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# Update in Anaesthesia

Education for anaesthetists worldwide

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The Journal of the World Federation of Societies of Anaesthesiologists

Update in
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# Editorial

## The Lancet Commission on Global Surgery and Anaesthesia

Dr Iain Wilson, Consultant Anaesthetist and Commissioner, Exeter, UK

In the United Kingdom today, access to skilled surgical and anaesthetic care is considered as a basic component of a highly functional healthcare system. UK anaesthetists deliver much of this care directly, equipped with a modern range of drugs and equipment and funded by a range of payment systems. Safety is recognised as a key concern for patients, and the UK's National Health Service is improving all the time in the variety of surgical procedures available and in its ability to get patients safely through them.

However, in low and middle income countries (LMIC) an estimated two billion people worldwide are without adequate access to surgical consultation, investigation or treatment. The reasons are often obvious – low numbers of trained surgeons and anaesthetists for the population, chronic under-resourcing of healthcare, lack of basic drugs and equipment and inadequate healthcare facilities. Affordability is a major issue for patients, with many being without insurance or free national healthcare systems.

Although the challenge of healthcare delivery in LMIC has been recognised for many years, the specific issues regarding the provision of surgery and anaesthesia have rarely received the recognition they deserve at the local hospital, healthcare planning, political or international level. It is recognised that many of the common diseases (HIV, pneumonia, malaria etc) that are considered major killers in LMIC have received huge publicity and funding, resulting in major improvements in care. Other diseases such as injury, cancer and congenital conditions are now recognised as causing substantial numbers of deaths and morbidity and are major concerns. Surgery is a key component in therapy and anaesthesia yet despite this, apart from Caesarean section, surgery is rarely promoted even at WHO level. This is a major catastrophe for patients, resulting in many unnecessary deaths and much suffering. Much of the surgery required is basic, inexpensive and extraordinarily effective in transforming lives – cataract, hernia, club feet, cleft palate and fracture care, for example.

Many surgeons and anaesthetists have published academic articles describing the deficiencies in care, so the issues are known, but there has never been an international united, high-profile campaign to portray the issues involved to Ministries of Health, governments, major foundations, healthcare planners and training institutions.[1]

The internationally renowned medical journal The Lancet has run a series of Commissions on issues such as the Health Effects of Climate Change, the Future of Medical Education, Antibiotic Resistance and others.[2] Commissions are essentially peer reviewed 25,000 word reviews of a major healthcare topic, which describe the underlying issues and make practical recommendations to healthcare planners to resolve them.

The opportunity of a Commission allows a global consultation with experts from all backgrounds to contribute, producing a unique collaboration with an overall message. This Commission will be followed by global advocacy to encourage investment in the ideas described and an improvement in affordable access to surgical care.

The Commission on Global Surgery was formally launched by Dr Jim Yong Kim, President of the World Bank, on 17–18 January 2014 at the Harvard School of Public Health, Boston, USA. Over 90 contributors sat together to discuss the issues from a variety of perspectives including workforce, training, measurement and finance. (www.gscommission.com). At the same time, the Lancet published the first comment on the work inviting contributions from anyone and everyone with a view.[3]

This is perhaps the first time that global surgery and anaesthesia will have the support of a major international journal to help us make our case – we hope the commission will be published in April in the Lancet.

As the Commissioners state: 'Surgery and anaesthesia are integral, indivisible components of any properly functioning health system. Our vision is that all people should have access to safe, high quality, affordable surgical and anaesthesia care: universal surgical care with financial protection.'[3]

**References**

1. Walker IA, Wilson IH. Anaesthesia in developing countries – a risk for patients. Lancet 2008; 371: 968–969.
2. The Lancet Global Health Journal (www.the lancet.com/global-health) (accessed 25 April 2014).
3. Meara JG, Hagader L, Leather AJM. Surgery and global health: a Lancet Commission. Lancet 2014: 383: 12–13.

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# Editor's Notes

Welcome to edition 29 of *Update in Anaesthesia*, the WFSA's educational journal.

