Core Airway Skills

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Second Edition, 2017

Adapted from the original "Advanced Airway Management" by Dr John Roos.
Updated copies of this manual can be found online from www.OpenAirway.org

ADVANCED AIRWAY MANAGEMENT
IN THE PREHOSPITAL, AVIATION, ANAESTHESIA AND EMERGENCY MEDICINE ENVIRONMENTS

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This work is based nearly entirely on the phenomenal volume "ADVANCED AIRWAY MANAGEMENT IN THE PREHOSPITAL, AVIATION, ANAESTHESIA AND EMERGENCY MEDICINE ENVIRONMENTS" by Dr John Roos. Dr Roos has been a leader in cross-disciplinary airway education in South Africa for many years, and has created a wealth of material – and the backbone of this entire workshop – almost singlehandedly.

In this revised edition for the Core Airway Skills workshop, the formatting and referencing of the manual have been updated. Some changes to the protocols and editorial changes have been made, but credit for the content still falls firmly to Dr Roos, in respect for his years of effort.

We hope that this is a useful resource, which will continue to grow and expand over time. Regular updates on airway topics, video tutorials, links to journal articles and discussion of all topics regarding the airway can be found on the open-access medical education website, www.OpenAirway.Org to accompany this volume.

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WORKSHOP OVERVIEW

The two-day workshop covers all aspects of airway management, from basic face-mask ventilation to tracheal intubation, and includes difficult and failed airway management, as well as the use of rescue airway devices. Delegates will gain insight into the true morbidity and mortality, the risk-benefit analysis and the appropriateness of emergency tracheal intubation.

Delegates will similarly gain an understanding of the under-estimated and even unrecognised difficulties associated with facemask ventilation, and will learn how to avoid the nightmare of the missed oesophageal intubation, and the unrecognised misplaced tracheal tube.

This workshop places emphasis on many basic concepts, surprisingly not routinely taught during intubation training. It revisits many core concepts, dispels myths, highlights pitfalls and offers many useful tips to smooth airway interventions.

Standard tracheal intubation is examined in terms of the pre-intubation, intubation and post-intubation phases, and emphasis is placed on rapid sequence intubation, and confirmation of tracheal tube placement.

Candidates will learn how to predict the difficult airway, and will learn how to manage the difficult and failed airway, including the "can't intubate, can't oxygenate" scenario. Candidates will also gain insight and skill in the use of alternative and "assist" airway devices, supraglottic "rescue" airway devices, and emergency surgical airway procedures.

'Special cases' are discussed in detail – namely the paediatric, pregnant, obese and the trauma patient – including the head-injured patient.

The pharmacology of induction agents, neuromuscular paralysis and pressor agents is similarly discussed in great detail. The management of the associated concepts of ventilation, hypotension, sedation, analgesia and neuromuscular blockade during the post-intubation phase will be discussed at length.

In the three-day workshop, the additional third day will be spent on a ventilation workshop, which will include the following:

- Ventilation in the aviation (and/or transport/emergency medicine) environment:
    - Pitfalls and tips in emergency ventilation
    - Ventilators and ventilatory modes
    - Physiology and pathophysiology of positive pressure ventilation
    - 'T-piece' ventilation and the alveolar gas equation
    - Sedation and analgesia in the ventilated patient
    - Monitoring of the ventilated patient
- Neuromuscular blockade in the ventilated patient
- How long will your oxygen cylinder last?
- Ventilatory strategies – how to set the ventilator
- Pulse oximetry
- Capnography
- The misplaced tracheal tube
- Traumatic brain injury (as an optional extra – time-permitting)
- Arterial blood gas analysis (as an optional extra – time-permitting)

WORKSHOP MATERIAL APPROPRIATE TO AIRWAY MANAGEMENT COVERED IN SEPARATE LECTURES

This lecture series presumes that the delegates have an adequate grasp of the lecture material below, which knowledge is considered fundamental to the practice of Advanced Airway Management:

- Pulse Oximetry
- Capnography
- Mechanical Ventilation in the Emergency Medicine Environment
- Sedation, Analgesia and Neuromuscular Blockade in the ventilated patient
- Arterial Blood Gas Analysis
- Traumatic Brain Injury

ILLUSTRATIONS

All aspects of these notes are fully illustrated with photographs, diagrams and graphs in the accompanying slide presentations on the same subject matter.

Cover image by Dr Mark Barley, courtesy of the Difficult Airway Society:
https://www.das.uk.com/content/anaesthetic-emergency-signage

THE ARGUMENT FOR AND AGAINST: SAN DIEGO

The magnitude of the problem…

It is well-established that endotracheal intubation in the prehospital environment is associated with a considerably greater degree of difficulty, and a much higher complication and failure rate than intubation undertaken in the controlled operating-theatre environment.   The reasons for this are many-fold and will be discussed in detail below.

There have been many conflicting international studies over the past decade that have examined prehospital endotracheal intubation in terms of difficulty and failure, adequacy of training, skills maintenance, and most importantly, on patient outcome.   Notwithstanding the volume of research on the subject, there remains a quagmire of controversy.

The "San Diego Paramedic Rapid Sequence Intubation Trial" was a landmark study in the field of prehospital endotracheal intubation.[1]   This study made the prehospital emergency medicine fraternity sit up and take notice, since its damning results led its researchers to terminate the study early, on ethical grounds.

The authors discovered a significant degree of:

- Inadvertent hyperventilation,
- Oxygen desaturation,
- Bradycardia and…
- Hypotension…

　　　　…during and after intubation.

A number of further studies found that mortality in the field-intubated groups was significantly greater than in the non-intubated groups.   All of the authors concluded that endotracheal intubation conferred **no demonstrable survival benefit** or functional advantage over BVM ventilation.[2-4]

Worse still, not only have these studies failed to demonstrate benefit, but have demonstrated evidence of actual harm!

The more recent OPALS Major Trauma Study showed **no overall benefit in survival to hospital discharge** in patients receiving Advanced Life Support prehospital care.[5]   This was a before-and-after controlled trial across 17 cities, and included 2867 major trauma patients, controlled for age, injury severity and physiologic measures.   Of the 598 patients with a GCS score of less than 9, mortality was actually greater during the ALS phase (79% ALS vs 86% BLS survival)!!
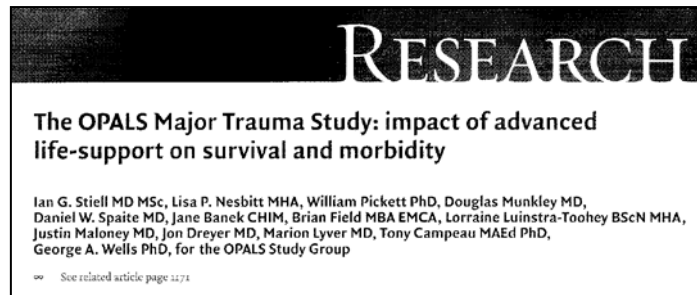
It is not only prehospital practitioners that have been placed under the spotlight.   Bowles et al studied in-hospital (but out-of-theatre) intubations in one large UK deanery, covering nine hospitals, over one month.[6]   Doctors with at least six-months full-time anaesthesia training, the majority of whom identified anaesthesia as their base specialty, undertook 136 intubations.   At least one severe adverse incident was identified per patient in 39% of intubations, despite specialist presence in more than 40% of cases.   These included death, cardiac arrest, significant hypotension or hypoxaemia.   It is clear from the above that even anaesthetists are not very good at out-of-theatre intubation.

Whilst the benefits of field intubation appear 'intuitively obvious', my observations and reading around the subject have forced me to ask the question, "Are paramedic field intubation techniques and conditions optimal, is our emergency practitioner training adequate, and do we undertake prehospital endotracheal intubation appropriately?"

Or, more simply put, "Are we helping or hurting our patients?"

Spaite and Criss, in an editorial comment on the landmark San Diego Paramedic Rapid-Sequence Intubation Trial, stated very aptly:

"…except for defibrillation, essentially no EMS intervention has been prospectively evaluated with robust methodologies that allow us to conclude that safety has been established… the vast majority of EMS systems have implemented interventions based on the 'Mount Everest' approach – because it's there.   We can no longer assume that, because we believe we are improving patient outcomes, this excuses us from verifying that we are in fact improving patient outcomes.   The blind Mount Everest approach to EMS must end."[7]

A further example of the "Mount Everest approach" in EMS would be that of the use of emergency lights and sirens in high speed responses.   Whilst the benefits once again appear intuitively obvious, there is *no evidence* that the employment of these devices reduces morbidity or mortality.

In fact, the London Metropolitan Police have demonstrated that high-speed car chases significantly increase injuries and deaths from motor vehicle accidents, without decreasing loss of life from reduced violent crime.   High-speed car chases by law enforcement authorities in the United Kingdom have thus been banned!

Wang and colleagues, accomplished researchers in the field, have left us with the following sage wisdom:

"Unfortunately, we cannot begin to fix the sinking ship until we shine bright lights on the holes in the hull.   Perhaps more alarming is the fact that we often do not recognise that the ship is slowly sinking.   Many EMS systems do not bother to track intubation errors or success rates. Is it possible that we lack the courage to look because we are afraid of what we may find? Or is it that we do not care to know?"[8]

One needs to bear in mind the adage, "*Absence of evidence is not evidence for absence*". ie. The fact that we continue to embrace a practice without rigorous supportive evidence (on the basis of expert opinion, 'common sense', or whatever) does not **necessarily** imply bad practice.   We should therefore not always elect to throw the baby out with the bathwater, but seek to gain the requisite supportive evidence required to justify the continuation of such practice.

There are multiple reasons to explain the anticipated observation that endotracheal intubation in the field is very much more difficult than intubation in the operating-theatre environment. These would include:

- The inherent dangers of a chaotic casualty scene;
- Concerns for personal safety;
- A working environment that comprises:
    - poor lighting;
    - high noise levels;
    - adverse weather conditions;
    - bright sunlight (obscuring laryngoscopy);
    - paramedics working on their knees or lying flat on the ground;
    - the ubiquitous presence of such hazards as broken glass, leaking motor fuel and oil, and body fluids on the ground.
- Patients are often found in challenging locations – crumpled on the floor of a cramped bathroom, trapped inside motor vehicle wreckage, or lying face-down in a muddy ditch, etc.
- 'Jump bags', oxygen cylinders, portable suction units and cardiac monitor-defibrillator units often have to be carried some considerable distance from the response vehicle, up flights of stairs, across busy freeways, or down elevator shafts, to name but a few of a paramedic's daily challenges.
- In order to begin resuscitation, equipment first has to be unpacked from 'jump bags', and laid out as best as possible under the circumstances.
- Paramedics work alone or with personnel who are not trained in advanced life support, and are unfamiliar with the practice of, and demands imposed by, endotracheal intubation.
- Patients often have fluctuating levels of consciousness, and notwithstanding, are more often than not combative.   Paramedics have an extremely limited pharmacological armamentarium, and are unable to institute neuromuscular blockade.

- Added to this are the difficulties imposed by:

    - cervical spine immobilisation;

    - the potential full stomach and aspiration;

    - craniofacial trauma;

    - neck, facial and upper airway burns;

> o   upper airway haemorrhage.

Many studies report a 10 to 15 percent (or higher) failure rate in prehospital intubation, compared to a 1% accepted in-hospital failure rate.   This high failure rate has been suggested by numerous authors to be the result of placing RSI in the hands of relative 'beginners' in airway management.   Further to this, the shocking study by Katz and Falk, which revealed a staggering 25% of intubations to be missed oesophageal intubations, has been alleged to be the consequence of inadequate training and medical oversight.[9]

If this is indeed the case, this study underscores the fundamental importance of close medical supervision and clinical governance by an experienced specialist in airway management.

We are thus reminded that RSI is not a simple procedure, and can result in significant morbidity and mortality when placed in the wrong hands (as has been evidenced by the San Diego trial).   Further to this, RSI is a specialised technique applied to a small subset of patients, as not all patients intubated in the field require paralysis.

RSI is not the complete answer to difficult intubation, as up to 40% of failed intubations in many studies are ***not*** attributable to inadequate relaxation!

Blind nasal intubations are associated with much longer intubation times, a failure rate of 25 to 40 percent, epistaxis and a high incidence of missed oesophageal intubation.[10-12] Considering the results of numerous studies, blind nasal intubation cannot be recommended in the prehospital or emergency environments, and should in fact be outlawed.

There are numerous studies reporting dismal missed oesophageal intubation rates of 1 to 2 percent, many of them inappropriately prefixing their alarming percentage-proclamation with the disarming adverb '*only*'. Three studies reported rates at 5.8%, 9% and (shockingly) 25% respectively.[9,13,14]

Bag-valve-mask ventilation has been proven to be a vastly safer practice, with ***no deaths*** attributed to complications of face-mask BVM ventilation in any of the studies cited.[15-18]

The North American anaesthetic mortality rate is believed to be between 3.3 to 5 deaths per million anesthetics, or at worst 1 death per 200 000 anaesthetics.[19]

If we were to accept an unrecognized oesophageal intubation rate of "***only one percent***", and presuming that the patient died from this oversight (which would certainly be the case if neuromuscular blocking drugs were used universally), this would translate into 2000 deaths per 200 000 prehospital intubations.

To rework these statistics into more meaningful figures, we would have to accept that the practice of intubation undertaken by American paramedics in the field was 2000 times more likely to kill a patient than if that same intubation was undertaken by an American

anaesthesiologist in an operating theatre.   This clearly makes prehospital intubation by North American paramedics an extremely hazardous and often-times **lethal** procedure.

If we extrapolate these *"success"* rates to the airline industry: if we were to accept a failure rate of *"only one percent",* we would have to accept no less than 30 airline crashes at London's Heathrow Airport alone, every single day!

To further illustrate this silent epidemic, or massacre, inflicted on the unwitting American public by its paramedics: There are approximately 200 000 EMTs in the USA, of which about 10% (20 000) are paramedics (or EMT-Ps).   If each paramedic intubates only one person per week, and if each paramedic has a missed oeophageal intubation rate of *"only one percent"*, this translates into 10 400 missed oesophageal intubations per annum – the equivalent of three "9/11" catastrophes every year!

Thus, a missed oesophageal intubation rate of *"only one percent"* should invoke a state of abject horror in the minds of paramedics and administrators alike, and should not be fobbed off with the idle complacency of, *"a small but important experience with failed prehospital airway management which remains to be addressed"*.[20]

To rub further salt into EMS's wounds, Bledsoe has reported recent studies which show misplaced ETTs amongst American paramedics to be, not in the order of one percent, but in the order of **_FIVE PERCENT!_**[21]

The EMS community needs to take full ownership of this highly lethal epidemic, and needs to introduce every measure to urgently intervene in this inexcusable cause of utter shame to the profession.

LESSONS LEARNED FROM THE INTERNATIONAL LITERATURE

1. There exists a high incidence of unrecognised peri-intubation oxygen desaturation and bradycardia reported in the literature.

2. A very high incidence of inadvertent hyperventilation has been consistently reported in the literature.

3. This hyperventilation and hypocarbia is strongly associated with poor outcomes in head-injured patients.

4. There is a high incidence of emergency tracheostomy in the prehospital environment in comparison with the operating-theatre environment.   This practice often precedes other less invasive airway rescue procedures such as LMA or Combitube placement.

5. There is an extremely high incidence of unrecognised oesophageal intubation, misplaced or dislodged endotracheal tubes, and there exists an unacceptable level of complacency towards this problem.

6. A high incidence of pulmonary aspiration is reported in the literature, occurring both before intubation, and during the intubation attempt.

7. Non-pharmacologically-facilitated intubation is associated with a dismal, almost imperceptible success rate.   We should be directing our efforts elsewhere.

8. Inadequate relaxation accounts for only 20 to 40% of difficult and failed intubations. Consequently, paralysing drugs are not a panacea to the problems faced with difficult intubation in the prehospital setting.

9. Prehospital RSI is regularly associated with failure rates of 10% and higher, whereas the in-hospital RSI failure rate is reported to approximate1% in many studies.

10. Basic airway management is not to be under-estimated.   There is no substitute for a good face-mask seal and adequate bag-valve-mask ventilation.

11. Blind nasotracheal intubation is associated with prolonged intubation times, low success rates, an increased incidence of oesophageal intubation, and high complication rates.   It is not to be encouraged in the prehospital setting.

12. There exists a general paucity of 'difficult airway' equipment amongst paramedic personnel, which includes alternative airway devices.

13. Failed intubation algorithms and protocols need to be developed and implemented.

14. Suxemethonium is associated with potentially lethal complications in the prehospital environment, as yet to be fully quantified.

15. Suxemethonium's relative short duration of action cannot be relied upon to restore ventilation before terminal desaturation, in a "can't intubate, can't oxygenate" (CICO) scenario.

16. The still experimental reversal agent Sugammadex (Org 25969) may yield rocuronium a safe alternative to suxemethonium in the future.   Its appropriateness in the field will have to be established.

LESSONS LEARNED FROM THE WESTERN CAPE PREHOSPITAL INTUBATION STUDY

1. We have a very high frequency of intubation amongst our paramedics, when compared to North American statistics.

2. We have a very favourable success rate of 90% in non-paralysed patients, which mirrors the best rates published in the USA, and is certainly better than their norm.

3. We have documented an extremely high rate of difficult and failed intubation (in comparison to the operating theatre environment).

4. Pulmonary aspiration of gastric contents occurs in more than 50% of patients before the first attempt at intubation.

5. There is a very high incidence of pulmonary aspiration occurring during the intubation procedure itself.

6. Difficult intubation carries an especially high risk of aspiration.

7. Accordingly, it has become clear that the wisdom of intubating non-paralysed patients with depressed levels of consciousness, in an attempt to secure the airway against aspiration, is a dubious practice, as this may lead to an increased incidence of

aspiration. This indication for intubation needs to be revisited. We also need to emphasise the role of cricoid pressure in this regard.

8. We have shown in our study, which is supported by the international literature, that the single-most important factor relating to difficult intubation is patient effort.

9. However, not all difficult and failed intubations are related to combativeness. Accordingly, we must look beyond this single parameter to improve prehospital intubation success rates in a significant proportion of patients.

10. We have detected a disproportionately high incidence of Cormack & Lehane grade III and IV laryngoscopic views.

11. One technique at improving visualisation of the vocal cords, which is sorely overlooked by paramedics, is the application of cricoid pressure, as "optimal external laryngeal manipulation".

12. Cricoid pressure is also overlooked in terms of attempting to minimise aspiration.

13. In-line neck stabilisation is overlooked in a large percentage of patients who would benefit from this precaution.

14. The only drugs that paramedics have at their disposal, to ablate the intubation response, are morphine and midazolam. The efficacy of midazolam for this purpose must be seriously questioned. So, too, must the injudicious use of morphine in a cardiovascularly unstable head-injured patient. We need to look towards more efficacious and judicious approaches to ablating airway reflexes, and to teaching paramedics which patients *not* to intubate.

15. Our somewhat startling revelations on oxygen saturation have revealed that pre-oxygenation, assisted ventilation and the management of 'peri-intubation' oxygen saturation are matters that require urgent attention amongst those performing field intubation.

16. We have demonstrated a high incidence of pharangeal trauma post-intubation. It is highly likely that this is due to patient effort, and is a matter that needs to be addressed under the heading of 'reducing patient struggling'.

17. We have revealed a relatively high rate of emergency tracheostomy (in comparison to hospital statistics, and not the prehospital environment).

18. Paramedic experience and frequency of intubation do not appear to be important factors relating to failed intubation in our study, but geographic area certainly does – with a three times higher rate of failed intubation coming from beyond the greater Cape Town metropolitan area, ie. the more outlying or rural areas.

19. There is real cause for concern regarding erosion of skills for our paramedics working in outlying areas, some of whom have very little exposure to endotracheal intubation.

20. We have documented the very important finding that "day hospital" doctors, or more correctly, doctors working in clinics and the satellite emergency (casualty) departments of Community Health Centres on the Cape Flats, are calling upon

paramedics to intubate patients in their departments, before interhospital transfer to referral institutions.

21. Paramedics are managing critically ill and unconscious patients for lengthy periods (a mean of 60 minutes and a maximum of 5 hours, in our study), without having the necessary equipment to maintain an acceptable level of Advanced Life Support care. As evidenced from the U.S. data, pulse oximetry and capnography have become mandatory essential equipment, and not "luxurious extravagances". In terms of patient safety, this aspect of equipment provision requires urgent attention by the authorities.

22. We have highlighted some glaring deficiencies in essential equipment. In a small percentage of cases there was an absence of some items as fundamentally basic as oxygen and suction apparatus. But far more commonly, there was an absence of the more advanced equipment required for adequate and safe monitoring of patients, for adjuncts to assist with difficult intubation, and alternative airway devices for the management of difficult airways following failed intubation. Continuous waveform capnography has become recognised internationally as an *essential* monitoring device for the intubated, ventilated patient.

CONDITIONS FOR THE IMPLEMENTATION OF A SUCCESSFUL AIRWAY MANAGEMENT PROGRAMME

JRCALC POSITION STATEMENT:

United Kingdom Joint Royal Colleges Ambulance Liaison Committee, July 2008.[22]

JRCALC assessed three aspects of paramedic intubation:

- Assessment of benefit in terms of patient outcome

- Assessment adequacy of current paramedic student training

- Assessment of adequacy of ongoing competency

The group concluded that "paramedic intubation can no longer be recommended as a mandatory component of paramedic practice and should not be continued to be practiced in its current format", and that "for the majority of paramedics… greater emphasis should be placed on airway management using a supraglottic airway device(SAD)".

JRCALC has recommended that prehospital practice no longer emphasizes chasing the illusive "gold standard" of Tracheal Intubation, but rather concentrates on the rapid establishment of a clear airway and optimal gas exchange. The primary aim should not be the use of a particular airway device, but to ensure a secure and effective airway, oxygenation and ventilation of the patient.

A working knowledge of tracheal intubation will be required of UK paramedics, but they will no longer be required to be "signed-off" as competent in that intervention.

Where ambulance services permit tracheal intubation as a specialist skill for some providers, these individuals should be trained in the provision of drug-assisted intubation (DAI).

- There is a paucity of evidence to suggest that tracheal intubation without the use of drugs improves patient outcome.
- Tracheal Intubation should be developed as a specialist skill for a selected subset of providers and should include the provision of drug-assisted intubation.
- Supraglottic airway devices have been shown to be safe and effective in elective and emergency procedures.
- Current evidence suggests they are suitable alternatives in the prehospital environment.

PARAMEDIC INTUBATION: RISK-BENEFIT ANALYSIS

Unrecognised oesophageal intubation is almost universally fatal. The benefit of correctly placed tubes would have to improve outcome for a great many patients in order to show a statistical benefit to offset the detrimental outcomes of oesophageal intubation. Such a benefit for tracheal intubation in cardiac arrest is very unlikely, especially in that $ETCO_2$ may be unhelpful in confirming correct tube placement in cardiac arrest victims.

Additionally, Tracheal Intubation requires cessation of compressions during cardiac arrest resuscitation, whereas SAD placement does not. One drawback to SAD placement is that it is easier to dislodge an SAD than an ETT.

Intubation for trauma:

One study of anaesthesia registrars showed that a 90% in-hospital success rate was only achieved after 57 intubation attempts.[23] Furthermore, such elective intubation is much easier under the controlled conditions of the operating theatre environment.

The San Diego study, paramedics intubated with midazolam and suxemethonium, where intubation still failed in **13%** of patients! Compare this to the in-hospital accepted RSI failure rate of no more than 1%. This re-inforces the findings of other studies, where paralysis has been identified as *only one of many factors* influencing successful intubation. Suxemethonium is not the panacea (magic salve) for difficult and failed tracheal intubation.

TRAINING REQUIREMENTS:

Current EMS training programs require that 10 to 25 in-hospital intubations need to be done to certify a paramedic student as competent. The Western Cape Provincial Ambulance College requires a minimum 10 supervised intubations before graduation. These numbers

are not based on any scientific derivation from learning curves, but rather on what is subjectively thought to be a *reasonable number* of **achievable** intubations during the prescribed training period.

The scientific literature has indicated that 32 intubations are required to achieve a 75% success rate, and 53 for a 90% success rate. [24]
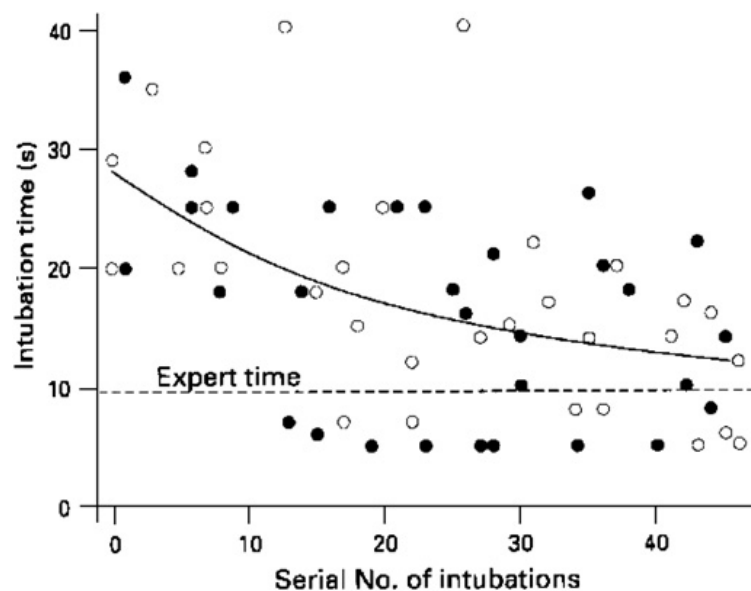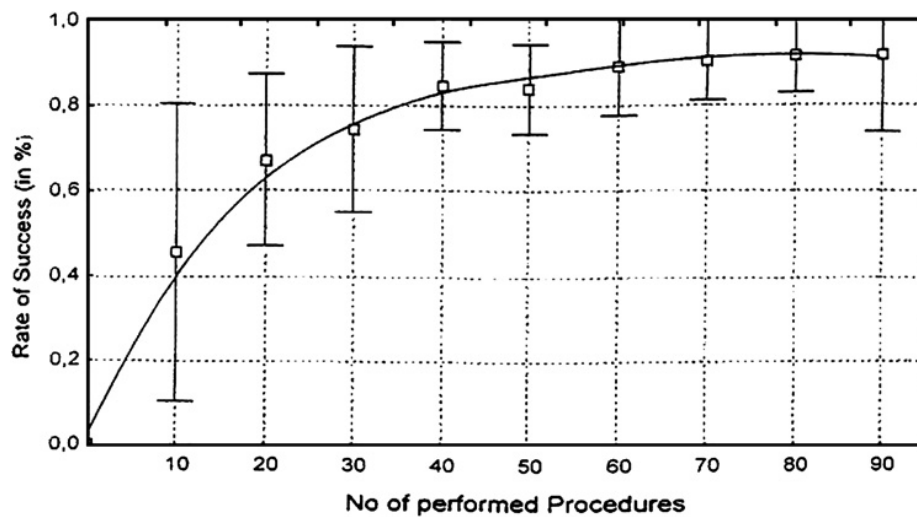
These figures are similar to those reported in other studies.[23,25]

Therefore, paramedics required to perform only 10 intubations during their training course may possibly achieve as little as a 50% or lower success rate once qualified. This guarantees the same chance of success as flipping a coin!

Even worse, Bledsoe [22] reports that the current USA National Standards Curriculum for 'Emergency Medical Technician – Paramedic' requires only *five* intubations to be performed prior to graduation![21]

Skill fade is faster with more complex tasks, and is accelerated by lack of practical experience.   In the UK, and many parts of the USA, paramedics intubate once to twice per year, and in one Pennsylvania study, individual paramedics intubate on average less than once in two years!   A significant fall-off in paramedic intubation success rates parallels lack of ongoing exposure to the procedure.

Training opportunities for initial and ongoing training are extremely limited, and it is now widely recognised that major challenges face exposure to practical procedures with respect to Continuing Medical Education and skill enhancement/retention.

Moreover, adequate initial and ongoing training would necessitate the removal of paramedics from frontline service for considerable periods, which would have major logistic, service delivery and financial implications.

It is suspected that a small but significant number of patients with partial or complete airway obstruction will not be able to be managed with SADs, and will require drug-assisted intubation.   A recent British NCEPOD review found that 13.7% of major trauma victims arriving at UK hospitals by ambulance did so with a partially or completely obstructed airway.[22,26]   The JRCALC report concluded that *"…if prehospital intubation is to be part of prehospital trauma management, then it needs to be in the context of a physician-based prehospital care system*".

In the South African context, patients often need to be transported long distances, within time-frames of many hours.   Many General Practitioners are unfamiliar and uncomfortable with tracheal intubation and rely on paramedics to undertake this procedure for them.   In addition, South Africa is a vast country in terms of travel distances and times, and we simply do not have the resources to provide country-wide physician-based prehospital Advanced Life Support with concomitant Advanced Airway Management.

NAEMSP CONDITIONS FOR A SUCCESSFUL PREHOSPITAL/EMERGENCY RSI PROGRAMME:

 Following the large number of disastrous American prehospital RSI studies reported in the literature, with very high rates of failed prehospital intubation and missed oesophageal intubations, the National Association of Emergency Medical Services Physicians (NAEMSP) in the United States published a *Position Paper* in January 2005[27] outlining their policy towards prehospital RSI thus:

- Drug-assisted intubation (DAI) should not be considered mandatory, and nor is it appropriate for many prehospital EMS systems;
- DAI should only be used in EMS systems where the medical director(s) identify a specific need and;
- Possess adequate resources to develop and maintain a DAI protocol.
- DAI is a powerful technique that can be harmful if not performed properly.
- EMS providers performing DAI must demonstrate ongoing competence in order to maximise patient safety and quality of care.
- EMS providers must have received specific training, and possess specific knowledge and experience in the technique and in the drugs used to perform DAI, as well as in the techniques confirming proper tube placement.
- Medical direction with concurrent and retrospective oversight and supervision is essential;
- Proper patient selection must be adhered to.
- Training of airway management in patients who cannot be intubated, as well as back-up 'rescue' airway methods and devices must be taught.
- Standardised DAI protocols must be in place.
- Resources for drug storage and delivery must be in place.
- Continuous monitoring of heart rate and rhythm, oxygen saturation and end-tidal carbon dioxide before, during and after DAI.
- Training and equipment to verify initial and ongoing tube placement.
- Continuous quality assurance, quality control, performance review and supplemental training where necessary.
- Research or clinical audit to clarify the role of DAI on improved patient outcome within the EMS system.

It follows from the above that:

- The same standards of training, ***scrutiny of practice*** and Performance Review as seen in the operating theatre setting ***must*** apply to the emergency and prehospital environment.
- Medical practitioner involvement in the process/programme is essential (according to the American model) for…
- Adequate Quality Assurance monitoring and intervention to occur.
- Multiple intubation attempts are associated with increased morbidity and risk of death in the field. Early recognition of defined difficult and failed intubation is essential to limit further futile and harmful attempts.
- Backup 'rescue' airway devices and protocols ***must*** be available.

- Precautions must be put in place to prevent ETT dislodgement, including the practice of physically documenting tube depth (referenced at the teeth, or less satisfactorily, the corner of the mouth).

- There must be <u>continuous</u> vigilance of tube depth and monitoring of ETT placement. Consequently, tracheal intubation should *not* be carried out in the absence of continuous (preferably wave-form) capnography.

- All attempts must be made to avoid oxygen desaturation, bradycardia and hypotension, and attention must be focused on the poor outcomes associated with these indices.

- Both hypo- and hyperventilation must be avoided at all costs (emphasising the importance of capnography again).

- The Paramedic who intubates the patient is the person who must take ultimate responsibility for the airway and the resuscitation attempt, and is the person who should hand over the patient (and attendant responsibility) to the Emergency Department doctor. (ETT placement should be assessed by the receiving doctor <u>before</u> the patient is transferred from the ambulance stretcher/trauma board to the hospital trolley).

- Paramedics should be taught to self-train their "airway assistants" at the beginning of a shift, in order that the assistant knows exactly what is required of them during the intubation procedure. This should include the pre-intubation equipment check and patient preparation. The assistant should be thoroughly familiar with the drugs and equipment used, the anticipated sequence of events, and the protocols in the case of a difficult or failed intubation. Tracheal intubation and especially Rapid Sequence Intubation is *not* a single-operator procedure!

- All patients must be assumed to have a full stomach, and all patients should have cricoid pressure applied by the assistant – either indicated as Sellick's manouvre or as OELM.

- All Paramedics undertaking tracheal intubation should be fully conversant with Difficult Airway and Failed Intubation drills, including the Can't Intubate – Can't oxygenate (CICO) scenario.

- All Paramedics utilising DAI or RSI should demonstrate proficiency of their skills and keep a record (log book) of successful tracheal intubations, and the circumstances under which those intubations occurred.

EDUCATION AND TRAINING IN AIRWAY MANAGEMENT

Until recently, airway management knowledge and skills were acquired hapharzardly 'on-the-job' wherever training opportunities arose. During the long course of an intense specialist training, most anaesthesiologists were exposed to a sufficient number of challenging airway

cases to enable them to become competent in advanced airway management. Taking into account the ASA closed claims analysis, it must be questioned whether this belief has *ever* been justified.[28]

Every anaesthesia department should set minimum standards for airway management to ensure that every trainee receives structured training to meet such minimum standards.

The overall requirements of such a programme should include:

- The patient's safety is the highest priority; and therefore, the patient must be protected from the total novice.

- Basic airway knowledge and skills should be taught throughout the specialist training programme.

- Advanced airway techniques should be taught as soon as possible in the training programme.

- New techniques and devices (especially supraglottic airway devices) should be taught selectively after the acquisition of basic airway skills.

- The decision-making processes (integration of skills and techniques and the application of a difficult airway algorithm) should be taught as soon as possible.

- Senior trainees need to be taught how to teach airway management skills to junior trainees.

To meet the above, there should be a defined syllabus of core knowledge and skills, and a definition of basic and advanced airway techniques, according to the following:

BASIC AIRWAY MANAGEMENT TECHNIQUES:
- Facemask ventilation – single and two-handed
- Simple manouvres to open the airway (head-tilt, chin-lift, jaw-thrust)
- Insertion of oropharangeal and nasopharangeal airways
- Oral intubation under direct laryngoscopy
- Pulling back the upper lip to aid visualisation
- Use of cricoid pressure (OELM)
- Use of stylet and gum elastic bougie
- Use of steerable introducer
- Rapid sequence induction/intubation
- Use of laryngeal mask airway and "Proseal-equivalent" LMA
- Use of King Airway a.k.a. Laryngeal Tube

ADVANCED AIRWAY TECHNIQUES:
- Nasotracheal intubation
- Use of alternative blades (Macintosh and Miller)

- Use of tube exchange catheters or gum elastic bougie
- Intubating laryngeal mask airway (I-LMA)
- Use of Airtraq® optical laryngoscope
- Use of light wands eg. Laerdal
- Fibre-optic and awake fibre-optic intubation
- Retrograde intubation
- Percutaneous cricothyroidotomy
- Transtracheal jet ventilation
- Placement of double-lumen endotracheal tubes
- Surgical cricothyroidotomy

FACE-MASK VENTILATION

Bag-mask ventilation is the foundation 'starting block' of airway management!

The single most important skill required of an '*airway technician*' is that of bag-mask ventilation, in conjunction with the use of oropharangeal and/or nasopharangeal airways.

Substantial skill is required to establish and maintain an adequate mask seal, position the head, and ensure a patent airway all with one hand, whilst ventilating with the other hand.

The above includes simultaneous chin lift, jaw thrust and head tilt – pushing the mask onto the face (maintaining a facemask seal), whilst at the same time lifting the face into the mask, and additionally ventilating the patient.

THE "STARFISH MANEUVER"

The index finger and thumb are used to apply the mask to the face, while the little finger is applied to the angle of the mandible, facilitating the jaw-thrust.   The two middle fingers are applied to the bony mandible (and not the soft tissue under the chin), facilitating the chin-lift. The entire hand is used to simultaneously apply the head tilt.   Avoid the application of the operator's fingers to the soft tissues, as this will occlude the airway, especially in paediatric patients.

Adequacy of mask ventilation is determined by mask seal, assessment of the chest rising, bag compliance and skin colour, in addition to pulse oximetry.   Pulse oximetry measures oxygenation and *not* ventilation – there is an importance difference between the two!   This is further discussed in detail under *Ventilation in the Transport Environment*.

Adequacy of ventilation in a non-intubated spontaneously-ventilating patient can also be assessed by placing the stethoscope over the angle of the mandible.   Tidal flow of air can be assessed by auscultating the tracheal breath sounds, whether or not an oro- or nasopharangeal airway has been inserted.   Such assessment can take place prior to intubation or after extubation, and provides an immediate and clear indication of airway

obstruction.   This method is especially useful in children, as children can display normal chest movement even when totally obstructed.   Remember that SpO_2 is a late warning device, and is foolhardy to rely on to inform you of airway obstruction!
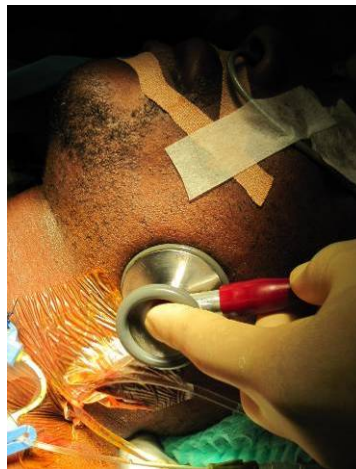
Remember that inadequate ventilation can be masked by an increased FiO_2, and an increased FiO_2 is almost universally administered during resuscitative attempts during airway, ventilation and other medical emergencies.   Inadequate ventilation results in hypercarbia, and this has serious implications in terms of cardiovascular stress, peripheral and cerebral vascular resistance.   This is discussed in detail under *Traumatic Brain Injury*, and *Ventilation in the Transport Environment*.

Bag-mask ventilation is not seen as a glamorous, impressive or challenging intervention, and *appears* easily done.   It therefore does not receive much focus of attention during airway and ventilation training.   Most healthcare providers under-estimate the degree of difficulty involved, and therefore over-estimate their own ability to adequately bag-mask ventilate patients.

Unless meticulous attention is paid to BVM ventilation, it is easy to deliver inadequate to non-existent ventilation without even realising it.   This is an uncomfortable truth that may never be discovered, as the vast majority of cardiac arrest resuscitation victims die, regardless of whether or not they are adequately ventilated.   Poor technique by an unwitting practitioner may thus never be discovered.

Unfortunately, training on live patients is an extremely limited privilege, and the artificial experience of ventilating a plastic manikin regrettably falls far short of a realistic true-to-life experience.

It is my anecdotal experience in training paramedic students in the operating theatre environment, that using a collapsible anaesthetic breathing bag (as opposed to a self-inflating BVM resuscitator), readily unmasks how surprisingly inadequate some ***senior and***

experienced ambulance personnel are at maintaining a good face-mask seal, and therefore adequate ventilation.

It may well be revealed in the future that poorly-executed BVM ventilation could be an even greater killer than missed oesophageal intubation! See later…

PREDICTORS OF DIFFICULT FACEMASK VENTILATION

Difficult facemask seal:

- Beards
- Edentulous patients
- Limited jaw protrusion

Difficult ventilation:

- Snoring patients
- Mallampati III and IV
- Asthma/chronic obstructive airways disease

In a recent study,[29] independent predictors of (i) difficult facemask ventilation and (ii) impossible facemask ventilation were identified thus:

Independent predictors of grade III (difficult to ventilate) facemask ventilation:

- Beard
- Mallampati III or IV
- 57 years or older
- Severely limited jaw protrusion
- Snoring

Independent predictors of grade IV (impossible to ventilate) facemask ventilation:

- Thyromental distance of less than 6cm
- Snoring

For those who like mnemonics, the following is a useful (but not comprehensive) list of predictors of difficult facemask ventilation:

MOANS:

Mask seal (bushy beards, edentulous patients)

Obesity/obstruction

Age greater than 55

No teeth

Stiff – limited neck mobility, stiff lungs: bronchospasm, pneumonia, etc.

Snoring – history of…

INTRA-ORAL FACE-MASK

For those patients with "collapsed" facial contours (such as the edentulous patient), and in bearded patients, in whom a good face-mask 'fit' is almost impossible to achieve, the intra-oral 'face-mask' is placed between the teeth and the lips.

The lips are positioned over the soft, pliable plastic once inserted, and pressure is applied with the operator's hand to create an air-tight seal.

An oropharangeal airway (supplied) can be inserted prior to insertion of the intra-oral face-mask.
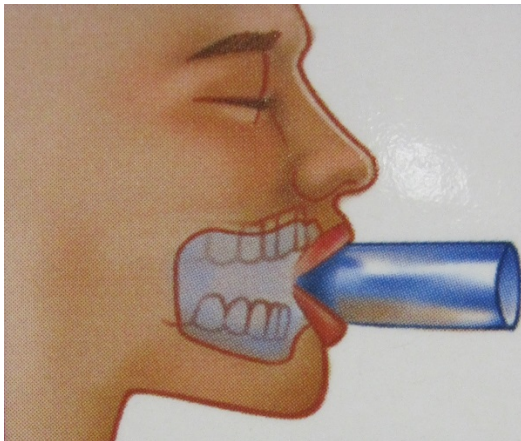
Nu-Mask

As it actually appears

Nu-mask in-situ – placed superficial to the teeth, but deep to the lips.

Nostrils must be occluded and pressure applied to lips to keep an air-tight seal.

The airway technician needs to have a planned approach to Emergency Department and prehospital intubation. This includes having a pre-thought-out "rescue plan" in the event of a failed intubation, and a "Can't Intubate, Can't oxygenate" scenario (CICO).

In addition, the airway technician needs to make an environmental assessment, a patient assessment, and a full assessment and preparation of equipment and drugs required for intubation.

Tracheal intubation training and practice can be conceptualized in terms of:

- The pre-intubation phase,
- The intubation phase itself, and
- The post-intubation phase

Furthermore, the sequence of action in terms of difficult laryngoscopy and/or failed intubation (according to UK Difficult Airway Society algorithms) can be broken down into:

- Plan A: Direct laryngoscopy

- Plan B: Laryngeal mask airway insertion

- Plan C: Revert to facemask ventilation

- Plan D: Cricothyroidotomy (needle or surgical)
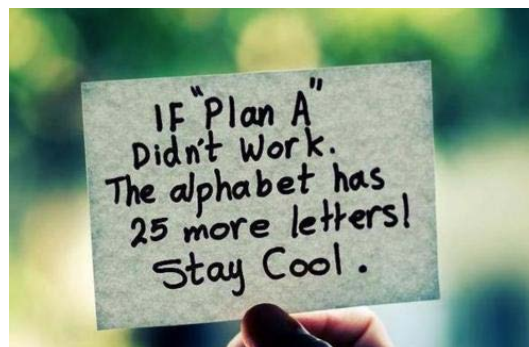
TRACHEAL INTUBATION – PREPARATION

THE DECISION TO INTUBATE

Emergency Medical personnel must be able to:

- Rapidly assess the **need** for intubation, and assess the **urgency** of intubation.
- Determine the most appropriate method of airway management.
- Use a *range* of airway devices.
- Decide which drugs are best to use.
- Manage the airway in the context of the patient's overall condition and projected clinical course.
- Consider the "fasting" status of the patient (see below) with respect to aspiration risk.
- Recognise the difficult and failed airway **early**, and institute an alternative "rescue" plan. (Have a "Plan B" up your sleeve).

Diligent maintenance of knowledge and skills, sound clinical judgement, and decisiveness to act are prerequisites in emergency airway management.   Indecision often leads to delayed intervention, a deteriorating clinical condition, and therefore increased difficulty and a greater likelihood of failure.

Optimal airway management, oxygenation and ventilation are the foundation upon which all subsequent resuscitative measures are based.

Indications for tracheal intubation:

- Failure of airway maintenance.
- Failure of ventilation.
- Anticipated clinical course (eg. multiple rib fractures, polytrauma patient with expected clinical decompensation, or burns to the head, neck or upper airway).

Any patient who requires an airway to be established, also requires that airway to be maintained.   Such patients almost invariably also require assisted positive pressure ventilation!   (This is discussed in detail in the lecture *Ventilation in the Transport Environment).*

The gag reflex does not correlate well with airway protection and is of no clinical value in assessing the need for intubation.

The assessment of spontaneous or volitional swallowing is a better tool for assessing the ability to protect the airway.

In cases where intubation is indicated to facilitate ventilation, assisted *active positve pressure ventilation **must** be administered.*   Consider the institution of "T-piece ventilation" for

conditions such as asthma or bronchiolitis.   This is further discussed under *Ventilation in the Transport Environment.*

PRE-INTUBATION FASTING

Pulmonary aspiration of gastric contents is associated with significant morbidity and mortality. ASA recommendations on pre-operative fasting in normal, healthy sub jects is as follows:

Clear (non-particulate) fluids:	2hrs
Breast milk:	4hrs
Light meals:	6hrs

The trauma patient is **never** considered to be fasted, as pain and other causes of sympathetic nervous system stimulation cause unquantifiable delayed gastric emptying, as does the administration of opiate medication.

Poorly-controlled diabetes mellitus, renal failure and sepsis have similar effects on delayed gastric emptying. Patients with gastro-oesophageal reflux and increased intra-abdominal pressure (such as occurs in pregnancy and obesity) are predisposed to passive regurgitation of gastric contents.

Gastric emptying is enhanced with prokinetic agents such as metoclopramide.   The IV route is better than the oral route, although no particular benefit has been demonstrated in the trauma patient.

H_2-receptor blockers and proton-pump inhibitors should be routinely administered through labour in high-risk patients, or should be adminstered the night before for elective surgery in pregnant and obese patients (eg. Ranitidine 150mg P.O.).

Sodium citrate is a non-particulate antacid, and is routinely administered as 30ml of 0.3M solution prior to intubation in pregnancy.

PRE-INTUBATION PHASE

ENVIRONMENTAL ASSESSMENT

It goes without saying that the immediate environment must be checked for hazards to the patient and rescuer before commencement of resuscitation.   In terms of intubation, special attention needs to be given to the presence of bright sunlight and other bright lights such as car headlights facing the airway technician.   As one's eyes accommodate to the surrounding bright light, the light of the laryngoscope may be inadequate to illuminate the posterior pharynx and glottic area.   Ambient light may need to be shielded by assistants holding up

blankets or trauma boards, or a blanket may need to be placed over the head of the airway technician and patient during intubation.

PATIENT ASSESSMENT

The patient must be assessed for:

1. The need and urgency for intubation.

2. Predictors of difficult intubation (see later).

3. Tracheal tube size.

4. Adequacy of patient positioning.

5. Ability to urgently reposition the patient if required.

EQUIPMENT ASSESSMENT AND PREPARATION

All necessary equipment and drugs must be checked and prepared, as follows:

1. Oxygen supply (cylinder). Checked full, with no leaks.

2. Bag-valve mask resuscitator (with masks and reservoir bag) attached to oxygen supply. Check that one-way valve is correctly assembled and that BVM can generate a positive airway pressure (by occluding the gas outlet and squeezing the bag).

