine has no effect on ropivacaine which has an intrinsic vasoconstrictor property.

Since epinephrine decreases the peak plasma concentration of the local anesthetic after a block, it would seem prudent to add epinephrine to the local anesthetic solution unless contraindicated. The obvious exceptions to this are blocks involving a periphery, such as digital or ankle blocks.

Epinephrine in a 1 in 200 000 concentration added to a local anesthetic solution also serves as a test of intravascular injection. A 5ml solution of 1 in 200000 epinephrine will produce tachycardia, hypertension and changes in T wave amplitude when injected intravascular.

Patient's clinical condition

Patients with liver or kidney disease require a reduction in the dose of local anesthetic because of impaired metabolism and excretion of the LA. Patients with congestive heart failure have decreased volume of distribution and clearance of local anesthetic resulting in higher plasma concentrations. Both acidosis and hypoxemia significantly increase local anesthetic toxicity. Neonates have a 2 to 3 fold prolongation in the elimination half life of amide local anesthetics.

PREVENTION OF LOCAL ANESTHETIC TOXICITY

Patient assessment

Perform a history and physical examination with careful attention to the patient's age and coexisting medical conditions. Ensure that the patient is an appropriate candidate for the regional anesthetic technique and the local anesthetic dose selected.

Chose a local anesthetic agent with the best safety profile and in an appropriate concentration and volume.

Preparation

Ensure availability of:

- Resuscitative equipment and drugs,
- Airway equipment: the means to provide bag mask ventilation, oral and nasal airways, laryngoscopes and endotracheal tubes, laryngeal mask airways.

Obtain consent for the procedure.

Attach standard monitors (ECG, pulse oximetry and non-invasive blood pressure).

Establish intravenous access.

Administer supplemental oxygen.

Consider pre-medication with a benzodiazepine.

Technique

- Choose the appropriate block and determine if the patient really needs a continuous block.
- If the patient needs a continuous block it is prudent to use an intermediate or short acting local anesthetic with less toxic potential. Ensure that the patient remains in a monitored setting until the catheter is removed.
- Check the dose and concentration of local anesthetic and epinephrine prior to performing the block.
- Draw up and label the local anesthetic and keep it with the nerve block equipment away from your anaesthetic drugs.
- While performing the block, aspirate before each injection and discard solution if discolored by blood.
- Inject the total volume in 5ml increments and monitor the patient for signs of toxicity between each injection.

Summary - Physicochemical properties of LA agents and toxicity

- Toxicity from local anesthetics depends on multiple variables and presents in various ways.
- The concept of maximum recommended dose of local anesthetic is not applicable to all patients.
- Cardiac toxicity of local anesthetics is potentiated by acidosis and hypoxemia.
- It is important to individualize the choice of drug, dose and concentration based on the patient's clinical condition and comorbidities.
- **It is also important to remember that toxicity from different local anesthetics is additive**. For example injecting a mixture of two different local anesthetics can produce toxicity even if the doses of the individual local anesthetics are under the recommended maximum dose.

- Maintain verbal contact with the patient during and after the injection.
- When possible perform blocks in mild to moderately sedated patients (i.e. maintaining verbal contact) so that they can report any symptoms of toxicity.
- There is no evidence that nerve blocks cannot be safely performed in patients under general anesthesia. If the patient really requires a block and is uncooperative, it may be safer to perform the block under anesthesia. It is very important in this situation to add epinephrine to the local anesthetic solution to be able to detect intravascular injection. The electrocardiogram should be closely monitored for T wave amplitude changes, which is a more sensitive indicator of intravascular injection in an anesthetized patient than heart rate changes alone.
- Do not leave the patient unattended after a regional anesthetic has been performed.

COMMENTARY ON ALGORITHMS

Box 1 – Recognition of LA toxicity

Recognition of LA toxicity may be difficult, since its mode of presentation is unpredictable and varies between individuals (Figure 2). In addition, presentation may occur at any time in the hour following administration. Onset of toxicity may also be late when LA is infused through a catheter, for example in paravertebral block or peripheral nerve catheters.

Systemic toxicity

Toxic reactions from local anesthetics primarily involve the central nervous system (CNS) or the cardiovascular system (CVS).

CNS toxicity

Symptoms start with lightheadedness, visual and auditory disturbances, perioral numbness and progress to disorientation, shivering, tremors, twitching and ultimately convulsions and coma. There is initial CNS excitation followed by depression. CNS depressant drugs (sedation and general anesthesia) can mask the initial CNS excitation. The potential for CNS toxicity is directly related to local anesthetic potency.

CVS toxicity

Local anesthetics have a direct depressant effect on both the myocardium and the peripheral vascular smooth muscle.