This edition covers a wide variety of areas within anaesthesia and intensive care medicine that we hope will be of interest and use to our readers. Our article on *Metabolism and biochemistry* should be a valuable aid to any anaesthetist currently embarking on postgraduate examinations, but also serves to highlight the importance of this area in understanding the metabolic response to fasting, surgery and critical illness. A similar blend of practical basic science and clinical practice is seen in *Apnoea and pre-oxygenation,* with compelling arguments presented that pre-oxygenation should be routine practice for all anaesthetists in all settings.

A competent anaesthetist should have a good grasp of areas of medicine that impact upon their daily anaesthesia practice, allowing them to both understand and predict events as they develop in their area of clinical practice. A good example is the ability to interpret the obstetrician's CTG, as this is heavily relied upon by our obstetric colleagues when deciding the urgency of caesarian section. *Foetal heart rate monitoring* explains this more clearly that I have encountered before. Two further articles describe *the anaesthetic management of patients with coronary stents, pacemakers and implantable defibrillators* – technologies that are encountered regularly in high-income countries, but will be seen increasingly in patients in low- and middle-income countries over the coming years.

*Simulation training* has gained both popularity and an evidence base for its use over recent years and the article on this topic explains the techniques that may be used, and how simulation training can be undertaken very effectively in areas where resources are stretched. Expensive high-tech mannequins are probably the least important aspect of this growing area of medical education.

These techniques offer huge potential for team training, for example the obstetric team of surgeon, anaesthetic officer, midwife and scrub nurse all practicing drills for events such as major obstetric haemorrhage or maternal cardiac arrest.

Marcel Durieux describes his extensive experience using *lidocaine* as an adjunct to traditional general anaesthesia, a technique that is gaining use worldwide and offers several benefits above standard drug combinations. His article is a through review of the evidence for this relatively novel practice, and also a thorough guide of 'how to do it'.

The educational section for this edition offers two installments of *Cerebral challenge*, collections of ECGs, chest Xrays and CT scans with accompanying case histories and interpretation questions.

I hope that this edition offers something for everyone, no matter what your area of interest and expertise, your stage of training or the resources of your workplace. This standard edition of *Update* will be followed early in 2015 by a larger special edition focusing on aspects of paediatric anaesthesia and I am very grateful for the ongoing efforts of the editors, Rachel Homer and Isabeau Walker, in this project

As always I would be delighted to receive feedback and comments on our journal and the articles within it, as well as suggestions for future topics. If you would like to submit an article or discuss potential topics for articles, please contact me at bruce.mccormick@nhs.net.

If you would like to receive the printed version of *Update*, please email Nichola Page at admin@wfsahq.org.

**Bruce McCormick**
***Editor-in-chief***
***Update in Anaesthesia***

# News from the WFSA

A lot has happened at the WFSA since the last edition of *Update in Anaesthesia* was published. Perhaps most important is the shift to a more strategic Programme Approach which puts Education & Training, Safety & Quality, Innovation & Research and, finally, Advocacy at the forefront of all that we do.

As this publication attests, it is still very much the continuing education of anaesthesiologists that provides a focus for the WFSA's efforts and we have a worldwide network of members and volunteers that help us respond to this challenge. Special mention must be made of Dr Wayne Morris (Chair, Education Committee) and Dr Isabeau Walker (Chair, Publications Committee) and members of their Committees for their untiring efforts in supporting our work. This is evidenced across our programmes and by the fact that, not only are we publishing a 31st *Update in Anaesthesia*, but also we have available more than 300 *Anaesthesia Tutorials of the Week* on the WFSA site - www.wfsahq.org

The value of these hundreds of educational articles and tutorials is being recognised with improved search facilities on the website (for example allowing archives to be searched by keyword and category), as well as by online sign up and mailing systems that ensures that our readers are informed as soon as new publications become available. We are also developing a more obvious "Resource" Section on the site that points the way, not only to WFSA publications, but also to open source materials from other publishers that we think might be useful to you. We hope that this will move us further along in our ambition for the WFSA site to become a "one stop shop" for anaesthesiologists everywhere, especially those who seek further learning or teaching within the specialty.