3. Oropharangeal airways.

4. Laryngoscope with range of blades, spare bulbs and batteries.   Check blade connection to handle and apply pressure in all directions, ensuring constant contact and no faltering of light.   (Remember, in the State sector you are usually using equipment provided on the lowest tender price!)

5. Range of endotracheal tubes (with cuffs checked).

6. Suction unit with soft suction catheters and Yankhauer or equivalent rigid suction catheters.

7. Alternative airway devices (LMAs, Comitubes, Layngeal Tubes, etc.)

8. Rubber coated malleable stylets, gum elastic bougies and/or steerable (Parker) introducers.

9. Patient monitoring equipment: ECG, NIBP, SPO_2, $ETCO_2$.

10. Intubation and resuscitation drugs and fluids as required (according to protocols and scope of practice): Atropine, adrenalin, benzodiazepines, neuromuscular blockade, IV fluids and vasopressors (ephedrine, phenylephrine) as appropriate.

    Note: syringes must be clearly labelled!

Whilst simultaneously pre-oxygenating the patient and assisting ventilation where necessary, drugs and equipment must be prepared, IV access and appropriate monitoring must be established, and the patient adequately positioned.

PATIENT POSITIONING

Non-trauma patients should be positioned in the classic "morning sniff" position, with neck extended and the occiput raised from the surface by approximately 5cm, achieved by placing a flattened blanket roll or similar under the head. A good rule of thumb is to ensure that the earlobes are higher than the sternal notch (or clavicles) in the supine patient.

Non-trauma patients should have extra blankets and/or padding placed under the head, neck and upper back to ensure that the lobe of the ear remains above the height of the clavicles. Obese and pregnant patients are almost impossible to reposition after induction, owing to their dead-weight. It is therefore critically important to position them properly before induction or intubation takes place. See the accompanying photographs of the "ramp" position.

"Morning sniff" and "ramp" positions illustrated:
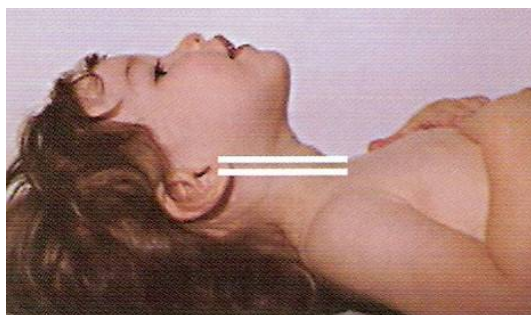
Note earlobe to sternal notch

(or to clavicle in mid-clavicular line).

This is not widely taught and is especially important in obesity!
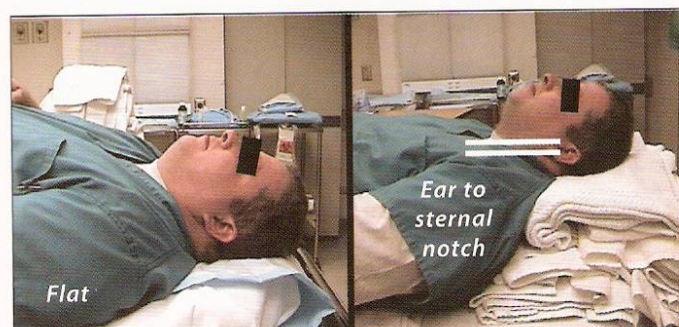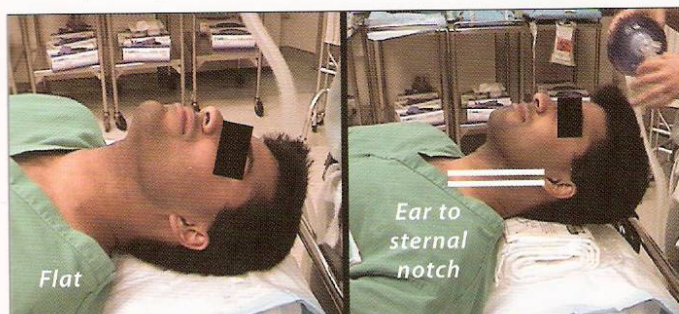
Positioning such as this (and middle right above) is known as the "ramp" position for obvious reasons.

How not to do it:
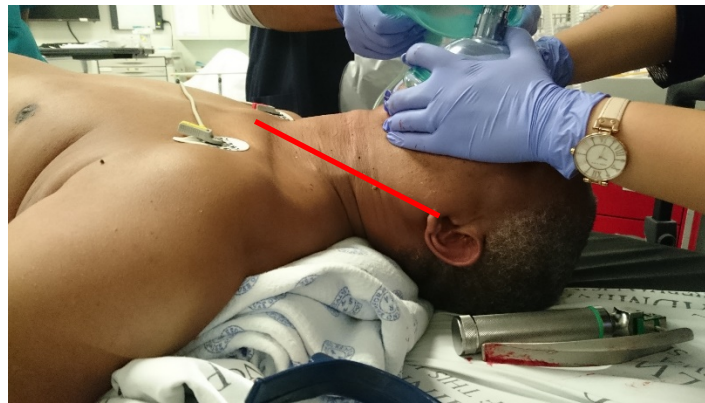
The practitioner here struggled for over 30 minutes to intubate this patient – notice the blood on the laryngoscope blade. Shifting the blanket roll from under the neck to under the occiput facilitated immediate intubation with ease.

Trauma patients should have all C-spine precautions observed, but should still place patient in "earlobe to sternal notch" position with appropriate occipital padding and cervical spine support. Slight movement of the cervical spine is not important as there is no evidence that slight movement of the cervical spine causes further injury.

Maiman *et al* showed that a force between 645 and 7439 Newtons is required to cause spinal injury. It is the force of a ***strong impact*** that causes injury and not minor post-injury movement of the cervical spine.[30]

OPTIMAL EXTERNAL LARYNGEAL MANIPULATION

The airway technician must be ready to apply or request the application of cricoid pressure (for rapid sequence intubation) or optimal external laryngeal manipulation (OELM) in order to ***protect the airway from aspiration <u>and/or</u> improve visualisation of the vocal cords***. The use of cricoid pressure or OELM is extremely valuable in transforming a Cormack & LeHane Grade III or IV view to Grade I or II. This is under-taught and under-emphasized, or not taught at all to junior doctors, paramedics, etc.

In a recent 2017 study of 2365 Emergency Department tracheal intubations in non-cardiac arrest patients, a significantly higher success rate was found in the RSI group vs non-RSI group (73% vs 63%), without any difference in complication rates detected.[31]   Could it be that the "OELM-effect" of cricoid pressure could have improved views of the glottis structures, improving intubation success rates?   This was not postulated by the authors, but was perhaps a significant oversight on their part.

GREENLAND'S CURVES

The conceptual model of Greenland's curves explain the reasons why the "earlobe to sternal notch" or "ramping" position works so well.   Greenland describes a primary and secondary curve, both of which need to be straightened out in order to obtain unobstructed glottis views.[32,33]
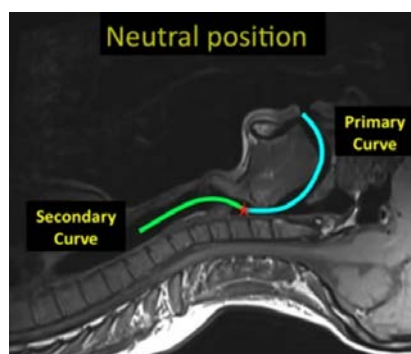
Greenland's curves
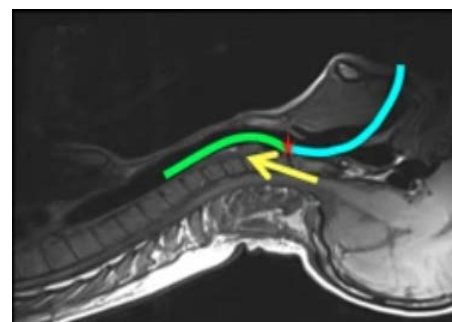
Hyperextension straightens out the primary curve.

Hyperextension straightens out the primary curve, assisted with a laryngoscope.   Note that this position still presents a difficult airway situation, as the tracheal tube must still be angled upwards in order to pass through the vocal cords and into the trachea.   (It is for these types of situation that the Parker steerable introducer was developed).
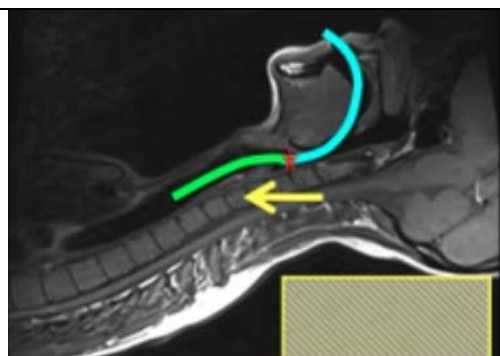
Heal elevation by itself helps to straighten out the secondary curve without affecting the primary curve.   Additionally, application of cricoid pressure (OELM) assists to straighten out the secondary curve.
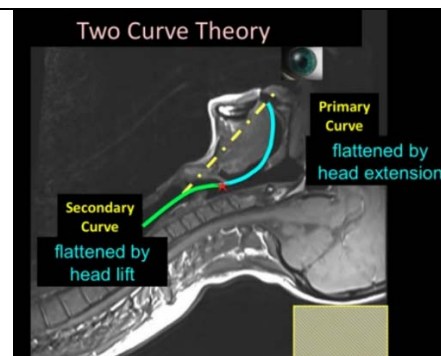
Combining head lift with head extension flattens out both curves, substantially improving direct visualization of the glottis structures.

A short-handled laryngoscope and an extra assistant may be required to provide downward (caudad) traction on large breasts in the case of pregnancy and/or obesity.

Patients should ideally be intubated on trolleys, stretchers or operating tables that can rapidly be placed into the Trendelenberg (head-down) position (where this luxury exists!). Trendelenberg position often improves visualisation of the vocal cords in its own right, but is essential in the event of a patient regurgitating or actively vomiting.   On regurgitation or vomiting, the patient should immediately be turned into the left lateral position and simultaneously placed in Trendelenberg position, to facilitate the gravitational flow of stomach contents from the posterior pharynx out of the mouth, rather than through the glottic opening and into the lungs.

The reason why left lateral position, as opposed to right lateral, is advocated, is because it is possible to shift the tongue out of the way and gain a view of the vocal cords with a left-handed laryngoscope, whilst in left lateral position (as the tongue falls away from the visual axis, owing to the action of gravity.   This is not easily achievable in the right lateral position, as the tongue must be lifted up against gravity, and balanced on the laryngoscope blade – an impossible task.

The easiest and quickest method of placing a supine non-trauma patient into a lateral position is to lift the arm furthest from the pactitioner into the vertical position, holding the arm at the wrist.   Grab the far leg at the knee, simultaneously bend the knee, and pull both the knee and the arm in the direction of the practitioner.   This method provides a great amount of leverage and even very large patients can easily be moved in this manner.   This technique will be illustrated and demonstrated during the accompanying Powerpoint presentation.

Trendelenberg position is also useful in countering residual hypotension following the intubation procedure, whilst waiting for IV fluid boluses and vasopressors or inotropes to take effect.

PRE-INTUBATION MONITORING

ECG, NIBP, SPO_2 and $ETCO_2$ should all be available and attached for measurements before, during and after intubation, where time and practicality exist.   Please note a word of caution – do not substitute a cardiac monitor with a pulse oximeter.   With the onset of a sudden bradycardia there will be loss of cardiac output, and the pusle oximeter will give the practioner no more information than to alarm "searching for pulse"!

Pulse oximetry and capnography are dealt with in great detail in separate lectures on these two subjects, respectively.

In addition, the following times should be monitored for clinical audit purposes:

1. Time at which decision is taken to intubate.
2. Time at which practitioner is ready to intubate (equipment prepared and checked).
3. Time of first attempt at laryngoscopy.
4. Time of confirmed successful placement of ET tube.

PHARMACOLOGY

Depending on circumstances, protocols and scope of practice, the airway practitioner may elect to draw up certain drugs prior to intubation.   Whether drawn up or not, emergency intubation and resuscitation drugs should at least be within arm's reach.   It is critically important to label syringes clearly.   It may be worthwhile for individuals or services to compile quick-reference pre-printed cards with dosages according to age and/or weight, (as is done with the Breslow® tapes).   Please see the addendum for a list of essential and other useful peri-intubation drugs.

Please note that there is currently *no evidence of benefit* for the administration of lignocaine 1 to 2mg/kg IV to ablate the cardiovascular response to intubation.

50th centile Body Weight (kg) = Age (yrs) x 2 + 9

CALCULATING ET TUBE DIAMETER AND DEPTH

As with drug dosages, it is very useful to have quick-reference pre-printed cards with ET tube internal diameter and depth guidelines.   My experience of using the small finger as a guide to ET size is highly inaccurate.   The following formulae are far more useful, and should be committed to memory.

ETT size (internal diameter in mm) = 4 + $\underline{\text{Age (yrs)}}$

$$4$$

ETT depth in cm (at teeth/gums) = 12 + $\underline{\text{Age (yrs)}}$

$$2$$

Another more user-friendly formula for ETT depth at the teeth (as taught by ATLS) is:

Internal diameter (mm) divided by 3 = distance at the teeth in cm.

It has been classically taught that, as a rule of thumb, tube distance at the teeth is approximately 21cm in adult females and 24cm in adult males.   However, new evidence suggests **20cm** in the adult female and **22cm** in the adult male as a more accurate and safer guideline.[34]   Please see the section on "referencing tracheal tube depth" (p64) under "clinical tests for confirming correct placement of the tracheal tube".

As an additional rule of thumb, a 'small' adult female will accept a size 7mm ETT, and an 'average' female will accept a 7.5mm ETT.   A 'small' adult male will accept a size 7.5mm ETT, and an 'average' adult male will accept a size 8mm ETT.

PRE-OXYGENATION

Pre-oxygenation is achieved with gentle (low pressure) assisted ventilation as necessary. High airway pressures (above 15cmH$_2$O) lead to gastric inflation, splinting of the diaphragm with consequent ventilation difficulty, and increased risk of aspiration.

In a Position Paper on Prehospital Intubation, the U.S. National Association of EMS Physicians recommended that "pre-oxygenation should preferably be performed by permitting the patient to breathe using a non-rebreather mask until oxygen saturation is 100%".[10]

They make no mention of assessing adequacy of *ventilation*.   Similar protocols are followed in numerous other studies, not least of which is the San Diego trial.   I am surprised at this (what I consider to be a seriously flawed) recommendation, since hypoventilation is extremely common in this group of patients, (especially following traumatic brain injury, the administration of benzodiazepine sedatives and/or opiate analgesics) and would almost certainly be masked by the administration of high concentration supplemental oxygen.   Of additional concern is that hypercapnia and its consequences would not be addressed.

Baillard and colleagues studied critically ill hypoxaemic patients requiring intubation in the Intensive Care Unit.[35]   They demonstrated a mean SpO$_2$ of 98% in those receiving non-invasive (face mask) assisted ventilation, in contrast to a mean SpO$_2$ of 93% in the non-positive pressure ventilation group.   They also recommended the use of PEEP valves to maintain positive pressure (see PEEP and Functional Residual Capacity in the next section below).

High-concentration oxygen can be more effectively administered via a BVM resuscitator with reservoir bag whilst cautiously providing assisted ventilation.   Be aware that some BVM resuscitators will not deliver supplemental oxygen whilst lightly held over the face of a spontaneously breathing patient, and especially without a good face mask seal.

If and when ventilation is judged to be inadequate, ventilation can easily be assisted without wasting critical seconds and taking the trouble to change oxygen delivery devices. Moreover, a BVM resuscitator will be required post-intubation, in any event.   There is an indisputable and irresistible logic to having a BVM resuscitator at hand and connected to a portable oxygen supply prior to intubation!

Patients should preferably be pre-oxygenated in a 20 degree head-up position, as opposed to lying in the flat supine position.   A slight head-up position decreases the pressure of the

abdominal contents on the diaphragm, thereby increasing Functional Residual Capacity (see below).

Lane and colleagues demonstrated that, in pre-oxygenated patients, time to desaturation to an SpO_2 of 95% took 386 seconds in the head-up group, as opposed to 283 seconds in the supine group.[36]   The head-up position thus increases time to desaturation by 36%.

SUPPLEMENTAL OXYGEN AND FIO₂

There is considerable confusion about the actual percentage of oxygen that can be delivered through a non-rebreather oxygen mask.   Many well-conducted studies have shown that the maximum percentage oxygen that can be delivered through a "non-rebreather" mask is 70% to 75% at a flow rate of 15L/min, since the mask does not form an effective seal to prevent the entrainment of ambient air, there are no one-way valves and the reservoir is too small to meet the large flow requirements during inspiration.   The "non-rebreather" mask is therefore erroneously named.[37]

APNOEIC OXYGENATION AND NO DESAT

Oxygen is taken up from the alveoli at a rate of 250ml/min to meet basal requirements whilst at rest.   As a result, the alveolar partial pressure of oxygen (P_AO_2) drops during periods of apnoea.

This partial pressure gradient generates a mass flow of oxygen along a concentration gradient from pharynx to alveoli.

At the same time, only 8 to 20ml/min of carbon dioxide crosses from the bloodstream into the alveoli.[37]   This is because the considerably higher solubility of carbon dioxide in the blood allows it to stay within the bloodstream.

As a result of the above, the arterial partial pressure of oxygen (P_aO_2) can be maintained above 13.3kPa for up to 100 minutes without a single breath being taken.   This is, however, associated with marked hypercapnia and respiratory acidosis.[38]

Taha *et al.* and Ramachandran *et al* demonstrated in two separate studies that nasal prongs (NPO_2) oxygenation at 5L/min can significantly prolong time to desaturation, and that the mean SpO_2 remains significantly higher in apnoeic patients during the intubation process.[39,40] Taha demonstrated that no desaturation takes place for up to 6 minutes with NPO_2 at 5L/min, following the onset of apnoea, where his control group (no NPO_2) desaturated to 95% within 3.65 minutes.

NO DESAT: **N**asal **O**xygen **D**uring **E**fforts at **S**ecuring **A** **T**ube

A pharyngeal FiO_2 of almost 100% can be achieved with NPO_2 at a flow rate of 15L/min. Mouth opening does not affect FiO_2 is not affected by mouth-opening. Nasal prongs can be left in place during face mask ventilation and intubation attempts. This is referred to as the "NODESAT" technique.[37]

<u>THRIVE: Trans-nasal Humidified Rapid-Insufflation Ventilatory Exchange:</u>

Significantly extended apnoea times of 25 patients with difficult airways, who were undergoing general anaesthesia for hypopharyngeal or laryngotracheal surgery, was achieved through continuous delivery of trans-nasal high-flow humidified oxygen at 70L/min.[41]

The high-flow trans-nasal oxygen was initially intended to provide pre-oxygenation, and continued as post-oxygenation during intravenous induction of anaesthesia and neuromuscular blockade until a definitive airway was secured. Upper airway patency was maintained with jaw-thrust while trans-nasal humidified high-flow oxygen was administered. There were 12 obese patients and nine stridulous patients in the group.

The median apnoea time was 17 (5 – 65) minutes. No patient experienced arterial desaturation < 90%.

Mean post-apnoea end-tidal carbon dioxide level was 7.8 kPa; the rate of increase in end-tidal carbon dioxide was 0.15 kPa/min.

This procedure combines the benefits of 'classical' apnoeic oxygenation with continuous positive airway pressure and gaseous exchange through flow-dependent deadspace flushing.

THRIVE works by more than simply passive apnoeic oxygenation:

- Positive Airway Pressure (PEEP) of $7cmH_2O$.
- Splints upper airways enhancing patency.
- Reduces shunt (through reduced deadspace).
- High flows facilitate oxygen delivery and carbon dioxide removal.

The disadvantage of THRIVE is that it requires highly-specialised equipment in order to administer high-flow transnasal oxygen. However, investigations are in progress which seek to examine if this can be achieved for shorter periods using normal nasal cannulae.
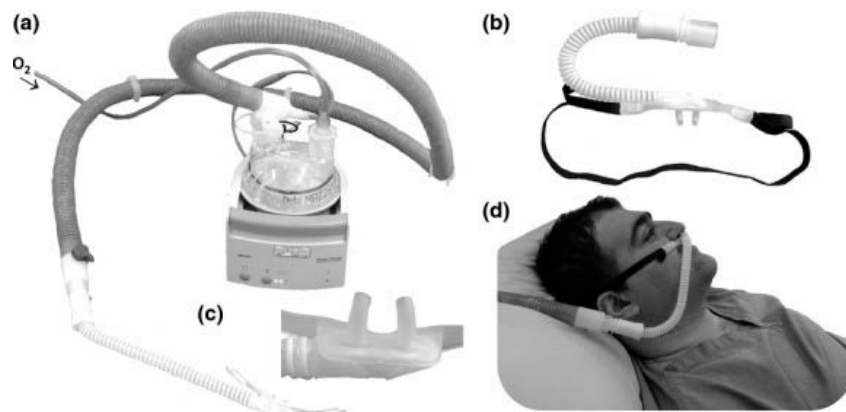
The OptiFlow high-flow humidified oxygen delivery system. The oxygen humidification unit (a) receives oxygen from a standard oxygen regulator and delivers humidified oxygen to a custom-built trans-nasal oxygen cannula (b and c) like a standard nasal oxygen cannula (d).
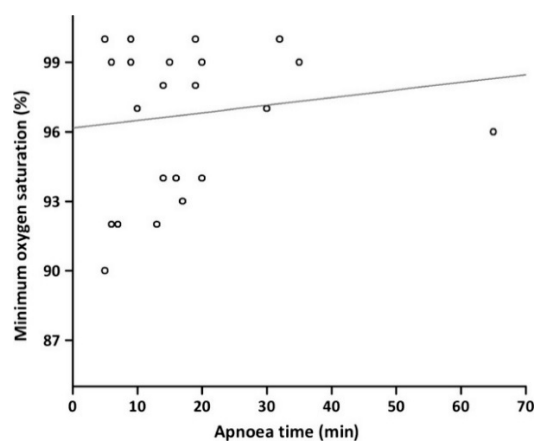
Apnoea time vs SaO$_2$
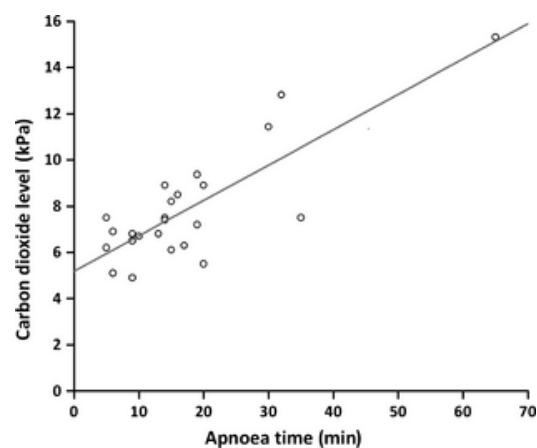
Apnoea time vs ETCO$_2$

FUNCTIONAL RESIDUAL CAPACITY AND TIME TO TERMINAL DESATURATION:

Pre-oxygenation is essential during the pre-intubation phase, during both RSI and non-RSI intubation.   Desaturation is related to Functional Residual Capacity (FRC), which is the sum of Expiratory Reserve Volume and Residual Volume.

FRC is normally in the order of 30ml/kg, and is markedly reduced in pregnancy, obesity, restrictive pulmonary disease, and instances where the stomach has been inflated with air (as occurs with high-pressure face-mask ventilation).   When FRC is reduced, oxygen desaturation occurs rapidly, for reasons explained below:

41

FRC can be considered the "reserve oxygen tank inside the chest".   In other words, FRC determines the amount of oxygen left in the lungs at the end of passive exhalation, and this determines the time to desaturation.
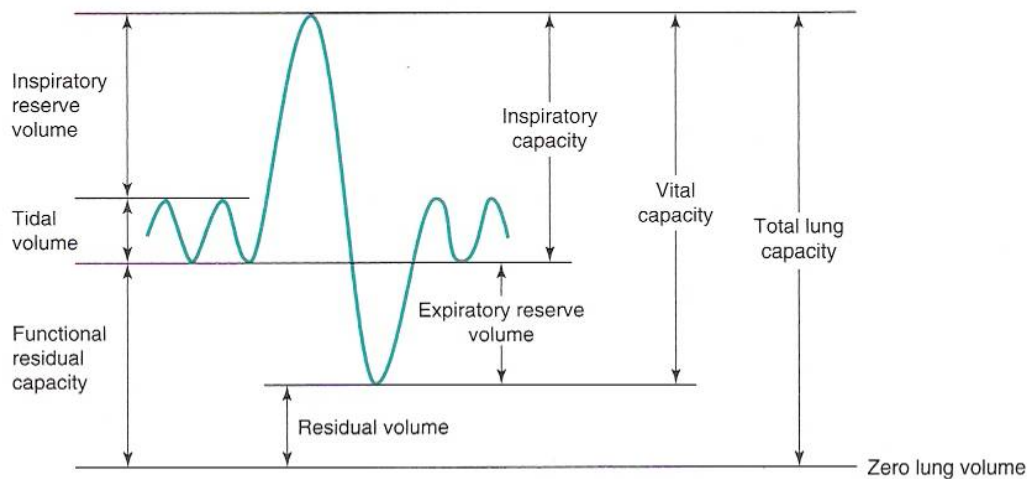
For example, if FRC is 2000ml and a patient breathes 20% oxygen, "oxygen reserve" is 400ml.   If basal oxygen consumption is 200ml/min, then desaturation occurs approximately 2 minutes after the last breath (in a non-preoxygenated patient). If the patient now breathes 100% oxygen, the "oxygen reserve" is now 2000ml.   At basal oxygen consumption of 200ml/min, desaturation will now take 10 minutes.

Please note that the above is an over-simplified mathematical model, as FRC and basal oxygen consumption have been rounded off for the sake of mathematical simplicity, and water vapour pressure (6.3kPa) has not been factored into the above calculation.

Administration of 100% oxygen for 3 minutes, whilst breathing normally, displaces nitrogen from the lungs, allowing several minutes of apnoea (in healthy lungs, with normal FRC) before desaturation occurs.   Similar preoxygenation can be achieved with eight Vital Capacity breaths.

Preoxygenation does not only serve to "top up" the FRC, it also creates an oxygen surplus in the bloodstream (dissolved in plasma) and in metabolically active tissues.

The below graphs demonstrates two important facts.   The first is that oxygen desaturation from 100% to 90% is a relatively slow process; however, desaturation from 90% to 0% occurs extremely rapidly.

The second fact is that it is a false belief that most patients will achieve neuromuscular recovery from suxemethonium before reaching terminal desaturation (saturation at which unconsciousness and no respiratory drive occurs).   This dispels the fallacy of the notion that suxemethonium is a short-acting muscle relaxant under these circumstances!   This is further discussed under *Neuromuscular Blockade*.
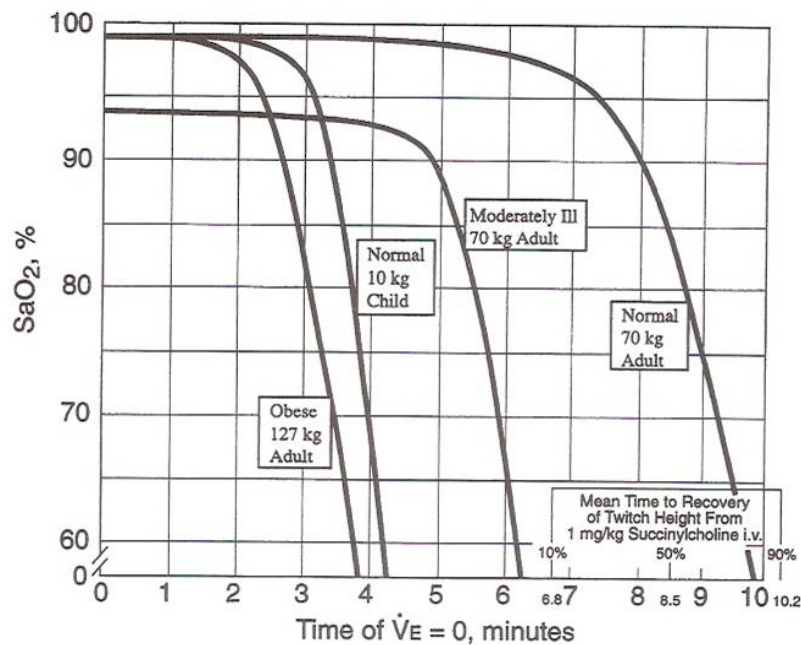
CLOSING CAPACITY

Another important consideration, in terms of lung mechanics, is that of Closing Capacity. Small airways, lacking rigid cartilagenous support, depend upon outward radial traction – provided by elastic recoil of lung parenchyma – to keep them open.   Patency of these airways is highly dependent on lung volume.   The volume at which these smaller airways begins to close (in dependent areas of the lung) is known as closing capacity.   At lower lung volumes, alveoli continue to be perfused, but not ventilated, and intrapulmonary shunting occurs.

Closing capacity is normally well below FRC, but rises with age.   At the age of 44, closing capacity equals FRC in the supine position, and by age 66 equals FRC in the upright position.
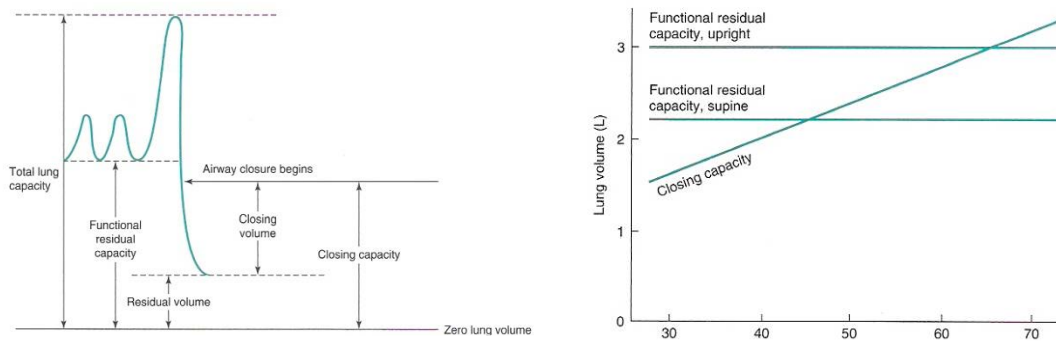
INTUBATION PHASE

Normal intubation non-RSI, non-trauma) will be discussed in a stepwise approach, followed by a discussion of Rapid Sequence Intubation (RSI).

All of the following steps are illustrated by means of detailed photographs and diagrams in the accompanying Powerpoint slide presentation.

1. The patient's head and neck must be manipulated into the classic "morning sniff" position.
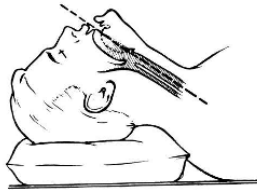
2. The patient must be pre-oxygenated with the mask gently applied to the face.

3. Supplemental ventilation may be administered if the patient's tidal volumes and or respiratory rate is judged to be inadequate.

4. Once the patient is sedated and/or relaxed, prepare for laryngoscopy.

5. The head and neck are extended with the right hand manipulating the head.

6. The laryngoscope blade is placed in the right-hand corner of the mouth, and gently introduced deeper into the oral cavity.

7. The tongue is picked up with the flange of the blade (left curled edge) and then manipulated into the centre of the oral cavity.

8. *Do not fulcrum the blade on the teeth!!*

9. The blade is introduced further into the oral cavity, whilst looking for the epiglottis.
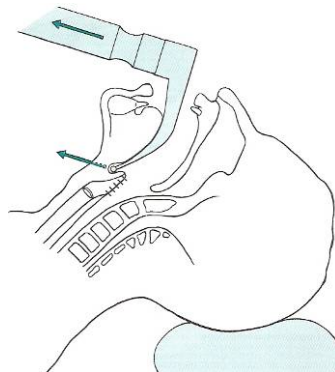
Tip of blade inserted into vallecula

Laryngoscopic anatomy

Macintosh blade inserted into valecula

Vocal cords

Arytenoid cartilages

epiglottis

glottis

esophagus
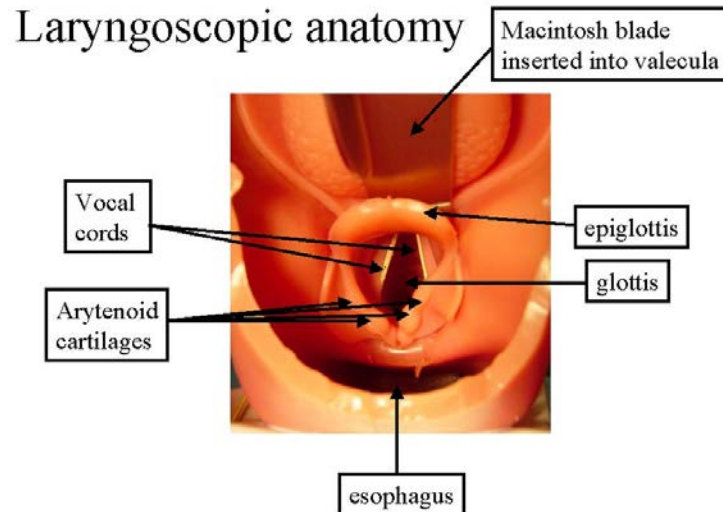
10. If the upper lip obscures the view into the oral cavity/hypopharynx, ask the assistant to retract the upper lip with a finger.

11. If the patient has a "passion gap", consider the placement of a "reversed" oropharangeal airway into the gap (as illustrated).   Degree of difficulty associated with a passion gap is wholly under-estimated, as either the laryngoscope blade or the ET tube tends to track into this gap, making manouvreability of both laryngoscope and ET tube extremely difficult.

12. If the epiglottis is not visible, the blade may not be advanced far enough, or conversely it may be advanced too far (and often is).

13. Under such circumstances, consider withdrawing the blade slowly, until the epiglottis "flops" into view.   (Some courses teach students to intentionally advance the blade 'too deeply', and then to slowly withdraw.

The Rock of Gibralter: Sailors of old, in trying to find the entrance to the Mediterranean Sea never looked for the actual entrance to the Mediterranean Sea – they focused on the Rock of Gibralter instead.   By finding the Rock of Gibralter, they knew – by relation – exactly where the entrance would be.   The same can be said about the epiglottis and the vocal cords.   Always first look for the epiglottis before looking for the vocal cords – because even if the vocal cords cannot be seen, if you can see the epiglottis then you will know where the vocal cords should be!
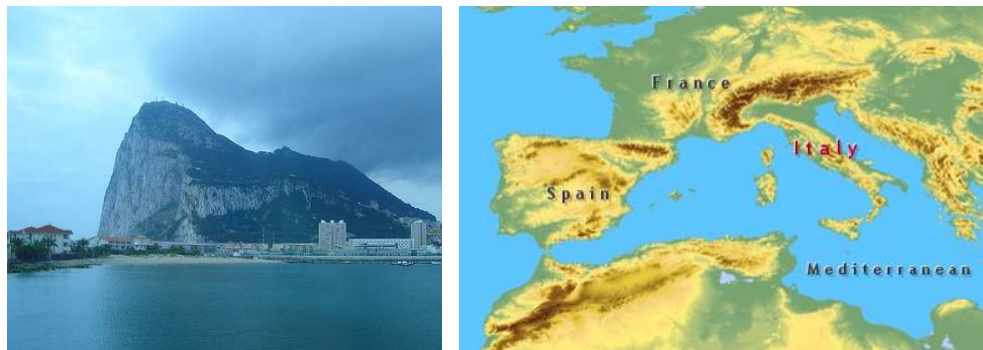
The Rock of Gibralter heralding the entrance to the Mediterranean Sea

14. Aim to introduce the tip of the blade into the vallecula.

15. Once the tip of the blade is in the vallecula, minimal further advancement and lifting of the blade will lift the epiglottis and bring the vocal cords into view.

16. If you can see the epiglottis, but can't visualise the cords, consider the application of cricoid pressure as Optimal External Laryngeal Manipulation.

17. If cricoid pressure is already being applied, consider the slow progressive release of pressure, as the over-zealous administration of cricoid pressure itself can distort the anatomy of the hypopharynx and glottic opening, completely obstructing the view of the cords.

18. You can often "talk the cords into view" by giving verbal commands to your assistant, eg. "Apply more pressure to the left/right, apply more/less pressure now".

19. Failing talking the cords into view, you can apply OELM with your right hand whilst holding the laryngoscope in the left, and then asking your assistant to mimic exactly what you have done.

20. If you can see the cords, but are unable to adequately guide the ET tube through the cords, consider repositioning of the patient, the use of a rubber-coated malleable stylet, steerable (Parker) introducer, or the use of a gum elastic bougie.

21. Inflate the ETT cuff once the tube has been placed through the cords.

22. Inflate the cuff slowly, whilst listening at the mouth for an air leak, and simultaneously applying positive airway pressure.   Inflate the cuff to a pressure that just prevents an air leak.   This precludes against over-inflation.

23. Confirm ETT placement clinically and with the use of adjunct devices.

24. Release of cricoid pressure is only allowed *after* ET tube placement is confirmed and the cuff has been inflated.

25. Note the tube distance at the teeth (or corner of the mouth) and **document** this.

It has been classically taught that, as a rule of thumb, tube distance at the teeth is approximately 21cm in the adult females and 24cm in adult males.   This is not a 'hard and fast' rule. Please see the section below where 20cm in females and 22cm in males has been advocated – and which doctrine is supported on this course.

26. Secure the ET tube.   ***The airway is not secure until the ET tube is secure!   (And the ETT is not secure until the head and neck are immobilised).***

27. ***Re-confirm*** ET tube position after securing the tube.   Beware migration of the tube into the right mainstem bronchus.

28. Do ***not*** strap an oropharangeal airway into the mouth whilst securing the ET tube – this can cause considerable swelling, distortion and pressure sores in the mouth and of the tongue.   This practice is frowned upon and is to be strongly discouraged, except where armoured tubes are used (discussed elsewhere).

RAPID SEQUENCE INDUCTION/INTUBATION

Some people seem to forget that the 'R' in 'RSI' stands for RAPID, and there's a 'R'eason for that!   The purpose of RSI is to minimise the exposure of an unprotected airway to aspiration of gastric contents.   The emphasis is on *"rapid"*, and therefore **time** is of critical importance during RSI.

The classic Rapid Sequence Induction is described as follows.   Any variant on this theme is known as "Modified Rapid Sequence Induction".

- The patient is pre-oxygenated (but not ventilated).
- A precalculated dose of sodium thiopentone is given as a rapid IV bolus, followed by a rapid precalculated dose of suxemethonium.
- **Cricoid pressure is immediately applied** and maintained (until the verbal command to release cricoid pressure is given, following confirmation of correct ET tube placement).

- The patient is observed for fasciculation of tongue, face and neck muscles.
- Once fasciculations have faded (or 30 to 45 seconds have passed if no fasciculations are observed), the patient is swiftly intubated.
- The ET tube cuff is inflated.
- ET tube position is confirmed (auscultation and capnography).
- Cricoid pressure is released _**after**_ correct tube placement is confirmed.
- The ET tube is secured.
- Tube placement is reconfirmed after strapping.
- Distance markings at the teeth are noted and documented.
- The patient is ventilated.

Caution: Note that in obese patients, and/or those with large breasts, it may not be possible to insert a standard laryngoscope, as the handle often snags on the hand of the assistant applying cricoid pressure.   Under these circumstances a short-handled laryngoscope can be used, if available.   Polio blades are an alternative, but in practice are seldom available outside of the obstetric operating theatre suite, and even then are sometimes little more than wishful thinking.

   If a short-handled laryngoscope or polio blade is not available, the standard laryngoscope is inserted sideways, with the handle pointing to the patient's right shoulder rather than to the patient's feet.   Once the blade is inserted as deeply as possible, the laryngoscope is correctly orientated, with the handle turned downwards towards the feet.

Caution: Note that cricoid pressure is capable of displacing the cervical spine approximately 5mm in normal patients, suggesting that it might present a hazard in patients with unstable cervical spines.   Notwithstanding this, cricoid pressure is still routinely applied to all trauma patients considered to be at risk of aspiration.

There are three large studies assessing the effects of cricoid pressure on c-spine movement in trauma patients, and there are no documented cases of secondary iatrogenic neurological injury recorded in the literature.[42-44]

Modified Rapid Sequence Induction includes the use of Fentanyl, Midazolam, Propofol, Etomidate, Ketamine and Rocuronium.

The use of gum elastic bougies is dealt with in detail in the section on alternative and 'assist' airway devices.

Additionally, when the BURP manoeuvre is applied (cricoid ring directed backwards, upwards and rightwards), airway obstruction is **28 times more likely** than when simple backward pressure is applied.[45]

CRICOID PRESSURE AND ASPIRATION

Aspiration is the largest cause of death associated with airway management in UK anaesthesia.   The NAP4 audit demonstrated that, of 29 aspiration events, eight patients died and two were left permanently brain-damaged – yielding a long-term morbidity and mortality of one third.[46,47]

The Royal College of Anaesthetists and the Difficult Airway Society therefore strongly recommend the application of cricoid pressure whilst intubating patients with potential full stomachs.

CONFIRMING CORRECT PLACEMENT OF THE TRACHEAL TUBE:

Confirmation of tube placement is done firstly by:

1. Clinical tests
    a. Auscultation
    b. Misting of the tube
    c. Referencing tube depth
2. Non-clinical adjuncts:
    a. Oesophageal detector device (EDD)
    b. Colorimetric devices
    c. Capnography

Caution:

Note that **all** clinical and non-clinical adjuncts to confirming correct ET tube placement are "**single snapshots in time**" and become irrelevant historical data immediately following completion of the test.   The **only** exception to this rule is the institution of **continuous end-tidal capnography**.

Note, however, that $ETCO_2$ will not reliably warn of tube dislodgement into the hypopharynx (above the cords) or into the right mainstem bronchus. $ETCO_2$ may also be unreliable in confirming tube placement in the trachea (false negative) where there exists either inadequate circulation or ventilation during CPR.

CLINICAL TESTS:

Observing the tube passing through the vocal cords:

It is not always possible to observe the tube passing through the vocal cords, even when the cords are easily viewed (Cormack and LeHane Grades I and II), as in some instances, the tube itself obscures the vocal cords as it is advanced through the cords.

Seeing the tube passing through the cords is always reassuring, but is ***NOT*** a reliable method of assessing tube placement, as it occurs fairly frequently in the controlled environment of the operating theatre that the practitioner "swears blind" that he or she saw the tube pass through the cords, yet further confirmatory tests prove oesophageal placement! The lesson to learn from this is that, no matter how good you think you may be, or however confident you are, ***NEVER*** fully trust your own eyes! Never pit your personal pride against a patient's life, and ***always*** use further clinical and non-clinical adjuncts to confirm tube placement.

a) Auscultating the left axilla:

Some advocate the second step to confirming tube placement should be to auscultate the epigastrium. Whilst this not a bad dictum to teach, I prefer auscultating the left axilla first, as breath sounds in the left axilla immediately tells you two important things:

1. The tube is correctly placed in the trachea.

2. The tube has not been advanced into the right mainstem bronchus, and is therefore correctly placed.

I believe it is better to be able to confirm correct ET tube placement on ***first auscultation***, in a situation where seconds count, rather than depending on the second and third auscultation under such circumstances. After auscultating the epigastrium, the practitioner still has to auscultate both lung fields in order to confirm tube placement – which can result in considerable delays in identifying a misplaced tube, during which time the patient is rendered apnoeic and the airway potentially unprotected. This is especially important in patients with reduced FRC and increased closing capacities.

Caution:

Be careful to auscultate the left axilla and not the left apex. Auscultating the apices of the lungs creates the danger of interpreting transmitted sounds from the oesophagus or trachea

as breath sounds, as the stethoscope diaphragm is placed very close to the mediastinum. Placing the stethoscope as far from the mediastinum as possible (ie. the axilla), minimises the incidence of incorrect interpretation of lung sounds.
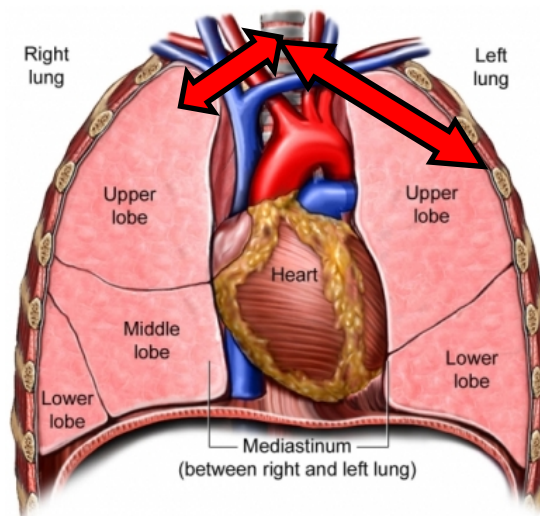
Note the distance between (a) trachea and the apex and (b) the trachea and axilla

A further word of caution involves interpretation of lung sounds in the paediatric age group. The smaller size of the paediatric chest means that much smaller distances between the mediastinum and axilla exist, and accurate interpretation of chest sounds may not be possible.   To illustrate this point, if you place a stethoscope on the top of a toddler's head, you will hear perfectly transmitted breath sounds!   Believe it or not!

Auscultation of the epigastrium:

Following auscultation of the left axilla, I auscultate the epigastrium.   Ventilation of the stomach results in pathognomonic auscultatory sounds.   The presence of air in the stomach mandates immediate removal of the ET tube and recommencement of face-mask ventilation.

Auscultation of the right axilla:

This step is theoretically unncessary, as the presence of breath sounds in the left axilla alone confirms correct tube placement.   Auscultation of the epigastrium therefore serves as a double safety check, and auscultation of the right axilla a triple safety check.   I, however, never skip this third check, as confirmation of correct tube placement is of such critical importance that every attention to every detail cannot be over-emphasised.

   b)   Misting of the tracheal tube:

Misting of the tube is to be ***completely discounted*** as providing any value to confirmation of tube placement.   Since the stomach is a warm and moist environment (as are the lungs), ventilation of the stomach through a relatively colder ET tube will produce as much tidal

misting as occurs with lung ventilation. ***Misting of the tube must be <u>thoroughly</u> <u>'unlearned'</u> as a method of confirming correct ET tube placement!***
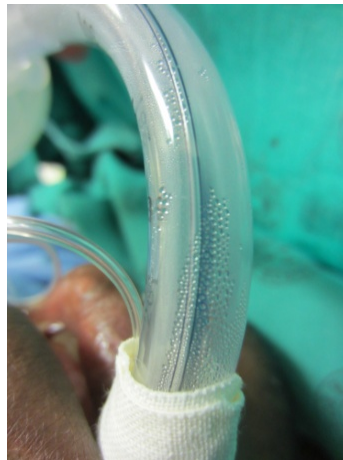
Tidal "misting" of the ET tube

"Tickling" the carina:

If the tube is too deep, it may come into contact with the carina (the bifurcation of the trachea into the left and right mainstem bronchi).   The carina is richly innervated with parasympathetic fibres, and an overly-deep tube may cause unexplained bronchospasm, bronchorrhoea and bradycardia.

c) <u>Referencing tracheal tube depth:</u>

The "rule of thumb" of placing the tracheal tube at a depth of 21cm at the teeth in females and 24cm in males must be revised.   In a recent study, it has been shown that **20cm** in females and **22cm** in men is safer and more reliable at preventing endobronchial intubation [33].