Cardiac effects

Local anesthetics cause a dose dependent prolongation in myocardial conduction which manifests as a prolonged PR interval and QRS duration. In high concentrations LA cause depression of spontaneous pacemaker activity in the SA node resulting in sinus bradycardia and arrest. They also depress the AV node and can cause AV dissociation. They also have negative inotropic effects on the myocardium.

The cardiotoxicity of bupivacaine is unique in that the ratio of the dose required for irreversible cardiovascular collapse (CC) and the dose that will produce CNS toxicity is lower for bupivacaine than other agents. Cardiac resuscitation is more difficult after bupivacaine induced cardiac arrest.

Peripheral vascular effects

With the exception of cocaine, local anesthetics exert a biphasic effect on vascular smooth muscle. They cause vasoconstriction at lower concentrations and vasodilation at higher concentrations. Cocaine produces vasoconstriction at most doses due to its inhibition of norepinephrine reuptake.

Summary

Local anesthetics will cause initial tachycardia and hypertension progressing to bradycardia and a variety of dysrhythmias leading to cardiac arrest.

Local toxicity

Nerve and muscle damage could occur at the site of injection. Skeletal muscle is usually more sensitive to the local irritant properties of local anesthetics than nerve tissue. These reactions are usually reversible.

Side effects of specific LA agents

Methemoglobinemia is seen with large (>600mg) doses of prilocaine. It is clinically insignificant in healthy adults with normal oxygen carrying capacity but can cause hypoxemia in infants.

Cocaine has significant potential for addiction.

Allergic reactions

These are usually more common with esters since they are derivatives of paraaminobenzoic acid which is a well recognised allergen. Allergy to amides though extremely rare can occur. The reactions range from hypersensitivity to anaphylaxis.

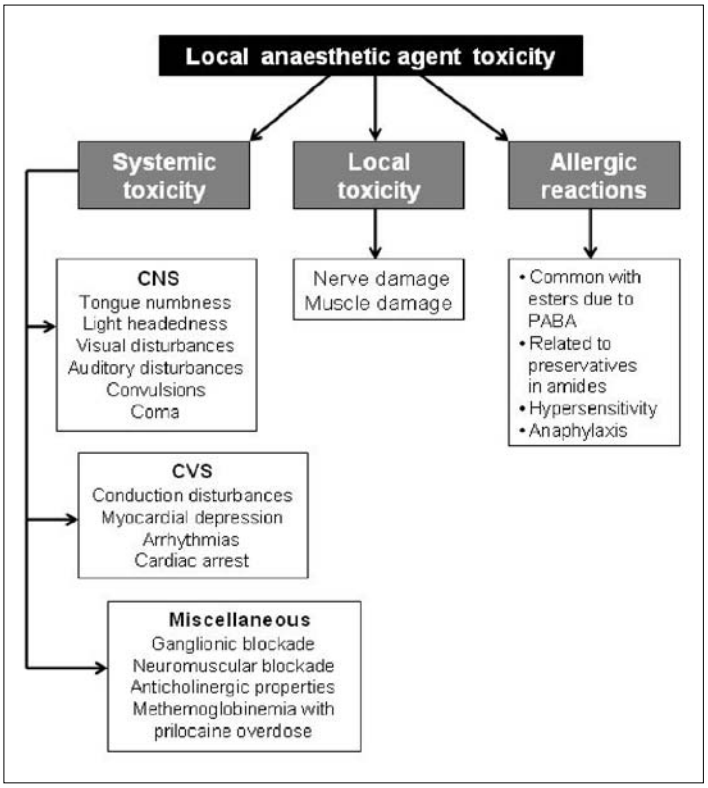

Figure 2. *Adverse effects of LA agents*

Box 2 - Immediate management

Local anesthetic toxicity from direct intravascular injection is usually immediate and transient. The first step is to stop injecting more local anesthetic. Supportive measures to maintain the airway and treat seizures are usually sufficient. For worsening symptoms or hemodynamic instability proceed to Box 3.

Box 3 - Lipid emulsion infusion for treatment of LA toxicity

- "Intralipid kits" should be available in all locations where local anesthetics are used.[2]
- The "Intralipid kit" consists of two 500ml bags of Intralipid 20%, infusion tubing, and dosing information.
- Where available, hospital pharmacies carry Intralipid 20%, and so it should be possible to stock it at all locations where local anesthetics are administered and easy to replace when the bags near expiry.
- Intralipid 20% is the formulation that has been used in the majority of the cases to treat cardiac arrest from local anesthetic toxicity. The use of other lipid emulsions is not well documented. Recommended maximum cumulative dose is $12ml.kg^{-1}$.
- Although there are many potential side effects of Intralipid infusion the only one likely after acute short term use to reverse local anesthetic toxicity is allergy.[2]
- It is therefore reasonable to administer Intralipid after conventional therapies have been initiated even in the absence of cardiac arrest.[3]
- **Propofol is not a substitute to Intralipid 20%** because it is a profound myocardial depressant and because it is formulated in 1% lipid emulsion (rather than 20%).
- There is evidence to suggest that epinephrine in doses greater than $10mcg.kg^{-1}$ impairs lipid resuscitation from bupivacaine overdose, possibly by inducing hyperlactatemia.[4]
- In cardiac arrest from local anesthetic overdose, it may be prudent to avoid escalating doses of epinephrine.