Change can also be measured by the increasing number of fellowships now on offer each year, with more than 40 being available through the WFSA at the last count. These fellowships are offered in a range of specialist areas within anaesthesia and each lasting between two and twelve months, depending on the host institution and the subject matter. Most importantly they offer young anaesthesiologists from low and middle income countries extraordinary opportunities to improve their knowledge and skills in a cost effective and impactful manner, making fantastic use of the WFSA global network. Those that are interested in hosting or applying for a fellowship can find out more at http://www.wfsahq.org/our-work/education-training

Worthy of celebration is the fact that we have now awarded over 100 WFSA-Baxter scholarships. Since 2008 these scholarships have allowed young doctors, who would otherwise not be able to attend, the opportunity to present posters and take part in World and Regional Congresses of Anaesthesiologists. The scholars themselves attest to the value of these awards with 94% identifying positive impact for patients and 98% saying they would encourage their colleagues to apply. This is remarkable and I can add from my own experience of meeting scholars that these awards bring very real value to the outstanding individuals that are selected.

Moving beyond the education programme, the WFSA is also very proud of the recent publication of the book *Occupational Wellbeing in Anaesthesiologists* which can be downloaded for free from the website by visiting http://www.wfsahq.org/our-work/safety-quality. On the same webpage you can find the *International Standards for a Safe Practice of Anaesthesia*, and the checklist that goes with the Standards (in English and Spanish). There are also some very useful guidelines for tendering for Anaesthesia machines – especially relevant for those of you that do not have access to reliable supplies of oxygen and / or electricity. We also feature Lifebox, the UK based charity of which WFSA is a founder, which is doing so much for Safe Surgery through the distribution of, and training on, pulse oximeters and the WHO checklist.

Other things to look out for include the launch of our Innovation Awards with a focus on initiatives that demonstrate or strengthen the role of anaesthesia in improving surgical patient outcomes. These awards will be made at the World Congress of Anaesthesiologists in Hong Kong 2016, and details about criteria and how to apply can be found at http://www.wfsahq.org/our-work/innovation-research. We are also very proud and excited about a new partnership with the International Anesthesia Research Society (IARS) which foresees the establishment of a Global Health Section in the highly respected journal *Anaesthesia and Analgesia* (A&A). This affiliation between WFSA and the IARS aims to focus the attention of the best minds in our discipline on advancing the healthcare of patients worldwide and particularly in resource poor environments.

Finally, let me mention an area of our work that is both a challenge and a major opportunity for anaesthesiology around the globe. In 2015 the World Health Assembly will vote on a resolution entitled, "Strengthening emergency and essential surgical care and anaesthesia as a component of universal health coverage". In his letter to all WFSA member societies WFSA President, Dr David Wilkinson wrote, "If approved, the resolution would change the situation of our profession, and of surgical patients (and those who need surgery) around the world for the better. It is likely that it would also significantly influence the amount of resource made available for anaesthesia, shifting national and donor budgets towards the 11%+ of the global burden of disease that could be addressed by surgery" Dr Wilkinson went on to say: "With the poorest 30% of our world accessing just 3% of the surgery, and with mortality rates from surgical intervention 1,000 times higher in some parts of the world than in others, it is my sincere hope that we are all bound together behind this resolution." This challenge, together with plans to draft Sustainable Development Goals (to replace the Millennium Development Goals or MDGs) therein recognising the impact of surgery and anaesthesia on global health, merits the attention of all of us http://www.wfsahq.org/our-work/advocacy.

We hope you enjoy and benefit from this edition of *Update in Anaesthesia*. Of course we have the contributing authors and the editor, Dr Bruce McCormick, to thank for putting such a valuable edition together. They are amongst a highly prized WFSA team that demonstrates, on a daily basis, the impact of volunteers and volunteering. I salute them and am honoured to be part of it all.