The 21/24cm (or 21/23cm) rule risks carinal stimulation and endobronchial intubation in 20% of patients.

Additionally, the study by Sitzwohl et al,[34] which compared (1) auscultation, (2) observation and palpation of symmetrical chest expansion and (3) tube depth, demonstrated that:

- Inexperienced clinicians missed detection of endobronchial intubation in 55% of cases, in comparison to experienced clinicians;
- Referencing tube depth alone achieved far more reliable results than auscultation, overall.
- Utilising all three (auscultation, observation of chest movements and referencing of tube depth) resulted in ***100% detection*** of right mainstem bronchus intubation by both inexperienced and experienced clinicians.

The take-home message from the above is that:

- The new tube depth rule should be a 20/22cm rule.
- Inexperienced clinicians should rely on monitoring of tube depth more than on auscultation.
- Utilising all three parameters provides 100% efficacy at preventing endobronchial intubation.

This is an important practice-changing lesson, and should be adhered to by all clinicians.

Right mainstem bronchus intubation:

Right mainstem bronchus intubation will *not* be detected by:

(1) end-tidal capnography, as carbon dioxide is 40 times more soluble than oxygen, and is fully vented through one-lung ventilation.

Similarly, right mainstem bronchus intubation may *not* be detected by:

(2) airway pressure monitoring, as the excessive right lung tidal volume is displaced into the non-ventilated collapsed left lung. The left lung collapses as it is in direct communication with the atmosphere (no tracheal tube cuff isolating the left mainstem bronchus, trachea or pharynx).

Right mainstem bronchus intubation may also *not* be evident from:

(3) pulse oximetry whilst adminstering a high F_iO_2, as such high F_iO_2 masks the resultant 50% V/Q mismatch.

The *only* guard against right mainstem bronchus intubation is a high degree of *clinical vigilance* through auscultation of the left axilla, and monitoring and recording of tube depth at the lips, teeth or gums.
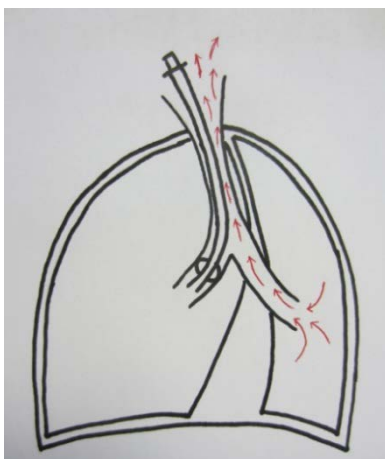
Schematic representation of right mainstem bronchus intubation:

Note that the increased right lung volume (and pressure) is displaced into the left lung. The left lung undergoes collapse, as it is in direct communication with the atmosphere, and experiences the pressure

NON-CLINICAL ADJUNCTS AND DEVICES FOR CONFIRMING CORRECT ET TUBE PLACEMENT

Non-clinical adjuncts to confirmation of correct tube placement are generally done *following* clinical tube confirmation.   It must be stressed that tube placement is initially a *clinical* assessment requiring additional back-up confirmation through other objective means.

a)   Oesophageal Detector Devices (EDDs or ODDs):

Oesophageal detector devices (known as either the EDD or ODD; depending on American or British spelling of the word "oesophagus"):

Oesophageal detector devices (ODD) are designed to aspirate air via the endotracheal tube and depend on the structural differences between the trachea and oesophagus to indicate ETT position. The ability to aspirate air easily when connected to an ETT indicates tracheal intubation as the trachea and main bronchi have a rigid structure and do not collapse when a negative pressure is applied. Failure to aspirate air indicates oesophageal intubation as the oesophagus collapses around the end of the ETT.
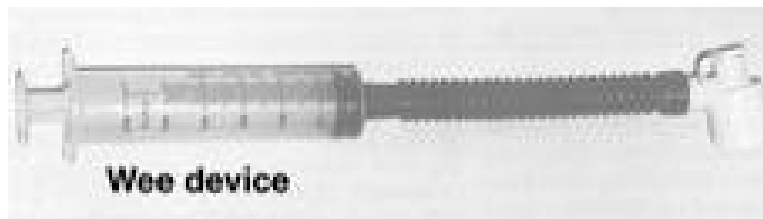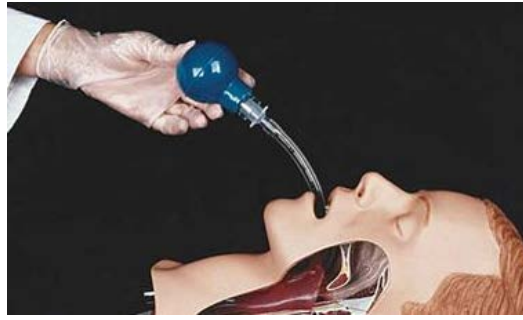
Wee device

The ODD is made by connecting a 60 ml catheter-tip syringe to a right-angled endotracheal tube connector by a short length of rubber tubing. The device is attached to an ETT and the syringe aspirated. If resistance is encountered when the syringe is aspirated i.e. with an oesophageal intubation, when the plunger is released it usually rebounds to its original position. O'Leary regarded the aspiration of 30mls of air as indicating tracheal intubation.[48]

Nunn described an adaptation using an Ellick's evacuator (a rubber bulb) and a connector. The bulb is squeezed and attached to the ETT.[49] Passive re-inflation indicates a tracheal intubation, while a failure to reinflate occurs with an oesophageal intubation. The bulb from a disposable bulb syringe may also be used.

Advantages of the ODD:

1. ODDs can be easily assembled using inexpensive and readily available equipment. They are easy to use (even by non-anaesthetists), portable, non-electronic, and provide a highly reliable assessment of ETT position. They are ideal for use in countries where capnography is not routinely available. They may also be useful for intubations performed outside the operating room (e.g., in the recovery room, emergency room, intensive care unit, and out in the field).
2. ODDs provide a rapid assessment of ETT position. In Wee's original study, the average time to perform the test was 6.9 seconds (range 5 - 16 seconds).[50] Nunn obtained a result with the Ellick's bulb in 3 - 5 seconds.[49] When the bulb from a disposable bulb syringe was used, full re-inflation of the bulb took up to 30 seconds in only 6% of tracheal intubations. The result of the ODD test is obtained more rapidly than that from capnography, and relies solely on observation.
3. ODDs are useful in patients in cardiac arrest as the test result does not depend on carbon dioxide being present in exhaled gas.
4. ODDs are useful when a Combitube has been used. They can indicate whether the Combitube is positioned in the trachea or oesophagus, and whether or not the airway is patent.
5. ODDs can be re-used after cleaning or sterilisation.

The disadvantages of the ODD include:

1. Some false results may occur.   However, the incidence of this is low.
2. Regurgitation of gastric air, distension of the oesophagus with air, or an ODD that is not airtight may give a false impression of tracheal intubation when the tube is in fact in the oesophagus.
3. Thick secretions may occlude a tracheal tube and give a false impression of oesophageal intubation. Occlusion of the bevel of a reinforced ETT by the wall of the trachea has been described to cause failure of bulb refill. Bronchial intubation, bronchospasm, tracheal compression, obesity, and chronic obstructive airways disease, may also cause resistance to aspiration or delayed refill of the bulb-type ODD.
4. Wee had no problem in identifying tracheal intubation in two patients with moderate bronchospasm (peak airway pressures of 3.0 - 4.2kPa). However, delayed refill of the bulb-type ODD has been observed in an asthmatic patient. Th[50]e slow re-inflation of the bulb seen in the presence of bronchospasm represents the slow exhalation that is characteristic of acute asthma.

False positive results (ETT in oesophagus but interpreted as in trachea):

- Regurgitation of gas from the stomach.
- Oesophageal distension with gas.
- Oesophageal detector device not airtight.

False negative results: (ETT in trachea but interpreted as in oesophagus):

- Thick secretions occluding the ETT.
- Occlusion of the end of an ETT (with no Murphy eye) by the mucosa of the tracheal wall.
- Endobronchial intubation.
- Bronchospasm.
- Tracheal compression.
- Morbid obesity and pregnancy.
- The under one-year old age group.
- Chronic obstructive airways disease.

Whilst there are multiple studies confirming 100% efficacy of the bulb and syringe-type EDDs, the following cautions exist:

Haynes and Morton found the device unreliable in the under one-year old age group.[51]   They hypothesized this failure was due to either air reflux from a hiatal hernia, a relatively non

collapsible oesophagus due to the ETT splinting it open, inadvertent intubation of the stomach with free air return, or a relatively immature/collapsible trachea which collapsed on aspiration. The esophageal detector device therefore cannot be recommended in children under one

Lang, Wafai et al found a 30% false negative rate for confirmation of tracheal intubation in morbidly obese patients.[52] Tracheal responses to the EDD were observed simultaneously by fiberoptic bronchoscopy. This observation revealed invagination of the tracheal wall as negative pressure occurred. They hypothesize that the mechanism is due to a reduced airway calibre in obese patients combined with a collapse of large airways resulting in reduced FRC and less air available for aspiration.   The same applies to pregnant patients.

Tanigawa et al found in a study of out of hospital cardiac arrest patients that both the bulb and syringe-type esophageal detector device (EDD) failed to confirm endotracheal tube placement in more than 25% of tracheal intubations in their study.[53]

One must therefore not rely too heavily on the results of the EDD alone, and proper clinical judgment in conjunction with all available modalities should be used to confirm ETT placement in out-of-hospital cardiac arrest patients.

Conclusion:

ODDs are best used where capnography is unavailable.

It must be stressed that ODDs do not replace capnography, but are the best alternative method to capnography in differentiating oesophageal from tracheal intubation.   The ODDs must not be used on their own, but always in conjunction with clinical methods to assess endotracheal tube position.

b) Colorimetric end-tidal carbon dioxide detection devices:

Colorimetric devices make use of the fact that carbon dioxide forms acidic solutions in water. Paper impregnated with an aqueous solution of a pH-sensitive dye can be maintained inside a hydrophobic filter.   When CO_2 containing gas comes into contact with the test paper, the pH drops and the indicator dye changes colour.   Disposable devices such as the Easycap or Stat-Cap can analyse CO_2 semi-quantitatively and can be used to confirm tracheal intubation in the field or emergency department.

These devices are not readily available due to expense and unreliability.   Anecdotal reports have found them to be extremely sensitive to moisture & fluids, rendering them inaccurate and inconsistent.

Colorimetric devices do not give accurate values of expired CO_2 levels, but simply give a "yes" or "no" colour change indication. The normal colour is purple, and changes to yellow on exposure to CO_2. (Remember **Y**ellow stands for **Y**es, and **P**urple stands for **P**roblem!). The colour change is reversible and changes with tidal breathing.

False-positive readings (CO_2 is detected but the tube is located in the oesophagus) can occur if the device becomes contaminated with gastric contents during oesophageal intubation), and can *theoretically* occur if a patient has ingested large amounts of carbonated drinks.

False-negative readings (in this context defined as failure to detect CO_2 despite correct tube placement in the trachea) can occur under the following circumstances:

- During cardiac arrest; as blood flow and delivery of CO_2 to the lungs is low.
- Pulmonary embolus; because pulmonary blood flow and carbon dioxide delivery to the lungs are reduced.
- The detector is contaminated with gastric contents or acidic drugs.
- During severe airway obstruction (eg. status asthmaticus), where exhalation of CO_2-rich expired air is markedly reduced.
- Intubation of the hypopharynx (ETT sitting above the vocal cords).

c) Continuous waveform end-tidal capnography:

The advantages of continuous waveform end-tidal capnography are discussed in detail in the lecture *Capnography* and will not be dealt with further in this section, except to say that ETCO$_2$ monitoring is the true gold standard of airway and ventilation monitoring and is considered a 'non-negotiable' *essential* monitor for the care of intubated, ventilated patients.

The NAP4 audit (which analysed 2.9 million anaesthetics across the UK over a period of one year), demonstrated that 74% of deaths or serious injury under anaesthesia are associated with the absence of capnography![46]

Advantages include:

1. The gold standard in confirming endotracheal tube placement.
2. Continuous breath-by-breath monitor of tube placement/dislodgement.
3. Ventilator or circuit disconnect/obstruction alarm.
4. Apnoea alarm.
5. Monitors adequacy of ventilation (hyper- and hypoventilation).
6. Fresh gas flow/rebreathing monitor.
7. Monitor of bronchospasm/expiratory obstruction (slope of expiratory phase).
8. Indirect monitor of sedation/paralysis/patient fighting ventilator.

9. Indirect (crude) monitor of metabolic rate (malignant hyperthermia, thyroid crisis, sepsis).
10. Crude monitor of cardiac output (gradually diminishing expired CO_2 level predicts imminent cardiovascular collapse).

Disadvantages include:

1. $ETCO_2$ may provide false-negative information (tube in the trachea but no $ETCO_2$ return) in a cardiac arrest situation where there is either inadequate circulation, inadequate ventilation, or both.
2. "Saddle" pulmonary embolus.
3. $ETCO_2$ will not detect an ETT in the right mainstem bronchus.
4. $ETCO_2$ will not reliably detect an ETT in the hypopharynx.
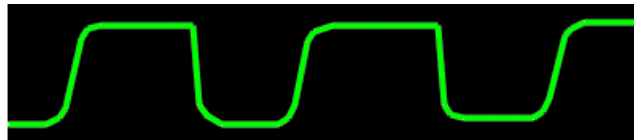
Capnography may provide a "false negative" result to correct tracheal tube placement in the situation of cardiac arrest, where there is a low to absent cardiac output (ineffective CPR) and inadequate ventilation.

All other confirmatory procedures for correct tube placement are "*single snapshots in time*" and become historical data ***immediately*** after being performed:

*   Visualisation of ETT passing through cords
*   Auscultation of left axilla and epigastrium
*   Oesophageal detector devices
*   Colorimetric carbon dioxide detection devices
*   Chest x-ray
*   Ultrasound

Tracheal intubation and the management of intubated patients should not be undertaken without the presence of end-tidal capnography. Failure to do so will be construed as <u>negligent practice</u> by a court of law.

A simple addition to SOPs requiring ***all*** ETT patients to have their head/neck immobilized by any means possible will play a significant role in reducing the number of accidental extubations, inadvertent oesophageal intubations and secondary right main stem bronchus intubations in the EMS environment.

Pulse oximetry:

It must be stressed that there is **no place** for pulse oximetry in confirming ET tube placement. Pulse oximetry is a **late warning device**, and as such, patients effectively ventilated with 100% oxygen may only desaturate 10 to 15 minutes after oesophageal intubation, circuit disconnection or other malfunction.   This is discussed in detail in the lecture *Pulse Oximetry*.

DOPES pnemonic for assessing tracheal tube placement:

Dislodged tube

Obstructed tube

Pneumothorax

Equipment failure – ventilator, circuit, ETCO$_2$

Stacking – breath-stacking

SECURING THE TRACHEAL TUBE

It seems an obvious thing to state, but securing the tracheal tube:

1. Secures the airway – note that *the airway is not secure until the ETT is secure*.
2. Securing the tube additionally frees up both hands and
3. Allows the practitioner to move more than one arm's radius from the patient's mouth! (I have noted on more than one occasion where a paramedic desperately holds onto the tube with one hand, whilst managing the rest of the resuscitation with the other hand!).

There are many ways to secure the ETT, and any method which provides secure and reliable fixing is acceptable.   Meticulous detail should be paid to monitoring and documenting tube distance at the teeth, and tube placement should **always** be rechecked after securing the tube, as the tube has a high tendency to **migrate deeper**, into the right mainstem bronchus, on strapping.

Please note that the airway is not secure until the ETT is secure, and the ETT is not secure until such time as the ***head and neck are immobilised!!***

See below – "*The post-intubation misplaced ETT*".

All methods of tube fixation will be illustrated in the accompanying Powerpoint presentation.

Please see below for illustrations.

METHOD 1: GAUZE ROLLER BANDAGE

A gauze roller bandage (Kling®-type or equivalent) can be 'larks-footed' over the tube, and secured around the back of the head.   Ensure the bandage passes over one ear and below the other.   This prevents slippage of the bandage.   Knot the bandage on the side of the head and not at the back, over the occiput. Gauze bandages tend to erode into the corners of the mouth when in place for an extended period.

Lark's-footed gauze bandage.

METHOD 2: ZINC OXIDE TAPE

Zinc oxide tape can also be used to secure the ETT, as shown in the accompanying Powerpoint presentation.   Two strips of gauze are used.   One strip is wrapped around the tube and the two limbs placed over the maxilla and upper cheek as shown.   A second mirror-image strip is secured to the tube, with the limbs placed over the mandible and lower cheek.
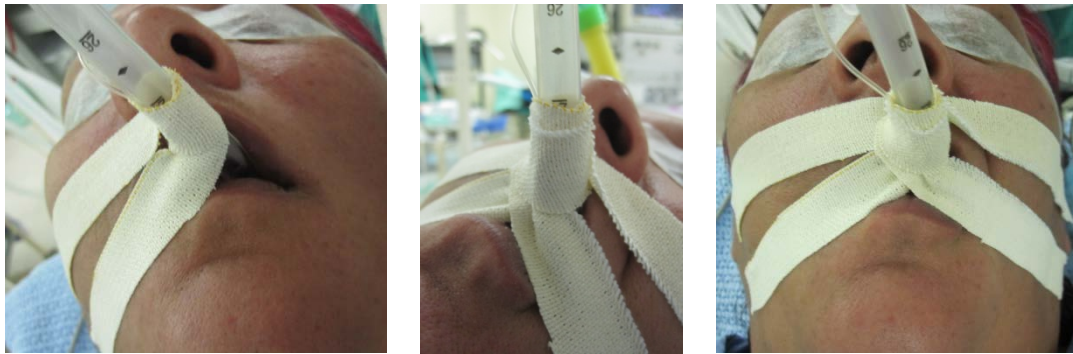
Tube strapping with zinc oxide tape – see text above for explanation.

Make sure not to fix the tube too high on the shaft, as tightening the tube will pull it deeper into the trachea, and may result in carinal stimulation or right mainstem bronchus intubation.
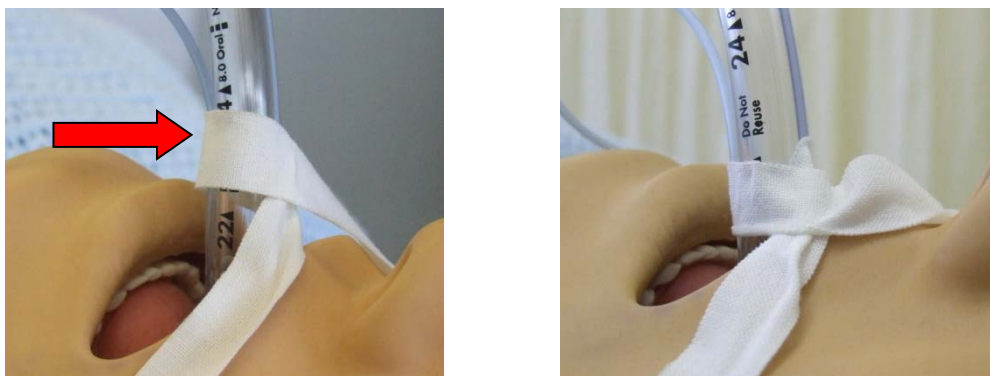
Left: Strapping fixed *too high* on the shaft of the tube – on tightening the strapping, the tube will be pulled deeper into the trachea.   Right: Fixing the strapping at the correct height around the tube ensures the tube is not pulled deeper into the trachea.

METHOD 3: ELASTOPLAST (TWO TAILS)

Wide Elastoplast® or equivalent strapping can also be used, as demonstrated in the accompanying Powerpoint illustrations, a few of which are included here.   Two "tails" are cut into either end of a wide Elastoplast strip.   The strip is placed behind the patient's head, over the occiput.

One tail is wrapped around the tube in one direction, and then the second tail wrapped around the tube in the opposite direction, as shown below.

The same is done with the other side of the Elastoplast, creating a very secure ETT.   The only disadvantage of this system is that tube depth at the level of the lips or teeth cannot be monitored.

METHOD 3: ELASTOPLAST (THREE TAILS)

A variation on the above theme is to cut three tails into the broad Elastoplast tape, as shown below.   The top (cephalad) tails are wrapped over each other, over the top lip (maxilla).   The second two tails are used to secure the tube, in a manner similar to that described above. The third two tails are used to secure the oro-/nasogastric tube.

Cut three tails at each end of the broad Elastoplast tape.

The top (cephalad) two tails wrap over each other, over the top lip (maxilla).

Each limb of the second tail is strapped in opposite directions around the tube.

Each limb of the third (caudad) tail is used to secure the orogastric tube.

METHOD 4: COMMERCIAL DEVICES

Commercially available tube holders such as the Thomas Tube Holder, distributed by Laerdal® are the most ideal, but are reasonably expensive.

Thomas tube holder

METHOD 5: HOME-MADE DEVICES

Cable-ties and PVC tubing can be used to make an ingenious home-made tracheal tube holder, which is very cheap to make (under R2 each) and is extremely effective.

Home-made tracheal tube ties, using cable ties and "fish tank" tubing – obtainable from any hardware store, are cheap and very effective.

When securing tubes on burns patients, when one can't fix the tube to the skin, one of the following two methods may be employed:

METHOD 6: SINGLE FEEDING TUBE

The method of fixation involves introducing a feeding tube into the nostril, over the soft palate and retrieving it through the mouth, with the use of a Magill forceps.   The tracheal tube is then secured by tying it in position with the feeding tube, as shown below.   Beware tying the tube too tightly, as the feeding tube can have something of a "cheesewire" effect, cutting into the tissues of the soft palate.
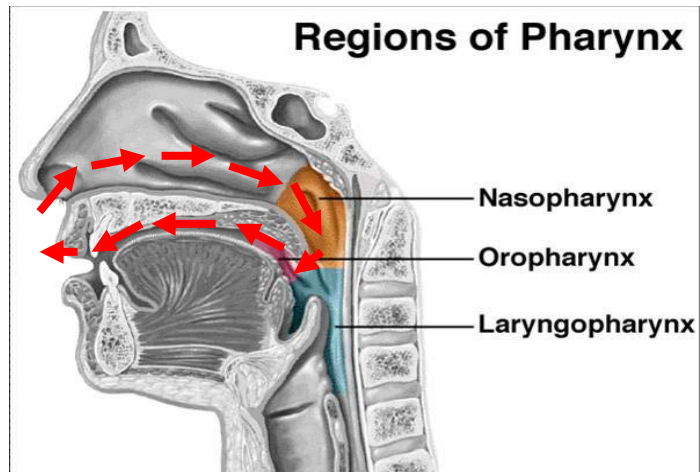
A feeding tube or suction catheter is passed through the
Nostril over the soft palate and out of the mouth.
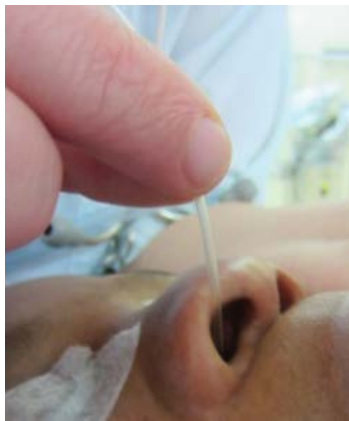
Feeding tube fed through the nostril.

Feeding tube retrieved through the mouth.

Feeding tube tied around the tracheal tube.

Feeding tube knotted securely.

METHOD 7: DOUBLE FEEDING TUBE

In the second method, one feeding tube is fed through each nostril, and the tips retrieved through the mouth, with a Magill forceps, as above.   The two proximal tips are then tied to each other with thin nylon suture, as shown.   It is better not to tie a simple knot, as the knot may cause an area of pressure necrosis in the posterior nasopharynx (soft palate).   The distal ends are then pulled outwards, and the tracheal tube (and/or nasogastric tube) can be secured to the two ends of the feeding tubes by tying simple knots, using tape or by using small cable ties.
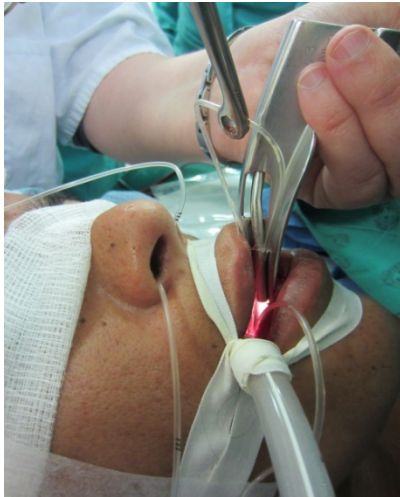
The two proximal ends of the feeding tube tied together with nylon suture.

Two feeding tubes, one through each nostril, proximal ends brought out through the mouth.

The distal ends of the feeding tube are seen protruding from the nostrils. The proximal ends (sutured together) are apposed against the posterior nasopharynx.

The distal ends are used to tie the tracheal tube or nasogastric tube (in this case nasogastric tube). Surgical tape/plaster or cable ties can be used.

Two or more methods of tube fixation can be used simultaneously where deemed necessary and appropriate.

CAUTION: ARMOURED ET TUBES

Beware the use of armoured Et tubes!   'Armoured' or 'reinforced' ET tubes have a wire coil spiralling through the outer wall of the tube.   They are specially designed to prevent kinking of the tube, and are used predominantly in ENT, facial, head and neck surgery, and where patients are operated on in the prone position.

Armoured tubes are far more "floppy" than ordinary PVC ET tubes, and most require the use of a stylet, gum elastic bougie or other introducer.   An unwitting practitioner attempting to intubate a patient with an armoured tube, without an introducer, is analagous to trying to pick one's nose with a piece of cooked spaghetti!

Another caution with armoured tubes is that if a patient bites an armoured tube, the wire coils will be crushed into a flattened configuration, and will not spring open again!   This constitutes a true airway emergency as the tube then represents a total airway obstruction.   A gum elastic bougie cannot be introduced into the tube, and hence a fresh tube cannot be railroaded over the old one.   A bite block or oropharangeal airway should be used as a precaution when armoured ET tubes are used in non-paralysed patients outside of the operating theatre.   Since the practice of securing bite blocks and oropharangeal airways into the mouths of intubated patients is a dubious practice, there exists very little place for the use of armoured ET tubes in the emergency environment, outside of the operating theatre.

Armoured or "reinforced" tracheal tube.   Bitten armoured tracheal tube – stays collapsed!

POST-INTUBATION PHASE

The post-intubation phase commences with confirmation of correct ET tube placement and securing of the ET tube – the following will have occurred:

- Tube placement is confirmed as being correct.
- The tube is adequately secured, and placement re-confirmed.
- Tube distance at the teeth is noted and documented.

In addition, the following must be managed during the post-intubation phase:

- Ventilation
- Hypotension
- Sedation
- Analgesia
- Neuromuscular blockade
- Nasogastric or orogastric tube placement
- Documentation

VENTILATION

Either the patient must be connected to a mechanical ventilator with appropriate tidal volume, rate and airway pressure settings, or the practitioner should continue (or should instruct and guide a fellow practitioner) in appropriate manual BVM ventilation. Correct ventilation technique is beyond the scope of this text, and is discussed in detail in the lecture *Ventilation in the Transport Environment*.

HYPOTENSION

Hypotension should be sought and managed by raising the legs, administering an appropriate IV fluid bolus, or administering vasopressors and/or inotropes, according to protocol and scope of practice. Please see the appropriate pharmacology sections for guidance on the use of inotrope infusions and pressor agents.

SEDATION AND ANALGESIA

The patient must receive appropriate sedation and analgesia (remember that sedation is not analagous with analgesia in the unconscious patient). Please see the appropriate sections for guidance on sedation and analgesia.

NEUROMUSCULAR BLOCKADE

Neuromuscular blockade should be considered simultaneously, where there is any chance of the patient fighting the ventilator.

The above issues are dealt with in detail in separate lectures on *Ventilation in the Transport Environment* and *Neuromuscular Blockade.* Head injury issues are specifically dealt with in the lecture *Traumatic Brain Injury*.

NASOGASTRIC/OROGASTRIC TUBE PLACEMENT

Gastric tube placement may be indicated for numerous reasons, not least of which is deflating an over-distended air-filled stomach. Traumatic brain injury with basal skull fracture is a relative contra-indication to nasogastric tube placement.

Placement of the NGT with a Magill's forceps under direct laryngoscopy can be a difficult and time-consuming procedure. One must ensure guidance of the NGT into the posterior hypopharynx, and check that it does not coil upon itself while advancing it. Keeping NGTs in the deep freeze helps to keep the point rigid, which aids initial placement into the oesophagus. It is useful to have an assistant guide and advance the NGT, as it has a tendency to slide back out each time one releases the NGT following advancement into the oesophagus (in a "three steps forward, two steps backwards" nature).

A further 'trick' is to grip the larynx (anterior neck) with one hand and simultaneously lift it upwards (anteriorly) whilst simultaneously inserting the NGT with the other hand. (See pictures).

Grasping the larynx and applying anterior (upward) traction

Another simpler and quicker trick to rapid placement of a gastric tube involves the intentional placement of a second large-bore ETT into the oesophagus and then simply channelling a well-lubricated gastric tube through the ETT.

It is critically important to continue with general patient monitoring and management, such as ECG, NIBP, SPO_2, $ETCO_2$, fluid status and urine output, core temperature, pressure area vigilance, etc.

Repeated regular ETT position checks and a state of 'hypervigilance' cannot be over-emphasised. Multiple clinical rechecks must be done every time the patient is moved.

"Just because you've never been hurt in a car accident doesn't mean you don't need to wear your seatbelt".

DOCUMENTATION

The following parameters must be noted in the documentation relating to airway management:

- Reason for intubation
- Intubated by whom
- Intubated where (location)
- Time intubated
- Method used (RSI or non-RSI, application of OELM, positioning, etc)
- Equipment used – blade type and size, gum elastic bougie, other adjuncts
- Drugs used
- Methods of confirmation of ETT placement, use of $ETCO_2$
- Observations before, during and after tracheal intubation
- ETT distance at teeth
- Intubation views (Cormack & LeHane), difficulty of intubation and reasons for any difficulties encountered
- Ventilation

Note: It is very useful to mark a difficult intubation by wrapping a short strip of red electrical insulation tape around the ET tube once placed.   This warns practitioners involved in the care that the tube was difficult to place.
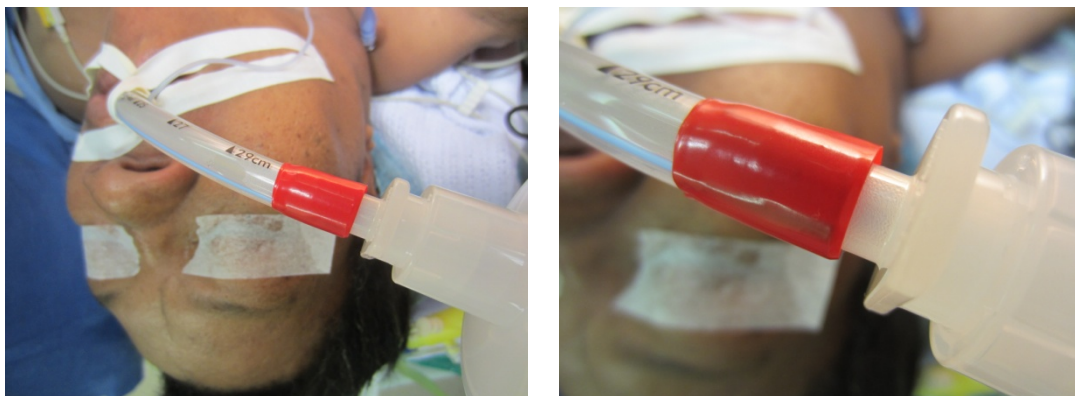
Red insulation tape signposts a difficult intubation.

UNRECOGNISED OESOPHAGEAL INTUBATION

The most catastrophic complication of RSI is unrecognised oesophageal intubation, which can occur either at the time of intitial intubation, or subsequently as a result of tube displacement into the pharynx and/or oesophagus.   Continuous end-tidal carbon dioxide monitoring is the *only* modality to reliably warn against this complication.

Unrecognised oeophageal intubation occurs with alarming regularity in some prehospital studies, and is regularly reported at between one and five percent, and in one notorious study, by Katz and Falk[9], as high as twenty-five percent![9]

One might not consider a failure rate of one percent as high, but considering the universal fatal outcome associated with this complication: If we translated a failure rate of "*only one percent*" to the airline industry, this would extrapolate to no less than 30 airline crashes per day at Heathrow airport alone.   As can be seen, one percent is in reality an extremely high percentage!

The Fourth National Audit Project [52] of major complications of airway management in the UK highlighted the fact that airway interventions outside of the operating theatre were more likely to result in adverse events with serious outcomes.   *Seventy-four percent of deaths or serious neurological injury were associated with the absence of capnography![46,47,54]*

THE POST-INTUBATION MISPLACED TRACHEAL TUBE

The ETT may relocate to the oesophagus, right mainstem bronchus or posterior pharynx), under certain circumstances.   Note well that missed oesophageal intubation carries a **100% mortality** in the paralysed patient!

Be aware that all tracheal tube position checks provide a "single snapshot in time", and become historical information immediately after the check has been been completed.   The only exception to this rule is provided by continuous end-tidal carbon dioxide monitoring. However, $ETCO_2$ will not alert the airway technician to either a tube placed in the posterior pharynx (just above the glottic opening) or right mainstem bronchus intubation.   Clinical vigilance is the only guard against these misplaced tubes.

In a study of ETT movement in secured ETTs by Matera, the average displacement of the fixed ETT tip away from the carina during neck extension was almost 30mm.[55] Biphasic (proximal and distal) movement of the ETT was observed to a varying degree on extension of the head from neutral (owing to the biomechanics of neck extension).   Neck flexion similarly

revealed that the ETT tip moved away from the carina up to 10 mm, and moved towards the carina up to 25mm from a neutral position.
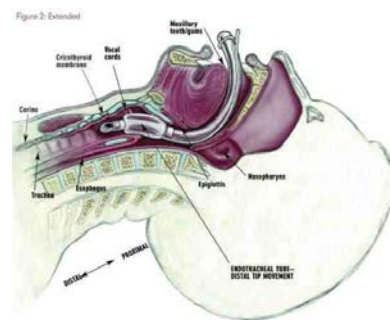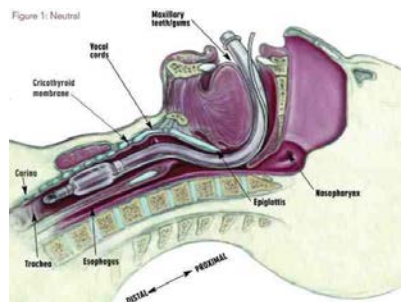
Another critical issue to understand is that the lung root is not a rigid structure. The lung root moves when a patient coughs or gags. Therefore, even with the proximal ETT secured at the teeth/nose level, a C-collar in place and the patient fully immobilized, the lung root (carina) can still move in relation to the ETT tip. This occurs *because* the proximal end of the ETT is secured to the mouth/nose, and not in spite of it.

Right mainstem bronchus intubation:

Right mainstem bronchus intubation is also 'technically' a misplaced post-intubation tracheal tube, and may have imported consequences, such as hypoxaemia, right lung barotrauma and left lung atelectasis.

Please review this topic, under the section "*Confirming correct placement of the tracheal tube*".

RE-CONFIRMATION OF TRACHEAL TUBE PLACEMENT

Re-confirmation of proper ETT placement should occur at various times during transport [46] of any intubated patient, and include:

1. Following initial intubation.
2. After securing the ETT in position.
3. After completion of packaging the patient for transport from the scene;
4. Prior to loading the patient into a transport unit;
5. After loading the patient into a transport unit;
6. After removing the patient from the transport unit at the receiving facility; and
7. Before placing the patient on a receiving facility bed or trolley.
8. After placing the patient on a receiving facility bed or trolley.
9. After any significant patient movement (e.g., following attachment or removal of a BVM, defibrillation, a sudden stop or bump in the road, etc.).

10. At each time that a new practitioner takes over responsibility for patient care and airway management.

TRANSFERRING PATIENTS

When transferring patients from one stretcher, trolley or bed to another, it is safest and wisest to completely disconnect the patient circuit or BVM resuscitator from the ETT.   There are many things that can snag an ET tube or ventilator circuit whilst transferring a patient.   Most patients ventilated with supplemental oxygen will withstand short periods of apnoea with ease.

However, few patients can withstand a life-threatening event such as an inadvertent extubation, lost airway and emergency re-intubation – especially where the intubation is known to be difficult, or where there is a considerable amount of airway swelling.

When transferring a patient from one stretcher/trolley to another, the receiving doctor should be asked to confirm ETT placement before the patient is transferred to the receiving bed/trolley.

This provides protection for the transferring doctor, paramedic or registered nurse, in that they cannot then be blamed for a missed oesophageal intubation, in the event that the tracheal tube dislodges during the actual transfer from one bed to another.

The same principle applies when responsibility for patient care is transferred from one doctor, paramedic or registered nurse to another.

LARYNGOSPASM

Management of laryngospasm can present a major challenge to the airway practitioner. Laryngospasm can manifest itself before intubation in the non-RSI or drug-assisted intubation.   Laryngospasm is not encountered during RSI, owing to the use of muscle relaxant drugs which paralyse the vocal cords.

Laryngospasm is most often encountered following extubation, in the *"twilight zone of emergence"* – where the patient is in that plane of anaesthesia that is somewhere between deep and awake.

Laryngospasm usually occurs due to an error of judgement in the timing of extubation.   (It has been said that good judgement comes from experience; and experience comes from bad judgement!).   The most reliable assessment of a sufficiently light plane of anaesthesia to enable extubation without encountering laryngospasm, is the application of "Aunty Gwen's rule".

"Aunty Gwen's rule" states simply that once spontaneous upper limb (elbow) flexion is observed (without purposeful movement being necessary), it will be safe to extubate the patient. In my anecdotal experience, Aunty Gwen's rule is truly 100% reliable!

Obviously, an awake patient with eyes open who swallows on, or coughs against, a tracheal tube in situ can also be safely extubated.

The most important resources in the management of tight laryngospasm are a high level of vigilance, nerves of steel, an unshakeable resolve, and judicious patience.

Tight laryngospasm may or may not present with obvious signs of airway obstruction, such as stridor, severe dyspnoea, use of accessory muscles, etc. Laryngospasm may present with a silent airway and the appearance of an unobstructed breathing pattern with normal inspiration and expiration. This is especially so in the paediatric age group. Placement of the stethoscope over the angle of the mandible will reveal absent bronchial breath sounds, and therefore no air movement in the trachea, despite the *appearance* of normal breathing.

A silent airway to the naked airway means either unobstructed clear air entry, or complete airway obstruction. In the context of laryngospasm, noise is good! Although loud stridor can be frightening very intimidating, it is generally much better than soft stridor (or silence), as the louder the stridor the better the air movement through the vocal cords. If stridor is not obviously apparent, the vibrations generated by obstructed air movement can often be perceived through the hand which applies the face-mask to the face during positive pressure ventilation.

Spirometry (the measurement of tidal volume) is of no value in assessing the course of laryngospasm. During tight laryngospasm, spurious tidal volumes (and airway pressure traces) will be generated by ventilation of the upper airway, oesophagus and stomach.

Capnography, however, remains a very useful monitoring modality, as any amount of end-tidal CO_2 (no matter how little) indicates true air movement and therefore yields the (slow) breaking of laryngospasm. Generally, the greater the $ETCO_2$ return, the greater the relief of laryngospasm.

Vigilance and early recognition are essential as, in the well pre-oxygenated patient, desaturation may not occur for several minutes. The shape of the oxygen-haemoglobin dissociation curve dictates that once saturation starts to fall – once the patient is on the "slippery slope of hypoxia" – saturation will _rapidly_ crash to frightening depths!

Remember, too, that pulse oximeters display averaged readings, which, depending on the make and model of pulse oximeter, may take up to 30 seconds to average. Therefore, saturation values may not change on the pulse oximeter for some time, even after effective ventilation has been established – and hence the requirement for "nerves of steel".

Poorly managed laryngospasm will result in prolonged hypoxia and hypercarbia, and the attendant problems associated with each. However, negative pressure pulmonary oedema is a very real likelihood following prolonged tight laryngospasm, and understandably exacerbates both hypoxia and hypercarbia.

Treatment includes the use of positive pressure ventilation and the judicious use of morphine and frusemide (as in the management of pulmonary oedema of other aetiologies).

Management of tight laryngospasm:

- Change to 100% oxygen or as high an FiO_2 as possible (with BVM and reservoir bag).
- Maintain gentle, yet firm and constant airway pressure with a good face-mask seal. High airway pressure itself helps to relieve laryngospasm, but be very mindful that gastric sphincter opening pressure occurs at approximately $15cmH_2O$, with consequent insufflation of the stomach, splinting of the diaphragms and increasing difficulty with face-mask ventilation.
- Propofol by incrementally increased IV boluses – propofol ablates airway reflexes very effectively. Boluses of 0.5 to 1mg/kg (up to 2mg/kg) should completely alleviate laryngospasm. Propofol will obviously deepen the patient as well, and must therefore be used with great caution in the difficult airway situation.
- Suxemethonium by incrementally increased IV boluses – 0.25mg/kg (or smaller dose) to 1mg/kg will also ablate airway reflexes, but comes with the attendant side-effects related to suxemethonium administration. Beware the administration of suxemethonium following the administration of neostigmine (for non-depolarising muscle relaxant reversal). Neostigmine works against both pseudo- and plasma cholinesterase, and since suxemethonium depends upon the action of pseudocholinesterase for its reversal, the administration of suxemthonium following neostigmine may delay the offset of suxemthonium by up to six hours. Be prepared for prolonged ventilation under such circumstances.

THE DIFFICULT AND FAILED AIRWAY

Never lose sight of the fact that the patient needs oxygen, and not a tracheal tube!

There are four key questions that must be asked, when it comes to difficult airways:

1. Is the airway difficult? (anatomic concerns)
2. Is the patient compromised? (physiologic concerns)
3. What is your primary approach?
4. What is your secondary approach – ie. what is your "bail-out" or rescue plan?

An airway 'strategy' vs an airway 'plan':

An airway "plan" suggests a single approach to airway management. A "strategy" is a co-ordinated logical sequence of plans – which aim to achieve good gas exchange and the prevention of aspiration. Anaesthetists should approach airway management with strategies rather than with plans.[46,47,56]

Planning for failure:

Where failed airways were managed with multiple repeated attempts of doing the same thing over and over, the situation regularly deteriorated into a 'can't intubate, can't oxygenate' situation.

It is well recognised that, rather than repeating a technique that has already failed, a change of approach is required.

The NAP4 airway audit highlighted a general failure to 'plan for failure' – for when airway management was unexpectedly difficult. Where the response was unstructured, the outcomes were poor (which occurred often). This reinforces the adage that "a good anaesthetist is always surprised by an easy airway" – implying that it is good practice to expect, and plan for difficulty with all routine airway management.

NAP4 revealed that unanticipated difficult airway often occurred in young, fit, healthy patients undergoing elective surgery, where a specialist anaesthetist was often involved. One quarter of cases occurred on emergence from anaesthesia and in the recovery room. Similarly, quarter of cases occurred in the Emergency Department and Intensive Care Unit.

DEFINING THE DIFFICULT AND FAILED AIRWAY

The failed airway can be defined as:

1. Failure to maintain acceptable oxygenation saturation during or after one or more failed attempts at laryngoscopy.
2. Three failed attempts at oral intubation by an experienced intubator, even when oxygen saturation can be maintained.

Benumof defines the six components of the 'best attempt' as:[57]

- Experienced endoscopist
- No significant muscle tone
- Optimal morning sniff position
- OELM
- A range of blade lengths
- Different types of blade (Magill –curved; Miller – straight).

PREDICTORS OF DIFFICULT INTUBATION (PATIENT ASSESSMENT)

HISTORY

A history of difficult airway management may not always be a luxury afforded the emergency practitioner.   However, it is still worth discussion.

Previous anaesthetics: If the patient has had previous anaesthetics, and significant airway difficulties were encountered, it is very likely that the patient would have been told of this.

Previous neck surgery, radiotherapy or burns to the neck could result in scarring and contracture formation, which could result in difficult intubation.

Diabetes mellitus (chronic hyperglycaemia) leads to glycosylation of tissue proteins, and a 'limited mobility joint syndrome'.   Evaluation of temporo-mandibular joint and cervical spine mobility will help to predict difficult intubations, which occur in 30% of type I (insulin-dependent) diabetics.

Rheumatoid arthritis, ankylosing spondylitis and other auto-immune diseases: Rheumatoid arthritis is characterised by immune-mediated joint destruction with chronic and progressive inflammation of synovial tissue.   All synovial tissues are involved, including those of the cervical spine and temporomandibular joint.   Atlanto-axial subluxation may lead to the protrusion of the odontoid process into the foramen magnum during intubation, compromising vertebral blood flow and compressing the spinal cord or brainstem.   Additionally, crico-arytenoid arthritis can cause narrowing of the glottic opening, leading to post-extubation airway obstruction.

EXAMINATION

- Neck masses, scars
- Small mouth
- Prominent underbite (recessed mandible)
- Large tongue

- Bull neck

- Morbid obesity

- Large breasts

- Prognathic mandible (protruding, over-riding upper teeth)

- Loose teeth

- "Passion gap"

RELIABILITY OF CLINICAL TESTS

No single test predicts intubation difficulty with certainty. However, combining tests increases the positive predictive value, but no combination of tests are failsafe! It must be emphasised that it is not unknown for a Mallampatti I score to correlate with a Cormack & Lehane grade III or IV view.

"A good anaesthetist [paramedic] is always surprised by an easy airway".

CLINICAL TESTS

1. Ability to push out mandible:
   a. Lower incisors brought edge to edge with upper incisors.
   b. Lower incisors cannot be brought edge to edge with upper incisors.
2. Inter-incisor distance of less than three centimetres.
3. Thyromental distance of less than 6cm has a 75% positive predictive value.
4. Short fat ("bull") neck.
5. Range of motion of head and neck.
6. Mallampati score (ties in very closely with Cormack & LeHane grading of vocal cord visualisation).

It is important to consider the respiratory functional reserve (Functional Residual Capacity) of the patient, when a difficult or prolonged intubation is anticipated.

Benumof has summarised the clinical tests thus:

Pre-operative airway examinations, acceptable end-points and significance of end-points. ASA Difficult Airway Algorithm. Benumof JL, San Diego, California.