CONCLUSIONS

Given the potentially serious consequences of local anesthetic toxicity even if successfully treated, it is prudent to prevent toxicity by following the guidelines for prevention. Early diagnosis of toxic signs and symptoms is important. Most signs of local anesthetic toxicity will respond to supportive measures. For worsening symptoms of toxicity unresponsive to conventional measures, it is reasonable to initiate Intralipid 20% infusion, even in the absence of cardiac arrest.

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FURTHER READING

- Visit www.lipidrescue.org for more information on Intralipid.
- Cousins and Bridenbaugh's Neural Blockade in Clinical Anesthesia and Pain Medicine, Fourth edition, Philadelphia, Lippincott, Williams and Wilkins

Correspondence

Dear Editor,

We write with reference to the article on Paediatric Spinal Anaesthesia by Troncin & Dadure.[1]

The article is well written and gives practical advice regarding, in particular, the anatomical considerations in spinal anaesthesia in children, and references our article of ultrasound measurements of spinal canal depth from 105 neonates aged 24 - 42 weeks gestation.[2]

Our article devised the formula of "Mid-spinal canal depth (mm) = 2 x weight (kg) + 7mm" as a reasonable estimation for lumbar puncture needle depth insertion, which could be used in clinical practice. Unfortunately, Troncin's article misquotes our advice in two respects.

First, Troncin uses this formula to describe the distance from skin to the subarachnoid space, not the distance from skin to mid-spinal canal depth. Our measurements demonstrate that the neonatal subarachnoid space varies in depth, with average spinal canal depth of around 6.7mm.[2] This means that the true formula for calculating skin to "nearest" subarachnoid space depth would be 2 x weight (kg) + 3.4mm, and skin to "furthest" subarachnoid space depth would be 2 x weight (kg) + 10mm. We were careful to give a formula for estimating mid-spinal canal depth for lumbar puncture; we presume that the same index is required for spinal anaesthesia?

The second is a typographical slip in the units of measurement, in that Troncin quote measuring "Distance from skin to subarachnoid space (centimetres)" where this is clearly in millimetres. Immediately prior to this, the authors correctly reference an alternative guide using height as an estimator of lumbar puncture depth: Mean depth of insertion (cm) = 0.03 x height of child (cm)[3] which may have lead to this error being carried forward.

We are anxious to ensure that your readers are aware of these possible sources of error in the manuscript, to encourage safest clinical practice!

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Update in Anaesthesia

Guide for Contributors

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Articles for consideration by the Editorial Board should be submitted as Word documents (Rich Text Format is preferred) to the Editor-in-chief, Bruce McCormick, by email at Bruce.McCormick@rdeft.nhs.uk or post on CD-ROM or paper copy to Dr Bruce McCormick, Department of Anaesthesia, Royal Devon and Exeter Hospital, Barrack Road, Exeter, EX2 5DW, UK.

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- Some readers' first language may not be English. Please keep your text straightforward and avoid long sentences and complex terminology. Explain words and abbreviations that may not be universally standardised. Aim to include the full range of therapies available worldwide, but provide most detailed descriptions of those therapies available in resource-poor settings (see 'Management of sepsis with limited resources' in *Update 23* – www.worldanaesthesia org/component/option,com_docmantask,cat_view gid,67 Itemid,49/). Discuss older drugs as well as newer ones; halothane, thiopentone, ketamine and ether are widely used around the world.
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Authors' details

Please supply the full forename and surname of all authors, stating their title (Anaesthetic Clinical Officer, Dr, Professor etc) and the name and address of their institution. One author should be identified for correspondence, with an email address provided.

Drug doses

Please use the international units, e.g. $mg.kg^{-1}$ rather than mg/kg. Use SI notation for g, mg, mcg etc. Please use internationally accepted non-proprietary drug names, e.g. furosemide, epinephrine and avoid trade names.

Headings

Three levels of heading may be used CAPITALS, **bold** and *italic*. Please do not employ different fonts within the text. Bullet points can be helpful.