**Julian Gore-Booth**
***Chief Executive Officer, WFSA***

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# Fetal heart rate monitoring – principles and interpretation of cardiotocography

*This article was originally published as Anaesthesia Tutorial of the Week 294 (2013)*

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**Summary**

Continuous electronic fetal monitoring is commonly performed by cardiotocography (CTG). The CTG monitor records both fetal heart rate (cardio) and maternal uterine contractions (toco). An understanding of the principles of CTG monitoring and a systematic approach to CTG analysis may enable anaesthetists to better appreciate why obstetricians make specific clinical decisions. This understanding may aid communication and timely delivery especially when the fetus is considered at high risk.

## CARDIOTOCOGRAPHY

The CTG monitor records the fetal heart rate (FHR) either from a transducer placed on the woman's abdomen or an electrode placed on the fetal scalp. An additional transducer placed on the woman's abdomen simultaneously records uterine muscle contraction. These variables are plotted graphically so that variations in FHR can be viewed over time and interpreted in the context of the contractile state of the uterus (Figure 1).

## INDICATIONS FOR CONTINUOUS CTG MONITORING

Intermittent auscultation of the FHR is recommended for women considered at low risk of complications during labour. The UK National Institute of Health and Clinical Excellence (NICE) make recommendations for continuous CTG monitoring which include:[1]

1. Meconium staining of liquor
2. Maternal pyrexia – defined as 38.0°C or 37.5°C on two occasions two hours apart
3. The use of oxytocin for labour augmentation
4. Fresh bleeding developing in labour
5. At the woman's request
6. Abnormal FHR detected during intermittent auscultation:
   - FHR <110 beats per minute (bpm)
   - FHR >160 bpm
   - Any decelerations after a contraction
7. Women receiving regional anaesthesia/ analgesia. Continuous electronic fetal monitoring is recommended for at least 30 minutes during establishment of regional analgesia and after administration of a further bolus of local anaesthetic agent. In most UK centres, continuous CTG monitoring is performed after the insertion of a labour epidural.

## CHARACTERISTICS OF THE CTG

A combination of several abnormalities increases the likelihood of fetal distress.

Suspicious or abnormal features include:

- Baseline FHR outside normal range of 110 – 160 beats per minute (bpm)
- Baseline variability <5 bpm
- Reduced or absent accelerations
- Presence of decelerations.

### Baseline rate

The normal baseline fetal heart rate is defined as 110 – 160 bpm. Fetal bradycardia is a baseline rate of <110 bpm. Fetal tachycardia is a baseline rate of >160 bpm.

Many fetal baseline bradycardias have no identifiable cause but may occur as a result of:

- Cord compression and acute fetal hypoxia
- Post-maturity (> 40 weeks gestation)
- Congenital heart abnormality.

Fetal tachycardia is associated with:

- Excessive fetal movement or uterine stimulation
- Maternal stress or anxiety
- Maternal pyrexia
- Fetal infection
- Chronic hypoxia
- Prematurity (<32 weeks gestation).

### Fetal heart rate variability

Variability refers to the normal beat to beat changes in FHR. Normal variability is between 5-15 bpm. Variability can be measured by analysing a 1-minute portion of the CTG trace and assessing the difference between the highest and lowest rates during that period. Variability can be defined as:

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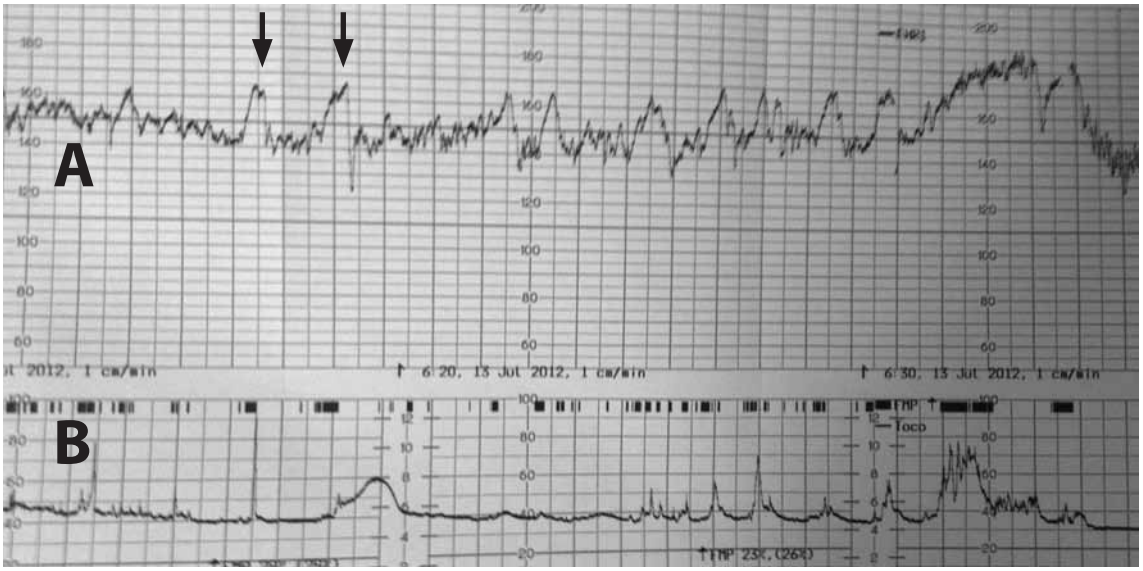


**Figure 1.** *Normal CTG with fetal heart rate uppermost (A) and the tocogram, showing uterine contractions below (B). The fetal heart rate is within the normal range and has normal baseline variability. The arrows demonstrate healthy fetal heart rate accelerations.*

| | |
|---|---|
| Normal | 5-15 bpm |
| Increased | >15 bpm |
| Decreased | <5 bpm |
| Absent | <2 bpm |

Fetal hypoxia may cause absent, increased or decreased variability. Other causes of decreased variability include normal fetal sleep-wake pattern, prematurity and following maternal administration of certain drugs including opioids.

### Accelerations (Figure 2)

Accelerations are periodic, transient increases in FHR, defined as an increase in FHR >15 bpm for more than 15 seconds. When accelerations are present, the CTG is said to be reactive. Accelerations are often associated with fetal activity and are considered an indication that the fetus is healthy.

### Decelerations

Decelerations are periodic, transient decreases in FHR, usually associated with uterine contractions. They can be subdivided into four main types by their shape and timing in relation to uterine contractions. Uterine contractions must be monitored adequately in order for a deceleration to be correctly classified.

Decelerations may be:

- Early
- Late

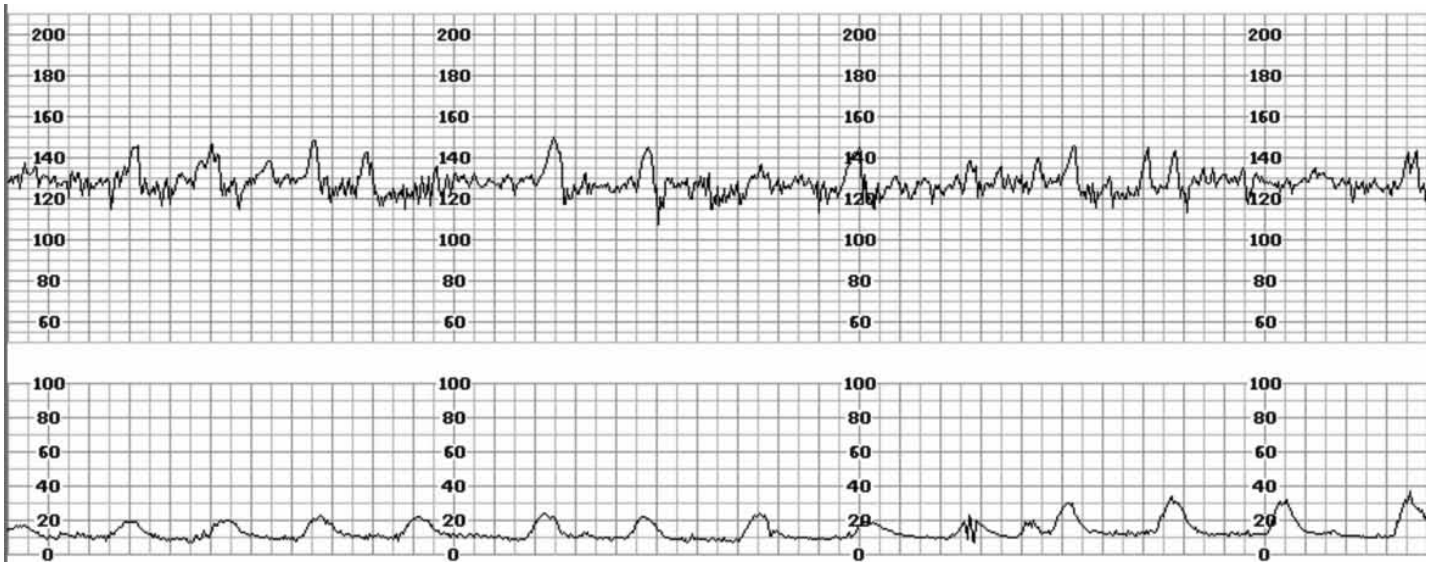

**Figure 2.** *CTG demonstrating fetal heart rate accelerations*

- Variable
- Prolonged.

### Early decelerations (Figure 3)

Early decelerations tend to occur with each contraction and are uniform in shape. Early FHR decelerations appear as a mirror image of the uterine contraction trace. The onset of the deceleration occurs at the onset of the contraction and the baseline FHR recovers by the end of the contraction. The FHR usually does not fall by more than 40 bpm during an early deceleration.

Early decelerations are caused by compression of the fetal head during a contraction. They are often relieved by changing maternal posture and are a normal finding in the second stage of labour. They are not associated with a poor fetal outcome.

### Late decelerations (Figure 4)

Late decelerations are uniform in shape on the CTG, but unlike early decelerations start after the peak of the uterine contraction. A deceleration in which the lowest point occurs more than 15 seconds after the peak of the uterine contraction is defined as a late deceleration. They are often associated with a decrease in the variability of the baseline FHR.

Late decelerations are associated with decreased uterine blood flow and can occur as a result of:

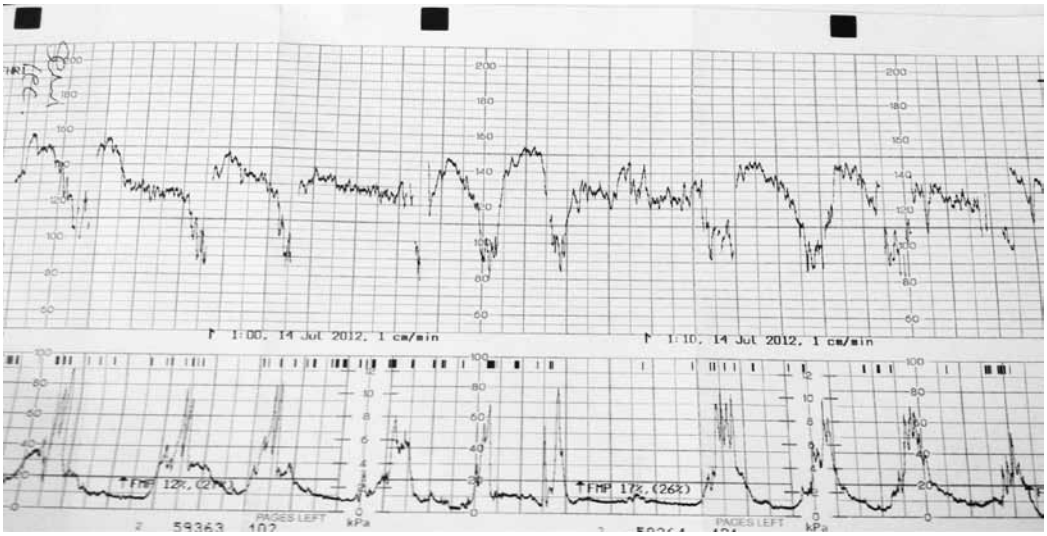

**Figure 3.** *CTG demonstrating early decelerations. Note the onset of the deceleration occurs with the onset of the uterine contraction*