	Pre-op examination	Acceptable end-points	Significance of end-points
1	Length of upper incisors	Qualitative/short incisors	Long incisors → blade enters mouth in cephalad direction

2	Involuntary: maxillary teeth anterior to mandibular teeth	No over-riding of maxillary teeth anterior to mandibular teeth	Over-riding maxillary teeth → blade enters mouth in more cephalad direction
3	Voluntary: Protrusion of mandibular teeth anterior to maxillary teeth	Anterior protrusion of mandibular teeth relative to maxillary teeth	Test of TMJ function – means good mouth opening and mandible will move anteriorly on laryngoscopy
4	Inter-incisor distance	>3cm	2cm phlange on blade can be inserted between teeth with ease
5	Oropharangeal Class	< or = to Class	IITongue is small in relation to size of oropharangeal cavity
6	Narrowness of palate	Should not appear very narrow or highly arched	A narrow palate desreases the oropharangeal volume and room for both blade and ETT
7	Mandibular space length (thyromental distance)	>5cm or > 3 ordinary sized finger-breadths	Larynx is relatively posterior to other airway structures
8	Mandibular space (MS) compliance	Qualitative palpation of normal resilience/softness	Laryngoscopy retracts tongue into MS. Compliance of MS determines whether or not tongue will displace into MS
9	Length of neck	Qualitative. A quantitative index is not yet available	A short neck decreases the ability to align the upper airway axes
10	Thickness of neck	Qualitative. A quantitative index is not yet available	A thick neck decreases the ability to align the upper airway axes
11	Range of motion of head and neck	Neck flexed on chest 35° and head extended on neck 80° = 'morning sniff' position	The 'morning sniff' position aligns the oral, pharangeal and laryngeal axes to create a favourable line of sight

None of the above tests should be considered a fail-safe predictor of airway difficulty, and most are not applicable in the emergency setting.   Many studies show that the more predictors used, the better the prediction.   It is not uncommmon for a Mallampati score of 1 to be associated with a Cormack & LeHane Grade IV view!   Pathological states such as cancer, bleeding, oedema, presence of a beard, large breasts and obesity are very important determinants of difficult facemask ventilation and intubation.

MALLAMPATTI AND CORMACK & LEHANE SCORE CORRELATION
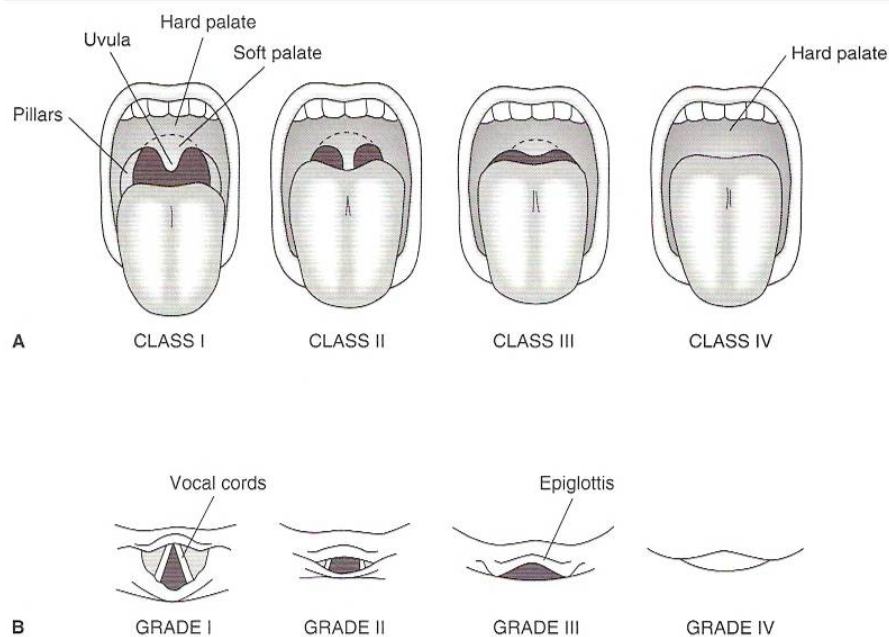
LEMON (MNEMONIC FOR DIFFICULT LARYNGOSCOPY AND INTUBATION):

The 'lemon law' is fine for those who like to remember facts through mnemonics.   However, I am not keen on the 'lemon law' as it is hardly comprehensive.   It is included here for the sake of completeness (…or the lack thereof!).

Look externally – if it looks like a difficult airway, it most likely will be.

Evaluate 3-3-2

 - Accommodate three fingers in mouth;

 - Three fingers under mandible – between tip of mentum (chin) and thyroid cartilage

 - 2 fingers to identify larynx to base of tongue (thyrohyoid distance)

Mallampati score

Obstruction – upper airway obstruction

Neck mobility.

Measurement of thyromental (left) and thyrohyoid (right) distances.

PNEMONIC FOR DIFFICULT PLACEMENT OF A SUPRAGLOTTIC AIRWAY DEVICE: RODS

- **R**estricted mouth opening
- **O**bstruction/**O**besity
- **D**istorted anatomy
- **S**tiff lungs or c-spine

DIFFICULT INTUBATION STATISTICS

- Failed intubation in general population: 1:3000
- Failed intubation in pregnancy: 1:300
- Difficult laryngoscopy: 1:50 to 1:100
- Can't intubate can't oxygenate: 1:5000

(LMA provides "rescue" ventilation in 94% of cases of CICO).[58]

If one does one intubation per week, one will experience:

- One difficult laryngoscopy per year.
- One failed intubation every 60 years.
- One CICO every 100 years.

If one does 25 intubations per week, one will experience:

- One difficult laryngoscopy every 2 weeks.
- One failed intubation every 2.5 years.

- One CICO every 4 years.

Incidence of difficult and failed airway:

- 0.05% in surgical patients (1:2000)
- 0.36% in parturients (~1:300 - 7.2 times higher than in the general population).
- Failure of in-hospital RSI: 1% (1:100)
- Failure of cricothyroidotomy: 0.5% in medical and 2.3% in surgical patients.

Sources: Clinical Anesthesiology, 3rd edition. Morgan & Mikhail. McGraw-Hill, and Manual of Emergency Airway Management, 2nd edition, Walls & Murphy, Lippincott Williams and Wilkins.

MANAGEMENT OPTIONS FOR THE ANTICIPATED DIFFICULT AIRWAY

- Rapid Sequence Induction (with 'rescue' airway devices on hand) and…
- ENT surgeon scrubbed and ready, and patient prepared for emergency tracheostomy.
- Propofol "quick-look" – the problem with this technique is that it is one of those situations in which you will "only know if you can fly once you've jumped over the cliff".   Recovery from propofol (as with suxemethonium), may take longer than time to terminal desaturation, in the event that you find yourself in failed airway "can't intubate, can't oxygenate" situation.   Please see the section on suxemethonium for a further discussion of this concept.
- Inhalation induction – to be undertaken by an experienced anaesthesiologist in an operating theatre.   There is great wisdom in maintaining spontaneous ventilation in an adult or paediatric threatened airway situation.   Inhalation induction allows for spontaneous breathing whilst deepening anaesthesia to a depth at which laryngoscopy can be undertaken.
- Classic LMA and Proseal LMA – see elsewhere – there is strong evidence that the 'standard' LMAs will provide rescue from a "can't intubate, can't oxygenate" scenario more than 90% of the time.   Remember that the LMA will not allow adequate ventilation during "tight" laryngospasm or other glottic or subglottic obstruction.
- Intubating LMA – as above for 'standard' LMAs.   In order to successfully pass a tracheal tube through the I-LMA, flaccid (paralysed) vocal cords are required.
- Awake flexible fibre-optic nasal intubation – undertaken by an anaesthesiologist in an operating theatre.   This is an elective procedure undertaken in a patient previously identified to have a difficult airway, and is not suited to the emergency "failed airway".
- Airtraq optical laryngoscope – also requires flaccid (or paralysed) vocal cords in order to pass a tracheal tube.
- Awake 'elective' tracheostomy under local anaesthetic.

MANAGEMENT OF THE UNANTICIPATED DIFFICULT OR FAILED AIRWAY

There are numerous algorithms that have gained widespread international acceptance, the two commonest being the American Society of Anesthesiologists and the UK Difficult Airway Society algorithms.   These tend to be convoluted and confusing at first glance, and can be quite intimidating to the uninitiated.   They have been designed more for elective general anaesthesia in the operating theatre environment than for emergency medicine.   One seldom has the luxury of pre-intubation examination and preparation in the emergency setting, and so not all limbs of the algorithm are entirely relevant under emergency airway circumstances.

The Resuscitation Council of South Africa has a simple linear algorithm which is very easy to follow, and is well worth reviewing.   However, the 2015 UK Difficult Airway Society guidelines are without a doubt the guidelines of choice.

Benumof has described "optimal" intubating conditions thus:

1. Experienced Endoscopist
2. No significant muscle tone
3. Optimal 'morning sniff' position
4. Optimal external laryngeal manipulation
5. Change of blade length x 1
6. Change of blade type x 1

I believe that the following can be added to the above:

1. Extra padding for optimal positioning of the patient (or repositioning in the case of a failed intubation) so that the ear lobe lies above the clavicles in a horizontal plane, at the mid-clavicular line ("ramping").
2. The early use of a gum elastic bougie.
3. Trained and experienced *assistant*.

The problem with multiple repeated attempts at direct laryngoscopy is the creation of laryngeal oedema and bleeding, which can evolve into a '*Can't Intubate, Can't oxygenate*' situation.   Laryngoscopy should be limited, and the "best attempt" at layngoscopy should be made as early as possible to prevent a Difficult Airway situation from evolving into a CICO scenario.

Benumof has clearly demonstrated that it is a complete fallacy that a patient will achieve neuromuscular recovery sufficiently, from a dose of suxemethonium 1mg/kg, to establish spontaneous ventilation, before terminal desaturation and death occur.   This is fully discussed in the lecture on *Neuromuscular blockade*.

Thus, a CICO situation must be recognised as *early as possible*, and 'rescue' airway interventions implemented *immediately* upon such recognition.

THE FAILED EMERGENCY AIRWAY

Under normal circumstances the patient will be pre-oxygenated and ventilation gently assisted where appropriate.

Each following step presumes failure of the former step:

1. Difficult facemask ventilation:

- Call for assistance – or declare the situation to your assistant.
- Ensure adequate head tilt, jaw thrust, chin lift (non-trauma).
- Consider oropharangeal and nasopharangeal airway insertion.
- Consider two-person (two-handed) facemask ventilation.
- Consider suctioning patient.
- Consider paralysis – with caution.
- Consider LMA insertion if difficult facemask seal.

2. Failed intubation:

- Call for assistance – or declare the situation to your assistant.
- Revert to facemask ventilation.
    - If difficult facemask ventilation, revert to above (1).
- Maintain cricoid pressure if RSI.
- Hand over to more experienced endoscopist if present.
- Ensure optimal positioning and reposition patient if necessary (remember – earlobes higher than clavicles).
- Ensure adequate sedation/relaxation.
- Ensure optimal external laryngeal manipulation:
    - Consider releasing cricoid pressure (may be causing distortion of normal anatomy)
    - Cricoid pressure might not be adequate.
    - Consider applying cricoid pressure yourself.
- Cant' see epiglottis?
    - May be too deep? – consider withdrawing laryngoscope blade slowly.
    - May not be deep enough? – consider larger blade/inserting deeper.
- Can see epiglottis, can't see cords?
    - Consider more OELM.
    - Consider "blind" introduction of gum elastic bougie by 'feel'.
    - Consider railroading with Parker Flex-tip ETT (if available) or 'regular' ETT.

- Consider standard LMA, or ProSeal equivalent if available.
- Consider Laryngeal Tube (King Airway).
- Consider surgical cricothyroidotomy.

Once airway established with above, consider the following in order to create a definitive endotracheal airway:

- Consider I-LMA if available.
- Consider Airtraq optical laryngoscope if available.
- Consider flexible fibre-optic laryngoscopy.

Flexible fibre-optic laryngoscopy is not a good first option in a failed RSI or CICO situation. Fibre-optic intubation precludes ventilation, and visualisation of the cords is extremely difficult in the presence of blood and secretions (which are usually present by the time the FO-scope is brought into action!). I-LMA is a better option, as it is possible to ventilate the patient through an I-LMA before 'blind' placement of the ETT.

ALGORITHMS FOR DIFFICULT AND FAILED INTUBATION

Please see appendices for American Society of Anesthesiologists, Resuscitation Council of South Africa and United Kingdom Difficult Airway Society algorithms. All of these algorithms are self-explanatory.

Practice guidelines and algorithms are systematically developed recommendations, based upon available evidence and expert opinion, that assist the practitioner. These may be adopted, modified or rejected according to clinical need, constraints and challenges. They do not necessarily constitute minimum standards or requirements, and do not guarantee outcomes when closely followed.

The Resuscitation Council of South Africa algorithm is refreshingly simple and 'linear' in its approach to the difficult and failed airway.

DAS GUIDELINES

The Difficult Airway Society of the United Kingdom's new 2015 guidelines (and algorithms) for unanticipated difficult airway have been simplified, and are undoubtedly the most up-to-date and current. These guidelines are based on published evidence and expert opinion, emphasise *early recognition* and *declaration* of airway difficulty, and provide a simplified, single algorithm for routine and emergency (RSI) intubation.[59]

The DAS 2015 guidelines focus on preoperative patient assessment, including front of neck access, adequate preparation, patient positioning, mandatory pre-oxygenation, maintenance of oxygenation throughout the difficult airway scenario, and minimising airway trauma which could potentially lead to a can't intubate can't oxygenate situation.

Management of unanticipated difficult tracheal intubation in adults

Plan A: Facemask ventilation and tracheal intubation

Optimise head and neck position
Preoxygenate
Adequate neuromuscular blockade
Direct / Video Laryngoscopy (maximum 3+1 attempts)
External laryngeal manipulation
Bougie
Remove cricoid pressure
Maintain oxygenation and anaesthesia

If in difficulty → call for help

Succeed → Confirm tracheal intubation with capnography

↓ Declare failed intubation

Plan B: Maintaining oxygenation: SAD insertion

2nd generation device recommended
Change device or size (maximum 3 attempts)
Oxygenate and ventilate

Succeed →

STOP AND THINK
Options (consider risks and benefits):
1. Wake the patient up
2. Intubate trachea via the SAD
3. Proceed without intubating the trachea
4. Tracheostomy or cricothyroidotomy

↓ Declare failed SAD ventilation

Plan C: Facemask ventilation

If facemask ventilation impossible, paralyse
Final attempt at facemask ventilation
Use 2 person technique and adjuncts

Succeed → Wake the patient up

↓ Declare CICO

Post-operative care and follow up
• Formulate immediate airway management plan
• Monitor for complications
• Complete airway alert form
• Explain to the patient in person and in writing
• Send written report to GP and local database

Plan D: Emergency front of neck access

Scalpel cricothyroidotomy

This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Features of the DAS 2015 guidelines:

This is essentially linear algorithm, starting with direct laryngoscopy, moving on to supraglottic device placement in the event of failure, reverting to facemask ventilation in the event of failure of SGA placement, and then proceeding directly to surgical airway intervention in the event of facemask ventilation failure.

Human Factors are acknowledged as a causative or contributory aspect of the failed airway, and receive appropriate attention: judgement and decision-making, team briefing, avoiding tunnel-vision, communication and assertiveness training, and planning for the event of failure. There is also a focus on adequate rehearsal and simulation training.

Graded assertiveness model – PACE:

Probe: *"Are you sure you can ventilate adequately?"*
Alert: *"It doesn't appear to me that you're oxygenating the patient!"*
Challenge: *"This is an airway emergency. It doesn't appear that you're in control of the airway".*
Emergency: *"Please provide 100% oxygen while I ready the Difficult Airway cart".*

Plan A: Facemask ventilation and tracheal intubation:

Optimise position - Morning sniff, ramp and 20 degrees' head-up.

Focus on pre-oxygenation with gentle facemask ventilation.

Ensure full neuromuscular paralysis.

No more than three attempts at laryngoscopy/intubation, with one additional attempt by an experienced colleague, minimises the likelihood of airway trauma and swelling.

Use of optimal external laryngeal manipulation (cricoid pressure), and the appropriate relaxation of cricoid pressure is advocated.

The use of bougies is encouraged.

Maintenance of oxygenation through nasal prongs oxygenation (NODESAT) is encouraged.

Plan B: Maintenance of oxygenation – SGA insertion:

Cricoid pressure must be relaxed whilst inserting the supraglottic airway device.

Second-generation SGAs (ProSeal LMA) are encouraged, since they provide more reliable first-time placement, have higher sealing pressures and separate gastric ports for drainage, reducing the likelihood of aspiration.

Enhancing the chances of first-time placement is important, as this decreases the chances of desaturation and further airway trauma.   Each successive attempt at placement is associated with a decreased likelihood of success – only 4% of SGAs are successfully placed on the third or fourth attempt.

Bougie-assisted placement of the ProSeal LMA is associated with 100% success rate, and should be considered.

Placement of the Fastrach Intubating Laryngeal Mask Airway may be considered.

Successful oxygenation and ventilation during this stage provides the team with the opportunity to stop and think, and review the immediate plan to follow.

Plan C: Facemask ventilation:

Ensure full paralysis.   If sugammadex has been given, a muscle relaxant other than rocuronium or vecuronium must be administered.

Use oropharangeal and nasopharangeal airways and a two-person, four-handed technique if necessary.

Plan D: Emergency front of neck access:

Cognitive processing and motor skills decline in high-stress situations, and so a simple rescue plan, with a high chance of successful outcome, is advised.   Equipment needs to be familiar, readily available, and the procedure needs to be rehearsed.   A cuffed tube provides an airway seal, allows normal minute ventilation, provides protection against aspiration, allows coupling to a standard ventilator circuit, provides a secure route for exhalation and facilitates $ETCO_2$ monitoring.   Needle cricothyroidotomy facilitates none of the above.

100% oxygen is administered to the upper airway by whatever means possible (facemask, nasal cannula or SGA).   The procedure is done under complete neuromuscular paralysis with the neck fully extended.

The "laryngeal handshake" is performed as follows:
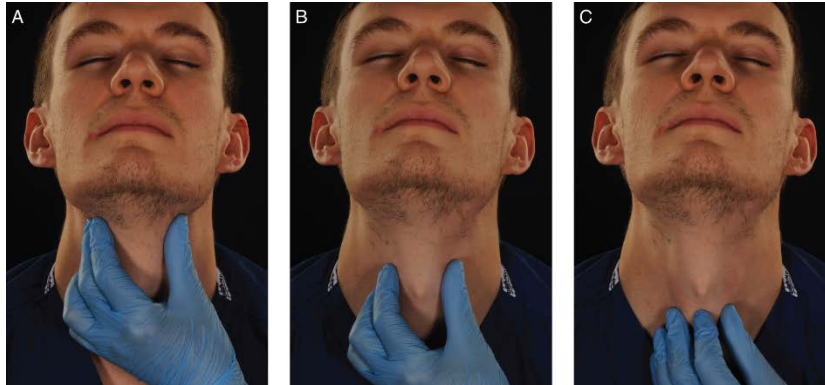
A: The non-dominant hand *identifies* hyoid bone & thyroid cartilage (laminae).

B: The larynx is *stabilised* between thumb and middle finger.

C: The cricothyroid membrane is *palpated* with the index finger.

A scalpel with No. 10 blade, a bougie with coude (angled) tip and a 6.0mm cuffed ET tube is all that is required to perform the procedure.

The scalpel-bougie-tube surgical airway technique is performed as follows:

- Stand on patient's left if you are right-handed

- Vice versa if you are left-handed

- Perform laryngeal handshake

- Stabilise larynx

- Scalpel cutting edge faces you

- Scalpel blade perpendicular to skin

- Transverse stab incision

- Rotate scalpel 90° – cutting edge faces down

- Rotate scalpel 90° – cutting edge faces down

- Swap hands – hold scalpel in non-dominant hand

- Feed bougie (horizontal) through incision 10 – 15 cm

- Remove scalpel

- Railroad lubricated ET tube (rotate as required)

- Avoid endobronchial intubation

- Remove bougie

- Inflate cuff

- Ventilate and confirm $ETCO_2$

In the event of obesity and an impalpable cricothyroid membrane:

- Perform an 8 – 10 cm vertical midline incision

- Use blunt finger-dissection to identify larynx

- Proceed with scalpel technique as above

THE VORTEX

FOR EACH LIFELINE CONSIDER:

MANIPULATIONS:
- HEAD & NECK
- LARYNX
- DEVICE

ADJUNCTS

SIZE / TYPE

SUCTION / O_2 FLOW

MUSCLE TONE

MAXIMUM THREE ATTEMPTS AT EACH LIFELINE (UNLESS GAMECHANGER)
AT LEAST ONE ATTEMPT SHOULD BE BY MOST EXPERIENCED CLINICIAN
CICO STATUS ESCALATES WITH UNSUCCESSFUL BEST EFFORT AT ANY LIFELINE

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The **Vortex** Airway Management Checklist

Prepare Interventions	Prevent Hypoxia	Promote Teamwork
Intravenous Access ☐ Adequate ☐ Running **Drugs:** Agent Dose Labelled Intubation: ☐ Induction ☐ Paralysis ☐ Adjuncts ☐ Emergency Post Intubation: ☐ Anaesthesia ☐ Analgesia ☐ Paralysis **Monitoring:** Confirm Optimise ☐ ETCO₂ ☐ SaO₂ ☐ BP ☐ ECG ☐ Alarms	**Safe Apnoea Time** PreOx: ☐ 100% O₂ Connected/Flowing ☐ Optimise FRC ☐ PreOx Complete ApOx: ☐ O₂ Connected/Flowing ReOx: ☐ BVM/Ventilator ☐ Ventilator Settings ☐ Consider PEEP **Vortex Approach** ☐ Position ☐ Suction Face Mask: ☐ Adjuncts ☐ FM: size/type Supraglottic Airway: ☐ SGA: size/type Endotracheal Tube: ☐ Adjuncts ☐ Laryngoscope: size/type (incl VL), functioning ☐ ETT: size/type ☐ Syringe/Cuff Test ☐ Tape/Ties Emergency Surgical Airway: ☐ ESA Kit	**Additional Staff:** ☐ Supervision ☐ Help: Senior/Anaesthetics/ENT **Roles:** Capable Briefed ☐ Team Coordinator ☐ Vortex ☐ Airway Operator ☐ Airway Assistant ☐ Cricoid ☐ Manual In-Line Stabilisation ☐ Drugs ☐ Monitors/Time ☐ Emergency Surgical Airway **Plan:** Discussed ☐ Anticipated Difficulties: FMV/ETT/SGA/ESA ☐ Vortex Sequence ☐ Vortex Optimisations ☐ Green Zone Options ☐ Questions/Concerns?

VORTEX APPROACH

Factors influencing performance during emergency airway management can be divided into:

1. Preparation:
   - Training
   - Experience
   - Consultation
   - Planning

2. Implementation:
   - Cognitive overload
   - Poor evaluation
   - Poor Situational Awareness
   - Poor recall of options
   - Poor decision-making
   - Omission, fixation
   - Failure to act

Problems occur with implementation far more than with preparation. Psychological barriers to surgical airway interventions may reduce performance.

Guidance tools are comprised of two types; foundation tools used for training such as protocols and guidelines, and implementation tools such as algorithms, checklists, SOPs, manualsand visual aids.

Well-constructed implementation tools have the potential to facilitate team situational awareness, improve communication, prompt decision-making, and guide key actions during high-acuity high-stress situations.   A low-content, graphic design is more suited to real-time use during crisis situations.

The Vortex model makes two assumptions.   The first is that it will be used by competent clinicians – Vortex is not a training substitute; it is there to prompt decision-making, not provide core knowledge.   The second assumption is that teams will have prior training in the tool's use – such as is provided in a simulation environment.

Vortex attempts to address impaired decision-making:
- Failure to consider all available techniques
- Failure to consider all the strategies available for optimizing success at each technique
- Excessive airway instrumentation
- Failure to recognize the significance of achieving oxygenation
- Failure to recognize the 'can't intubate, can't oxygenate' (CICO)
- Lack of priming for emergency front-of-neck access
- Delayed implementation of emergency front-of-neck access
- Impaired teamwork

Vortex provies three means or 'lifelines' for securing a non-surgical airway:
- Face mask
- Tracheal Tube
- SGA

It does not matter where one starts in the vortex:
- Failure of 'best effort' at one modality necessitates moving to the next 'lifeline'.
- 'Best effort' is defined by context – acknowledgement of failure prompts the whole team to move to the next option.
- A maximum of three attempts at each lifeline; but fewer attempts are acceptable, before moving on to the next lifeline.
- Any repeat attempt must include 'optimisation' methods, rather than simply doing "more of the same".

The green zone of the vortex implies that the patient is adequately oxygenated.   One can use the green zone to consider options, and formulate a plan for the immediate next steps.

Narrowing of the funnel implies diminishing time and options.   The horizontal green surfaces represent time to pause and plan, whereas the deepening blue colour lower down in the funnel suggests imminent hypoxaemia if airway patency is not restored.

If best efforts at all lifelines have been unsuccessful, CICO rescue is a crucial intervention to make the patient safe. The visibility of the green zone at the bottom of the funnel is intended to convey this path to safety and to encourage clinicians to proceed in this circumstance. CICO status should not reach 'go' unless best efforts at all three lifelines have been unsuccessful.

CICO STATUS ESCALATES WITH AN UNSUCCESSFUL 'BEST EFFORT' AT ANY LIFELINE!

- Consider additional escalation in CICO status if:
- Predicted difficult airway
- $SpO_2 < 90^o$
- Rapidly deteriorating SpO_2
- Consecutive unsuccessful attempts at any two lifelines

ENSURE BEST EFFORT IN ALL THREE LIFELINES BEFORE DECLARING 'GO' STATUS

Team Situational Awareness and team communication enhanced by Vortex model:

Cardiac arrest management includes verbal declarations, such as 'no pulse' or 'shockable rhythm', which convey critical moments of situational awareness to the clinical team. The Vortex model encourages similar verbal declarations, enhancing a shared team situational awareness. Vortex introduces standardized terminology, such as 'completed best effort', 'in the green zone', 'CICO status', and 'sucked into the Vortex'

SURGICAL AIRWAY INTERVENTIONS

The first thing to say about surgical airway intervention, is that its contemplation loudly heralds the ***ultimate failed airway***.   The second thing to say is that it is a technique or intervention that is used very seldom indeed, and is therefore often doomed to failure. Supraglottic rescue airway devices should <u>always</u> be attempted before surgical airway intervention is considered.

Surgical airway interventions must be considered <u>after</u> repositioning of the patient (earlobe/sternal notch or ramping), application of external laryngeal manipulation, the use of gum elastic bougies, stylets, etc., and after attempting airway rescue with supraglottic airway devices.

In the wrong hands, surgical airway intervention following a 'failed airway scenario' leads to no more than a '*bleeding* failed airway scenario'.

The NAP4 study (which assessed 2.9 million anaesthetics undertaken in the UK over the course of one year) revealed a very high failure rate (60%) of emergency cannula **cricothyroidotomy**.   Causes of failure were related to equipment, training, insertion technique and ventilation technique.

In contrast, emergency **surgical airway** intervention (tracheostomy) was almost universally successful.

The NAP4 investigators concluded that (1) all anaesthetists (and others managing difficult airways) should be trained to perform surgical airway techniques (2) that cricothyroidotomy must be taught to the highest standards to ensure success in emergency situations, and that (3) we must consider that cannula cricothyroidotomy **may be an intrinsically inferior procedure (and therefore a poorer option)** in comparison to a formal surgical airway.[46,47]

Non-cuffed cricothyroidotomy tubes are prone to high leakage rates past the tube, especially in obese or heavily gravid obstetric patients, where the abdominal contents push up on the diaphragm, preventing the delivery of adequate tidal volumes to the lungs.

The use of such devices is presently discouraged, but if such devices are used, practitioners must remember to place a large wet (preferably abdominal) swab in the posterior pharynx, and to occlude the nostrils, if the tidal volume generated by positive pressure ventilation is to be delivered to the lungs and not lost to the atmosphere.

Furthermore, anaesthetists, emergency physicians and paramedics are generally not very comfortable with surgical procedures (wielding cold steel), but may be very comfortable utilising the Seldinger guidewire technique.   The easy, unobstructed, threading of the guidewire helps in confirming correct tracheal placement.

Remember that in many cases of a failed airway scenario, a poorly-executed attempt at surgical airway placement results in nothing more than a bleeding failed airway scenario (as already mentioned above). We therefore need to teach, and maintain currency in safe and effective emergency surgical airway management through (at minimum) bi-annual simulator training.

However, the DAS 2015 guidelines very strongly advocate the scalpel-bougie-tube surgical airway technique, and this is our primary focus, with the Seldinger technique as an additional option for those practitioners comfortable and practiced in its use. Needle cricothyroidotomy with uncuffed 'cannula over needle' type of devices is strongly discouraged!

Needle 'cric' sets such as the VBM Quicktrach II and Rusch TracheoQuick sets are now discouraged by DAS 2015, owing to the very high rate of failure.

The advantages of the DAS 2015 scalpel-bougie-tube surgical airway technique is that this technique's reliability has been <u>unquestionably proven</u>, and it affords standardized training, using familiar, cost-effective and readily available equipment.   The technique is covered in great detail in the chapter on the Difficult and Failed Airway in this manual, under the DAS guidelines.

The disadvantage of using the Seldinger-guidewire techniques is that it may be more time-consuming, and that cognitive processing and motor skills decline in high-stress situations, reducing the likelihood of first-time success.
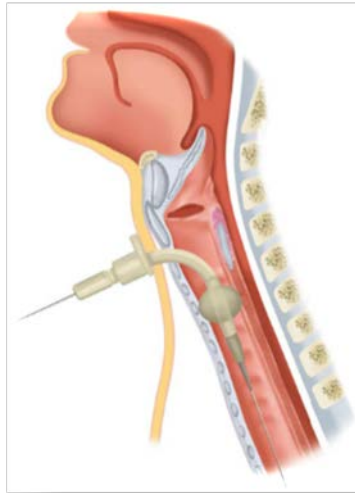
Some patients have such gross neck pathology (obesity, Ludwig's angina) that surgical airway interventions are simply not possible.   In these instances, awake fibre-optic intubation or semi-elective retrograde intubation (at least, placement of a guidewire) may be the only viable safe alternative.

Morbid obesity and Ludwig's angina can preclude surgical airway intervention.

The patient on the left is undergoing awake fibre-optic intubation – for obvious reasons.

Statistically speaking, most anaesthesiologists, during the course of their professional careers, will never undertake a single surgical airway procedure.

If you find that in your practice that you are undertaking many surgical airway procedures, then it is probably safe to say that **you are doing something wrong!**   If surgical airway intervention precedes the use of repositioning of the patient, employment of optimal external laryngeal manipulation, the use of gum elastic bougies, and the use of sugraglottic rescue airway devices such as the LMA, LTA or combitube, then it is safe to say that you are definitely doing something wrong!

Emergency Physicians and Paramedics generally carry out surgical airway interventions far more frequently than anaesthesiologists. This could be due to the fact that, owing to a more challenging working environment, they have a lower threshold for proceeding to surgical airway intervention. It could also be due to the fact that many paramedics do not routinely carry airway "assist" devices and supraglottic rescue airways.

A caveat to the above could be the postulated existence of a special cohort of threatened or obstructed airway patients, seen in the prehospital (and emergency unit) environment, who would not survive long enough to get to hospital without 'heroic' surgical airway interventions carried out in the field.

Additionally, surgical interventions should only be undertaken in the "can't intubate, can't oxygenate" scenario, and should not be used to create a definitive airway (in the emergency setting) where bag-valve-mask or supraglottic airway ventilation is successful, until all DAS 2015 options have been exhausted.

CRICOTHYROIDOTOMY

Cricothyroidotomy should be considered a last-resort rescue airway technique, and is indicated once a "failed away" (can't intubate, can't oxygenate) scenario has been declared.

One must consider the following:

- Will the technique be effective – is the obstruction above or below the level of the cricothyroid membrane?
- Will the anatomy allow for a successful procedure to be performed – eg. obesity or Ludwig's angina?
- Which type of surgical technique should be used – open surgical vs percutaneous?

MNEMONIC FOR DIFFICULT CRICOTHYROIDOTOMY: SHORTY

- Surgery – previous (scars)
- Haematoma, incl. abscess or infection (swelling) – Ludwig's angina.
- Obesity
- Radiation distortion and other deformity
- Tumour
- Young – patients under 20 kg

Surgical cricothyroitomy is contra-indicated in children below the age of 12 years (unless they are of large size), as this age-group has a soft larynx with small diameter, and a soft and pliable cricoid cartilage. Transtracheal jet ventilation only should be used in this age group.

The cricothyroid membrane, rather than the trachea, is the site of access for surgical intervention because it is more anterior than the trachea, has less overlying thyroid tissue and generally has less vascularity (but can still be a site of considerable haemorrhage!).
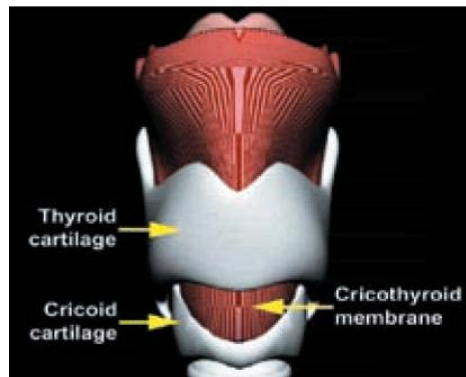
The cricothyroid membrane is identified by locating the laryngeal prominence (notch) on the thyroid cartilage.   Below this, another prominence can be felt – the cricoid cartilage. Approximately one finger-breadth below the thyroid notch, and immediately above the cricoid cartilage, a distinct "valley" can be palpated.   This is the cricothyroid membrane.
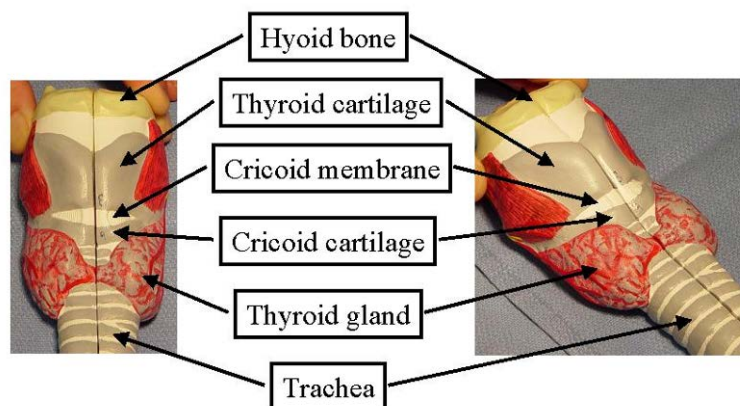
In the event that laryngeal anatomy cannot be identified as above, the cricothyroid membrane can be relatively accurately delineated by placing the four fingers of the flat hand longitudinally against the anterior aspect of the throat.   The little finger is placed in the sternal notch, and the area of skin underlying the index finger indicates the height of the cricothyroid membrane.

The little finger is placed in the suprasternal notch whilst the flat of the hand is placed over the throat as shown.   The index finger delineates the cricothyroid membrane as shown.

MELKER/COOK SURGICAL AIRWAY KITS

The Melker Universal Emergency Cricothyrotomy sets offer the opton of either a purely surgical (scalpe-based) technique, or a Seldinger technique, and provide a cuffed tracheostomy tube.

Melker/Cook cuffed Seldinger technique cricothyroidotomy set.   This set is considered the gold standard or "Rolls-Royce" of emergency surgical airway devices.

COMPLICATIONS OF NEEDLE AND/OR SURGICAL CRICOTHYROIDOTOMY:

- Paratracheal insertion and continued 'failed airway'.
- Haemorrhage – can be catastrophic.
- Aspiration of blood.
- Laceration of posterior trachea wall and oesophagus.
- Pneumomediastinum.
- Barotrauma.
- Infection.
- Subglottic stenosis (later complication).
- Vocal cord trauma/paralysis and hoarseness

RETROGRADE INTUBATION

Retrograde intubation cannot really be considered an emergency procedure, as it can be fairly time-consuming, and would therefore not be fully appropriate in a failed airway situation, in an already-desaturated patient.

It really is an elective procedure, which implies pre-planning, preparation and the setting up of equipment and trays beforehand.

However, in a true emergency situation, it is a technique that can be considered.   As with cricothyroidotomy, it is a technique that is seldom undertaken, and practitioners are invariably poorly prepared to undertake such a procedure – and the less so the more emergent the circumstances.

The initial approach to the technique is essentially similar to the cricothyroidotomy procedures discussed above, and is detailed below.

- Landmarks are palpated and identified.
- The cricothyroid membrane is punctured with needle and syringe.
- The Seldinger technique is used to place needle and guidewire into the trachea (with "bubble technique" as previously described), but the guidewire is angled or aimed upwards (cephalad) and not downwards (caudad).
- The guidewire is advanced and the distal end retrieved with a Magills forceps from the mouth or posterior pharynx, or from the nose – whichever is desired.
- The insertion needle (at the cricothyroid membrane) is removed, and an artery forceps is attached to the guidewire (clamped in position) at the distal (cricothyroid) end, preventing the guidewire from being pulled right through and out the mouth or nose.
- A tracheal tube can be loaded directly onto the oral or nasal end of the guidewire (pulled fairly taught), although the guidewire can act as a "cheese-wire" and cut into the soft tissues of the posterior pharynx, with this technique.
- It may be better to load an Aintree tube-exchange catheter onto the oral or nasal end of the guidewire first, and then load the tracheal tube over the tube-exchange catheter.
- The tracheal tube is then advanced as far as possible – through the vocal cords.
- The guidewire is then removed first, facilitating further advancement of the tracheal tube, by railroading the tube along the tube-exchange catheter.
- The tube-exchange catheter is then withdrawn after the tracheal tube has been placed at an adequate depth.
- The tracheal tube cuff is inflated, the patient is ventilated, and tube placement is confirmed through clinical means and capnography.
- The tube is then strapped in position and tube placement reconfirmed.

Please see the section on the paediatric airway under "Special Cases" for more information and illustrations on both needle cricothyroidotomy and retrograde intubation in the paediatric population.
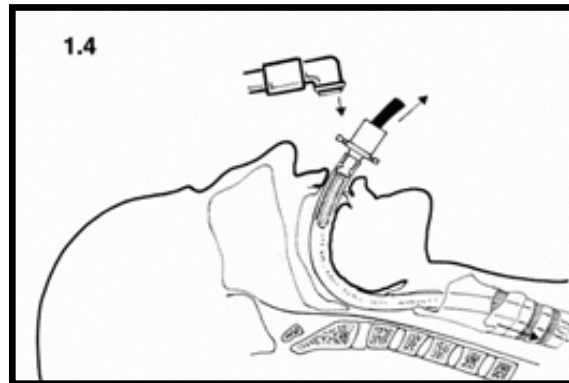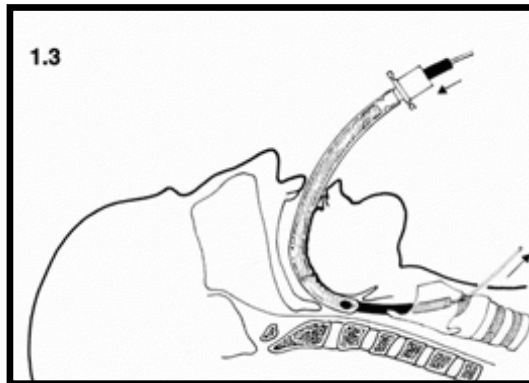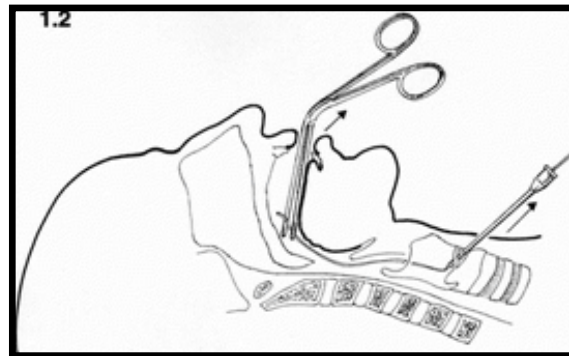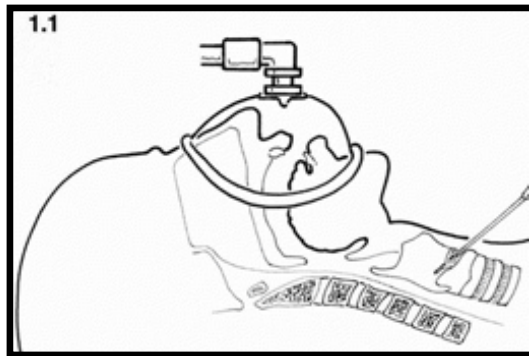
1.1 Needle is inserted through the cricothyroid membrane and angled upwards.
1.2 Seldinger wire is inserted, advanced and retrieved from the posterior pharynx
1.3 Tube-exchange catheter and ETT are advanced over the Seldinger wire.
1.4 The tube-exchange catheter and Seldinger wire are removed, the tracheal tube is connected to the ventilator circuit or BVMR, tube placement is confirmed and the tube secured.
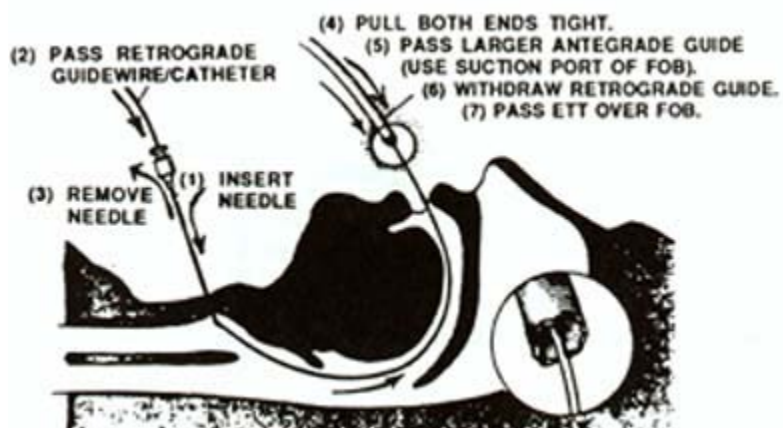
Useful tip:[60]

If one finds one's self in the unfortunate situation of having to anticipate a cricothyroidtomy as a ""Plan B" in a potential failed airway situation, it might be prudent to place a central venous catheter through the cricothyroid membrane, under local anaesthetic, prior to commencement of the intubation procedure itself.

CT scan of the neck and airway will help to assess airway patency and the value of this technique.
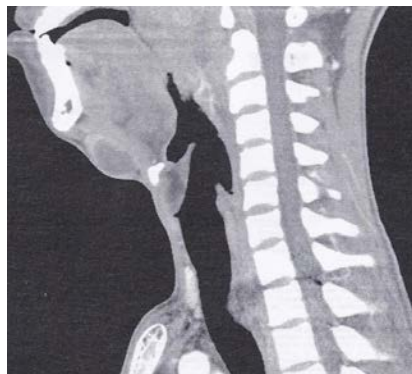
Prior placement of the central venous catheter provides a guide to the surgical 'track' and clearly delineates the anatomy, which is especially useful in obesity or in neck distortion due to tumour.

Jet ventilation can be carried out through the central venous catheter if needed.
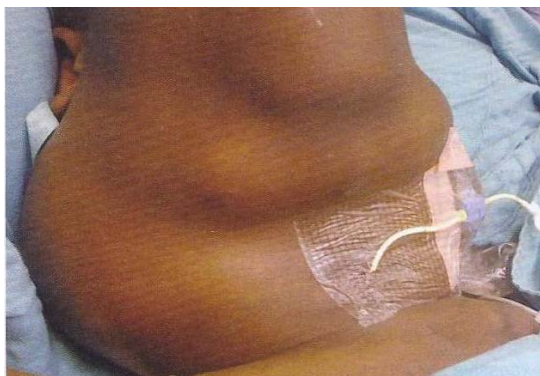
Single-lumen CVP catheter placed prior to induction of anaesthesia.

Tracheostomy in situ in recovery room.

ALTERNATIVE AND 'ASSIST' AIRWAY DEVICES:

MCCOY LARYNGOSCOPE

The McCoy laryngoscope has a lever on the handle that retracts a hinged tip Magill blade. This provides added leverage in order to lift the epiglottis to gain a better view of the cords. Whilst being a good idea on paper, personal anecdotal experience with the McCoy blade during difficult airway situations has revealed that whilst there may be some benefit, the overall additional performance of the McCoy laryngoscope is unremarkable.

McCoy Laryngoscope

SHORT-HANDLED LARYNGOSCOPE

Short-handled laryngoscopes are especially useful when intubating obese or pregnant patients, especially those with large breasts, who require RSI. The assistant's hand, which applies cricoid pressure, can completely obstruct the handle of a conventional laryngoscope, while inserting the laryngoscope.

Short-handled compared to normal laryngoscope

A trick to placing a conventional laryngoscope under such circumstances is to turn the laryngoscope 'sideways', with the handle perpendicular to the head-to-foot plane of the patient, insert the blade, and then turn the blade back into the normal orientation. This is demonstrated in the accompanying Powerpoint presentation.

POLIO MACINTOSH BLADE

The polio blade was designed many years ago when polio was an endemic disease. Polio resulted in significant chest wall contracture and deformity, which resulted in difficult intubation. The Polio blade attaches to the laryngoscope handle at 45 rather than the standard 90o, and this makes it possible to wield the laryngoscope and place the blade in the vallecula in situations such as patients with large breasts, or in obesity.

The first problem with the polio blade is that there are very few around, even though they are reasonably easy to obtain. The second problem isthat they are rather disconcerting to use when used for the first time, as the 45o angle throws the operator considerably "off balance", when 'brought up' on a 90o angle   The last place to introduce yourself to a Polio blade is your first Can't Intubate Can't oxygenate situation!

Polio Macintosh blade   Heine "polio" angled handle

NASOPHARANGEAL AIRWAY

Nasopharangeal airways are very useful adjuncts to oropharangeal airways. They can be used in conjunction with OPAs during difficult mask ventilation, or can be used as stand-alone devices in patients who will not tolerate OPAs, or in non-paralysed patients with clenched jaws (as occurs in head injury). They are constructed of soft latex, are available in different sizes, and are unlikely to induce epistaxis, except in patients with hyperaemic nasal mucosa, such as pregnant women at term!

In the absence of NPAs, ETTs can be 'cut down' to serve as NPAs. The primary disadvantage of this makeshift arangement is that epistaxis may be caused by the relatively hard ETT, which could further compromise airway management.

Unlike the McCoy blade, the correct use of the GEB makes this simple device indispensible on the difficult airway trolley.   Under most circumstances, a patient with a Cormack & LeHane grade III or IV view can be reliably intubated, without requiring a direct view of the vocal cords.

The GEB can be 'preloaded' onto the ETT (with lubrication) or can be placed without loading the ETT.   The ETT can then be 'rail-roaded' in a Seldinger-technique once the GEB has been correctly placed in the trachea.   Rail-roading is easier and less traumatic if a Parker Flex-tip is used instead of a standard ETT.

If the ETT is to be preloaded, the tube and GEB should be kinked by holding it forcibly between the index and middle finger, and the opposing thumb (see picture).   This prevents slipping of the GEB in the tube as it is advanced into the trachea.