Illustrations / figures

These may be sent to us as drawings (black on white), which we will scan into the text, or as picture files in jpg (JPEG) format. Black and white photos are also suitable. If you do not have facilities to produce drawings, contact the editor for help. If you copy illustrations from another publication please obtain copyright permission from the publishers or author. If patients appear in a photo please ensure that they have consented to this. Text accompanying illustrations should be supplied on a separate piece of paper.

Tables or figures reproduced from other published texts should be accompanied by a statement that permission for reproduction has been obtained from the author or publisher. An acknowledgment should be included in the caption and the full reference included in the reference list.

Tables

These should be prepared using the Microsoft Word table facility whenever possible.

Graphs

Graphs should be supplied using the Microsoft graph-compiling feature within Microsoft Word, or as a figure on paper.

References

A minority of Update readers have access to journals and therefore references should in general be limited to those that would be considered as 'further reading'. Please format your references as shown. Number the references in the order they appear, using the reference number as a superscript at the relevant point in the text.

References should include: names and initials of all authors (unless more than 6, when only the first 6 are given followed by 'et al.'), title of the paper; Medline abbreviation of the journal title (in italic); year of publication; volume number; first and last page numbers.

Papers accepted but not yet published should be included in the references, with the abbreviated journal name, followed by '(in press)'.

Those in preparation (including any submitted for publication), personal communications and unpublished observations should be referred to as such in the text.

1. Reynolds F, O'Sullivan G. Lumbar puncture and headache. 'Atraumatic needle' is a better term than 'blunt needle'. *Br Med J* 1998; **316**: 1018.

2. Costigan SN, Sprigge JS. Dural puncture: the patients' perspective. A patient survey of cases at a DGH maternity unit 1983–1993. *Acta Anaesthesiol Scand* 1996; **40**: 710–14.

3. Spriggs DA, Burn DJ, French J, Cartlidge NE, Bates D. Is bedrest useful after diagnostic lumbar puncture? *Postgrad Med J* 1992; **68**: 581–3.

References to books should give book title, place of publication, publisher and year; those of multiple authorship should also include chapter title, first and last page numbers, and names and initials of editors. For example:

1. Roberts F. Chapter 22: Ear, nose and throat surgery. In: Allman KG, Wilson IH, eds. Oxford handbook of Anaesthesia (1st edition) Oxford: Oxford University Press, 2001: 506-39.

UPDATE SHORT REPORTS

The scope for publication of articles describing original research and audit conducted in, and specifically relevant to, poorly-resourced settings is limited. Successful publication in major journals is rare and the distribution and accessibility of the national and regional journals that currently publish these articles is often poor. As the official journal of the World Federation of Societies of Anaesthesiologists, *Update in Anaesthesia* is the appropriate forum for publication of these manuscripts and offers a wide distribution.

The guidance above for clinical overview articles applies, with the following additional considerations.

Legal considerations

- Papers based on clinical investigation on humans should include the consent of patients and a statement of approval from an appropriate Ethics Committee. In those institutions where Institutional Review Board consent is required for the performance of audits, this should be obtained and referred to in the text.
- Avoid use of identifiable names, initials and hospital numbers of patients.
- Human subjects of case reports, research or audits should not be identifiable. Manuscripts should not disclose patients' names, initials, hospital numbers (or other data that might identify the patient(s)).
- Guides for use of tables, figures and illustrations are as described above for Clinical Overview articles.

Brief Communications

- Original investigative articles or audits of patient outcome or clinical techniques.
- Up to 1500 words (approximately 2 pages of *Update in Anaesthesia).*
- Subdivided into:
  - Summary (maximum five sentences) and key words
  - Introduction
  - Patients and methods
  - Results
  - Discussion
  - Acknowledgements
  - References – maximum 15
  - Tables and/or figures - limited to two per article.

Case Reports

- Suitable for presenting descriptive studies (a series of cases), personal experience or individual case reports of particular interest.
- Up to 800 words. Three tables or figures is allowed in addition to text.
- A summary may be included (up to five sentences). Division into sections is optional.
- Up to seven references may be given.

Correspondence

- Welcomed on any subject, including editorials or articles that have appeared in *Update in Anaesthesia*.
- Letters may also be a suitable vehicle for presenting items of experience or observation that are too brief for Brief Communications.
- Papers describing procedures, techniques or equipment adapted by readers to their own conditions of work are welcomed.

Proofs

- Proofs are sent to the author designated to receive them. Corrections should be kept to a minimum and the proofs returned within 7 days of receipt.

The editorial team will be delighted to help with the preparation of articles. The best way of doing this is via email - Bruce.McCormick@rdeft.nhs.uk

Dr Bruce McCormick
Editor-in-chief
Update in Anaesthesia, July 2008

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