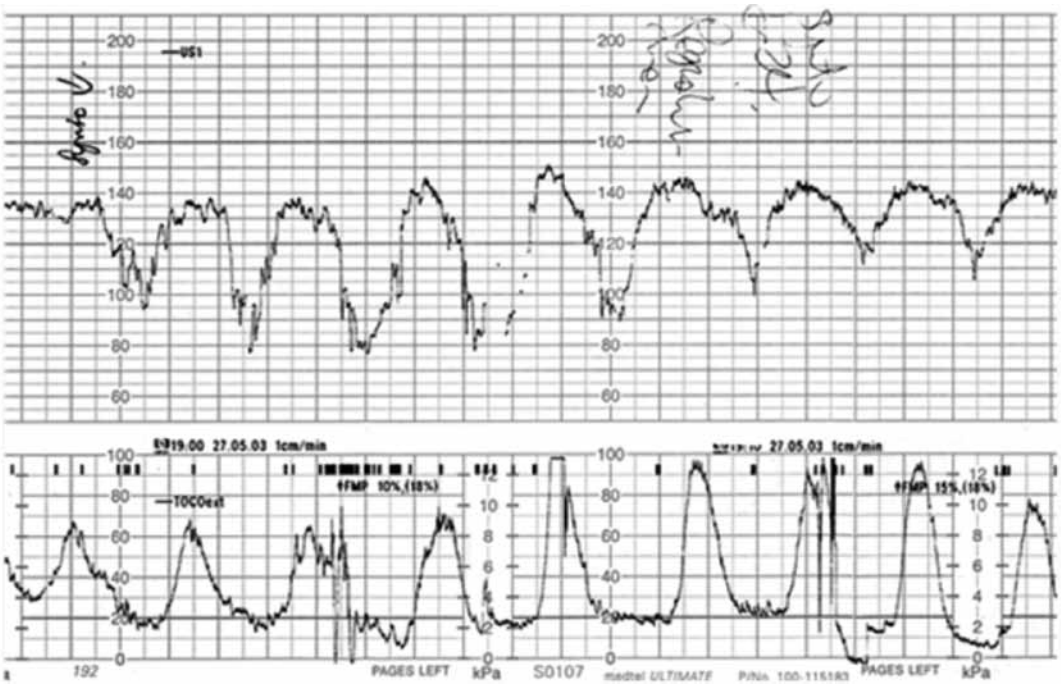

**Figure 4.** *CTG demonstrating late decelerations resulting from cord compression*

- Hypoxia
- Placental abruption
- Cord compression / prolapse
- Excessive uterine activity
- Maternal hypotension / hypovolaemia.

### Variable decelerations (Figure 5)

Variable decelerations describe FHR decelerations that are both variable in timing and size. They may be accompanied by increased variability of the FHR. They are caused by compression of the umbilical cord and may reflect fetal hypoxia.

### Prolonged decelerations/ bradycardia (Figure 6)

A deceleration with a reduction in FHR of greater than 30 bpm that lasts for at least 2 minutes is termed a prolonged deceleration They are caused by a decrease in oxygen transfer to the fetus, so can arise as a consequence of a wide variety of disorders including:

- Maternal hypotension
- Umbilical cord compression
- Uterine hypertonia.