Note that the GEB is intentionally 'floppy', and has a kink at the anterior tip (Coude tip).   The tube can be roughly preformed, and blindly passed into the hypopharynx with the kinked tip is angled upwards towards the "roof" of the trachea in the supine patient.   The GEB is then blindly advanced, and usually tracks upwards into the trachea, as opposed to downwards into the oesophagus.   It is helpful if one can visualise the tip of the epiglottis, as this indicates to the practitioner where the vocal cords are, roughly, even if they are not in direct vision.

An added advantage of the GEB is that the technique of blind placement can be practiced on elective patients, by intentionally creating a suboptimal view of the cords, in elective patients with otherwise normal laryngoscopic views.

Gum elastic bougie

Tips on railroading the ETT over the gum elastic bougie:

A standard ETT held in the "neutral" position, poised for intubation, has the bevelled opening oriented towards the left, with the 'leading edge' of the tube facing to the right.   This often results in impingement of the tube against the right arytenoid cartilage.   The tube also commonly impinges on the posterior ("bottom" in the supine patient) aspect of the glottic

opening.   This is a function of the shape of the tube, the mechanics of placing the tube through the vocal cords, and the action of gravity.   By rotating the tube 90° in the anti-clockwise direction *("quarter-round anti-clockwise"),* the bevelled opening faces 'downwards' (or posteriorly), and the 'leading edge' now faces upwards (or anteriorly).   This orientation of the bevelled edge assists in freeing up any impingement on either arytenoid and on the posterior aspect of the glottic opening.   Clockwise rotation would orientate the bevelled opening anteriorly (facing 'upwards'), and would therefore place the 'leading edge' posteriorly, where it can once again impinge on posterior glottic structures.   It is very unlikely that the tube will impinge in the anterior glottis.

Use of the Parker Flex-tip tubes largely overcomes the problems of impingement, as discussed below.
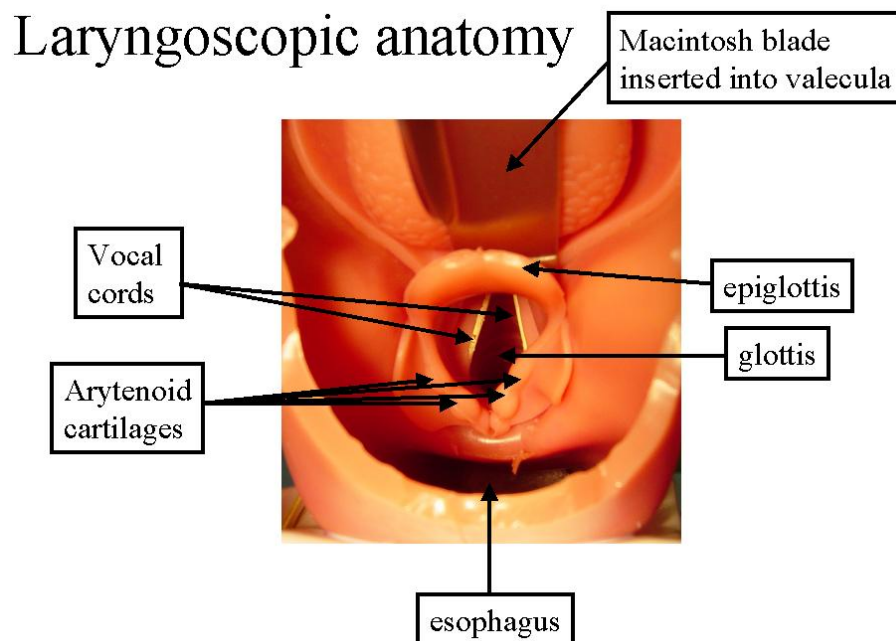
Standard ETT held in neutral position in right hand. View from left.

Close up of ETT tip, viewed from left. Note bevel opens to left; Murphy eye on right; leading edge on right

ETT held in neutral position, tip viewed from above

90° anti-clockwise rotation of tube places places leading edge anteriorly, bevel angled to allow easy 'slippage' over the posterior aspect of the glottis:

View of rotated tube from left

View of rotated tube from above. Notice rounded 'shoulders' allow slippage past arytenoid cartilages and through vocal cords.

PARKER FLEX-TIP® ETTS

'Regular' ETTs create a 'step' when railroading over a gum elastic bougie, which can cause snagging of the tube on hypopharangeal structures and on the vocal cords.   The Parker Flex-tip is specially designed with a soft atraumatic tip with a three-dimensional taper, creating a gradual transition from gum elastic bougie surface to ETT surface.   This aids snag-free railroading through the cords, and is also extremely advantageous for nasal intubations. Parker Flex-tip ETTs are minimally more expensive than regular ETTs, and are available through SSEM in South Africa.
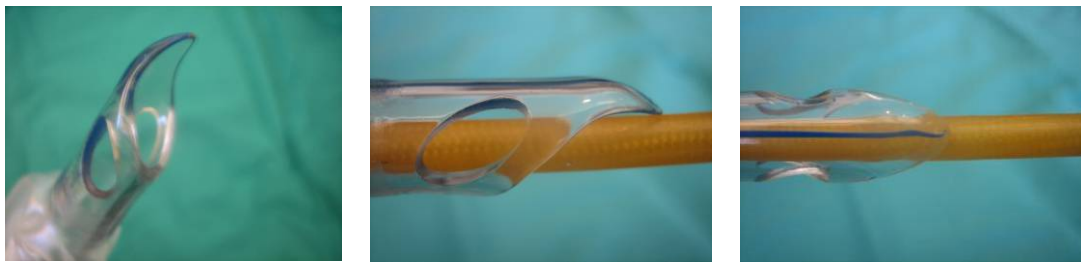
Parker Flex-tip endotracheal tube

PARKER STEERABLE INTRODUCERS

These steerable introducers are very simple, inexpensive and ingenious plastic devices, which facilitate up-down movement of the tip of the tube, on pressing the "plunger".   By turning the ETT sideways, left-right movement can also be achieved.   If the patient is adequately positioned, it is seldom necessary to use such a steerable introducer.   However, in the trauma situation, where in-line neck stabilisation must be maintained, these introducers may be of great value.

Parker steerable introducer

Parker steerable introducer

RUBBER-COATED MALLEABLE STYLET

The rubber-coated malleable stylet is a standard part of any intubation kit, as it provides rigidity and allows for the "hockey-stick" shaping of the ETT, in order to facilitate intubation. Malleable stylets are particularly useful when using armoured (re-inforced) ETTs.   Non-rubber coated malleable stylets must be used with great caution as they can cause considerable trauma if the tip inadvertently projects beyond the tip of the ETT.

Rubber-coated malleable stylet

AINTREE TUBE EXCHANGE CATHETERS

The Aintree catheter looks very similar to a gum elastic bougie, except that it is hollow, has a Murphy eye, and a detachable 15mm universal connector.   This allows ventilation, or at least entrainment of oxygen through the catheter when it is placed in situ.

The Aintree catheter is placed inside the ETT due to be exchanged.   The ETT is then removed by slipping it over the catheter.   The new ETT is then railroaded over the catheter and the catheter removed.

The Aintree catheter is a very useful device, as it can be 'loaded' onto a flexible fibre-optic scope, and can be introduced into the trachea via either a standard LMA or I-LMA with the aid of the scope.   The LMA is then removed, leaving the catheter in place.   A standard or Parker Flex-tip ETT can then be railroaded over the catheter.

This is a very useful technique during emergency intubation where blood and sectretions prevent an adequate view with the fibre-optic scope.   By placing the scope through the LMA, blood and secretions are isolated, it is possible to still ventilate the patient, and the tip of the scope is taken to the "doorstep" of the glottic aperture, minimizing the potential for a fibre-optic navigational nightmare.

Aintree exchange catheter

15mm connector detaches

Note the catheter is hollow

AIRTRAQ® OPTICAL LARYNGOSCOPE

The Airtraq is a very useful and relatively inexpensive disposable optical laryngoscope (inexpensive in comparison to a law suit or flexible fibre-optic bronchoscope).   These devices cost in the region of R500 each and are strictly single-use.   They essentially work on the principle of a "reverse" periscope, and are very useful for elective intubation where the vocal cords are not visible.   The Airtraq is marketed by Teleflex Medical in South Africa.
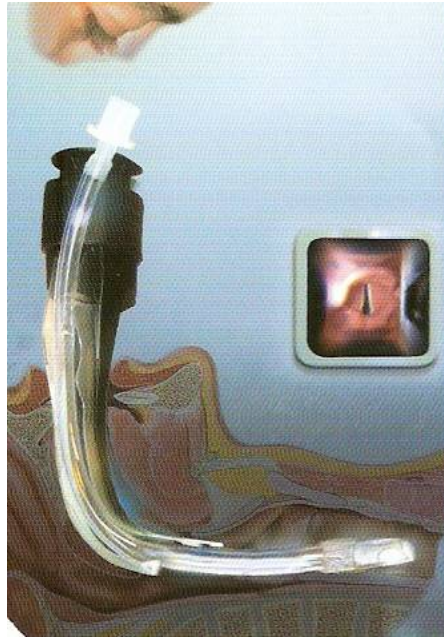
Airtraq Optical Laryngoscope

One problem that we have encountered with the Aitraq is that the smaller the tracheal tube used, the more it tends to angulate downwards.

Sometimes the manoeuvrability of the Airtraq in the mouth is somewhat limited, which does not enable 'upward' displacement of the Airtraq and the ETT. As a consequence, the tube cannot be manoeuvred upwards to clear the arytenoid cartileges, causing the tube to snag and prevent the tube from passing between the cords.

The solution to this is to pull the tube tip back to the distal opening of the Airtraq, and insert a gum elastic bougie (with kinked tip) into the tube, just protruding through the distal end. The bougie can then be swivelled between the index finger and thumb, allowing for full rotation and steerage of the bougie. The bougie is then advanced through the cords, and the tracheal tube railroaded over the bougie.

The above technique works extremely well.

The tip of the tracheal tube is pulled back to the distal outlet of the Airtraq and the gum elastic bougie inserted into the tube, with the kinked tip protruding through the tube tip
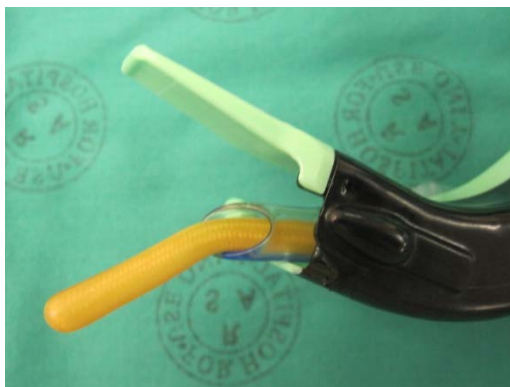
By swiveling the gum elastic bougie between the tips of the index finger, it is fully steerable and can be directed through the cords under direct visualization.   The tube is then railroaded off the bougie, through the vocal cords.

PENTAX AWS P-BLADE

The Pentax AWS P-Blade is marketed by Marcus Medical and costs more than R60 000. The disposable blades cost around R250 each, but can be sterilised and re-used. It has an adjustable video screen for a fibre-optic camera, and is compatible with a wide range of tracheal tubes. It looks like a very promising device, but the very high cost is obviously a hugely limiting factor.
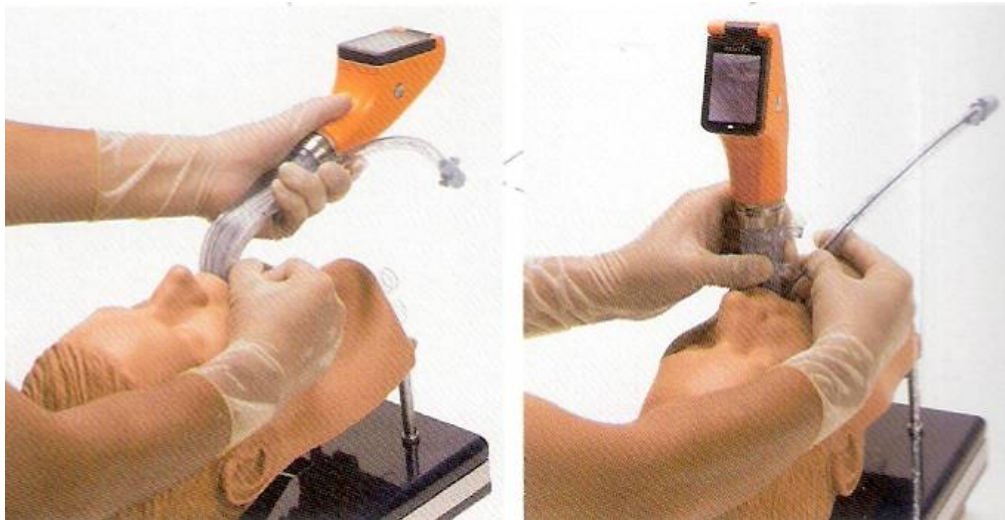
Pentax AWS P-Blade

KING VISION VIDEO LARYNGOSCOPE

The King Vision video laryngoscope is marketed by SSEM in South Africa. It has a simple two-piece design which comprises a reuseable monitor that attaches to disposable blades. In some respects this is similar to the Pentax AWS P-blade above, which also has a reuseable monitor and disposable blades. Except for the addition of the video screen, it is almost identical to the Airtraq.

This is an excellent option for getting started in video laryngoscopy due to low-pricing, quality imaging and simplicity of use. It is well-suited to the prehospital or aeromedical environment owing to its extremely light-weight and compact design, and will easily fit into a jump bag.

Like with the Pentax AWS P-blade, the operator does not need to lean over the patient, thereby allowing one to keep one's face a reasonable distance from the patient's airway, and allowing improved situational awareness.

The King Vision video laryngoscope

Left above: The King Vision with gum elastic bougie pre-loaded

Right above: Incredible views obtained – bougie passing through cords, soon to be followed by rail-roaded tracheal tube

One word of caution, though – patients must not be placed in the standard "morning sniff" – earlobe to sterna notch – position when using the King Vision. Patients should be placed with their necks in a hyper-extended position, as shown in the accompanying photographs below.
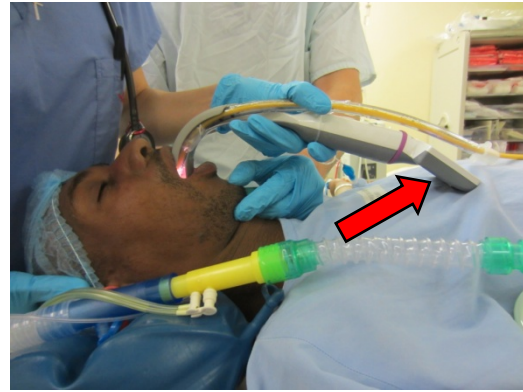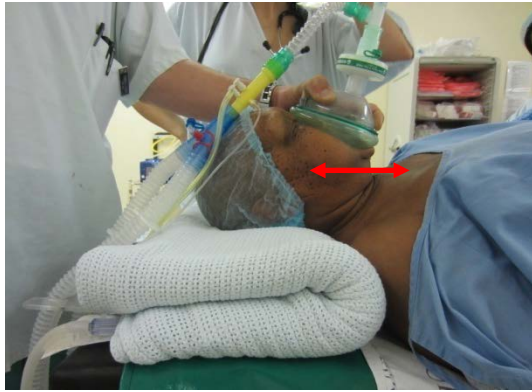
Left above: The normal morning sniff position – earlobe to sterna notch.

Right above: Neck hyper-extension is necessary to accommodate insertion of the King Vision.

The same technique of using a 'steerable' gum elastic bougie (as described with the Airtraq above) works exceptionally well with the King Vision.

Steerable introducer loaded into the King Vision

SUPRAGLOTTIC AIRWAY DEVICES

SADs under discussion here will include the following:

- Cuffed Oropharangeal Airways (COPAs)
- LMA Classic and variants (Ambu® LMA)
- Intubating LMA and Air-Qsp disposable intubating LMA
- Proseal LMA & LMA Supreme
- Combitube
- King LT Airway

Devices such as the Pharyngeal Airway Express and the Glotttic Aperture Seal Airway will not be discussed here.

The first supraglottic airway device was invented by Archie Brain in 1981, and is known as the Laryngeal Mask Airway or LMA. The LMA has been commercially available since 1988, and has been used on more than 200 million occasions.

SADs are generally easy to use, require relatively little training, produce little in the way of adverse cardiovascular responses, and have a significant role in emergency airway management.

SADs are only tolerated in patients with adequately anaesthetised airways, or in patients sufficiently obtunded (Traumatic Brain Injury, adequate sedation) to accept such an airway device.

Placement of an SAD does not constitute definitive airway management, as the airway is not fully protected until an ETT has been passed into the trachea with the cuff inflated. Whilst SADs cannot guarantee prevention of aspiration of gastric contents, they are efficacious at preventing aspiration of blood and saliva from the pharynx.

The lower sealing pressures of SADs compared to tracheal tubes may result in a leak when ventilating during chest compressions – which may cause gastric inflation and regurgitation.

One disadvantage of the LMA is that it is relatively easy to dislodge, with loss of airway patency and ventilation, and sometimes triggers tight laryngospasm.

CRICOID PRESSURE AND LMA INSERTION

There exists considerable controversy with regard to the application of cricoid pressure whilst inserting an LMA as a 'rescue' airway device under the circumstances of a failed intubation. Takashi et al [57] claim that the application of cricoid pressure makes LMA insertion 'much more difficult', and advocate the release of cricoid pressure while placing the LMA.[61] Brimacombe et al quote numerous references where they suggest that the application of cricoid pressure creates 'minimal' increased difficulty and that it should be applied.

Pnemonic for difficult placement of a supraglottic airway device: RODS:

- **R**estricted mouth opening
- **O**bstruction/**O**besity
- **D**istorted anatomy
- **S**tiff lungs or c-spine

CUFFED OROPHARANGEAL AIRWAYS

Cuffed oropharangeal airways (known as COPAs), have an inflatable cuff at the distal end, which helps to seal the pharynx. They also have a standard 15mm ISO attachment for a breathing circit or BVM at the proximal end, through which the patient can be ventilated without the need for a face mask. COPAs do not reduce the risk of pulmonary aspiration of stomach contents.

Cuffed OPA

LMA CLASSIC® (OR 'STANDARD' LMA)

There are a number of maufacturers now providing extremely competitively priced disposable LMAs.

Much of which has already been mentioned above applies to the LMA.   It is worth repeating that the LMA Classic (and variants) will only be accepted in sufficiently obtunded patients, and is relatively easy to dislodge, with consequent loss of airway patency and/or the onset of laryngospasm, and/or the risk of aspiration of blood, saliva and secretions from the upper airway.

It is well worth emphasizing that as a 'rescue' airway device, the LMA Classic has been reported to successfully ventilate patients 94% of the time, in a CICO situation.[58]

The LMA Classic will only provide a seal up to peak airway pressures of 25 to 30 cmH$_2$O. Pressures beyond these will result in leak, and/or gastric insufflation.   Pressures above 15cm cmH$_2$O may result in gastric insufflation.

Insertion techniques:

The tip of the LMA can fold either forward or backwards on insertion, and the epiglottis can herniate into the tube portion of the LMA.   Whilst LMA insertion is usually straight-forward and trouble-free, successful insertion and achievement of an adequate seal is sometimes unpredictable.

 The LMA can be inserted fully deflated or partially inflated.   My preference is to use a partially inflated cuff, as this lends stability to the cuff, reducing the incidence of the tip "folding over" upon itself.   An advantage of the Ambu® LMA is that the tip has been intentionally reinforced, and the angle of the tube has been anatomically angled to assist insertion.

Three methods of insertion have been described:

1. One holds the device in the left hand, like a ballpoint pen.   The index finger presses the tubing against the hard palate, inducing a curve in the device.   This necessitates the operator placing their finger/hand into the patient's mouth.   Rubber gloves are therefore essential.   The LMA is then 'slid' as far as it will go, into the hypophaynx. The device is then released, and the cuff fully inflated.   "Re-seating" of the device is seen on inflation (the tip of the tube moves one to two cm in a cephalad direction). This is usually an indication that the tube is correctly placed and will provide a good

seal.   Re-seating is not always observed with the Ambu® LMA, owing to the preformed curvature of the tube.   The patient is then ventilated through the device.

2. The device is held by the tip of the tube, and inserted into the mouth, angled at about the seven o'clock position (with the operator positioned at the patient's head).   The cuff is then slid down over the left hard palate and down into the posterior pharynx, simultaneously angling into the midline as the LMA is advanced.   The palate and posterior pharynx thus provide support and guidance for the cuff, assisting in (but not guaranteeing) the prevention of the cuff folding over.   This precludes the need for inserting a finger or hand into the patient's mouth, although gloves should still be worn.   Re-seating is observed on full inflation of the cuff.

3. If the above two methods are unsuccessful in providing adequate insertion and mask seal, the third and last method usually proves successful (third time lucky), a method which I call the "reverse-plop" technique:

The LMA is reversed on initial insertion, in exactly the same way that an oropharangeal airway is often anatomically reversed on initial insertion, and then "cork-screwed" into correct anatomical position as it is advanced.   Once the LMA is fully inserted in the reverse position, it is then held by the tip of the tube and given a strong twist.   This usually results in a clear "plop" being felt or heard as the LMA aligns itself anatomically.   The LMA is then fully inflated and the patient ventilated, after observing re-seating.
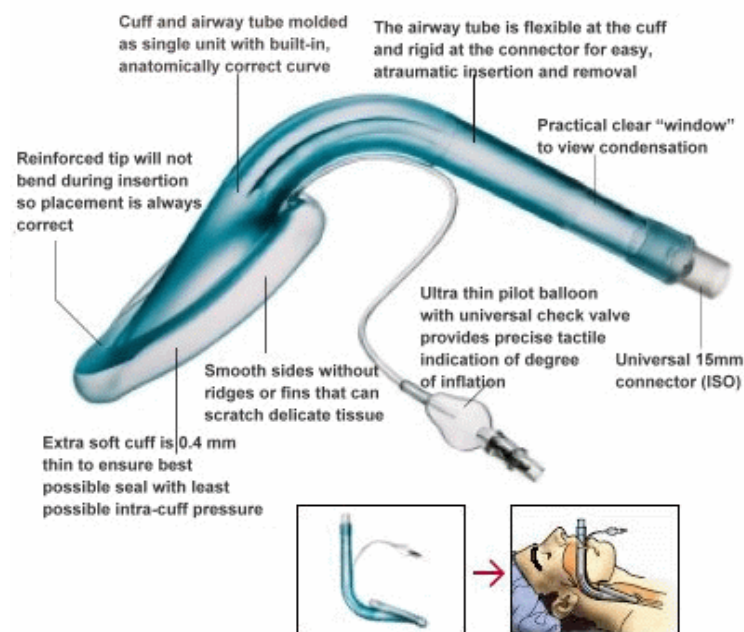
The 'Ambu' LMA – variation on the LMA Classic

PROSEAL® LMA AND LMA SUPREME®

The Proseal LMA (P-LMA) has an additional posterior cuff to improve the seal around the glottis to facilitate higher positive pressure ventilation.   An oesophageal drain tube guides positioning of the device and facilitates gastric deflation.   The Proseal LMA can be considered the "Rolls-Royce" of all supraglottic airway devices.

The Proseal LMA or its disposable equivalent, the LMA Supreme, is perhaps the most suitable supraglottic device for pregnant patients, obese patients, and those with potentially full stomachs.   The higher sealing pressures allow for high ventilatory pressures in these patients and provides better protection against aspiration.   The gastric port allows for insertion of a gastric tube for deflation and drainage of the stomach.   Additionally, passage of a gastric tube through the port confirms correct placement and 'seating' of the LMA in the posterior pharynx.

Face mask ventilation with high airway pressures leads to inflation of the stomach, consequent splinting of the diaphragms, and increasing difficulty in ventilation.   Deflation of the stomach improves ventilation significantly, and reduction of gastric fluid volume reduces, but does not eliminate, the risk of pulmonary aspiration of gastric contents.

Note that the inability to aspirate gastric contents from a naso- or orogastric tube does not necessarily imply an empty stomach!

The LMA Supreme® has a rigid structure, which can create challenges in the correct placement of the device – take care to ensure correct 'seating'. Note that an ill-fitting P-LMA may result in leakage of air through the gastric drainage port, amounting to a significant circuit leak, with consequent under-ventilation!!

(The disposable equivalent of the P-LMA is the 'LMA Supreme®', marketed by Marland Medical in South Africa).

Proseal LMA: The "Rolls-Royce" of Laryngeal Mask Airways
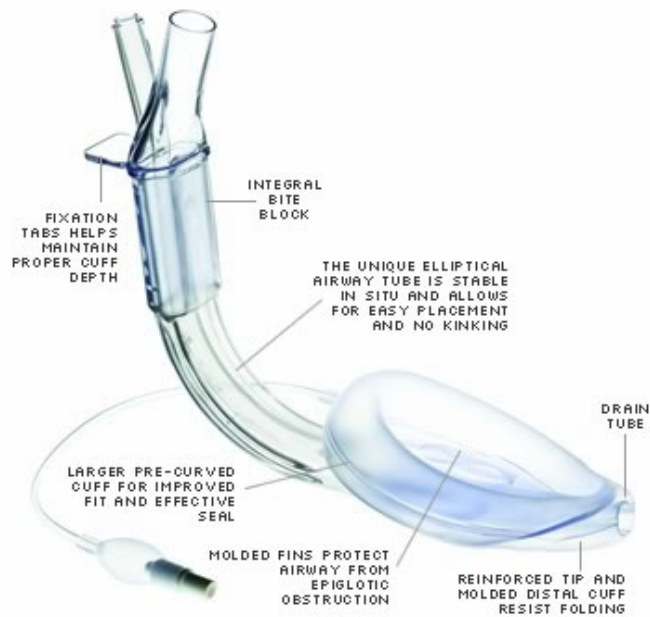
LMA Supreme – disposable version of the Proseal LMA.
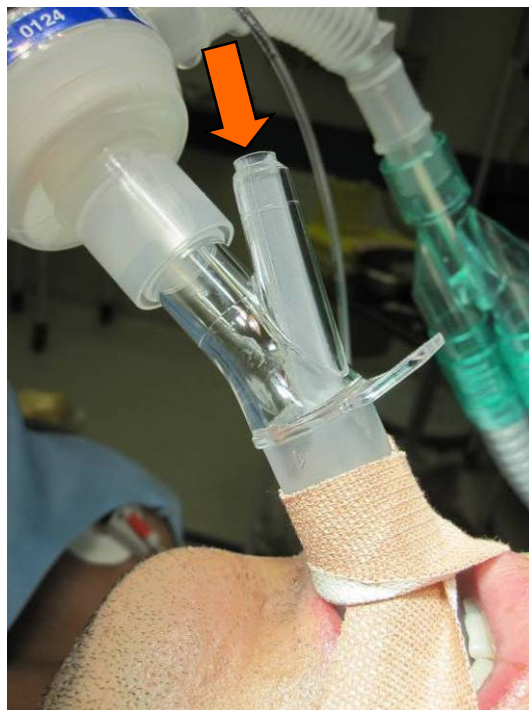
The LMA Supreme in situ – notice the large gastric drainage port – assists in deflating the stomach and confirming correct positioning through successful passage of a NG tube – but also a potential cause of a large air leak in a poorly-fitting LMA.

INTUBATING LMA (FASTRACH®)

The Intubating LMA (or I-LMA), commercially available as the Fastrach, is available in sizes 3, 4 and 5, through Marland Medical in South Africa. Disposable and re-usable versions are available. The re-usable version costs in the order of R4500 each, and the disposables in the order of R1500 each. Despite their expense, these devices are invaluable and should be considered an <u>essential</u> item in any difficult airway kit.

The obvious advantage of the I-LMA over flexible fibre-optic intubation is that, firstly, the patient can be ventilated once the LMA is inserted. This is not the case with the flexible fibre-optic scope.

Secondly, intubation is undertaken as a fully "blind" procedure. Usually, as a difficult or failed intubation scenario unfolds, an increasing amount of blood and secretions complicate one's efforts to successfully place an ET tube. Fibre-optic intubation can be rendered impossible under these circumstances, as one reaches a point where "pink froth" completely obliterates any view of the vocal cords.

Additionally, flexible fibre-optic intubation requires a considerable amount of skill and experience on the part of the operator, whereas this is not the case with I-LMA intubation.

In a study of 254 patients with recognised difficult airway, 100% were successfully ventilated with the I-LMA and 96.5% were successfully intubated.[62] In another study of 100 patients with anticipated difficult airways, randomised to fibre-optic and I-LMA, 92% were successfully intubated by fibre-optic laryngoscopy and 94% were successfully intubated using the I-LMA, both groups within three attempts.[58]

The I-LMA is a truly amazing device, as it can be used to rescue the patient from a "can't intubate – can't oxygenate" situation, in addition to <u>reliably</u> placing a tracheal tube <u>completely blindly</u>.

The intubating LMA™ airway

Selected by NASA and included in the Difficult Airway Algorithms of the AHA and ASA, the LMA Fastrach™ is the ideal choice for the anatomically difficult airway. Designed to facilitate blind intubation without moving the head or neck, the LMA Fastrach™ allows continuous ventilation between intubation attempts.

- Ideal for the anticipated and unexpected difficult airway
- Proven use in difficult to intubate patients
- Ventilation is possible between intubation attempts
- Dedicated ET tube
- Designed for one handed insertion
- All sizes accommodate ET tube up to 8mm
- Latex free

ET tube: With a silicon cuff.* Designed to exit the LMA Fastrach™ at the correct angle to enter the trachea
*this product is available to purchase separately

Handle: Allows one handed insertion with the patient in any position

Airway tube: Anatomical curvature allows fast insertion into the correct position without moving the patient's head or neck

Epiglottic elevating bar: Designed to protect the airway tube from obstruction and raises the epiglottis out of the way when an ET tube is passed through

Fastrach Intubating LMA

COMBITUBE®

The Combitube is strictly a 'rescue' airway device.   It is a double-lumen device with two cuffs or balloons.   It is only available in two sizes (37FR and 41FR), for small and large adults.   It is placed blindly, with the intention of placing the distal tube into the oeophagus, although tracheal placement is equally acceptable (as it will function as an ETT).

Once placed, both the distal and pharyngeal cuffs are inflated through separate ports.   The operator must then test both lumens to check whether the distal tip is placed in the oesophagus or trachea, and through which lumen to ventilate.

The portion of the tube between the pharyngeal and the oesophageal cuffs is fenestrated. When placed in the oesophagus, the proximal and distal cuffs seal the pharynx and

oesophagus respectively, and air is directed through the fenestrations and through the vocal cords.

As with all SADs, the Combitube does not protect against pulmonary aspiration, and may not ventilate effectively in the presence of laryngospasm or upper airway obstruction.

The Combitube has fallen out of favour since the advent of the more "user-friendly" King Airway or Laryngeal Tube.   The Combitube can be fairly difficult to insert, as it is a fairly rigid device.   There have been reports in the literature of oesophageal perforation and lethal pneumomediastinum with the use of the Combitube.

Combitube

KING AIRWAY® OR LARYNGEAL TUBE®

The King Airway is synonymously known as the Laryngeal Tube, or as the King Laryngeal Tube.   It is a simplified modification of the Combitube, with only one lumen, and two cuffs, but with only one inflation port for both cuffs.   It is latex free, and is considerably more compliant than the Combitube, lending itself to atraumatic and easy insertion.

It is available as both disposable and re-usable devices.   The re-usable device has a gastric tube port (similar to the Proseal LMA).

It is placed blindly into the oesophagus, the cuffs are inflated, and the patient ventilated through a port placed midway between the two cuffs, in the same way as the Combitube. The distal lumen is too large to be placed in the trachea, so tracheal placement is highly unlikely.

There are numerous reports in the literature, and much favourable comment in the literature on the advantages of using this device as a first-line airway device in the prehospital setting.

From limited personal experience with this device, I must caution that patient positioning is important, with adequate neck extension being important for efficacious ventilation.   This may limit its value in the emergency and trauma settings.

King Airway or Laryngeal Tube

The Laryngeal Tube (King Airway; LT) provides a better seal than the LMA, with leak pressures of 26 cmH$_2$O +/- 7.3 cmH$_2$O compared to the LMA of 19.2 cmH$_2$O +/- 8.6 cmH$_2$O. In 20% of cases, the P-LMA leak pressure exceeds 40 cmH$_2$O, and has been used successfully in ventilating morbidly obese and pregnant patients. (The LT is marketed by SSEM in South Africa).
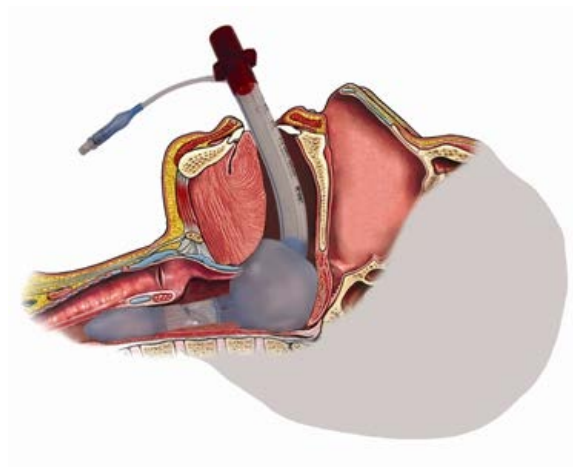
Placement of the King Airway aka the Laryngeal Tube Airway

THE I-GEL® SUPRAGLOTTIC AIRWAY DEVICE

Like the Cobra device, the i-Gel confers no special advantage over the standard LMA.   The i-Gel does not need to be inflated, and has a more rigid "stem" than the various LMAs, minimising torsion following insertion.   The largest size i-Gel should always be selected as, unlike the LMA, if an air leak occurs, the device cannot be further inflated.   A limitation in the prehospital environment is that the gel-rubber compound becomes very sticky at high ambient temperature – such as occurs in the boot (trunk) of a response vehicle.   The i-Gel is still

trying to gain market-share in the supraglottic airway market, and therefore – once again – experience with this device is somewhat limited.

The latest advent with the i-Gel is a port for a supplemental oxygen supply – enabling the entrainment of oxygen whilst placing the device in emergency airway situations.   The value of this additional port is currently untested.

The additional oxygen port on the latest version of the i-Gel

Rigid thick stem of the i-Gel limits torsion

It has been suggested that the i-Gel can be used as a conduit for blind intubation, in a manner similar to that of the Fastrach intubating LMA.   This can be demonstrated with remarkable reliability in airway manikins, but ***clinical results in live patients have proven dismal***, and we cannot recommend this practice.

Back to the humble LMA:

There is no doubt that the LMA (in all its guises) is an extremely efficacious supraglottic airway device, has been extensively studied, and for which most anaesthetists have a vast experience base.

The undisputed adage remains, *"If it ain't broke, don't fix it".*

These devices are extremely expensive, costing in the order of R120 000 and above. This device comprises essentially an endoscopic camera placed at the tip of a laryngoscope blade, with the image displayed on a plasma screen. It is very useful for intubating patients in whom direct laryngoscopy is not possible.

Glidescope also provide a standard Macintosh blade (for intubation training) in addition to their patented difficult airway blade.

Above left: Glidescope Ranger – portable machine ideal for prehospital fieldwork.

Above right: The shape of the Glidescope blade (straight tip) and the pre-formed accompanying tracheal tube introducer (shaped to the Glidescope blade), facilitates easy intubation in the "impossible" airway.

Above left: The Glidescope blade compared to a standard Macintosh blade. Note the increased angle of the Glidescope blade, and the straight (non-curved) tip.

Above right: The Glidescope blade with it's pre-formed commercially-supplied introducer.

C-MAC VIDEO-LARYNGOSCOPE

The C-Mac by Karl Storz is conceptually very similar to the Glidescope. The advantage of the C-Mac is that a flexible fibre-scope can be attached through an external port, allowing the C-Mac video screen to be used as the fibre-scope screen. This is a potentially significant cost-saving, and increases the portability of the fibre-scope. The range of Karl Storz flexible scopes are all compatible with the C-Mac. The Glidescope unfortunately has no such external port for "enslaving" an external camera and scope.

A regular Macintosh blade, as well as a D-blade for difficult intubation (pictured below) is available for the C-Mac.

The curvature of the D-blade lends itself to excellent visualisation of the glottic structures, but placement of the tracheal tube through the vocal cords can be exceedingly difficult, as the curvature is extreme. Guiding the tube upwards, to follow the curvature of the blade, can be extremely challenging in a truly difficult intubation. The C-Mac is unfortunately not supplied with a commercially available introducer pre-shaped to the blade contour.

The curvature of the D-blade provides excellent glottis views in truly difficult intubations, but it can be exceedingly difficult to coax the tracheal tube through the glottic opening owing to the extreme curvature of the blade.

FLEXIBLE FIBRE-OPTIC (ENDOSCOPIC) AWAKE AND ASLEEP ORAL OR NASAL INTUBATION

Flexible fibre-optic intubation is the subject of an entire lecture in its own right, and will be expanded upon in due course.   In short, flexible fibre-optic intubation is not a viable first-line option in the emergency situation, as it can be a time-consuming and difficult process in the face of blood and secretions in the upper airway.   It is especially inappropriate in situations where one is faced with a difficult airway in an hypoxic patient that is difficult to ventilate.

Fibre-optic intubation is largely an elective procedure, but may be useful when guided through an LMA in the emergency setting, with the additional use of an Aintree tube exchange catheter (see later).   This technique requires considerable training, and comparable results are obtained with the blind placement of an ETT through an I-LMA.
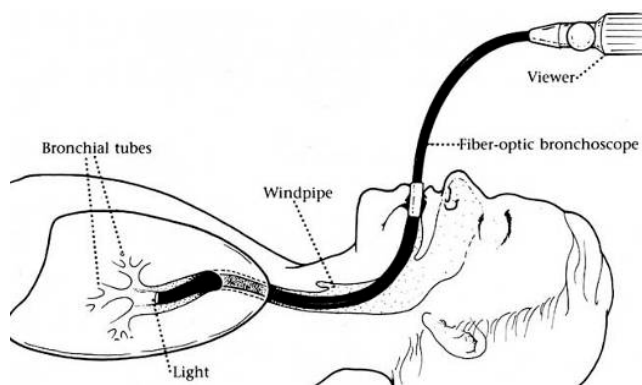
DOUBLE-LUMEN ENDOTRACHEAL TUBES

Double-lumen endotracheal tubes have very specific indications, such as in thoracic surgery where single-lung ventilation is undertaken, and is primarily the domain of specialist anaesthesiologists.

Correct placement must be confirmed before placement of patients in the lateral position, as misplaced tubes under these circumstances can be catastrophic.   Confirmation of correct endobronchial tube placement will be expanded upon at a later date.
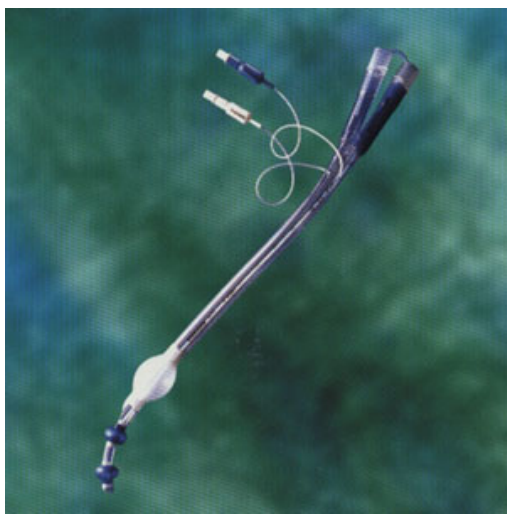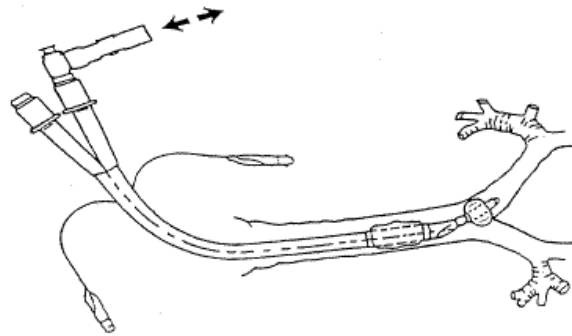
BRONCHIAL BLOCKERS

Bronchial blockers can be used through standard tracheal tubes, in order to isolate one lung by blocking off one mainstem bronchus.   The clinical setting in which bronchial blockers may be used includes haemorrhage into the lung from a stabbed chest – where the patient bleeds copiously into the tracheal tube, with blood spillage into the opposite lung.

A word of warning is that, whilst bronchial blockers may isolate one lung, they cause a massive shunt (V/Q mismatch), as ventilation to the blocked lung is stopped, but that non-ventilated lung is still fully perfused, thereby returning de-oxygenated blood to the central circulation.   Low oxygen saturation would therefore be the order of the day under such circumstances.

Blood from one lung (stabbed chest) over-flowing into the tracheal tube, contaminating the other lung and filling the HME device with coagulated blood – resulting in near-total airway obstruction.

Left: The Rusch EZ Blocker placed into a standard tracheal tube. Right: the "business end" of the EZ blocker showing two inflatable cuffs. The EZ Blocker is advanced to the carina and one cuff inflated at a time. This should ideally done under fibrescope guidance.

NEBULIZER ATTACHMENTS

There are three types of in-circuit nebulizer attachments.   Please note that the unit featured below requires the addition of a respirator solution, and requires a separate (additional) oxygen supply as driving gas.

The attachment featured below is designed to accept a metred-dose inhaler, and does not require a separate oxygen supply.   The MDI is activated (squeezed) during inspiration.

Note that HMEs (heat and moisture exchange devices) must be removed from the circuit when these devices are used, or else the 'atomised' nebules will simply be trapped in the HME.

The Mucosal Atomisation Device (MAD) featured below has a standard Luer connection at the proximal end.   This allows the attachment of a standard syringe through which a salbutamol (or other) respirator solution can be directly injected into the tracheal tube.   The distal end has an atomising nozzle, creating a fine nebulised spray.

THE PAEDIATRIC PATIENT

The infant airway is different to the adult airway for the following reasons:

- The infant has a large tongue and epiglottis, which make airway obstruction more likely, more difficult to manage, and makes laryngoscopy more difficult.

- The larynx is situated more cephalad (C3 in the preterm infant, between C3 and C4 in the term infant, and between C4 and C5 in the adult.   This changes the angle of insertion of the laryngoscope, rendering the straight blade more useful than the curved blade.
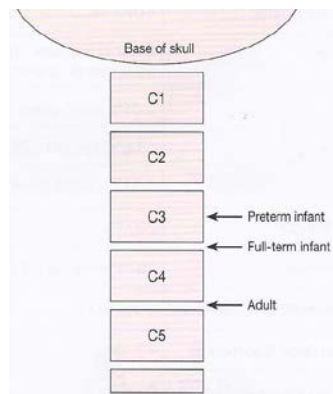
Position of the larynx [62]

- In general, children obstruct far more easily than adults do, owing to smaller airway diameters and also due to more pliable and collapsible airways.
- The importance of assisting ventilation by applying gentle PEEP cannot be over-emphasised.   This cannot be done with a standard BVMR and will require the employment of a paediatric Ayres T-piece (Mapleson F) anaesthetic breathing circuit.
- It must also be noted that children may completely obstruct, yet it may appear to the casual observer as though the patient is breathing normally.   One cannot wait for oxygen desaturation to signal airway obstruction in this group, and so a state of hyper-vigilance is always necessary.   Continuous waveform capnography is <u>always</u> a good idea.
- The epiglottis is short and stubby, and is angled over the laryngeal inlet, making it more difficult to control with the laryngoscope blade.
- The vocal cords are angled – increasing the chances of a blindly-passed ETT lodging in the anterior commissure.
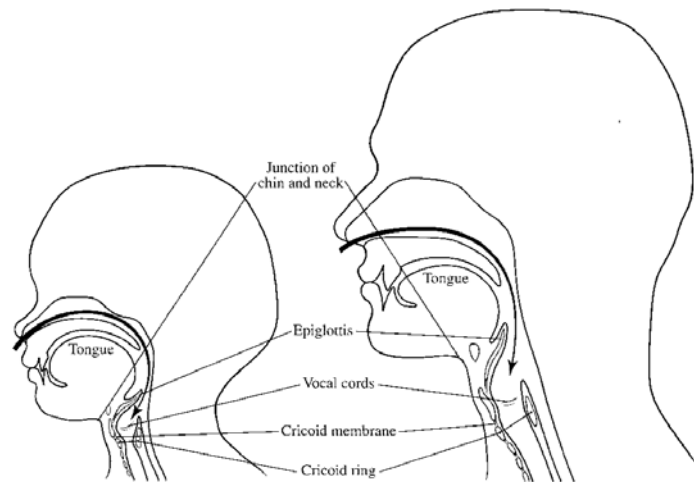
Anatomic differences between the child and adult.

Traditionally, it was thought that the infant larynx is funnel-shaped with the narrowest portion is at the cricoid cartilage.   Newer evidence has shown that this is not correct, but an important principle is that the least stretchable portion of the airway is at the level of the complete cricoid ring. Thus, tracheal tubes that may pass through the vocal cords easily may sit very tightly in the subglottic area. It is this appropriate to use high volume, low pressure cuffed endotracheal tubes of appropriate size in small children, provided that a leak is present with the cuff deflated, and a cuff pressure manometer is used to confirm cuff pressure within the safe range (<30 cmH$_2$O). See below for more discussion.

The paediatric trachea is remarkably short in comparison to the adult. This fact, in combination with the associated relief in successfully placing the tube through the cords during difficult intubations, results in the subconscious tendency to advance the tracheal tube too far!

This results in right mainstem bronchus intubation and carinal stimulation presenting as bradycardia and bronchospasm through intense vagal stimulation.

Most traditional paediatric tracheal tubes are uncuffed, to prevent the development of circumferential mucosal necrosis from an over-inflated cuff.   Consequently, paediatric tubes are associated with high leak rates, and therefore may require higher tidal volumes and gas flow rates to compensate for this.

Aspiration is usually not a problem, as the glottic air leak generated by positive airway pressure blows secretions out of and away from the glottic opening.

The paediatric trachea is much more susceptible to dynamic collapse than the adult trachea. Such airway collapse can be countered with the use of Positive End Expiratory Pressure (PEEP).
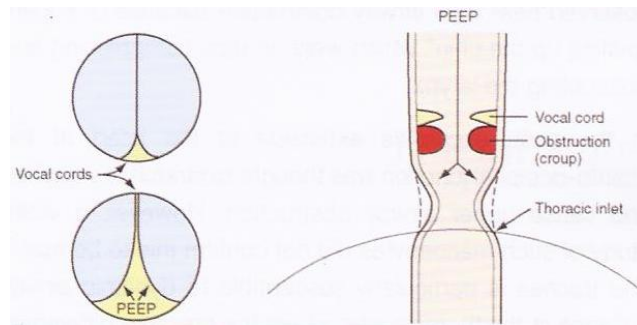
Effect of PEEP on paediatric airways [52]

Airway oedema has a very significant effect on paediatric airflow resistance. The Hagen-Poiseuille equation (page 152) dictates that resistance to flow is inversely proportional to radius to the 4th power. This means that halving airway diameter increases airway resistance by sixteen times. The mucosal lining of the paediatric airway makes up a greater proportion of airway diameter up to the age of five years. Mucosal oedema and airway trauma (laryngoscopy) can therefore have a profound effect on work of breathing up to this age.

Type 1 muscle fibres are very much reduced in the infant diaphragm and intercostals muscles, under the age of two years, and is even less in the neonate. Type 1 fibres allow for repetitive motion without fatigue. This accounts for apnoea seen in infants with an increased respiratory workload.[63]

Paediatric airway resistance under different conditions [52]

- A large occiput makes for difficult positioning of the head during airway manipulation. Placing a towel under the torso and the use of a head-ring greatly assists in stabilising the head.
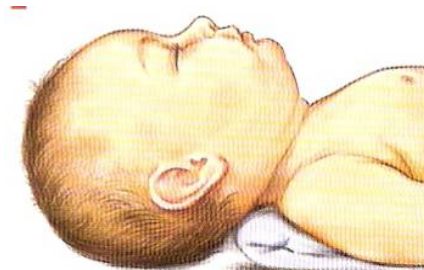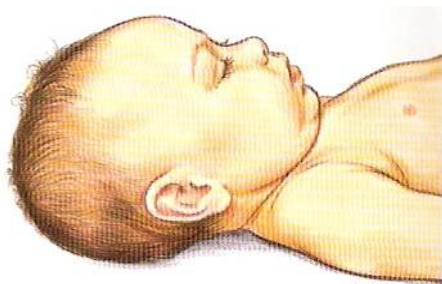
Owing to the large occiput, intubation is often made easier by placing a towel or blanket under the torso of the patient, and not under the head, as in adults.

(These illustrations are from the APLS manual).

- Placing a nasogastric tube pre-intubation assists with deflating a hyper-inflated stomach – which is almost an inevitable consequence of face-mask ventilation, especially during bag-valve-mask resuscitation.   Deflating the stomach reduces the risks of aspiration, and reduces diaphragm splinting – rendering ventilation easier.

- Furthermore, the nasogastric tube in situ delineates the oesophageal 'track' – directing the airway technician *away* from "where you don't want to go"!

- Fasting reduces gastric volume but does not guarantee an empty stomach.   Breast milk is cleared more rapidly than formula milk.   In infants, prolonged fasting does not further decrease aspiration risk, but increases the risk of dehydration and hypoglycaemia – hence the recommendation of clear apple juice 2 hours pre-operatively.

- Infants are at greater risk of aspiration, as they have reduced gastro-oesophageal sphincter tone.   They also have an increased tendency to distend the stomach during mask ventilation, and therefore splinting of the diaphragm.   However, the incidence of pneumonitis following aspiration is lower in infants than in adults.

Red Cross Childrens' Hospital routinely uses a gentle and careful inhalational induction technique for non-fasted children without IV access, and consequently has a very low risk of pulmonary aspiration.

The ASA pre-anaesthetic fasting guidelines in healthy patients are as follows:

Clear (non-particulate) fluids:	2hrs
Breast milk:	4hrs
Light meals:	6hrs

Drug dosages are often higher in children (for example, suxemethonium is administered at 2mg/kg in the paediatric age group.   From toddlers upwards, propofol dosage requirements may be two to four times higher than that required for adults.

Resuscitation aids based on age, weight and height are invaluable in paediatric resuscitation, and pre-constructed tables are of immense value to those practitoners who do not regularly work with children.

The formulae for estimating tracheal tube size and depth are discussed elsewhere in this manual.

IV access in the neonate and in the 'chubby toddler' can be extremely challenging and may require specialist intervention.

Always keep in mind that it may be better to refer to an anaesthesiologist (where possible) for an inhalation (gas) induction in the operating theatre, when faced with a threatened airway in a paediatric patient (epiglottitis or croup).

CUFFED VERSUS UNCUFFED TRACHEAL TUBES IN PAEDIATRIC PATIENTS

Uncuffed tracheal tubes have been seen as the gold standard in paediatric anaesthesia for more than 50 years, as cuffed tubes (high-pressure low-volume cuffs used many years ago) were thought to damage airway mucosa, resulting in much-feared subglottic stenosis.

There is a growing body of evidence that newer low-pressure high-volume cuffed tubes are not associated with increased airway morbidity, provided that tube sizes are carefully selected and cuff pressures carefully monitored [63, 64].[64,65]

In fact, smaller-diameter cuffed tubes actually reduce the pressure exerted on the non-distensible cricoid region, in comparison to larger-diameter uncuffed tubes.

It has now been recognised that circular uncuffed tubes placed to fit 'snugly' into the non-circular (elliptoid) cricoid area may cause pressure damage and ischaemia on the airway mucosa at this point, as the region of the cricoid ring remains the smallest functional part of the infant airway.

The Broselow tape predicts the appropriate tracheal tube size to within 0.5mm in 98.5% of patients.[66]   However, some tube replacements will still be needed, especially if uncuffed tubes are used.

Cuffed and uncuffed tracheal tubes – advantages and disadvantages:[67]

Advantages of cuffed tubes	Disadvantages of uncuffed tubes
- Reliably sealed airway. - Better control of ventilation. - Better capnograph tracing. - Reduced pollution – volatile agents, infectious droplets. - Lower fresh gas flows. - Protection against aspiration. 'Close' subglottic fit unnecessary. - Centralises tube in trachea – takes pressure off of mucosa. - Difficult airways – reduced need for tube exchanges. - Limited tube movement. - Cuff lifts tube from tracheal wall.	- Clinically significant loss of tidal volume. - Unreliable oxygenation and ventilation. - Higher gas flow (consumption). - Greater incidence of reintubation to change tube size. - Increased risk of aspiration. - Increased pressure on laryngeal structures with over-sized uncuffed tubes. - Airway injury from repeated tube exchanges. - Higher risk of dispersion of infectious droplets in communicable diseases.

When using the modified Cole's formula (tube internal diameter = age/4 + 4), reduce tube size by 0.5 to 1.0 mm when using cuffed tubes, in order to compensate for the volume of the cuff. Beware of using a tube size that is too small, which may result in increased resistance to gas flow.

We tend to calculate tube size based on internal diameter, but the external diameter of different manufacturers may vary considerably. Over-sized outer diameters, poorly positioned tubes, lack of depth markings and cuff over-inflation have been identified with airway injury.

Cuff deflation results in sharp folds and edges in the cuff membrane, which can lead to mucosal damage through 'cutting' the mucosa through cuff movement. Cuff deflation should only be carried out immediately prior to extubation, and should not be used as a means to create an air leak in an over-sized cuff.[67]

Folds of the membrane of a deflated tracheal tube cuff result in 'sharp' edges that can cut tracheal mucosa.

PAEDIATRIC PERCUTANEOUS CRICOTHYROIDOTOMY

1. The cricothyroid membrane is located with the index finger-tip, between the thyroid and cricoids cartilages.   This space is very narrow (1mm) so that only a fingernail can discern it.

2. A large IV cannula (14G) is then inserted through the membrane and air aspirated.

3. The cannula is advanced and the needle removed.

4. A 2ml syringe is attached and air aspirated again, confirming intraluminal placement.

5. A 3mm tracheal tube adaptor can be attached to the hub of any IV cannula.

6. Alternatively, the 2ml syringe can be left in place (connected to the hub of the cannula) and the adaptor of a 7.5mm tracheal tube inserted into the barrel of the syringe.

7. A standard 22mm airway connector is then attached, and ventilation achieved through this

Technique for paediatric percutaneous cricothyroidtomy:[63]

Components of a paediatric cricothyroidotomy set: (1) 14g IV cannula; (2) 2ml syringe;

(3) 3mm ET tube connector; (4) 7.5mm ET tube connector.

Syringe connects to IV cannula.

B
Locate cricothyroid
space with fingernail

A
Stabilise
trachea

Cricoid   Thyroid cartilage

D
Aspirate air

Large IV
catheter

C
Puncture
cricothyroid
membrane

E
Advance
catheter

F
Withdraw and
discard needle

H
Aspirate air and
discard plunger

J
Attach to
ventilating bag

G
Attached
syringe

I
Attach 8-mm
adapter to barrel
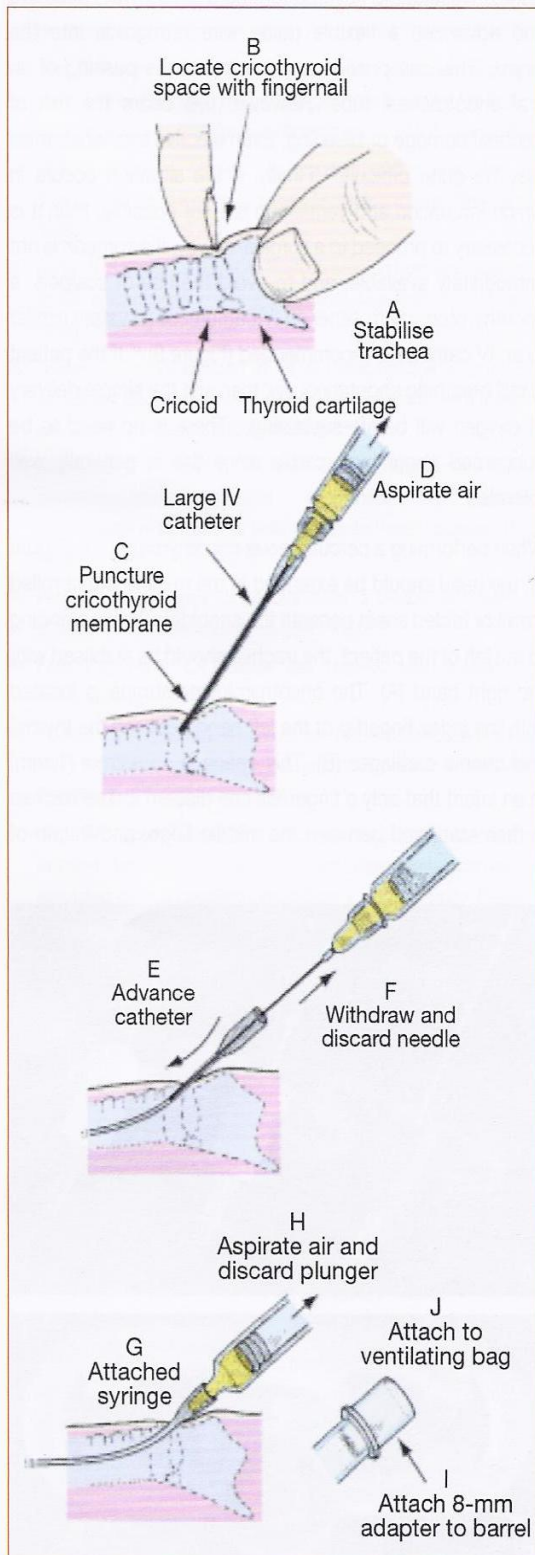
IV: intravenous

7.5mm ET tube connector fits into barrel of syringe. Breathing circuit or bag-valve-mask resuscitator can be connected to facilitate ventilation.

Alternatively, a 3mm ET tube connector can be connected directly to the cannula, and the ventilation system attached to the connector.

Case report – paediatric retrograde intubation:[68]

This seven month old child with congenital trismus was anaesthetised with sevoflurane and oxygen, and kept breathing spontaneously while cricothyroid puncture was carried out under local anaesthesia.

Figure 1: An infant with congenital trismus and no mouth opening

The patient was positioned with a shoulder roll and head ring.

The epidural catheter was rail-roaded using a 5F feeding tube, and brought out of the right nostril from the oral cavity.

Figure 2: Retrograde intubation using a Tuohy needle

The tip of the epidural catheter was tied to the Murphy's eye on a 4mm ET tube.

After being well-lubricated, the tube was then guided into the oral cavity and then pulled through the larynx and into the trachea.

Resistance was overcome with the application of gentle cricoid pressure, lubrication and manipulation.

Figure 3: Tuohy needle inserted into the cricothyroid membrane

Note:

An alternative method would have been, instead of tying the tube through the

Murphy's eye, to have rail-roaded the tube over the epidural catheter, pulled tight in a "cheese-wire" type of technique.

Figure 4: The epidural catheter was pulled out through the oral cavity

Figure 5: The epidural catheter was pulled out through the nasal cavity

THE PREGNANT PATIENT

Anatomic challenges related to pregnancy, airway swelling, decreased gastric motility, and the need to rapidly secure an emergency airway in the face of rapid oxygen desaturation, can prove an enormously daunting task to the airway technician.

Backup rescue airway devices and alternative laryngoscopes such as the Airtraq and KingVision optical laryngoscopes, video-laryngoscopes, polio Macintosh blades and Mc Coy laryngoscopes are very strongly recommended.

The ProSeal LMA is the supraglottic rescue airway device of choice, as it provides a more reliable seal, which reduces the risk of aspiration and facilitates ventilation at higher pressures[68, 69].[69,70]

The pregnant patient has a markedly reduced FRC (up to 20%), and a Closing Capacity exceeding FRC in up to *one half* of patients.   This results in increased atelectasis and shunt, with consequent rapid desaturation.

Thoracic breathing predominates over abdominal (diaphragmatic breathing), which is markedly impaired in the supine position.

A relative anaemia (reduced oxygen carrying capacity) is countered by an increased cardiac output and a rightward shifted oxygen haemoglobin dissociation curve.

An increased metabolic rate, increased myocardial oxygen demand, increased work of breathing (minute ventilation is increased by 50% at term) and foetal oxygen requirements mean that *oxygen consumption* is greatly enhanced!

The additive effect of the above factors leads to early, rapid and profound oxygen desaturation on administration of induction agents and muscle relaxants, **despite** '*adequate*' pre-oxygenation.

Face-mask ventilation is more difficult in the obstetric patient, as a result of the higher intra-abdominal pressures experienced during pregnancy. Insufflation of air into the stomach, with splinting of the diaphragm, increasing difficulty with ventilation and an increased risk of aspiration are the order of the day!

In addition, the pregnant patient is **always** considered to have a **full stomach,** due to decreased gastric emptying. This leads to a greater risk of pulmonary aspiration. Please see the section above on *Pre-intubation Fasting*. Fasting during labour may reduce gastric volumes, but in no way guarantees an empty stomach.

Tracheal intubation in parturients is notoriously difficult, reflected by a **ten times** greater failure rate in pregnancy as compared to the general population.

The incidence of difficult intubation in the pregnant population is in the order of 5 to 15%,[71,72] and the incidence of failed intubation is in the order of 1:250, which is roughly ten times the failure rate in the non-pregnant population.[73]

The prevalence of Mallampati class IV scores increase with gestational age, and correlate directly with weight gain (34% at 38 weeks).[74] Additionally, oral and pharyngeal volumes decrease between the onset and end of labour.[75]

The incidence of difficult intubation in the morbidly obese obstetric population rises to 50%. In addition, the morbidly obese parturient is at increased risk of emergency caesarean section and failed neuraxial (spinal and epidural) anaesthesia.[76]

Large breasts may necessitate an assistant to retract the breast 'downwards' in a caudad direction. Large breasts may also make insertion of the laryngoscope into the oral cavity nearly impossible, and may require the use of a short-handled laryngoscope and/or a polio Macintosh blade and/or "sideways" insertion of the laryngoscope handle. Please see the chapter on difficult intubation.

Engorgement of the nasal mucosa precludes the use of blind nasal intubation, as the increased risk of epistaxis would be disastrous in a difficult or failed intubation scenario. Similarly, avoid the use of nasopharangeal airways if at all possible.

In terms of pre-oxygenation, three minutes of normal tidal volume breathing is superior to four maximal deep (vital capacity) breaths over 30 seconds, but equivalent to eight vital capacity breaths over 60 seconds.[77,78] Apnoeic oxygenation using high-flow nasal cannulas (THRIVE) has been reported with good success in this population.[79]

It must be stated here that, whether buying a house, kicking a ball, making love or intubating a pregnant or obese patient, POSITION is everything!!   The "ramp" position described previously in this manual is of critical importance in both face-mask ventilation and intubation of pregnant and obese patient.

The airway technician must simultaneously manage the effects of **aortocaval compression** in the supine pregnant patient.   Aortocaval compression results in profound hypotension from decreased venous return as the gravid uterus compresses the inferior vena cava.

This is commonly severe enough to result in maternal unconsciousness (and an unprotected airway) and foetal distress as the placental blood flow is significantly reduced.

Aortocaval compression is managed by the simultaneous insertion of a wedge under the right buttock (15° of left lateral tilt), lifting the uterus off the inferior vena cava) and the administration of "co-loaded" IV fluid boluses and vasopressor agents.

Note that aspiration of stomach contents **on emergence** from general anaesthesia or during recovery carries a relatively high risk, as revealed in the UK Confidential Enquiries into Maternal and Child Health for 2006 to 2008 [78].

Your problems are not over once the patient has been successfully intubated!.

78. Cooper GM, McClure JH. Anaesthesia chapter from Saving mothers' lives; reviewing maternal deaths to make pregnancy safer. Br J Anaesth 2008; 100:17.

Care must be taken when extubating the patient with a difficult airway. Airway compromise during emergence has become the main cause of airway tragedies in the obstetric population. This is especially important if the parturient is obese, has a history of obstructive sleep apnea, has received large amounts of intravenous fluids or blood products, has undergone a lengthy surgical procedure, or has an underlying disease process that increases the risk of airway edema, such as pre-eclampsia[79].

79. Mhyre JM, Riesner MN, Polley LS, Naughton NN. A series of anesthesia-related maternal deaths in Michigan, 1985-2003. Anesthesiology 2007; 106:1096.

It goes without saying that tracheal intubation of the pregnant patient in the field, by a non-anaesthetist, and without obstetric anaesthesia experience, is a decision not to be taken lightly.

THE OBESE PATIENT

All factors described above in the pregnant patient, excepting for aortocaval compression and foetal oxygen requirements, apply equally to the obese patient. Similarly, obese patients are generally very difficult to intubate, have a higher incidence of failed airway management, are difficult to ventilate by face-mask, and desaturate rapidly.

These patients are sometimes untruthful about their fasting status, as many struggle to forego food for six hours, and therefore are at higher risk of aspiration, even under elective circumstances.

Intubating the obese patient can, accordingly, be a frightening experience, and the risk: benefit ratio needs to be carefully assessed. In addition, IV access can be extremely difficult to attain in the obese patient.

Correct positioning <u>before</u> intubation cannot be over-emphasized, as it is almost impossible to reposition these patients after induction.
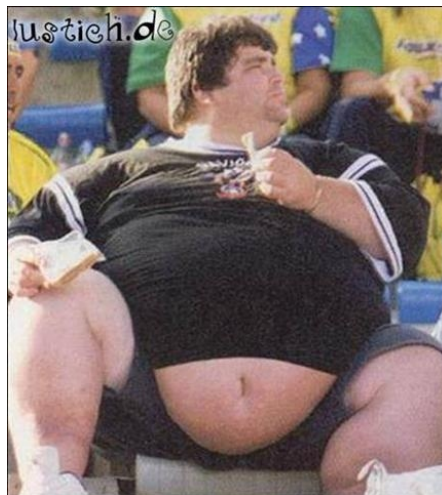
THE TRAUMA PATIENT

The primary and secondary surveys in the trauma patient are done as per normal.   As well as managing the airway as a priority, associated injuries and haemorrhage may have to be managed simultaneously.

Intubation in Traumatic Brain Injury (TBI) requires a separate discussion – please see the discussion of cerebral perfusion pressure under the section "Pressor Drugs" – ephedrine and phenylephrine.

The trauma patient (and especially the head-injured patient is always considered to have a cervical spine and/or other spinal injuries, requiring full spinal precautions, including in-line neck stabilisation during intubation.

Induction agents and muscle relaxant drugs must be carefully chosen in the haemodynamically unstable or shocked patient.   Please see the section on pharmacology, where this is discussed in detail.

Please note that the "six-hour rule" to patient fasting does not apply in the trauma patient, owing to delayed gastric emptying.   All trauma patients are considered at risk of aspiration, unless the traumatic event itself occurred six or more hours after the previous meal. All trauma patients are therefore intubated with the RSI technique, even though cricoid pressure can cause up to 5mm cervical vertebral displacement.

The incidence of associated C-spine injuries in major trauma has been reported to be in the order of 2%, and hence the routine use of in-line neck stabilisation.   However, despite the application of in-line neck stabilisation, face-mask ventilation and laryngoscopy are still associated with some C-spine movement (C_1 on C_2).   Under such circumstances, flexible fibre-optic intubation may be better, but the hardware and skills are seldom available, and the practice is also associated with a higher aspiration risk.   The patient may be better off with "doing what you do best" and resorting to the familiarity of direct laryngoscopy.

The Fastrach Intubating LMA, Airtraq optical laryngoscope or video laryngoscopy may provide safe alternatives to conventional laryngoscopy in the patient with the unstable C-spine.

In addition to C-spine immobilisation, the airway technician may be further challenged by bleeding from major vessels, and airway obstruction from haematoma and/or oedema formation.   Formal tracheostomy can be considered in the awake patient, if awake fibre-optic intubation is not possible.   In the true emergency setting, a needle or surgical cricothyroidotomy may be required.

The particularly challenging trauma airway is the 'gunshot' or stabbed neck, where there is direct local tissue damage, swelling and bleeding into the airway itself.   Here, the only option (besides surgical cricothyroidotomy) may be to "go for the bubbles"…

An assistant performs slow cardiac compressions of a lesser intensity and depth than undertaken during standard CPR.   This causes the expulsion of air from the trachea, and the appearance of bubbles at the glottic opening.   Either a tracheal tube or gum elastic bougie (or gum elastic bougie loaded with a tracheal tube) can be directed through the bubbles and into the trachea (with a little bit of luck!), and the tracheal tube 'rail-roaded' over the gum elastic bougie.

TRAUMATIC BRAIN INJURY:

The Monro-Kelly doctrine states that the cranium is a rigid box, containing, blood, cerebrospinal fluid and brain tissue.   An increase in the volume of any one of these three constituents requires a compensatory decrease in one or both of the other two constituents.

Once the compensatory mechanism is overwhelmed, there is an exponential rise in intracerebral pressure.   As intracerebral pressure rises progressively, cerebral perfusion decreases progressively.   Once intracerebral pressure reaches (or rises above) mean arterial pressure, then cerebral perfusion ceases.

Cerebral perfusion pressure is defined by the equation:

$$\textbf{CPP = MAP – ICP (or CVP)*}$$

CPP = Cerebral perfusion pressure

MAP = Mean arterial pressure

[* whichever of the two is greatest]

Mean Arterial Pressure is slightly less than halfway between systolic and diastolic BP.   This is because systole is shorter than diastole.   MAP can be approximated as DBP + 1/3 of pulse pressure, and can be accurately measured by integrating the area under the arterial pressure waveform
.

From the above equation it can be seen that a rise in intracerebral pressure (cerebral oedema, cerebral vasodilation or intracerebral haematoma) or a drop in blood pressure, will decrease cerebral blood flow.

An umbra of dense ischaemia occurs in stroke and traumatic brain injury, surrounded by a penumbra zone of partial and variable ischaemia.

Penumbra: moderate ischemia
delayed infarction

Core: dense icschemia
early infarction
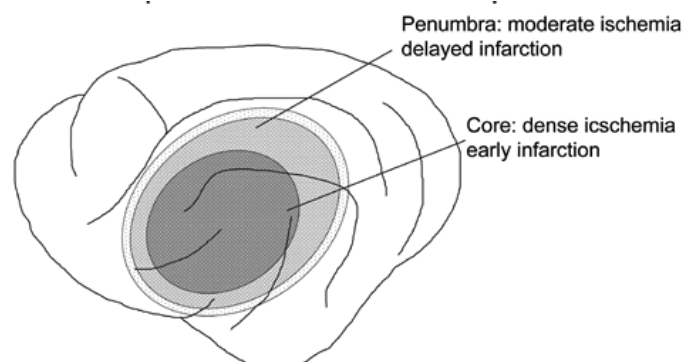
Umbra and penumbra: zones of cerebral ischaemia

Unwitting mismanagement of blood pressure and ventilation can result in an increased penumbra of partial ischaemia, and can result in encroachment of the umbra into the penumbra zone.

Considering that the brain consumes 10 times more oxygen than the rest of the body's tissues, even small decreases in perfusion are critical events and can have catastrophic outcomes.

Hence, maintaining end-tidal carbon dioxide within normal parameters is critically important. Hypoventilation and hypercarbia will increase intracerebral pressure through vasodilation, and conversely, hyperventilation and hypocarbia will increase ischaemia through vasoconstriction.

Furthermore, a significant decrease in blood pressure post-intubation, such as that resulting from the concomitant administration of benzodiazepines and opiates, or the administration of induction agents such as sodium thiopentone and propofol, will seriously compromise cerebral perfusion.

It is for this reason that drugs used in drug-assisted intubation must be selected judiciously, ventilation must be titrated against end-tidal capnographic endpoints, and the use of pressor drugs must be available to the airway technician.

This is especially important in the polytrauma patient with TBI, who is flown by helicopter in the feet-down (reverse-Trendelenberg) position.   It is equally important on initial ascent, when such a patient is flown by fixed-wing aircraft, when loaded head-first.

Beware reverse Trendelenberg position on helicopters

and fixed-wing aircraft when flying head-injured patients

Please refer to the lecture "Traumatic Brain Injury" for a detailed discussion of this topic. Please also see the pharmacology section "Pressor Agents".

Note also that Rapid Sequence Induction in severely hypertensive patients can result in catastrophic intracerebral haemorrhage.   Hypertension should thus be controlled pre-intubation if at all possible.

As well as the administration of large doses of opioids such as fentanyl or alfentanil, labetolol by IV bolus (5mg increments) every 3 to 4 minutes can be considered in non-asthmatic patients.

Magnesium sulphate is a calcium channel blocker, and can be used in dosages of 2 to 4g by rapid IV bolus to safely attenuate the intubation response.   It has been used in the obstetric setting of safely intubating pre-eclamptic and eclamptic patients with severe hypertension for many years.

Magnesium sulphate is a non-scheduled, cost-effective and freely available drug.   Additional advantages are that it is an anticonvulsant drug (always beneficial in TBI, and that it prolongs the action of non-deporarising muscle relaxants (a very convenient side-effect when transporting ventilated patients under neuromuscular blockade). Please see the Pharmacology section for more details.

Warning:

Lastly, beware the insertion of nasogastric tubes and nasotracheal tubes in patients with basal skull fractures:

Left: Intracerebral misplaced nasogastric tubes.

MIDAZOLAM

Midazolam is a benzodiazepine, which has the following five principle actions: sedation, anxiolysis, anticonvulsant properties, spinal cord-mediated muscle relaxation and retrograde amnesia.

All benzodiazepine actions are caused by facilitation of GABA (gamma-aminobutyric acid),which is the major inhibitory neurotransmitter of the CNS. Benzodiazepine receptors are found in the CNS in very close proximity to the GABA receptor, or may exist as part of the GABA receptor. GABA receptors are found exclusively on postsynaptic membranes within the CNS, with maximal density in the cerebral cortex.

The GABA Receptor is a large macromolecule with separate binding sites for GABA, barbiturates, alcohol, and benzodiazapines – explaining the synergistic effects amongst these agents.
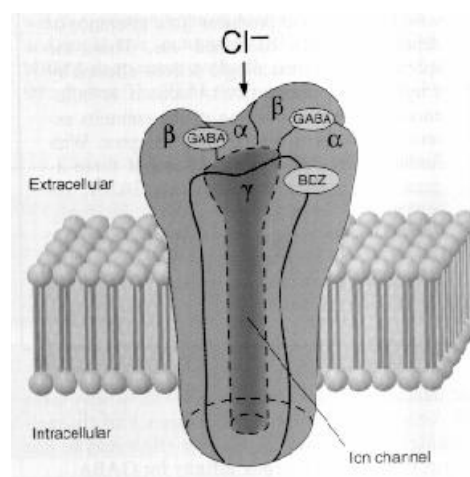
Binding of benzodiazepines to their receptors increases the affinity of GABA for its receptor. This results in enhanced opening of chloride (Cl⁻) channels; with a consequent influx of chloride. The consequent hyperpolarisation of the postsynaptic cell membrane causes resistance to excitation.

152
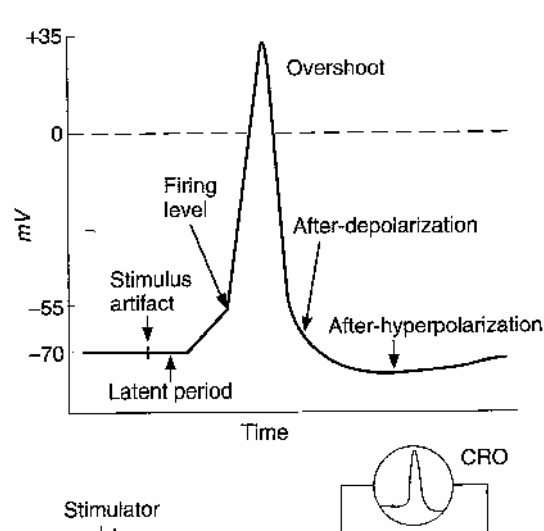
Midazolam is the only water-soluble benzodiazepine which is stable in aqueous solution at low pH.   At physiological pH it becomes lipid-soluble (due to ring closure), facilitating rapid crossing of the blood-brain barrier.   Because of this, there is no need for stabilising or solubilising preparations such as propylene.   There is therefore no no pain on injection (cf. diazepam).   Midazolam has two to three times the affinity of diazapam for the GABA receptor, and is therefore twice as potent.

Despite rapidly crossing the blood-brain barrier, midazolam, has a slow effect-site equilibration time varying between 0.9 to 5.6 minutes, with an average of about 3 minutes. The long and highly variable inter-individual effect-site equilibration time renders midazolam a less than ideal anaesthetic induction agent.

Midazolam is extensively bound to plasma proteins (96 to 98%), and rapidly redistributes from brain to other (inactive) storage sites.   Hepatic clearance is rapid (more than 10 times that of diazepam).   Water-soluble metabolites are excreted as glucuronide conjugates in the urine.

Midazolam decreases cerebral metabolic oxygen requirements, and decreases cerebral blood flow.   The cerebral vasomotor response to elevated CO_2 is preserved.   There is some evidence that patients with severe head trauma experience raised intracranial pressure when Midazolam is injected rapidly, but the clinical significance of this is not known.   Midazolam does NOT attenuate raised ICP associated with endotracheal intubation!!

Benzodiazapines do NOT possess neuroprotective activity, as do the barbiturates.

Midazolam causes dose-dependent decreases in ventilation.   Transient apnoea can occur with rapid injection of a large dose.   Synergistic effects with opioids and alcohol occur.

Blood pressure decreases and heart rate increases as compensation for a decreased systemic vascular resistance, although cardiac output remains unchanged.   There is usually no significant drop in blood pressure when midazolam is injected slowly in normovolaemic patients, but a significant drop in blood pressure occurs in hypovolaemic patients.

Midazolam is packaded in 3ml (15mg) and 5ml (5mg) ampoules.   The adult and paediatric dose is 0.1 to 0.2mg/kg as an IV bolus.

SODIUM THIOPENTONE

STP is a barbiturate which is readily soluble in water or saline.   It is highly alkaline, the pH of a 2.5% (25mg/ml) solution being 10.5.   The powder form is stable at room temperature indefinitely, and the prepared solution is stable for at least 6 days at room temperature and for two weeks in the refrigerator.   Unlike propofol, the alkalinity of the solution renders it bacteriostatic.

Thiopentone is packaged as 500mg pale yellow powder in a 20ml multi-dose vial, and is reconstituted with 20ml sterile water to a concentration of 25mg/ml.   The standard dose is 3 to 5mg/kg, usually erring on the side of the smaller dose.

The high pH renders this drug incompatible with highly acidic drugs such as opiates, non-depolarising muscle relaxants and adrenalin.   Vecuronium precipitates into a solid 'cement' when mixed with thiopentone, which blocks IV lines solid!   Thiopentone must never be mixed to a concentration greater than 2.5%.

The term "thiobarbiturate" denotes the inclusion of a sulphur atom on the number two carbon atom, which increases lipid solubility considerably.   This increased lipid solubility confers a greater potency, more rapid onset of action and a shorter duration of action.

Barbiturates produce their sedative-hypnotic (and anticonvulsive effects) through their action on the GABA receptor.   Activation of the GABA receptor results in an influx of chloride with consequent hyperpolarisation of the cell membrane.   The interaction of thiopentone (etomidate, propofol and midazolam) at the GABA receptor complex decreases the rate of dissociation of GABA from its receptor.

Barbiturates depress the reticular activating system and selectively depress impulse transmission in the sympathetic ganglia, leading to a drop in blood pressure.   They also decrease the sensitivity of the neuromuscular junction to the depolarising action of acetyl choline.

Rapid awakening is due to redistribution from the brain to inactive sites, rather than due to rapid metabolism.   Thiopentone undergoes hepatic metabolism and renal excretion.   It is highly lipid-soluble, and highly protein-bound, displacing other highly-protein bound drugs from their binding sites.

Dose requirements decrease with age.   Thiopentone decreases intracranial pressure by causing cerebral vasoconstriction, and by decreasing cerebral blood flow – due to a drop in blood pressure.

Thiopentone confers cerebral protection as it decreases $CMRO_2$ (by 55% with iso-electric EEG), but confer no protection during global ischaemia. It is a potent anticonvulsant.

Thiopentone depresses the medullary vasomotor centre, and causes transient vasodilation with a drop in blood pressure.   There is minimal to no myocardial depression, the drop in blood pressure overcome by an increased carotid sinus mediated increase in peripheral sympathetic nervous system activity.   However, a negative inoptropic effect is readily demonstrated in the isolated heart.   Histamine release can occur, but is seldom of clinical significance.   Thiopentone should be avoided in shocked patients, those on

antihypertensives and beta blockers, any patients maximally adrenergically driven or otherwise unable to compensate for drops in blood pressure (eg. hypovolaemia).

There is a dose-dependent depression of medullary and pontine ventilatory centres, and a decreased sensitivity to CO_2 drive.   These respiratory depressant effects are enhanced by other respiratory depressant drugs.

Laryngeal and cough reflexes are not suppressed.   Instrumenting the airway under thiopentone will result in laryngospasm and a profound sympathetic response. Bronchospasm can be accentuated (also histamine release).

Intra-arterial injection is catastrophic!   Immediate intense vasoconstriction occurs, with blanching of skin, followed by cyanosis and possibly gangrene.   The mechanism not fully understood.

ETOMIDATE

Etomidate is a carboxylated imidizole-containing compound – which means that it is chemically unrelated to any other drug used for IV induction of anaesthesia, yet like midazolam is water soluble at acidic pH and lipid soluble at physiological pH.   Like the other IV induction agents, it produces its inhibitory effect on the CNS through its action at the GABA receptor.

It is packaged as a 0.2% solution (2mg/ml), in 10ml ampoules.   The usual dosage is 0.2 to 0.3mg/kg IV.   It is formulated with propylene glycol, which causes considerable pain on injection, and is associated with occasional venous irritation.

Etomidate has a very large volume of distribution, and is therefore distributed widely throughout the body.   Etomidate penetrates the brain rapidly, reaching peak brain levels at one minute.   Like thiopentone and propofol, rapid awakening occurs due to redistribution. Hepatic clearance is five times that of thiopentone.   It is also a highly protein-bound, and therefore displaces other highly protein-bound drugs from their binding sites (eg. warfarin).

Clinically, etomidate is associated with myoclonic movements, which are due to disinhibition of subcortical structures that normally suppress extrapyramidal (involuntary) motor activity. Myoclonus, dystonia and tremor occur in more than 50% of patients.

There is significant suppression of adrenocortical function.   Even with a single bolus dose, cortisol production can be inhibited for 4 to 8 hrs.   This may be very important in sepsis and shock.   Etomidate has not escaped serious suspicion, but so far little evidence exists to implicate the drug directly as a cause of increased peri-operative morbidity in non-elective surgery.   For this reason, etomidate has been withdrawn in a number of countries such as the United States, Canada, the Republic of Ireland and Australia.

Etomidate's depressive effects on myocardial contractility are minimal, with minimal changes in heart rate, stroke volume and cardiac output. Mean arterial pressure may decrease by up to 15%, because of decreases in systemic vascular resistance. Administration of etomidate to severely hypovolaemic patients could still result in severe hypotension.

Despite the above concerns, etomidate is widely used as the agent of choice in cardiovascular instability (hypovolaemic shock, etc.). Until etomidate has either been fully implicated or freed from its suspicions, ketamine (or even low-dose thiopentone) may be a more judicious choice under these circumstances.

Etomidate is associated with an increased incidence of nausea and vomiting.

Etomidate is a potent cerebral vasoconstrictor, lowers intracranial pressure and decreases cerebral blood flow and $CMRO_2$ by 35 to 40%, in a similar fashion to comparable doses of thiopentone.

Etomidate produces an increase in the excitatory spikes seen on EEG, and may activate seizure foci. Etomidate should therefore be used with caution in patients with a history of seizures. Paradoxically, etomidate also possesses anticonvulsant properties, and has been used to terminate status epilepticus.

Etomidate causes less ventilatory depression than barbiturates, the effects lasting for three to five minutes. Decreases in tidal volume are offset by an increased respiratory rate. Etomidate may stimulate ventilation independently of CO_2-responsive medullary centres, and for this reason may be preferable when spontaneous ventilation is preferred.

More on Etomidate:

Etomidate used as a stand-alone agent in RSI carries only a 55% success rate, which is no better than that for midazolam, and is therefore not the answer to drug assisted intubation without paralysis.[80,81]

The biggest single criticism levelled against etomidate is its propensity to cause adrenocortical suppression. While this inhibition has previously not been thought to be significant in single induction-dose boluses of the drug, a 2005 editorial in the "Anaesthesia" journal has prompted a sobering revisitation of the subject.

Etomidate has escaped scrutiny in the examination of an unacceptably high mortality rate in emergency surgery, in which etomidate was used. The authors point to the moratorium placed on etomidate infusions in the ICU setting, where its use increased mortality from 28% to 77% in polytrauma patients. The authors thus question the allegedly benign effect of adrenocortical suppression following single bolus induction-dose etomidate administration in the context of critical illness, emergency surgery and septic shock.

Considering that etomidate has been withdrawn in the United States, Australia, Canada, and the Republic of Ireland, the authors caution against the blind administration of this drug, and urge the consideration of safer induction agents under the circumstances [82].[82]

Thus, the fact that the potentially lethal complications of etomidate are currently being debated in the international literature, and the fact that etomidate offers at best a 55% successful intubation rate in the combative patient, we are forced to look elsewhere for acceptable stand-alone alternatives.

PROPOFOL

Propofol is chemically distinct from all other sedative-hypnotics.   It is a substituted isopropyl phenol that is administered as an aqueous 1% solution (10mg/ml), comprising soybean oil, glycerol and egg lecithin.   Awakening is more rapid and complete than with the other induction agents, and there are minimal residual CNS effects.

Propofol is formulated without any preservative, and serves as a nutrient bacterial growth medium.   Propofol solutions must be used within six hours of opening, as sepsis and death have been linked to contaminated solutions.

Severe pain is often experienced on injection.   This can be attenuated by the addition of 10 to 20mg lignocaine (2ml 1% lignocaine added to 18ml propofol).

Propofol has a significant anti-emetic effect (by an unknown mechanism), is anticonvulsant on the basis of its GABA activity, and is antipruritic, thought to be mediated at a spinal cord level.

Propofol is packaged as a 20ml ampoule of a 1% solution (10mg/ml) and is administered at a bolus dose of 2mg/kg in adults.   Double to triple this dose is required for the paediatric age group from toddlers upwards, and half to one quarter (or less) is required for the debilitated and the elderly.

Propofol decreases the rate of dissociation of GABA from its receptor, with functional inhibition of the post-synaptic neuron, in a similar manner to thiopentone and etomidate.

Clearance exceeds hepatic blood flow, emphasising that tissue uptake as well as metabolism are important in removing the drug from the circulation.   There is rapid and extensive hepatic metabolism with renal excretion of inactive water-soluble metabolites.   Propofol is safe to adminster to cirrhotic patients, and despite 75% renal clearance, is safe to administer in renal failure.

Patients older than 60yrs have smaller volumes of distribution and slower clearance rates. Children require higher doses because of larger volumes of distribution and higher clearance rates.

Propofol decreases cerebral blood flow, intracranial pressure and $CMRO_2$.

Large doses may decrease systolic blood pressure to the point that cerebral circulation is compromised. Cerebral autoregulation, with respect to CO_2 response, is unaffected by propofol. Cortical EEG changes are similar to those induced by thiopentone.

Decreases in blood pressure are greater than that for comparable doses of thiopentone. This is due to (1) inhibition of sympathetic vasoconstriction, and decreased SVR, and (2) has a direct negative inotropic effect on the myocardium due to intracellular calcium antagonism. The baroreceptor mediated reflex is often suppressed – as heart rate often remains unchanged even in the presence of severe hypotension. Sympathetic activity may be suppressed more than parasympathetic activity, as profound bradycardia and asystole have been described in healthy patients. The blood pressure drop is exaggerated in hypovolemic patients, patients with impaired LV function, the elderly and debilitated.

Propofol suppresses the intubation response to a much greater degree than thiopentone. Ablation of upper airway reflexes allows for much easier airway instrumentation, most commonly seen with the ease of LMA placement following administration of propofol. Respiratory depression is dose-dependent. Apnoea occurs in 25 to 30%, and is enhanced by opiates and other respiratory depressants. There is a heightened ventilatory response to hypercarbia, and a decreased response to hypoxia. Propofol has significant bronchodilatory effects, and a decreased incidence of intra-operative bronchospasm is seen.

KETAMINE

Ketamine is a phencyclidine derivative producing "dissociative anaesthesia". It dissociates thalamic sensory input from the cortical and limbic systems. The patient may appear to be awake or semi-rousable with varying degrees of hypertonic and purposeful muscle movements, but is amnestic and experiences intense analgesia. Ketamine is a potent hallucinogen, has a very high abuse potential and is marketed as a street drug known as "Special K". Emergence delirium and long emergence times limit its use.

Ketamine has been reported to work at mu, kappa and delta opioid receptors, but its principle site of action is as a non-competitive antagonist at the NMDA (N-methyl D-aspartamate) receptor. The NMDA receptor is an excitatory calcium channel receptor of the glutamate receptor family.

Ketamine can be administered by the IV, IM, subcutaneous, oral, sublingual and intranasal routes. Ketamine is highly lipid-soluble (about five to ten times that of thiopentone), and therefore has a rapid onset of action and a relatively short duration of action. Peak plasma concentrations occur at about one minute after IV administration, and five minutes after IM administration.

Ketamine is transferred to highly perfused tissues, with rapid transfer across the blood-brain barrier.   It has a large volume of distribution and a high hepatic clearance.   It is not significantly protein bound.   Active metabolites may contribute to its duration of action, especially with multiple dosing.

Ketamine is administered at a dose of 2mg/kg IV and 5mg/kg IM.   The IM route is associated with longer onset and emergence times.   Intense analgesia occurs with doses of 0.2 to 0.5mg/kg.   Ketamine can be administered by the oral, sublingual and intranasal routes as well.

The following important drug interactions must be noted:

Ketamine used in combination with theophylline can precipitate seizures.   Co-administration in the presence of beta blockers can unmask myocardial depression.   Enhanced arhythmogenicity may be seen when ketamine is given with adrenalin.   The use of atropine increases the incidence and magnitude of emergence delirium.

Ketamine hallucinations, or emergence delirium, otherwise known as "bad trips" can last as long as 24 hours, and can be severe enough to leave the patient with post-traumatic stress disorder.   Hallucinations can be attenuated with the co-administration of a benzodiazepine, midazolam being the benzodiazepine of choice.   Please note that ketamine should never be adminstered without the concurrent administration of midazolam!

Ketamine is a potent cerebral vasodilator, increasing cerebral blood flow by 50% in normocapneic patients.   Benzodiazepines blunt this response.   Nonetheless, ketamine has been shown to be used without adverse effect in traumatic brain injury and raised intracranial pressure [83, 84].[83,84]

Ketamine raises systolic blood pressure, heart rate, cardiac output, cardiac work, and myocardial oxygen requirements.   These effects are progressive over 3 to 5 minutes, settle over 10 to 20 minutes, and are attenuated by the co-administration of benzodiazepines.   Myocardial depression may be unmasked in catecholamine-depleted (severely shocked) or beta-blocked patients.

Ketamine does not significantly depress ventilation, and the CO_2 ventilatory drive is maintained.   Upper airway reflexes remain relatively intact, but protection against aspiration is not guaranteed.   If a patient's airway is compromised, the patient should be intubated.   Salivary and mucous secretions are increased significantly, and the co-administration of an antisialogogue should be considered.   Ketamine is a potent bronchodilator.

EMA

Ketamine for rapid sequence induction in patients with head injury in the emergency department

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Abstract

Objective:	To examine the evidence regarding the use of ketamine for induction of anaesthesia in patients with head injury in the ED.
Method:	A literature review using the key words ketamine, head injury and intracranial pressure.
Results:	Advice from early literature guiding against the use of ketamine in head injury has been met with widespread acceptance, as reflected by current practice. That evidence is conflicting and inconclusive in regards to the safety of using ketamine in head injury. A review of the literature to date suggests that ketamine could be a safe and useful addition to our available treatment modalities. The key to this argument rests on specific pharmacological properties of ketamine, and their effects on the cerebral haemodynamics and cellular physiology of brain tissue that has been exposed to traumatic injury.
Conclusion:	In the modern acute management of head-injured patients, ketamine might be a suitable agent for induction of anaesthesia, particularly in those patients with potential cardiovascular instability.

MAGNESIUM SULPHATE

INTRODUCTION:

Whilst not being an anaesthetic induction agent, magnesium sulphate should be considered prior to intubation of patients with severe/uncontrolled hypertension.   Malignant hypertension, in conjunction with the sympathetic surge accompanying laryngoscopy and intubation, can cause cardiovascular effects and intracranial haemorrhage.   It is for this reason that it is included in this section on pharmacological induction agents.

MAGNESIUM PHYSIOLOGY:

Magnesium is the second-most important intracellular cation, potassium being the first.   The normal plasma magnesium concentration is 1 mmol/litre.

It plays an essential role in the regulation of most cell functions, activating over 300 enzyme systems.   It is critically important in energy metabolism, being essential to the production and functioning of ATP.   It is also important for DNA, RNA and protein synthesis.   It is a "natural" physiological calcium antagonist, and possesses potent membrane-stabilising properties.

It is THE drug of choice in exogenous (and endogenous) adrenalin overdose, or in adrenalin-induced dysrhythmias.

HYPOMAGNESAEMIA (<1MMOL/LITRE)

Most commonly seen in the ICU setting. Signs and symptoms are similar to that of hypocalcaemia.

Sx hypocalcaemia:

- Neuromuscular irritability and neuropsychiatric manifestations, viz. paraesthesiae, circum-oral numbness, muscle cramps, anxiety, tetany, convulsions, laryngeal stridor, dystonia, psychosis.
- Increased risk of dysrhythmias, and respiratory muscle impairment.
- CNS irritability – hyper-reflexia and convulsions.
- Skeletal muscle spasm: Trousseau's sign (inflation of BP cuff above diastolic pressure for 3 minutes causes severe tetanic spasm of fingers and wrist), and Chvostek's sign (gentle tapping over facial nerve area causes twitching of facial muscles).

HYPERMAGNESAEMIA: (>2.5MMOL/LITRE):

Magnesium is a CNS depressant (it was used as a genaral anaesthetic in the early 1900's).

Signs and symptoms of hypermagnesaemia are dose-dependent and include:

- Slowing of cardiac conduction – widened QRS and prolonged P-Q interval.
- Sedation, hypoventilation, muscle weakness, decreased deep tendon reflexes.
- Hypotension, bradycardia and diffuse vasodilation (flushing).
- Arreflexia, coma and respiratory paralysis.

Treatment of hypermagnesaemia:

- IV fluid loading, diuresis, dialysis.
- Temporary reversal with Calcium administration.

Calcium chloride is the antidote to magnesium sulphate overdose:

Calcium Chloride ($CaCl_2$) 10% solution = 100mg/ml.

Dosage: 8 to 16mg/kg (10% solution) given IV slowly (or 500 to 1000mg, or half to one full 10ml ampoule for a 70kg adult).

Note:

1. Calcium chloride releases three times more Ca^{++} ions than calcium gluconate;

2. Rapid administration can cause bradycardia;

3. Caution in patients on digitalis – can cause digitalis toxicity and ventricular irritability;

---

4. If given with $NaHCO_3$, Ca^{++} will precipitate out as carbonates;

5. Calcium may produce vasospasm in coronary and cerebral arteries;

6. Mechanism of action: Ca^{++} ions bind to sites on actin and myosin fibrils in the sarcoplasmic reticulum, causing positive inotrope and vasoconstrictor effects.

MAGNESIUM SULPHATE (MGSO₄) PROPERTIES

Magnesium penetrates the blood-brain barrier poorly, and does not have *direct* anticonvulsant properties. However, it does have anticonvulsant properties, but by an unknown mechanism. Anticonvulsant activity is thought to be due to cerebral vasodilation – breaking vasospasm – thought to be an important cause of convulsions in eclampsia.

Magnesium interferes with the release of neurotransmitters from ALL synapses, and therefore produces enhanced action of local anaesthetics, depolarising and non-depolarising muscle relaxants. It causes severe muscle weakness in Eaton-Lambert syndrome and Myasthenia Gravis.

Eaton-Lambert Syndrome: rare non-metastatic manifestation of small cell carcinoma of the bronchus, causing defective acetyl choline release at the neuromuscular junction.

Myasthenia Gravis: acquired condition of unknown cause, where antibodies to post-synaptic acetyl choline receptors lead to weakness and fatiguability of proximal limb, ocular and bulbar muscles.

- It has a direct vasodilatory action on blood vessels (as mentioned above).
- It reduces systemic vascular resistance by sympathetic blockade and inhibition of catecholamine release.
- It causes a marked decrease in contractile force (negatively inotropic) in the isolated heart. It also causes bradycardia in the isolated heart, but in the intact patient causes a reflex tachycardia because of vagal inhibition (inhibits acetyl choline release).
- It has no effect on respiratory drive, but is an effective bronchodilator, and may increase the efficacy of beta-agonists.
- It is a powerful tocolytic.
- It acts as a diuretic on the kidney, enhanced by its vasodilatory effect.

MAGNESIUM SULPHATE PHARMACOLOGY:

Packaged as 50% w/v = 500mg/ml in one millilitre ampoules.

Routes: IM and IV.

Indications:

- GPH – lowers blood pressure and anticonvulsant.
- Anti-arryhthmic: treatment of choice in Torsade de Pointes)
- Reduces peri-infarct VT and VF; has a cardioprotective effect in acute MI.
- Endogenous and exogenous adrenalin overdose.
- Magnesium deficiency (known or suspected), eg. patients on diuretics, alcoholics, chronic diseases and other poor nutritional states.
- (Prophylaxis 1 to 2g over 1 to 5 minutes).
- Status Asthmaticus.

Dose:

See the drug protocols at the end of this manual.

a) Obstetrics: 60mg/kg (4 to 6g). Halve dose if patient has already received MgSO$_4$. [Infusion rate: 4g in 250ml D$_5$W or saline at max 3ml/minute].

b) Myocardial Infarction: (VT) 1 to 2g or 2 to 4ml of 50% solution, diluted to 10ml, given over 1 to 2 minutes. If VF, give rapid IV.

c) Convulsions: IM 1 to 5 g as 50% solution (undiluted), or IV 1 to 4g as 10% solution given IV slowly.

d) Asthma: see drug protocol at the end of this manual.

Antidote:

Calcium chloride – see box above.

See drug protocols in the back of the AAM manual for exact dosages.

PRESSOR DRUGS

CEREBRAL PERFUSION PRESSURE:

CPP = MAP – ICP (or CVP)*

[* whichever of the two is greatest]

CPP = Cerebral perfusion press.
MAP = Mean arterial pressure

Mean Arterial Pressure is slightly less than halfway between systolic and diastolic BP. This is because systole is shorter than diastole. MAP can be approximated as DBP + 1/3 of pulse pressure, and can be accurately measured by integrating the area under the arterial pressure waveform graph.

Intracranial hypertension is defined as a sustained rise in intracranial pressure above 15mmHg.   Normal cerebral perfusion pressure is in the order of 60mmHg, where the MAP is about 75mmHg.

When intracranial pressure rises, the mean arterial pressure must be increased, in order to maintain an adequate cerebral perfusion pressure, or else cerebral perfusion rapidly becomes compromised.   Intracerebral pressures can rise well above 20mmHg following Traumatic Brain Injury or haaemorrhagic stroke, and it therefore becomes critical to maintain mean arterial pressures above 80mmHg.

The administration of benzodiazepines, opiates (often in conjunction with each other, and therefore with synergistic side-effects) and other anaesthetic IV induction agents, can drop mean arterial pressure considerably.

This results in the serious compromise of cerebral perfusion pressure.   A low mean arterial pressure in the face of a raised intracranial pressure can entirely obliterate cerebral perfusion!

It is for this reason that IV induction agents must be carefully administered to patients with intracranial hypertension, and that pressor drugs are available to be adminsistered in the presence of a low mean arterial pressure following tracheal intubation.

These concepts, including the Monro-Kelly doctrine, are fully discussed in the lectures Traumatic Brain Injury and Ventilation in the Emergency Medicine Environment.

EPHEDRINE

 Ephedrine is an indirect-acting alpha agonist and a direct-acting beta agonist.   It causes the release of noradrenalin to be released from sympathetic nerve terminals.   Ephedrine acts as a pure alpha agonist in the presence of beta blockade.

Administration of ephedrine thus results in an increased cardiac output (increased heart rate and stroke volume) and an increase in vascular tone. Both systolic and diastolic pressure are elevated.   The cardiovascular effects of ephedrine are similar to those of adrenalin, but the effects are 250 times less intense and last ten times longer than adrenalin.

Renal and splanchnic blood flow are decreased, while coronary and skeletal blood flow are increased.

Ephedrine is packaged as 50mg/ml one millilitre ampoules, and is usually diluted into a 10ml syringe, constituted as 5mg/ml in 10ml.   It is administered at bolus doses of 5 to 10 mg IV (and as high as 25mg) titrated to effect to restore blood pressure during sympathetic blockade.

Uterine blood flow is not greatly affected by ephedrine, as alpha-mediated vasoconstriction in the uterine vascular bed is less than that which occurs in the systemic vascular bed.

Ephedrine also has beta-2 stimulatory effects, and can be used as an adjunct to treat bronchospasm.   It also has an anti-emetic effect similar to that of droperidol.

Subsequent doses of ephedrine produce lesser responses to the drug, a phenomenon known as tachyphylaxis, thought to be due to the fact that some receptor sites are still bound by ephedrine at the time of subsequent doses, and also due to depletion of noradrenalin stores in sympathetic nerve terminals.

PHENYLEPHRINE

Phenylephrine is structurally almost identical to adrenalin, having predominantly a direct alpha agonist effect, with a slight indirect alpha agonist effect.   There is minimal effect on beta receptors.   The resulting venoconstriction is greater than arterial constriction.   Clinically, phenylephrine mimics the effects of noradrenalin, but is less potent and longer lasting.

Phenylephrine is packaged as 10mg in one millilitre ampoules, and is diluted as 10mg in 200ml saline (50mcg/ml).   It is administered IV in one to two millilitre (50 to 100mcg) boluses, or can be run as a continuous infusion through a 60drops/ml paediatric IV administration set, if regular top-up doses are required.

Phenylephrine is of great value in patients with coronary artery disease and aortic stenosis because, in contrast to other sympathomimetics, it increases coronary perfusion pressure without chronotropic side-effects.

Phenylephrine causes reflex slowing of the heart in response to blood pressure increases, mediated by the carotid body baroreceptors.   Cardiac output falls as a result of a decreased heart rate and an increased afterload.   Renal, splanchnic and cutaneous blood flows are decreased, while coronary blood flow and pulmonary arterial pressure are increased.

The reflex vagal effects mediated by phenylephrine can be used to slow the heart rate in haemodynamically significant tachyarrhythmias.

NEUROMUSCULAR BLOCKADE

INTRODUCTION

Muscle relaxant drugs are not formally taught to emergency medicine doctors, registrars and paramedics as part of their pharmacology syllabus.   This is a great pity since, even though these drugs are not included in the Advanced Life Support protocol, they are extremely useful

drugs, and as such are used regularly during patient management whilst in the Emergency Unit, resuscitation unit and during the interfacility transfer of ventilated patients.

As said, these drugs are extremely useful, and therefore powerful drugs.   Like all powerful drugs, they are also extremely dangerous, and should not be used without specific medical direction.   The knowledge imparted by way of this course manual does not suggest a tacit approval for the autonomous use of these drugs by paramedics, but is intended to render their use by paramedics safer, under circumstances where prescribed by a medical practitioner.

THE RATIONALE OF PARALYSING VENTILATED PATIENTS

In years gone by, for various reasons not least of which was the relative state of "unsophistication" of ventilators, patients were routinely paralysed with non-depolarising muscle relaxants.

This is no longer the case – in modern intensive care units, paralysing patients has become the extreme exception, rather than the rule, and a practice severely frowned upon.   Part of the reason for this trend is that modern ventilators have achieved a degree of sophistication which allows for synchronisation between the ventilator's inspiratory and expiratory cycling, and the patient's spontaneous efforts at breathing.

We have also realised that it is an extremely insensitive (and cruel) practice to paralyse patients without adequate sedation.   Long-term infusions of muscle relaxants have also been shown to have direct toxic effects on nerve tissue, causing a type of "intensive care neuropathy".   Muscle relaxant drugs also have other side-effects to which the patient is exposed.

TRANSPORT VENTILATORS

In order to keep transport ventilators compact, light-weight and simple, they have remained extremely basic machines, without the ability to synchronise breath cycles to the patient's respiratory efforts.   This often results in patients "fighting" the ventilator – ie. the patient attempting to exhale at the time that the ventilator is in its inhalation cycle, and vice versa.

Adequate sedation with an appropriate benzodiazapine or other sedative agent often overcomes this problem.   Sometimes, however, even deep sedation is unable to overcome the potent respiratory stimulus seen in some patients, and paralysis must be considered, or else adequate ventilation cannot be achieved.

It is also important to prevent patients from gagging or coughing against the tracheal tube, especially in cases of head injury.   This is discussed in detail in the lectures on "Ventilation in the Emergency Medicine Environment" and "Traumatic Brain Injury".

Paralysis without sedation must never be undertaken!

THE DANGERS OF PARALYSING A PATIENT

Paralysing patients is an extremely dangerous practice, and the decision to paralyse should not be taken lightly.   In the case of either a ventilator malfunction, a ventilator circuit "disconnect", or a dislodged tracheal tube, the patient will be rendered apnoeic.   Thus ventilator failure, disconnect and/or other apnoea alarms, as well as continuous (preferably waveform) capnography should be used at all times and without exception.

Under normal circumstances, in a non-paralysed patient experiencing ventilatory failure, the patient will be able to maintain at least some degree of spontaneous ventilation, albeit it inadequate.   In the case of a paralysed patient, he or she will not be able to take one single breath!   It sometimes occurs, through pharangeal and upper airway oedema, or due to natural anatomic variances, that a patient who is not intubated, cannot be ventilated despite the best face-mask seal, orophrangeal airway in situ, and ventilation technique.

In the case of a dislodged tracheal tube, a "Can't intubate, Can't oxygenate" (CICO) scenario is probably the single-most frightening event a medical professional can experience, and will rapidly lead to hypoxic brain damage and death by asphyxia, if not immediately detected and corrected.

Hence, under circumstances where a patient is going to be paralysed for the purposes of an interfacility transfer, full monitoring (preferably including capnography with disconnect alarm) and the utmost vigilance are mandatory.   It goes without saying that an emergency back-up system for ventilation, and equipment for emergency re-intubation must be at hand at all times.

For example, the transfer of a ventilated patient in a hospital elevator, or down a long passage, without intubation equipment, a spare oxygen cylinder, and a bag-valve mask resuscitator (with face mask, suction unit and oropharangeal airways), consitutes the most gross criminal negligence!!

DISTINCTION BETWEEN DEPOLARISING AND NON-DEPOLARISING BLOCKADE

(The physiology of the neuromuscular junction must to be understood before reading this section).

Depolarising muscle relaxants resemble Acetylcholine (Ach) and therefore bind to the post-synaptic nicotinic receptor, and generate an action potential.   They are therefore cholinergic in action.   They are not metabolised by Ach esterhase, and therefore produce a prolonged depolarisation of the muscle end-plate (competitive binding to the Ach receptor prevents repolarisation, and therefore subsequent depolarisation).

So, the big thing to remember is that initial depolarisation and a brief period of *intense* muscle contraction occur, followed by paralysis, lasting for three to five minutes (or longer).   These drugs can be thought of as "nicotinic agonists".

There is only one depolarising muscle relaxant in clinical use worldwide, and that is suxemethonium hydrochloride, also known as "sux", succinyl choline or Scoline (trade name). They are all the same drug.

Non-depolarising muscle relaxants (NDMRs), of which there are many, also bind to the nicotinic receptor, but bind in such a way that they do not trigger a response (depolarisation). Instead, they bind competitively, and then "hog" the receptor site, preventing the binding of Ach and consequent activation of the receptor.   Depolarisation and therefore muscle contraction do not occur.

NDMRs can therefore be thought of as "nicotinic antagonists",or "blockers", in the same way that beta blockers prevent the action of adrenalin at beta receptors.

The different NDMRs all have different characteristics: dose, onset and duration of action, histamine release, intrinsic sympathomimetic activity, metabolism and excretion.   I am not going to present a comprehensive or exhaustive breakdown of the various agents, but will offer only a summary of the more salient points.   A full discussion could be dedicated to each drug.

SUXEMETHONIUM HYDROCHLORIDE (SCOLINE)

"Sux" consists of two Ach molecules joined end to end, and hence the profound muscle contraction and cholinergic side-effects seen with the use of this drug.

It is common practice to keep suxemothonium in the fridge, to prevent its spontaneous breakdown, and it is a common belief that sux will degrade over a matter of hours.   However, the degradation rate of sux has been reported as 0.3% per month at 4^OC, 2% per month at room temperature, and 8% per month at 37^OC [85].   Sux can therefore be stored at room temperature for *five months* whilst losing only 10% efficacy, and at 37^OC for one month.[85]

Only a very small percentage of drug reaches the "effect site" -- the nicotinic receptor -- as it is rapidly hydrolysed (broken down) by an enzyme similar to acetylcholinesterase, called pseudocholinesterase.

In some people with genetic abberations, there is a deficiency of pseudocholinesterase, which results in a condition called "Scoline Apnoea". Paralysis can last for six to eight hours (where it is normally of only three to five minutes duration).

Onset of action of sux is about 30 seconds, or one arm-brain circulation time. This drug is the most ideal drug that we have for "rapid sequence induction" (RSI) – a method of emergency intubation performed where there is a high risk of aspiration of stomach contents.

Suxamethonium has a relatively short duration of action – in the region of three to five minutes in normal, healthy individuals. This fact lulls the unsuspecting into a false sense of security – or a fool's paradise. The uncomfortable truth is demonstated in Benumof's graph below:

TIME TO HEMOGLOBIN DESATURATION WITH INITIAL F_AO_2 = 0.87
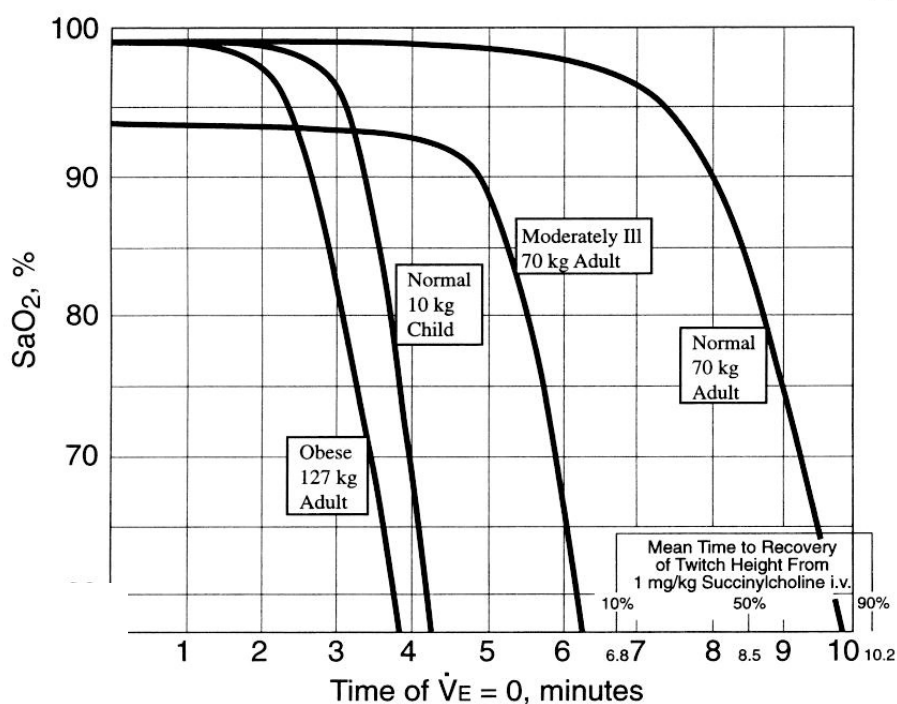
Benumof has demonstrated that ill or debilitated adults, obese and pregnant patients, and children will achieve terminal desaturation before attaining even 10% neuromuscular recovery![86] Terminal desaturation is defined as hypoxia leading to unconsciousness and death before the brainstem's respiratory centre is activated, and breathing occurs. The implication of this is that, in a can't intubate – can't oxygenate RSI scenario, the patient will asphyxiate and die *before* neuromuscular recovery is attained, regardless of the claim that suxemethonium is a short-acting neuromuscular blocker!

Additionally, conditions such as pregnancy, scoline apnoea, and organophosphate poisoning markedly prolong the action of suxamethonium. Organophosphates are cholinesterase inhibitors, and so inhibit the action of pseudocholinesterase as well as acetylcholinesterase.

Please see the next section, non-depolarising muscle relaxants, for a full discussion of rocuronium and sugammadex regarding neuromuscular reversal.

Because sux is literally "pure" acetylcholine, it has marked cholinergic side-effects: profound bradycardia, salivation, bronchial secretions and bronchoconstriction are seen. This is seen especially after a second dose, where atropine (at a dose of 20 mcg/kg) must always be administered concurrently.

Rapid and severe (and potentially fatal) hyperkalaemia is commonly seen after sux administration, owing to the muscle injury that follows the profound muscle contraction seen with sux administration. Patients always wake up with stiff and painful muscles (as though they had run a marathon) after sux administration.

The following patients are particularly at risk of fatal hyperkalaemia:

- Any kind of muscle disease, such as muscular dystrophy.
- Renal failure (already hyperkalaemic).
- Any burn injury or severe trauma (tissue damage).
- Neurological disorders or injury (up-regulation of nicotinic receptors occurs after a denervating injury, as a natural process, owing to lack of stimulation of those receptors -- and therefore profound muscle contraction occurs with subsequent life-threatening potassium release).

Owing to the generalised muscle contractions, patients experience raised intragastric pressure (risk of aspiration), raised intra-ocular pressure (hazardous in eye trauma), and raised intracranial pressure.

However, the risks of aspiration are generally thought to be more hazardous than the side-effects of the raised pressures described above, so sux is still administered with caution, where these patients present with a potential full stomach.

"The administration of suxemethonium, with all of its problems, is infinitely better than having a patient's lungs filled with vomit." – Prof. MFM James, Dept. of Anaesthesia, University of Cape Town.

Sux is a powerful trigger for a condition known as Malignant Hyperthermia, which is a catastrophic, life-threatening hypermetabolic state. Malignant Hyperthermia requires highly specialised in-hospital treatment and a specific antidote (Dantrolene hydrochloride), if the patient is to stand any chance of survival.

As can be seen, suxemethonium is not a drug that anyone should be using complacently, whether inside or outside of the controlled hospital environment.

NON-DEPOLARISING MUSCLE RELAXANT DRUGS

These are a group of drugs which share many common characteristics. They therefore share a common set of rules, although each drug has exceptions to these common rules.

NDMRs generally take three to five minutes before maximum paralysis is achieved, and are therefore not suitable for rapid sequence induction. A controversial exception is Rocuronium (Esmeron), which in high dose can be used for RSI. One of the other problems, in terms of RSI, is that their duration of action varies between twenty minutes and an hour – depending on the dosage and pharmacokinetics of the drug. Hence, a failed intubation can quickly turn into a nightmare situation with no light at the end of the proverbial paralysis tunnel.

All of the NDMRs have a prescribed induction dose, based on lean body mass. Top-up doses are generally administered on an "as-needed" basis, at 10% of the calculated induction dose. There is no maximum dose, as these drugs are constantly redistributed and metabolised.

All of the NDMRs are highly charged molecules and do not cross biological membranes easily. They therefore do not redistribute to other compartments (see notes on pharmacokinetics), do not cross the blood-brain barrier, and importantly, do not cross the placenta. The same is true for suxemethonium.

Some of these drugs need to be refrigerated, and others do not.

They are not metabolised by pseudocholinesterase (except for Mivacurium), and depend on being "washed away" from the synaptic cleft and metabolised over time. They are metabolised by the liver, and metabolites are excreted by the kidneys. Some have active metabolites. Renal and/or liver failure results in prolonged duration of action. A notable exception to this is Atracurium, which undergoes Hoffman elimination – this is a non-enzymatic degradation process; or a kind of "self-destruct" mechanism.

Reversal of the paralysing effects of NDMRs are brought about by (1) the passage of time and (2) the administration of an acetylcholinesterase inhibitor -- neostigmine. Neostigmine (similar to organophosphate poisons) allows for an excess of Ach in the synaptic cleft. Competitive binding of Ach to the nicotinic receptor then displaces the NDMR molecule, and its action is terminated.

However, neostigmine's action also has an effect at muscarinic receptors (parasympathetic), and so must be combined with either atropine or glycopyrrolate, to counter the cholinergic parasympathetic side-effects of Ach. Atropine has no effect at the nicotinic receptor site.

NDMRs are divided into two major groups, the amino steroids and the benzyl isoquinilones. The significance of this distinction is that the amino steroids are non-histamine releasers, while the benzyl isoquinilones release histamine.   Histamine can cause or exacerbate bronchospasm. Histamine also causes a transient vasodilation and drop in blood pressure, with a resultant baroreceptor- mediated reflex tachycardia.   Histamine releasers would be unsuitable for asthmatics and patients with ischaemic heart disease.

Pancuronium (Pavulon) is a non-histamine releaser, but has intrinsic vagolytic <u>and</u> sympathomimetic properties – it elevates heart rate and blood pressure, and is a very good bronchodilator.   It is also a long-acting NDMR (duration of action 45 to 60 minutes). Because of its bronchodilator effect, it is an ideal agent for paralysing ventilated asthmatics, and because of its increased cardiac output, is indicated for shocked patients (eg. hypovolaemia), that require prolonged paralysis for ventilation.   It is relatively contra-indicated in ischaemic heart disease and head injuries.   Pancuronium must be refrigerated. The usual induction dose is 0.1mg/kg.

Vecuronium (Norcuron) is very similar to Pancuronium, except that it is devoid of the bronchodilator and sympathomimetic properties of Pancuronium.   It is a haemodynamically stable drug, and is therefore safe to use in ischaemic heart disease and head injuries.   It must be reconstituted from its powder form before use, and does not need to be refrigerated. It is not a histamine releaser, and has an intermediate duration of action of about 30 minutes. Vecuronium is a very close cousin of Pancuronium, and is also dosed at 0.1mg/kg.

Mivacurium (Mivacron) is a short-acting agent, and as such is not suitable for longer term ventilation or interfacility transfers. Duration of action is ten to fifteen minutes.   Mivacurium is the exception in that it is the only NDMR to be metabolised by pseudocholinesterase. Mivacurium is a histamine releaser.   The usual dose is 0.2mg/kg.

Atracurium's claim to fame is its non-enzymatic Hoffman elimination – it does not depend on either the liver or kidneys for metabolism and/or excretion.   Atracurium is a histamine releaser.   Atracurium's usual dose is 0.5mg/kg, with an intermediate duration action of 20 to 30 minutes.

Cisatracurium (Nimbex) is one of ten isomers of Atracurium, which does *not* release histamine.   Cisatracurium should be kept in a cool place, but does not strictly need to be refrigerated.   Cisatracurium's usual dose is 0.15mg/kg.   With the advent of cisatracurium, atracurium has become more or less obsolete because of function, but is still widely used because of price.

Rocuronium (Esmeron) is the newest NDMR, and is marketed as the only alternative to suxemethonium for RSI.   It is still less ideal than suxemethonium, despite all of suxemethonium's problems.   Of concern is its long duration of action at the high dose (0.9 to

1.1 mg/kg) required for RSI.   This would be catastrophic in a Can't Intubate Can't oxygenate RSI situation. Rocuronium does not need to be refrigerated, and can be stored at room temperature (25°C or below) for up to eight weeks.   It is not a histamine releaser.   The usual induction dose is 0.6mg/kg.

Sugammadex is the agent-specific reversal agent to rocuronium.   This compound causes rapid reversal of neuromuscular blockade induced by rocuronium.   The rocuronium molecule becomes encapsulated by the Sugammadex molecule, bound through van der waal's and hydrophobic forces in the plasma.   This results in reversal from profound rocuronium-induced neuromuscular blockade within two minutes.

As previously mentioned, the alleged short duration of action of suxamethonium is somewhat of a myth, lulling unsuspecting clinicians into a false sense of security.   In a recent study comparing spontaneous return of neuromuscular function from suxamethonium versus reversal of an intubating dose of rocuronium by an "escape bolus" of sugammadex, the rocuronium-sugammadex combination won hands-down, as shown in the table below:[87]

Median time from tracheal intubation to:	Suxamethonium	Rocuronium/Sugammadex
Return of spontaneous ventilation:	406 seconds	216 seconds
90% recovery of first twitch of Train of Four:	518 seconds	168 seconds

Whilst the above information may be very exciting, the authors draw attention to the fact that an "escape bolus" of 16mg/kg of sugammadex for a 75 kg patient costs in the order of 760 Euros – or in the region of R9000 ZAR (South African rands).

Alcuronium (Alloferin) is an older NDMR, which confers no advantage over the more modern agents.   It is a histamine releaser, and is of intermediate duration of action (20 to 30 minutes).   The usual induction dose is 0.2mg/kg.

D-tubocurarine (curare) and older such agents are no longer available, and have been withdrawn from the market. However, many text books still refer to the practice of neuromuscular paralysis as "curarisation", although today curare carries little more than historical significance.

DRUGS REQUIRED FOR ADVANCED AIRWAY MANAGEMENT INCLUDING RSI:

- Sedation –midazolam, diazepam.
- Induction agents – propofol, etomidate, thiopentone, ketamine.
- Neuromuscular blockade – suxemethonium, rocuronium, pancuronium, atracurium, cisatracurium, vecuronium.
- Analgesia – opiates (morphine, fentanyl, alfentanil).
- Vasopressors – ephedrine, phenylephrine.
- Anticholnergics: atropine, glycopyrrolate.
- Adrenergic – adrenalin.

1. Safe drug administration depends on the practitioner having a full understanding of the pharmacological profile of a given drug.   These drug infusion protocols are not a substitute for knowledge of indications, contra-indications, side-effects, special precautions, kinetics and dynamics of each individual drug.
2. It is incumbent upon all practitioners to follow HPCSA protocols and institutional policies, and to seek advice and clarification when in doubt.
3. NOT ALL DRUG CAUTIONS HAVE BEEN DOCUMENTED IN THIS PROTOCOL.
4. Always label solutions clearly, documenting generic drug name, diluent, concentration, patient name, time and date of mixing, and the name of the practitioner who mixed the drug.
5. It is always a good idea to ask a colleague to double-check your drug infusion mixtures, and to document this in the patient's clinical record.
6. When documenting a drug infusion in the patient's clinical records, be sure to document the drug name, drug concentration mixed, rate administered, and time commenced.   It is an accepted standard of care and is institutional policy.   Failure to comply with this exposes the patient to clinical risk, and both the individual practitioner and the institution to medico-legal culpability.
7. When mixing drugs for infusion:
    a. Check that the diluent is compatible with the drug.
    b. Remember to first remove an equal volume of diluent from the total diluent volume, equal to the volume of 'neat' drug to be added to the diluent.
8. The drug dosages and volumes specified in these protocols are intended for use with a 50ml syringe driver.   If you are mixing solutions of 100ml, multiply drug dose/volume by two; and if mixing 200ml volumes, multiply by four.   Infusion rates remain unchanged.
9. Be cautious when flushing inotropes – very small volumes of relatively dilute inotrope can have catastrophic effects when even small boluses are administered in an uncontrolled manner.   When replenishing and reloading syringes into the syringe driver, disconnect the syringe from the administration tubing first.
10. When using very low infusion rates, check deadspace volumes in administration sets and IV catheters (esp. central lines).   It may be prudent to halve the concentration and double the flow-rate when deadspace volumes are large and/or when flow rates are low.

ADVANCED AIRWAY MANAGEMENT COURSE	
TITLE:	**MIDAZOLAM INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Dormicum.	5mg/5ml and 15mg/3ml ampoules.
Cautions:	
See SAMF.	
Loading dose:	
Adult: 0.1 to 0.2mg/kg IV.	**Paediatric:** 0.2 to 0.5mg/kg IV or IM. Administer 0.2mg/kg and repeat after 10 minutes if necessary.
Commonly for 70kg adult: 7 to 14mg Usually 5 to 10mg. May require higher dose.	**Neonatal:** As above.
Constant infusion:	
Adult dosage range: 1 to 4mcg/kg/min = 60 to 240mcg/kg/hr.	**Paediatric dosage range:** 1 to 4mcg/kg/min.
	Neonatal dosage range: As above.
Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Add 50mg in 50ml total volume = 1mg/ml. (Or 45mg in 45ml – 3 x 15mg ampoules).	**Mixture:** Add 3mg/kg into 50ml total volume.
Rate: For 70kg adult: 4.2mg/hr to 16.8mg/hr. 5 to 15ml/hr.	**Rate:** 1 to 4ml/hr.
Alternative adult regime:	**Alternative paediatric regime:**
Mixture:	**Mixture:** 10mg in 50ml = 0.2mg/ml.
Rate:	**Rate:** 1ml/kg/hr. [240mcg/kg/hr approximates 200mcg/kg/hr].
Additional notes:	
Double the strength and halve the rate if volume restriction is required.	

ADVANCED AIRWAY MANAGEMENT COURSE	
TITLE:	**MORPHINE INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Morphine.	10mg/1ml and 15mg/1ml ampoules.

Cautions:
See SAMF.

Loading dose:	
Adult: 0.1 to 0.2mg/kg.	**Paediatric:** 0.1 to 0.2mg/kg.
Commonly for 70kg adult: 7 to 14mg for a 70kg patient. Usually 5 to 10mg, but may be up to 15mg.	**Neonatal:** As above.

Constant infusion:	
Adult dosage range: 20 to 80mcg/kg/hr.	**Paediatric dosage range:** 20 to 80mcg/kg/hr.
	Neonatal dosage range: 10 to 30mcg/kg/hr.

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Add 20mg in 50ml total volume = 400mcg/ml.	**Mixture:** Add 1mg/kg into 50ml total volume.
Rate: 20 to 80mcg/kg/min = 1.4 to 5.6mg/hr. Run at 3.5 to 14ml/hr (20 to 80mcg/70kg/hr) Or for practical purposes: 5 to 15ml/hr.	**Paediatric rate:** 1 to 4ml/hr (20 to 80mcg/kg/hr). **Neonatal rate:** 0.5 to 1.5ml/hr (10 to 30mcg/kg/hr).

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: 40mg morphine in 50ml total volume.	**Mixture:**
Rate: Morphine concentration 1mg/ml at rate 1 to 10ml/hr.	**Rate:**

Additional notes:
Many units consider 2 to 5mg/70kg/hr adequate.   However, if a higher dose is required, the GSH C27 Respiratory ICU high-dose infusion at a concentration of 1mg/ml with a rate of 1 to 10ml/hr, titrated to effect, may be required.   This provides a maximum rate of 143mcg/kg/hr for a 70kg patient. Australian Royal Flying Doctor Service (Western Operations) use a morphine infusion regimen of concentration 1mg/ml (50mg/50ml), run at a rate of 2.5 to 15ml/hr.

Caution: Mixing of midazolam and morphine into the same syringe is frowned upon by some healthcare professionals, but the mixture is inherently stable and safe, and is used within the intensive care units of many academic hospitals throughout the world.   Ketamine and midazolam are also routinely mixed in the same syringe.   Check that local or institutional policy is accepting of this practice before implementation.

COMBINED INFUSIONS:

"Normal strength" combined infusion for adults (50ml syringe driver):
Add 50mg midazolam and 20mg morphine into 50ml total volume.
Run at 5 to 15ml/hr.

"Normal strength" combined infusion for adults (100ml ml infusion pump):
Add 100mg midazolam and 40mg morphine into 100ml total volume.
Run at 5 to 15ml/hr.
Or add 100mg midazolam and 40mg morphine into 200ml total volume.
Run at 10 to 30ml/hr.

"High dose" combined infusion for adults:
Add 50mg midazolam and 40mg morphine to a total volume of 50ml.
Run at 5 to 15ml/hr.

Australian Royal Flying Doctor Service (Western Operations):
Syringe driver: 50mg midazolam + 50mg morphine in 50ml total volume.
Mix 30ml volumes (1mg/ml) for shorter flights.
Run at 2.5 to 15ml/hr.
Infusion pump: 50mg midazolam + 50mg morphine in 500ml total volume.
Run at 25 to 150ml/hr.

Normal strength combined infusion for paediatrics:
Caution: this regime not applicable to neonates:
Add 3mg/kg midazolam and 1mg/kg morphine into a total volume of 50ml.
Run at 1 to 4ml/hr.

Normal strength combined infusion for neonates:
Add 3mg/kg midazolam and 0.5mg/kg morphine into a total volume of 50ml.
Run at 1 to 3ml/hr.

ADVANCED AIRWAY MANAGEMENT COURSE	
TITLE:	**MORPHINE AND MIDAZOLAM COMBINED INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Morphine and Dormicum	Midazolam: 5mg/5ml and 15mg/3ml ampoules. Morphine: 10mg/1ml and 15mg/1ml ampoules.

Cautions:
See SAMF.

Loading dose:	
Adult: Midazolam: 0.1 to 0.2mg/kg IV. Morphine: 0.1 to 0.2mg/kg.	**Paediatric:** Midazolam: 0.2 to 0.5mg/kg IV or IM. Administer 0.2mg/kg and repeat after 10 minutes if necessary. Morphine: 0.1 to 0.2mg/kg.
Commonly for 70kg adult: Midazolam: 1 to 4mcg/kg/min. 60 to 240mcg/kg/hr. For 70kg adult: 4.2mg/hr to 16.8mg/hr. Morphine: 7 to 14mg for a 70kg patient. Usually 5 to 10mg, but may be up to 15mg.	**Neonatal:** Midazolam: As above.

Constant infusion:	
Adult dosage range: Midazolam: 1 to 4mcg/kg/min = 60 to 240mcg/kg/hr. For 70kg adult: 4.2mg/hr to 16.8mg/hr. Morphine: 20 to 80mcg/kg/hr.	**Paediatric dosage range:** Midazolam: 1 to 4mcg/kg/min. Morphine: 20 to 80mcg/kg/hr.
	Neonatal dosage range: Midazolam: as above. Morphine: 0.5 to 1.5ml/hr (10 to 30mcg/kg/hr).

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Add 50mg midazolam and 20mg morphine to a total volume of 50ml.	**Mixture:** Add 3mg/kg midazolam and 1mg/kg morphine into a total volume of 50ml.
Rate: Run at 5 to 15ml/hr.	**Rate:** Run at 1 to 4ml/hr.

Alternative adult regime:	**Neonatal regime:**
Mixture: (higher-dose morphine infusion): Add 50mg midazolam and 40mg morphine into 50ml total volume.	**Mixture:** Add 3mg/kg midazolam and 0.5mg/kg morphine into a total volume of 50ml.
Rate: Run at 5 to 15ml/hr.	**Rate:** Run at 1 to 3ml/hr.

Additional notes:
To mix solution for infusion pump, double strength for 100ml IV bag and quadruple strength for 200ml IV bag.   Keep rate the same. Australian Royal Flying Doctor Service (Western Operations): Syringe driver: 50mg midazolam + 50mg morphine in 50ml total volume. Mix 30ml volumes (1mg/ml) for shorter flights. Run at 2.5 to 15ml/hr. Infusion pump: 50mg midazolam + 50mg morphine in 500ml total volume.Run at 25 – 150ml/h

TITLE:	**KETAMINE INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Ketalar. Brevinase.	200mg/20ml vial (10mg/ml). 500mg/10ml vial (50mg/ml). 1000mg/10ml vial (100mg/ml).

Cautions: see SAMF.
Ketamine is supplied in different concentrations.concentrations.   Ensure that you are working with the correct concentration to avoid major drug error.Use 100mg/ml or 50mg/ml vials for mixing 50ml infusions.   Always combine low-dose midazolam with ketamine to prevent emergence delirium.The mixture of ketamine and midazolam is safe and stable when mixed in the same syringe, and is an internationally accepted practice.

Loading dose:	
Adult: IV anaesthetic induction: 2mg/kg; For IM anaesthesia induction: 5mg/kg (onset 5 minutes). For sedation and analgesia: half to one quarter or less of above dose.	**Paediatric:** As for adult.
Commonly for 70kg adult: IV anaesthetic induction: 140mg. Usually 100 to 150mg IM anaesthesia induction: 350mg.	**Neonatal:** As for paediatric.

Constant infusion:	
Adult dosage range: For anaesthesia: ketamine 4mg/kg/hr. Add low-dose midazolam at 0.1mg/kg/hr. For sedation: ketamine 2mg/kg/hr and midazolam 0.05mg/kg/hr.	**Paediatric dosage range:** As for adult.
	Neonatal dosage range: As for paediatric.

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: 800mg ketamine + 20mg midazolam into 50ml total volume.	**Mixture:** 200mg ketamine + 5mg midazolam into 50ml total volume.
Rate: Run at 0.25ml/kg/hr for anaesthesia. Run at 0.125ml/kg/hr for sedation. Usually about 20ml/hr for anaesthesia and about 10ml/hr for sedation.	**Rate:** Run at 1ml/kg/hr for anaesthesia. Run at 0.5ml/kg/hr for sedation.

Alternative adult regime:	**Alternative paediatric regime:**
Mixture:	**Mixture:**
Rate:	**Rate:**

Additional notes:
Must use 50mg/ml or 100mg/ml multi-dose vials to mix infusions, therefore must have these in stock.   Can mix smaller volumes for shorter flights.

ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	PROPOFOL INFUSION
REVIEWED:	25 JANUARY 2012

Trade names:	Presentation and strength:
Diprivan	10mg/ml (1%) 20ml ampoules. 20mg/ml (2%) in 50ml vials.

Cautions: see SAMF.
Beware two separate strengths – source of drug error. Both strengths cause irritation (burning) when administered through peripheral veins. Propofol infusions must be administered with strict aseptic technique, and should be completed within 6 hours of opening the ampoule, as propofol is a preservative-free bacterial culture medium. Propofol can cause severe hypotension in the elderly, in dehydrated patients or those otherwise haemodynamically unstable. Doses may need to be doubled in children, or halved in the elderly. Propofol is incompatible with atracurium. Propofol should be used with great caution by those unfamiliar with the drug.

Loading dose:	
Adult: IV anaesthetic induction: 1.5 to 2.5mg/kg.	**Paediatric:** IV anaesthetic induction: up to 4mg/kg. (150 to 200% or more of adult dose in mg/kg).
Commonly for 70kg adult: 120 to 150mg slow IV bolus.	**Neonatal:** Not recommended.

Constant infusion:	
Adult dosage range: Maintenance of anaesthesia: 4 to 12mg/kg/hr. Sedation in ICU: 1 to 3mg/kg/hr.	**Paediatric dosage range:** Not recommended in children due to propofol infusion syndrome.
	Neonatal dosage range: Not recommended.

Regime for 70kg adult:	Regime for paediatrics:
Mixture: 1% solution = 10mg/ml. 2% solution = 20mg/ml.	**Mixture:** N/A
Rate: Range: 70 to 210mg/hr. 1% solution: 7 to 20ml/hr. 2% solution: 3.5 to 10ml/hr.	**Rate:** N/A

Alternative adult regime:	Alternative paediatric regime:
Mixture: (For administration through peripheral vein) 5mg/ml = 1000mg in 200ml saline. Add 100ml of 1% solution to 100ml saline or 50ml of 2% solution to 150ml saline.	**Mixture:** N/A
Rate: 14 to 48ml/hr.	**Rate:** N/A

Additional notes:
Total Intravenous Anaesthesia (TIVA) regimes are not recommended in the prehospital environment, and are beyond the scope of this protocol. Propofol 2% solution may not be available in Western Cape district-level government hospitals – in which case use propofol 1%. Propofol may be used for status epilepticus.
ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	ADRENALIN INFUSION
REVIEWED:	25 JANUARY 2012

Trade names:	Presentation and strength:
Adrenalin	1mg/1ml ampoules

Cautions: see SAMF.	
Be very cautious when flushing IV lines and bolusing even dilute adrenalin infusions. Very small boluses (inadvertently 'bumping' the syringe plunger) can precipitate severe tachycardia, hypertension, myocardial ischaemia/infarction and cardiac failure, even in the very young and very fit.	

Loading dose:	
Adult: IV 0.1 to 1mg for cardiac arrest. SC: 0.1 to 0.5mg.	**Paediatric:** Cardiac arrest: IV 0.1ml/kg of 1:10 000. Anaphylaxis: IV: 0.05 to 0.1ml/kg 1: 10 000 or IM 0.01mg/kg (0.01ml/kg of 1: 1000). Repeat if required.
Commonly for 70kg adult: IV: 1mg SC: 0.5mg	**Neonatal:** As above.

Constant infusion:	
Adult dosage range: 0.01 to 1mcg/kg/min titrated to response.	**Paediatric dosage range:** 0.05 to 1mcg/kg/min as below:
	Neonatal dosage range: As above.

Regime for 70kg adult:	Regime for paediatrics:
Mixture: Mix 1mg/50ml or 4mg/200ml = 20mcg/ml	**Mixture:** 0.3mg/kg in 50ml total volume.
Rate: 0.01mcg/kg/min = 2.1ml/hr 1mcg/kg/min = 200ml/hr. Usually start at 5 to 10ml/hr.	**Rate:** 0.5 to 10ml/hr.

Alternative adult regime (1):	Alternative adult regime (2):
Mixture: Double strength = 2mg/50ml or 8mg/200ml.	**Mixture:** Quadruple strength = 4mg/50ml or 16mg/200ml.
Rate: Maximum rate 100ml/hr.	**Rate:** Maximum rate 50ml/hr.

Additional notes:	
Lower doses tend to produce predominantly beta effects, while higher doses tend to produce predominantly alpha effects.	

Other:	
Maximum rates quoted above are very high and seldom, if ever, necessary. Usual dose is seldom more than 20ml/hr (0.1mcg/kg/min) single strength solution.	

ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	**DOBUTAMINE INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	Presentation and strength:
Dobutrex	250mg in 20ml ampoules (25mg/ml)

Cautions: see SAMF.	
Avoid in diluents containing sodium bicarbonate. Can be mixed with saline or D$_5$W. Administer via central line or large peripheral line – be extremely cautious of vasoconstriction, ischaemic pain and local necrosis.	

Loading dose:	
Adult: N/A	**Paediatric:** N/A
Commonly for 70kg adult: N/A	**Neonatal:** N/A

Constant infusion:	
Adult dosage range: 5 to 40mcg/kg/min.	**Paediatric dosage range:** 5 to 20mcg/kg/min:
	Neonatal dosage range: As above.

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: 250mg in 50ml total volume = 5mg/ml. 500mg in 100ml total volume = 5mg/ml.	**Mixture:** <30kg: 15mg/kg in 50ml.
Rate: 5mcg/70kg/min = 4.2ml/hr. 10mcg/70kg/min = 8.4ml/hr. 20mcg/70kg/min = 16.8ml/hr. 40mcg/70kg/min = 33.6ml/hr.	**Rate:** 1 to 4ml/hr.

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: (infusion pump) 500mg in 200ml = 2.5mg/ml.	**Mixture:** If greater volumes required for larger children, can stay with above, or: >30kg: 6mg/kg in 100ml.
Rate: 5mcg/70kg/min = 8.4ml/hr. 10mcg/70kg/min = 16.8ml/hr. 20mcg/70kg/min = 33.6ml/hr. 40mcg/70kg/min = 67.2ml/hr.	**Rate:** 5 to 20ml/hr.

Additional notes:	
Add heparin 1u/ml to the solution. Caution with dilutions – heparin is available as 1000u/ml and 5000u/ml. If 1000u/ml then: 0.1ml = 100u (use insulin syringe).	

ADVANCED AIRWAY MANAGEMENT COURSE

Trade names:	**Presentation and strength:**
Cordarone X. Amiotach.	150mg/3ml ampoules.

Cautions: see SAMF.	
Amiodarone is incompatible with saline – mix with D5W only. Correct hypokalaemia – amiodarone may be ineffective or arrythmogenic. Use a large vein or central line – thrombophlebitis of peripheral veins is common. IV bolus is associated with hypotension – infusion is preferred method. Unsafe in porphyria (avoid sunlight exposure). Contra-indicated in 2nd and 3rd degree AV block. Caution with beta blockers and calcium antagonists.	

Loading dose:	
Adult: Acute resuscitation: slow IV bolus of 150mg to 300mg over 1 to 2 minutes. Otherwise slow IV infusion: 5mg/kg over 20 mins. Follow with a constant infusion.	**Paediatric:** Not for paediatric use.
Commonly for 70kg adult: 350 to 400mg diluted in 100 to 200ml D5W over 20 to 30 minutes.	**Neonatal:** Not for neonatal use.

Constant infusion:	
Adult dosage range: 0.4 to 0.7mg/kg/hr.	**Paediatric dosage range:** N/A
	Neonatal dosage range: N/A

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Dilute 600mg (4 amps or 12ml) into 50ml D5W = 12mg/ml.	**Mixture:** N/A
Rate: 28 to 50mg/hr = 2.3 to 4.2ml/hr.	**Rate:** N/A

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: Mainenance dose for 70kg patient after loading-dose infusion completed: Mix 600mg (4 amps or 12ml) in 500ml D5W.	**Mixture:** N/A
Rate: Run at 23 to 42ml/hr.	**Rate:** N/A

Additional notes:	

ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	GLYCERYL TRINITRATE INFUSION
REVIEWED:	25 JANUARY 2012

Trade names:	Presentation and strength:
Tridil, Nitrocine.	1mg/ml as 10ml ampoules. 50ml vials.
Cautions: See SAMF.	
Up to 80% of GTN is absorbed by PVC giving sets and plastic IV fluid bags. Use minimum volume administration tubing. See additional notes below.	
Loading dose:	
Adult: Boluses of 0.1mg may be given.	**Paediatric:** N/A
Commonly for 70kg adult: 0.1ml bolus (use insulin syringe) for acute hypertensive emergencies. Beware precipitous BP drop.	**Neonatal:** N/A
Constant infusion:	
Adult dosage range: Start at 25 to 50mcg/min. Maximum rate 200mcg/min.	**Paediatric dosage range:** 0.5 to 5mcg/kg/min.
	Neonatal dosage range: N/A.
Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Use solution of 50mg/50ml.	**Mixture:** <30kg: 3mg/kg in 50ml D5W.
Rate: Start at 1.5 to 3ml/hr. Maximum rate 12ml/hr. Increase by 1ml/hr every 5 mins according to clinical response.	**Rate:** 0.5 to 5ml/hr.
Alternative adult regime:	**Alternative paediatric regime:**
Mixture:	**Mixture:** If greater volumes required for larger children, can stay with above, or: >30kg: 3mg/kg in 100ml D5W.
Rate:	**Rate:** 1 to 10ml/hr.
Additional notes:	
Due to GTN absorption by PVC tubing, use clinical response (blood pressure) and not calculated dose to establish a dose that is appropriate for the patient. Slow or stop infusion if patient becomes hypotensive.	

ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	**SALBUTAMOL INFUSION – OBSTETRIC INDICATION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Ventolin	5mg in 5ml ampoules.

Cautions: See SAMF.	
Salbutamol is incompatible with ketamine, aminophylline and magnesium sulphate. Do not use in severe antepartum haemorrhage. Beware hypokalaemia. Use with extreme caution in heart disease.	

Loading dose:	
Adult: N/A.	**Paediatric:** N/A.
Commonly for 70kg adult: N/A.	**Neonatal:** N/A.

Constant infusion:	
Adult dosage range: 10 to 50mcg/min = 600mcg to 3mg/hr.	**Paediatric dosage range:** N/A.
	Neonatal dosage range: N/A.

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: Add 5mg into 50ml saline = 0.1mg/ml.	**Mixture:** N/A.
Rate: Start at 6ml/hr. Maximum rate 30ml/hr.	**Rate:** N/A.

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: Add 5mg into 200ml saline = 25mcg/ml.	**Mixture:** N/A.
Rate: Start at 24ml/hr. Maximum rate 120ml/hr.	**Rate:** N/A.

Additional notes:
Consider stopping infusion if maternal heart rate goes above 130 beats/min.

ADVANCED AIRWAY MANAGEMENT COURSE

TITLE:	**SALBUTAMOL INFUSION – RESPIRATORY INDICATION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	Presentation and strength:
Ventolin	5mg/5ml ampoule.

Cautions: see SAMF.	
Salbutamol is incompatible with ketamine, aminophylline and magnesium sulphate. Beware hypokalaemia.	

Loading dose:	
Adult: 4 to 5mcg/kg over 10 minutes.	**Paediatric:** 5 to 10mcg/kg/min for 1 hour. Then 1 to 2mcg/kg/min.
Commonly for 70kg adult: 350mcg (3.5ml) over 10 minutes.	**Neonatal:**

Constant infusion:	
Adult dosage range: Start at 5mcg/min = 300mcg/hr. Maximum dose 20mcg/min = 1.2mg/hr.	**Paediatric dosage range:** 0.3mg to 0.6mg/kg/hr for 1 hour. Then 0.06 to 0.12mg/kg/hr.
	Neonatal dosage range: As above.

Regime for 70kg adult:	Regime for paediatrics:
Mixture: 5mg in 50ml saline = 100mcg/ml.	**Mixture:** Dilute to 10mg/50ml saline = 0.2mg/ml.
Rate: Start at 3ml/hr. Maximum dose 12ml/hr.	**Rate:** 1.5 to 3ml/kg/hr for one hour. Then 0.3 to 0.6 ml/kg/hr.

Alternative adult regime:	Alternative paediatric regime:
Mixture: 50mg in 500ml = 10mcg/ml.	**Mixture:**
Rate: Run at 30 to 120ml/hr.	**Rate:**

Additional notes:

TITLE:	**INSULIN INFUSION (FAST-ACTING)**
REVIEWED:	**25 JANUARY 2012**

Trade names:	Presentation and strength:
Actrapid Humulin R.	100u/ml (1000u/10ml vials).

Cautions: see SAMF.	
Some insulin is absorbed by the PVC admin tubing – delivery may vary. Colloid facilitates better insulin delivery than normal saline. Insulin infusions in transit are inherently unsafe and should be discouraged, unless deemed essential by the referring or receiving doctor. See additional notes below.	

Loading dose:	
Adult: 10u IV (0.1ml).	**Paediatric:** IV 0.025 to 0.1u/kg prn.
Commonly for 70kg adult: As above.	**Neonatal:**

Constant infusion:	
Adult dosage range: 5 to 15u/hr.	**Paediatric dosage range:** 0.025 to 0.1u/kg/hr.
	Neonatal dosage range:

Regime for 70kg adult:	Regime for paediatrics:
Mixture: Dilute 50u (0.5ml) in 50ml colloid.	**Mixture:** 2.5u/kg in 50ml colloid.
Rate: Commence at 5 to 10ml/hr. Maximum 15ml/hr.	**Rate:** 0.5 to 2ml/hr.

Alternative adult regime:	Alternative paediatric regime:
Mixture: 100u (1ml) in 500ml colloid = 0.2 IU/ml.	**Mixture:**
Rate: Start at 25ml/hr. Maximum 75ml/hr.	**Rate:**

Additional notes:
In DKA, "infection hunt" (antibiotics), hypovolaemia, acid-base and electrolyte disturbances (esp. serum potassium) are far more important than bringing blood sugar levels under control, in the short-term.   Insulin treatment without controlled potassium supplementation can result in potentially lethal hypokalaemia, and should not be undertaken without access to arterial blood gas and electrolyte monitoring facilities.

Other:
In DKA, if decrease in blood sugar level (BSL) is less than 4mmol/l/hr, double infusion rate every hour until decrease in BSL is greater than 4mmol/l/hr, or until BSL is less than 15mmol/l. When decrease in BSL is greater than 4mmol/l/hr, maintain insulin infusion at current rate until BSL is less than 15mmol/l.

ADVANCED AIRWAY MANAGEMENT COURSE	
TITLE:	**MANNITOL INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	**Presentation and strength:**
Mannitol	12.5g/50ml (25% m/v) = 250mg/ml.

Cautions: see SAMF.
Must insert urinary catheter before administering mannitol.

Loading dose:	
Adult: N/A.	**Paediatric:** N/A.
Commonly for 70kg adult: N/A	**Neonatal:** N/A.

Constant infusion:	
Adult dosage range: 0.25 to 1g/kg given 6 to 8 hourly (GSH). 0.25 to 2g/kg given 6 to 8 hourly (SAMF).	**Paediatric dosage range:** As for adult.
	Neonatal dosage range: As for paediatric.
Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: 12.5g/50ml (25% m/v) = 250mg/ml.	**Mixture:** 12.5g/50ml (25% m/v) = 250mg/ml.
Rate: 70 to 280ml rapidly (30 minutes).	**Rate:** 1ml/kg to 4ml/kg.

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: 12.5g/50ml (25% m/v) = 250mg/ml.	**Mixture:** 12.5g/50ml (25% m/v) = 250mg/ml.
Rate: 70 to 560ml (SAMF).	**Rate:** 1ml/kg to 8ml/kg (SAMF).

Additional notes:
Osmotherapy is the second-best option, after CSF drainage, for controlling raised intracranial pressure. Indications for mannitol infusion are: - Deteriorating LOC.   - New onset focal signs.   - Suspected significantly raised ICP on clinical grounds (assuming an intracranial pressure monitor has not been placed).   Give rapidly (GSH). [SAMF says give over 30 to 60 minutes.] Dose can be repeated if the patient's condition improves, remains improved, or if condition deteriorates after an initial improvement.

Other:

TITLE:	**MAGNESIUM SULPHATE INFUSION**
REVIEWED:	**25 JANUARY 2012**

Trade names:	Presentation and strength:
Magnesium sulphate.	1g/2ml ampoules (500mg/ml).

Cautions: see SAMF.	
Indicated in all eclamptics and severe pre-eclamptics. Contra-indicated in myasthenia gravis and heart block. Magnesium toxicity – stop infusion when: • Patella reflex (knee-jerk) disappears. • Respiratory depression. • Bradycardia. Antidote = 10ml of 10% calcium chloride by slow IV injection – slowly and cautiously (reverses therapeutic effects of magnesium sulphate and patient may have convulsions).	

Loading dose:	
Adult: 4g loading dose over 5 to 10 minutes.	**Paediatric:** N/A.
Commonly for 70kg adult: 4g (8ml) in 200ml saline over 5 to 10 minutes by infusion pump or gravity feed.	**Neonatal:** N/A.

Constant infusion:	
Adult dosage range: 1 to 2g hourly by constant infusion. Start at 1g/hour.	**Paediatric dosage range:** N/A.
	Neonatal dosage range: N/A.

Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: 1g/50ml saline = 20mg/ml.	**Mixture:** N/A.
Rate: 50 to 100ml/hr.	**Rate:** N/A.

Alternative adult regime:	**Alternative paediatric regime:**
Mixture: 4g/200ml = 20mg/ml.	**Mixture:** N/A.
Rate: 50 to 100ml/hr.	**Rate:** N/A.

Additional notes:
Eclampsia and pre-eclampsia: The above is known as the Zuspan regimen and is used by GSH. The Pritchard regimen describes the same loading dose, followed by 10g IM (5g into each buttock). Then 5g IM every 4 hours (alternating buttocks). This is continued for 24 hours (limited by some to 12 hours). Suppression of labour: Use 6g loading dose and 2g/hr for suppression of labour where salbutamol contra-indicated or ineffective. Acute severe asthma: Adult: 1 to 2g IV over 20 minutes. Paediatric: 40mg/kg to a maximum of 2g.

ADVANCED AIRWAY MANAGEMENT COURSE	
TITLE:	**ATROPINE INFUSION – ORGANOPHOSPHATE POISONING**
REVIEWED:	**25 JANUARY 2012**

ATROPINE SULPHATE INFUSION – ORGANOPHOSPHATE POISONING:

Trade names:	Presentation and strength:
Atropine sulphate.	0.5mg/ml and 1mg/ml ampoules.
Cautions: see SAMF.	
Organophosphate poisoning – avoid aminoglycosides, suxamethonium, phenothiazines and theophylline. Control convulsions with a benzodiazepine.	
Loading dose:	
Adult: SAMF: Initial test dose of 1mg in adults. Followed by 0.05mg/kg every 15 minutes until full atropinization is achieved. GSH:1-2mg IV initial dose. Repeat and double dose every 5 minutes until atropinised. Give initial dose IM while acquiring IV access if necessary.	**Paediatric:** SAMF: Initial test dose of 0.01mg/kg IV bolus. Followed by 0.05mg/kg every 15 minutes until full atropinization is achieved. GSH: (Unclear history or minimal symptoms: 0.01 mg/kg and then proceed with a doubling dose every 5 minutes titrated against response; if the child is clearly symptomatic or confirmed history start with 0.03 mg/kg and titrate using a doubling dose)
Commonly for 70kg adult: 1mg IV bolus test dose. Followed by 2 to 4mg every 15 minutes. Dose varies widely according to level of poisoning – if atropinised with only one or two doses then the poisoning is likely very mild. Can require very large doses.	**Neonatal:** N/A.
Constant infusion:	
Adult dosage range: 0.02 to 0.08mg/kg/hr constant infusion once full atropinization has been achieved. Higher doses may be required – can require 10-20mg/hr in severe poisonings.	**Paediatric dosage range:** Range from 0.01 to 0.03 mg/kg (or as for adult) [Commence at 10% of total loading dose per hour and titrate to keep patient atropinised.]
	Neonatal dosage range: N/A.
Regime for 70kg adult:	**Regime for paediatrics:**
Mixture: 10mg/50ml = 40mg/200ml = 0.2mg/ml.	**Mixture:** 1mg/50ml = 4mg/200ml = 20mcg/ml.
Rate: Start at 0.02mg/kg/hr = 1.4 to 5.6mg/hr. 7 to 28ml/hr.	**Rate:** 1 to 4ml/kg/hr.
Alternative adult regime:	**Alternative paediatric regime:**
Mixture: 40mg/200ml = 0.2mg/ml.	**Mixture:** 1mg/50ml = 4mg/200ml = 20mcg/ml.

Rate:	Rate:
Commence at 10% of total loading dose per hour and titrate to keep patient atropinised. See next… 10 to 20mg/hr = 50 to 100ml/hr.	0.5ml/kg/hr to 1.5ml/kg/hr titrated upwards to effect.

Additional notes:
The primary end point of atropinisation is drying of bronchial secretions (manifest as crepitations and wheeze).   Pupil size can only be used as an end point if miosis is present on admission.   Also: the dilation of pupils often lags behind other signs of atropinsiation. Generally, aim to keep heart rate above 100 bpm.   Dosage requirements are extremely variable may need to be doubled or quadrupled – double or quadruple the concentration rather than the rate. If fluid volumes are too great, double or quadruple the concentration, and administer half to one quarter of the rate.
Other:
See Eddleston et al for further guidance and for an example of an atropine monitoring sheet. Good notes at http://curriculum.toxicology.wikispaces.net/ ***Acknowledgements to Dr Ross Hofmeyr for contributing to this protocol.***

REFERENCES FOR ABOVE DRUG INFUSION PROTOCOLS:

South African Medicines Formulary.
9th edition.
Division of Clinical Pharmacology.
University of Cape Town.

Drug Doses.
14th edition.
Frank Shann.
Intensive Care Unit.
Royal Children's Hospital.
Pareville, Victoria, Australia.

Royal Flying Doctor Service.
Western Operations.
Clinical Manual.
Part 2: Drug Infusion Guidelines.
Version 5, June 2008.

Drugs in Anaesthesia and Intensive Care.
3rd edition.
Martin Susada and Susan Smith.
Oxford Medical Publications.

Oxford Handbook of Anaesthesia.
2nd edition.
Keith Allman and Iain Wilson.
Oxford Medical Publications.

1. BVM resuscitator, with reservoir bag and range of face mask sizes.
2. Oropharangeal airways – range of sizes.
3. Endotracheal tubes – range of sizes.
4. Suction unit with range of suction catheters (rigid and soft).
5. Oxygen supply.
6. Laryngoscope with range of blades, spare bulbs and batteries.
7. Gum elastic bougies and rubber coated malleable stylets.
8. LMAs – standard, Proseal®-equivalent, I-LMA in a range of sizes.
9. King Airway or Laryngeal Tube – range of sizes.
10. i-Gel supraglottic airway device – range of sizes.
11. Needle cricothryroidotomy and/or surgical cricothyroidotomy sets.
12. Sanders injector or other appropriate device for jet insufflation.
13. Appropriate drugs including: Atropine, Adrenalin, Midazolam, Flumazenil, neuromuscular blockade, etc.
14. ECG, NIBP, pulse oximetry and end-tidal capnography.
15. Acceptable method of securing endotracheal tube.

Endotracheal intubation should *not* be considered in the absence of continuous end-tidal capnography, and RSI should *not* be considered without the presence of at least two alternative backup 'rescue' airway devices.

A trained assistant should be available to assist the advanced airway practitioner with all aspects of patient management, but especially with advanced airway management.

APPENDIX 3: LIST OF SKILLS AN ADVANCED "AIRWAY TECHNICIAN" SHOULD BE ABLE TO PERFORM

- One-hand face-mask seal
- Two-handed face-mask seal
- Bag-valve-mask ventilation
- Insert OPA
- Insert NPA
- Cricoid pressure
- OELM – "talking the cords into view"
- Use of malleable stylet
- Gum elastic bougie – railroading an ETT
- Steerable introducer
- Use regular ETT
- Use Parker Flexi-tip ETT
- Reinforced ETT
- Double-lumen ETT
- Regular laryngoscopy
- Nasal intubation
- Throat pack insertion
- NGT/OGT insertion*
- Oral intubation
- Magill forceps use
- LMA insertion – three techniques: ballpoint pen, holding by tip, 'reverse-plop'
- Proseal insertion, nasogastric drainage.
- King Laryngeal Tube Airway insertion
- Combitube insertion*
- I-LMA insertion.
- Airtraq intubation.
- Fibre-optic intubation*
- Video laryngoscopy.
- Needle cricothyroidotomy.
- Jet insufflation – Sanders injector (trans-tracheal jet insufflation).
- Jet insufflation – prehospital devices.
- Surgical cricothyroidotomy and tracheostomy, cuffed trachy tube, cuffed ETT.
- Retrograde intubation.

ASA DIFFICULT AIRWAY ALGORITHM

AMERICAN SOCIETY OF ANESTHESIOLOGISTS

DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
   A. Difficult Ventilation
   B. Difficult Intubation
   C. Difficulty with Patient Cooperation or Consent
   D. Difficult Tracheostomy

2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management

3. Consider the relative merits and feasibility of basic management choices:

   A. Awake Intubation — vs — Intubation Attempts After Induction of General Anesthesia

   B. Non-Invasive Technique for Initial Approach to Intubation — vs — Invasive Technique for Initial Approach to Intubation

   C. Preservation of Spontaneous Ventilation — vs — Ablation of Spontaneous Ventilation
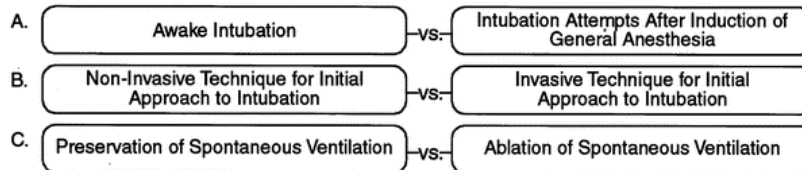
4. Develop primary and alternative strategies:

A. AWAKE INTUBATION

Airway Approached by Non-Invasive Intubation | Invasive Airway Access(b)*

Succeed* | FAIL

Cancel Case | Consider Feasibility of Other Options(a) | Invasive Airway Access(b)*

B. INTUBATION ATTEMPTS AFTER INDUCTION OF GENERAL ANESTHESIA

Initial Intubation Attempts Successful* | Initial Intubation Attempts UNSUCCESSFUL

FROM THIS POINT ONWARDS CONSIDER:
1. Calling for Help
2. Returning to Spontaneous Ventilation
3. Awakening the Patient

FACE MASK VENTILATION ADEQUATE | FACE MASK VENTILATION NOT ADEQUATE

CONSIDER / ATTEMPT LMA

LMA ADEQUATE* | LMA NOT ADEQUATE OR NOT FEASIBLE

NON-EMERGENCY PATHWAY
Ventilation Adequate, Intubation Unsuccessful

EMERGENCY PATHWAY
Ventilation Not Adequate, Intubation Unsuccessful

Alternative Approaches to Intubation(c)

IF BOTH FACE MASK AND LMA VENTILATION BECOME INADEQUATE

Call for Help

Emergency Non-Invasive Airway Ventilation(e)

Successful Intubation* | FAIL After Multiple Attempts

Successful Ventilation* | FAIL

Invasive Airway Access(b)* | Consider Feasibility of Other Options(a) | Awaken Patient(d)

Emergency Invasive Airway Access(b)*

*Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO_2

a. Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

b. Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy.

c. Alternative non-invasive approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

d. Consider re-preparation of the patient for awake intubation or canceling surgery.

e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal jet ventilation.

RESUSCITATION COUNCIL OF SA ADVANCED AIRWAY MANAGEMENT ALGORITHM

Advanced Airway Management Algorithm

(Adult and Child)

```
┌─────────────────────────────┐
│      Apnoeic Patient        │
│            or               │
│    Unprotected Airway       │
└─────────────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│  Ensure Anatomic Alignment  │
│ (Beware of Cervical Spine   │
│           Injury)           │
└─────────────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│    Apply Cricoid Pressure   │
└─────────────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│  Ensure Initial Oxygenation │
└─────────────────────────────┘
              │
              ▼
      ⬡ Secure Airway ⬡
   (Depending on Skill and
     Equipment Available)
```

```
┌──────────────────┐   ┌──────────────────┐   ┌──────────────────────┐
│ Laryngeal Mask   │   │     Tracheal     │   │ Combitube/Laryngeal  │
│    Airway        │   │    Intubation    │   │        Tube          │
│  (alternative)   │   │                  │   │    (alternative)     │
└──────────────────┘   └──────────────────┘   └──────────────────────┘
```

```
      ⬡ Failure to secure ⬡
        Airway and adequate
             ventilation
              │
              ▼
┌─────────────────────────────┐
│ Return to Basic Airway      │
│        Technique            │
└─────────────────────────────┘
              │
              ▼
       ⬡ Unable to ⬡
          ventilate?
              │
              ▼
┌─────────────────────────────┐
│  Consider Surgical Airway   │
└─────────────────────────────┘
```

```
┌──────────────────────────┐           ┌──────────────────────────┐
│ Needle Cricothyroidotomy │ ────────► │ Surgical Cricothyroidotomy│
└──────────────────────────┘           └──────────────────────────┘
                    ┌──────────────────┐
                    │    VENTILATE     │
                    └──────────────────┘
```

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DAS Difficult intubation guidelines – overview

2015

Plan A:
Facemask ventilation and tracheal intubation

Laryngoscopy — Succeed → Tracheal intubation

Failed intubation

Plan B:
Maintaining oxygenation:
SAD insertion

Supraglottic Airway Device — Succeed →

STOP AND THINK
Options (consider risks and benefits):
1. Wake the patient up
2. Intubate trachea via the SAD
3. Proceed without intubating the trachea
4. Tracheostomy or cricothyroidotomy

Failed SAD ventilation

Plan C:
Facemask ventilation

Final attempt at face mask ventilation — Succeed → Wake the patient up

CICO

Plan D:
Emergency front of neck access

Cricothyroidotomy

This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

Management of unanticipated difficult tracheal intubation in adults

2015

Plan A: Facemask ventilation and tracheal intubation

Optimise head and neck position
Preoxygenate
Adequate neuromuscular blockade
Direct / Video Laryngoscopy (maximum 3+1 attempts)
External laryngeal manipulation
Bougie
Remove cricoid pressure
Maintain oxygenation and anaesthesia

If in difficulty → call for help

Succeed → Confirm tracheal intubation with capnography

Declare failed intubation

Plan B: Maintaining oxygenation: SAD insertion

2nd generation device recommended
Change device or size (maximum 3 attempts)
Oxygenate and ventilate

Succeed →

STOP AND THINK
Options (consider risks and benefits):
1. Wake the patient up
2. Intubate trachea via the SAD
3. Proceed without intubating the trachea
4. Tracheostomy or cricothyroidotomy

Declare failed SAD ventilation

Plan C: Facemask ventilation

If facemask ventilation impossible, paralyse
Final attempt at facemask ventilation
Use 2 person technique and adjuncts

Succeed → Wake the patient up

Declare CICO

Plan D: Emergency front of neck access

Scalpel cricothyroidotomy

Post-operative care and follow up
• Formulate immediate airway management plan
• Monitor for complications
• Complete airway alert form
• Explain to the patient in person and in writing
• Send written report to GP and local database

This flowchart forms part of the DAS Guidelines for unanticipated difficult intubation in adults 2015 and should be used in conjunction with the text.

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**DAS
2015**

Failed intubation, failed oxygenation in the paralysed, anaesthetised patient

CALL FOR HELP

Continue 100% O_2
Declare CICO

Plan D: Emergency front of neck access

Continue to give oxygen via upper airway
Ensure neuromuscular blockade
Position patient to extend neck

Scalpel cricothyroidotomy

Equipment: 1. Scalpel (number 10 blade)
2. Bougie
3. Tube (cuffed 6.0mm ID)

Laryngeal handshake to identify cricothyroid membrane

Palpable cricothyroid membrane
Transverse stab incision through cricothyroid membrane
Turn blade through 90° (sharp edge caudally)
Slide coude tip of bougie along blade into trachea
Railroad lubricated 6.0mm cuffed tracheal tube into trachea
Ventilate, inflate cuff and confirm position with capnography
Secure tube

Impalpable cricothyroid membrane
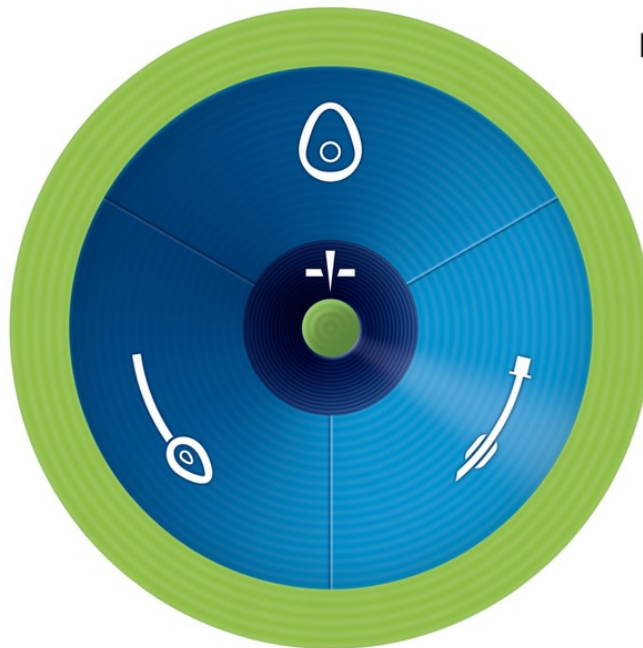Make an 8-10cm vertical skin incision, caudad to cephalad
Use blunt dissection with fingers of both hands to separate tissues
Identify and stabilise the larynx
Proceed with technique for palpable cricothyroid membrane as above

Post-operative care and follow up
- Postpone surgery unless immediately life threatening
- Urgent surgical review of cricothyroidotomy site
- Document and follow up as in main flow chart

THE VORTEX

FOR EACH LIFELINE CONSIDER:

MANIPULATIONS:
- HEAD & NECK
- LARYNX
- DEVICE

ADJUNCTS

SIZE / TYPE

SUCTION / O₂ FLOW

MUSCLE TONE

MAXIMUM THREE ATTEMPTS AT EACH LIFELINE (UNLESS GAMECHANGER)
AT LEAST ONE ATTEMPT SHOULD BE BY MOST EXPERIENCED CLINICIAN
CICO STATUS ESCALATES WITH UNSUCCESSFUL BEST EFFORT AT ANY LIFELINE

The Vortex Airway Management Checklist

Prepare Interventions → **Prevent Hypoxia** → **Promote Teamwork**

Intravenous Access
- [ ] Adequate
- [ ] Running

Drugs: Agent Dose Labelled

Intubation:
- [ ] Induction
- [ ] Paralysis
- [ ] Adjuncts
- [ ] Emergency

Post Intubation:
- [ ] Anaesthesia
- [ ] Analgesia
- [ ] Paralysis

Monitoring: Confirm Optimise
- [ ] ETCO₂
- [ ] SaO₂
- [ ] BP
- [ ] ECG
- [ ] Alarms

Safe Apnoea Time

PreOx:
- [ ] 100% O₂ Connected/Flowing
- [ ] Optimise FRC
- [ ] PreOx Complete

ApOx:
- [ ] O₂ Connected/Flowing

ReOx:
- [ ] BVM/Ventilator
- [ ] Ventilator Settings
- [ ] Consider PEEP

Vortex Approach
- [ ] Position
- [ ] Suction

Face Mask:
- [ ] Adjuncts
- [ ] FM: size/type

Supraglottic Airway:
- [ ] SGA: size/type

Endotracheal Tube:
- [ ] Adjuncts
- [ ] Laryngoscope: size/type (incl VL), functioning
- [ ] ETT: size/type
- [ ] Syringe/Cuff Test
- [ ] Tape/Ties

Emergency Surgical Airway:
- [ ] ESA Kit

Additional Staff:
- [ ] Supervision
- [ ] Help: Senior/Anaesthetics/ENT

Roles: Capable Briefed
- [ ] Team Coordinator
- [ ] Vortex
- [ ] Airway Operator
- [ ] Airway Assistant
- [ ] Cricoid
- [ ] Manual In-Line Stabilisation
- [ ] Drugs
- [ ] Monitors/Time
- [ ] Emergency Surgical Airway

Plan: Discussed
- [ ] Anticipated Difficulties: FMV/ETT/SGA/ESA
- [ ] Vortex Sequence
- [ ] Vortex Optimisations
- [ ] Green Zone Options
- [ ] Questions/Concerns?

REFERENCES

1. Davis, D.P., Hoyt, D.B., Ochs, M., et al., *The effect of paramedic rapid sequence intubation on outcome in patients with severe traumatic brain injury.* J Trauma, 2003. **54**(3): p. 444-53.

2. Sloane, C., Vilke, G.M., Chan, T.C., et al., *Rapid sequence intubation in the field versus hospital in trauma patients.* J Emerg Med, 2000. **19**(3): p. 259-64.

3. Murray, J.A., Demetriades, D., Berne, T.V., et al., *Prehospital intubation in patients with severe head injury.* J Trauma, 2000. **49**(6): p. 1065-70.

4. Bochicchio, G.V. and Scalea, T.M., *Is field intubation useful?* Curr Opin Crit Care, 2003. **9**(6): p. 524-9.

5. Stiell, I.G., Nesbitt, L.P., Pickett, W., et al., *The OPALS Major Trauma Study: impact of advanced life-support on survival and morbidity.* CMAJ, 2008. **178**(9): p. 1141-52.

6. Bowles, T.M., Freshwater-Turner, D.A., Janssen, D.J., et al., *Out-of-theatre tracheal intubation: prospective multicentre study of clinical practice and adverse events.* Br J Anaesth, 2011. **107**(5): p. 687-92.

7. Spaite, D.W. and Criss, E.A., *Out-of-hospital rapid sequence intubation: are we helping or hurting our patients?* Ann Emerg Med, 2003. **42**(6): p. 729-30.

8. Wang, H.E. and Yealy, D.M., *Out-of-hospital rapid sequence intubation: is this really the "success" we envisioned?* Ann Emerg Med, 2002. **40**(2): p. 168-71.

9. Katz, S.H. and Falk, J.L., *Misplaced endotracheal tubes by paramedics in an urban emergency medical services system.* Ann Emerg Med, 2001. **37**(1): p. 32-7.

10. Wang, H.E., O'Connor, R.E., Schnyder, M.E., et al., *Patient status and time to intubation in the assessment of prehospital intubation performance.* Prehosp Emerg Care, 2001. **5**(1): p. 10-8.

11. Colwell, C.B., McVaney, K.E., Haukoos, J.S., et al., *An evaluation of out-of-hospital advanced airway management in an urban setting.* Acad Emerg Med, 2005. **12**(5): p. 417-22.

12. Brown, J. and Thomas, F., *What happens with failed blind nasal tracheal intubations?* Air Med J, 2001. **20**(2): p. 13-6.

13. Vilke, G.M., Steen, P.J., Smith, A.M., et al., *Out-of-hospital pediatric intubation by paramedics: the San Diego experience.* J Emerg Med, 2002. **22**(1): p. 71-4.

14. Silvestri, S., Ralls, G.A., Krauss, B., et al., *The effectiveness of out-of-hospital use of continuous end-tidal carbon dioxide monitoring on the rate of unrecognized misplaced intubation within a regional emergency medical services system.* Ann Emerg Med, 2005. **45**(5): p. 497-503.

15. Eckstein, M., Stratton, S.J. and Chan, L.S., *Cardiac Arrest Resuscitation Evaluation in Los Angeles: CARE-LA.* Ann Emerg Med, 2005. **45**(5): p. 504-9.

16. Stockinger, Z.T. and McSwain, N.E., Jr., *Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation.* J Trauma, 2004. **56**(3): p. 531-6.

17. Liberman, M., Mulder, D. and Sampalis, J., *Advanced or basic life support for trauma: meta-analysis and critical review of the literature.* J Trauma, 2000. **49**(4): p. 584-99.

18. Cooper, A., DiScala, C., Foltin, G., et al., *Prehospital endotracheal intubation for severe head injury in children: a reappraisal.* Semin Pediatr Surg, 2001. **10**(1): p. 3-6.

19. Lema, M., *American Society of Anaesthesiologists' Newsletter.* 2004. **64**(March).

20. Criss, E.A., *Where we fail: research reveals our weaknesses in CPR delivery.* JEMS, 2005. **30**(12): p. 5-8.

21. Bledsoe, B.E. and Gandy, W.E., *The disappearing endotracheal tube: historic skill threatened by lack of practice & new devices.* JEMS, 2009. **34**(3): p. 88-99.

22. Clarke, T., Deakin, C., Blancke, W., et al., *Improving UK trauma care: the NCEPOD trauma report.* Anaesthesia, 2008. **63**(11): p. 1255; author reply 1255-6.

23. Konrad, C., Schupfer, G., Wietlisbach, M., et al., *Learning manual skills in anesthesiology: Is there a recommended number of cases for anesthetic procedures?* Anesth Analg, 1998. **86**(3): p. 635-9.

24. Cormack, R.S. and Smith, J.E., *Training requirements for tracheal intubation* in *Anaesthetic Research Society* 2006: Dundee.

25. West, M.R., Jonas, M.M., Adams, A.P., et al., *A new tracheal tube for difficult intubation.* Br J Anaesth, 1996. **76**(5): p. 673-9.

26. Findlay, G., Martin, I.C., Carter, S., et al., *Trauma: Who Cares*. 2007, NCEPOD.

27. O'Connor, R.E., *Drug-Assisted Intubation in the Prehospital Setting Position Statement of the National Association of Emergency Physicians.* Prehospital Emergency Care, 2006. **10**(2): p. 260-260.

28. Goldmann, K. and Ferson, D.Z., *Education and training in airway management.* Best Pract Res Clin Anaesthesiol, 2005. **19**(4): p. 717-32.

29. Kheterpal, S., Han, R., Tremper, K.K., et al., *Incidence and predictors of difficult and impossible mask ventilation.* Anesthesiology, 2006. **105**(5): p. 885-91.

30. Maiman, D.J., Sances, A., Jr., Myklebust, J.B., et al., *Compression injuries of the cervical spine: a biomechanical analysis.* Neurosurgery, 1983. **13**(3): p. 254-60.

31. Okubo, M., Gibo, K., Hagiwara, Y., et al., *The effectiveness of rapid sequence intubation (RSI) versus non-RSI in emergency department: an analysis of multicenter prospective observational study.* Int J Emerg Med, 2017. **10**(1): p. 1.

32. Greenland, K.B., Edwards, M.J. and Hutton, N.J., *External auditory meatus-sternal notch relationship in adults in the sniffing position: a magnetic resonance imaging study.* Br J Anaesth, 2010. **104**(2): p. 268-9.

33. Greenland, K.B., Edwards, M.J., Hutton, N.J., et al., *Changes in airway configuration with different head and neck positions using magnetic resonance imaging of normal airways: a new concept with possible clinical applications.* Br J Anaesth, 2010. **105**(5): p. 683-90.

34. Sitzwohl, C., Langheinrich, A., Schober, A., et al., *Endobronchial intubation detected by insertion depth of endotracheal tube, bilateral auscultation, or observation of chest movements: randomised trial.* BMJ, 2010. **341**: p. c5943.

35. Baillard, C., Fosse, J.P., Sebbane, M., et al., *Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients.* Am J Respir Crit Care Med, 2006. **174**(2): p. 171-7.

36. Lane, S., Saunders, D., Schofield, A., et al., *A prospective, randomised controlled trial comparing the efficacy of pre-oxygenation in the 20 degrees head-up vs supine position.* Anaesthesia, 2005. **60**(11): p. 1064-7.

37. Weingart, S.D. and Levitan, R.M., *Preoxygenation and prevention of desaturation during emergency airway management.* Ann Emerg Med, 2012. **59**(3): p. 165-75 e1.

38. Nielsen, N.D., Kjaergaard, B., Koefoed-Nielsen, J., et al., *Apneic oxygenation combined with extracorporeal arteriovenous carbon dioxide removal provides sufficient gas exchange in experimental lung injury.* Asaio j, 2008. **54**(4): p. 401-5.

39. Taha, S.K., Siddik-Sayyid, S.M., El-Khatib, M.F., et al., *Nasopharyngeal oxygen insufflation following pre-oxygenation using the four deep breath technique.* Anaesthesia, 2006. **61**(5): p. 427-30.

40. Ramachandran, S.K., Cosnowski, A., Shanks, A., et al., *Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration.* J Clin Anesth, 2010. **22**(3): p. 164-8.

41. Patel, A. and Nouraei, S.A., *Transnasal Humidified Rapid-Insufflation Ventilatory Exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways.* Anaesthesia, 2015. **70**(3): p. 323-9.

42.   Gabbott, D.A., *The effect of single-handed cricoid pressure on neck movement after applying manual in-line stabilisation.* Anaesthesia, 1997. **52**(6): p. 586-8.

43.   Donaldson, W.F., 3rd, Towers, J.D., Doctor, A., et al., *A methodology to evaluate motion of the unstable spine during intubation techniques.* Spine (Phila Pa 1976), 1993. **18**(14): p. 2020-3.

44.   Criswell, J.C., Parr, M.J. and Nolan, J.P., *Emergency airway management in patients with cervical spine injuries.* Anaesthesia, 1994. **49**(10): p. 900-3.

45.   Hartsilver, E.L. and Vanner, R.G., *Airway obstruction with cricoid pressure.* Anaesthesia, 2000. **55**(3): p. 208-11.

46.   Cook, T.M., Woodall, N., Harper, J., et al., *Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 2: intensive care and emergency departments.* Br J Anaesth, 2011. **106**(5): p. 632-42.

47.   Cook, T.M., Woodall, N., Frerk, C., et al., *Major complications of airway management in the UK: results of the Fourth National Audit Project of the Royal College of Anaesthetists and the Difficult Airway Society. Part 1: Anaesthesia.* Br J Anaesth, 2011. **106**(5): p. 617-31.

48.   O'Leary, J.J., Pollard, B.J. and Ryan, M.J., *A method of detecting oesophageal intubation or confirming tracheal intubation.* Anaesth Intensive Care, 1988. **16**(3): p. 299-301.

49.   Nunn, J.F., *The oesophageal detector device.* Anaesthesia, 1988. **43**(9): p. 804.

50.   Wee, M.Y., *The oesophageal detector device. Assessment of a new method to distinguish oesophageal from tracheal intubation.* Anaesthesia, 1988. **43**(1): p. 27-9.

51.   Haynes, S.R. and Morton, N.S., *Use of the oesophageal detector device in children under one year of age.* Anaesthesia, 1990. **45**(12): p. 1067-9.

52.   Lang, D.J., Wafai, Y., Salem, M.R., et al., *Efficacy of the self-inflating bulb in confirming tracheal intubation in the morbidly obese.* Anesthesiology, 1996. **85**(2): p. 246-53.

53.   Tanigawa, K., Takeda, T., Goto, E., et al., *The efficacy of esophageal detector devices in verifying tracheal tube placement: a randomized cross-over study of out-of-hospital cardiac arrest patients.* Anesth Analg, 2001. **92**(2): p. 375-8.

54.   Woodall, N., Frerk, C. and Cook, T.M., *Can we make airway management (even) safer?--lessons from national audit.* Anaesthesia, 2011. **66 Suppl 2**: p. 27-33.

55.   Matera, P., *The truth about ET tube movement continued.* JEMS, 2004. **29**(3): p. 52-9.

56.   Woodall, N.M. and Cook, T.M., *National census of airway management techniques used for anaesthesia in the UK: first phase of the Fourth National Audit Project at the Royal College of Anaesthetists.* Br J Anaesth, 2011. **106**(2): p. 266-71.

57.   Benumof, J., *Airway Management: Principles and Practices.* 1995, Philadelphia: Mosby, Inc.

58.   Parmet, J.L., Colonna-Romano, P., Horrow, J.C., et al., *The laryngeal mask airway reliably provides rescue ventilation in cases of unanticipated difficult tracheal intubation along with difficult mask ventilation.* Anesth Analg, 1998. **87**(3): p. 661-5.

59.   Frerk, C., Mitchell, V.S., McNarry, A.F., et al., *Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults.* Br J Anaesth, 2015.

60.   Bishop, D., Farina, Z. and Wise, R., *Difficult airways: a reliable "Plan B".* Southern African Journal of Anaesthesia and Analgesia, 2011. **17**(2): p. 186-189.

61.   Asai, T. and Morris, S., *The role of the laryngeal mask for failed tracheal intubation in the patient with a "full stomach".* Anesth Analg, 1994. **78**(4): p. 817-9.

62.   Ferson, D.Z., Rosenblatt, W.H., Johansen, M.J., et al., *Use of the intubating LMA-Fastrach in 254 patients with difficult-to-manage airways.* Anesthesiology, 2001. **95**(5): p. 1175-81.

63. Cote, J., *The difficult paediatric airway.* Southern African Journal of Anaesthesia and Analgesia, 2012. **18**(5): p. 230-238.

64. Sheridan, R.L., *Uncuffed endotracheal tubes should not be used in seriously burned children.* Pediatr Crit Care Med, 2006. **7**(3): p. 258-9.

65. Weber, T., Salvi, N., Orliaguet, G., et al., *Cuffed vs non-cuffed endotracheal tubes for pediatric anesthesia.* Paediatr Anaesth, 2009. **19 Suppl 1**: p. 46-54.

66. Shih, M.H., Chung, C.Y., Su, B.C., et al., *Accuracy of a new body length-based formula for predicting tracheal tube size in Chinese children.* Chang Gung Med J, 2008. **31**(3): p. 276-80.

67. Motlang, *Cuffed endotracheal tubes in paediatrics.* Southern African Journal of Anaesthesia and Analgesia, 2012. **18**(5): p. 263-265.

68. Krishna, R. and TV, S., *Airway management in an infant with congenital trismus: the role of retrograde intubation.* Southern African Journal of Anaesthesia and Analgesia, 2012. **18**(5): p. 267-269.

69. Kuczkowski, K.M., Reisner, L.S. and Benumof, J.L., *Airway problems and new solutions for the obstetric patient.* J Clin Anesth, 2003. **15**(7): p. 552-63.

70. Halaseh, B.K., Sukkar, Z.F., Hassan, L.H., et al., *The use of ProSeal laryngeal mask airway in caesarean section--experience in 3000 cases.* Anaesth Intensive Care, 2010. **38**(6): p. 1023-8.

71. Douglas, M.J. and Preston, R.L., *The obstetric airway: things are seldom as they seem.* Can J Anaesth, 2011. **58**(6): p. 494-8.

72. McKeen, D.M., George, R.B., O'Connell, C.M., et al., *Difficult and failed intubation: Incident rates and maternal, obstetrical, and anesthetic predictors.* Can J Anaesth, 2011. **58**(6): p. 514-24.

73. Samsoon, G.L. and Young, J.R., *Difficult tracheal intubation: a retrospective study.* Anaesthesia, 1987. **42**(5): p. 487-90.

74. Pilkington, S., Carli, F., Dakin, M.J., et al., *Increase in Mallampati score during pregnancy.* Br J Anaesth, 1995. **74**(6): p. 638-42.

75. Kodali, B.S., Chandrasekhar, S., Bulich, L.N., et al., *Airway changes during labor and delivery.* Anesthesiology, 2008. **108**(3): p. 357-62.

76. Hood, D.D. and Dewan, D.M., *Anesthetic and obstetric outcome in morbidly obese parturients.* Anesthesiology, 1993. **79**(6): p. 1210-8.

77. Baraka, A.S., Taha, S.K., Aouad, M.T., et al., *Preoxygenation: comparison of maximal breathing and tidal volume breathing techniques.* Anesthesiology, 1999. **91**(3): p. 612-6.

78. Chiron, B., Laffon, M., Ferrandiere, M., et al., *Standard preoxygenation technique versus two rapid techniques in pregnant patients.* Int J Obstet Anesth, 2004. **13**(1): p. 11-4.

79. Tan, P.C. and Dennis, A.T., *High-flow humidified nasal pre-oxygenation in pregnant women.* Anaesthesia, 2016. **71**(7): p. 851-2.

80. Werman, H.A., Schwegman, D. and Gerard, J.P., *The effect of etomidate on airway management practices of an air medical transport service.* Prehosp Emerg Care, 2004. **8**(2): p. 185-90.

81. Swanson, E.R., Fosnocht, D.E. and Jensen, S.C., *Comparison of etomidate and midazolam for prehospital rapid-sequence intubation.* Prehosp Emerg Care, 2004. **8**(3): p. 273-9.

82. Morris, C. and McAllister, C., *Etomidate for emergency anaesthesia; mad, bad and dangerous to know?* Anaesthesia, 2005. **60**(8): p. 737-40.

83. Himmelseher, S. and Durieux, M.E., *Revising a dogma: ketamine for patients with neurological injury?* Anesth Analg, 2005. **101**(2): p. 524-34, table of contents.

84. Sehdev, R.S., Symmons, D.A. and Kindl, K., *Ketamine for rapid sequence induction in patients with head injury in the emergency department.* Emerg Med Australas, 2006. **18**(1): p. 37-44.

85.   Adnet, F., Le Moyec, L., Smith, C.E., et al., *Stability of succinylcholine solutions stored at room temperature studied by nuclear magnetic resonance spectroscopy.* Emerg Med J, 2007. **24**(3): p. 168-9.

86.   Benumof, J.L., Dagg, R. and Benumof, R., *Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine.* Anesthesiology, 1997. **87**(4): p. 979-82.

87.   Sorensen, M.K., Bretlau, C., Gatke, M.R., et al., *Rapid sequence induction and intubation with rocuronium-sugammadex compared with succinylcholine: a randomized trial.* Br J Anaesth, 2012. **108**(4): p. 682-9.