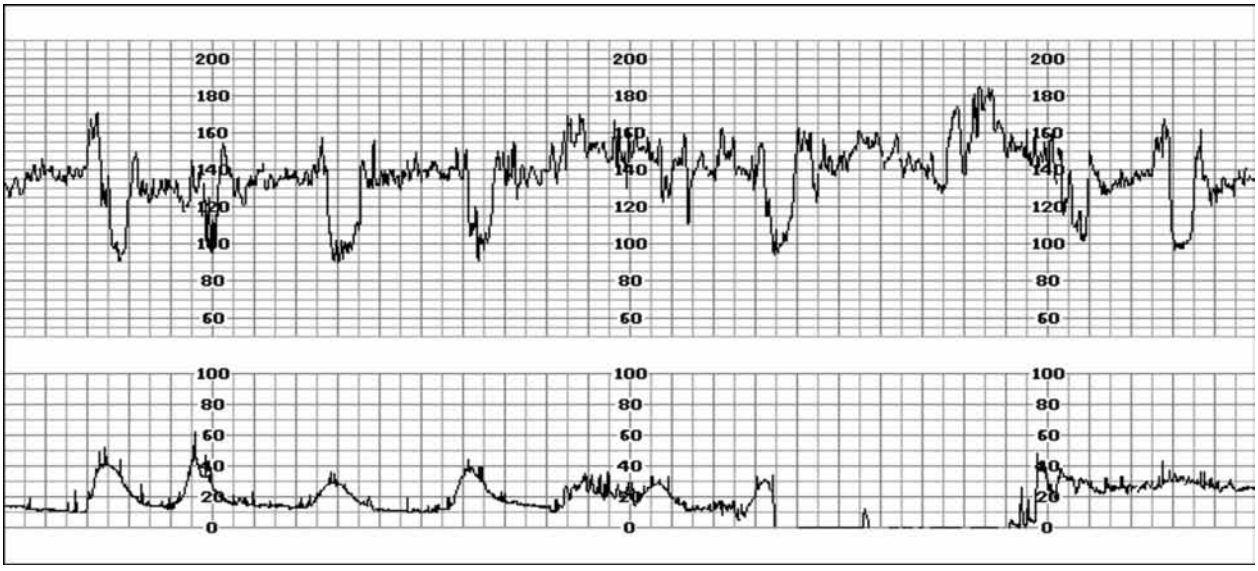

**Figure 5.** *CTG demonstrating variable decelerations*

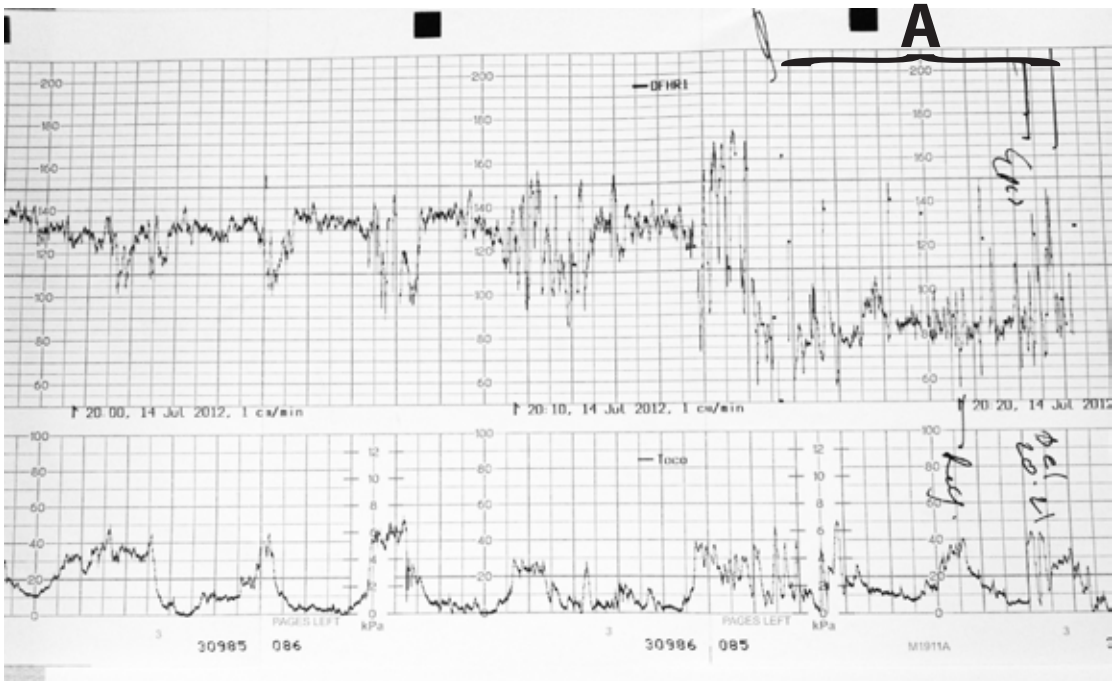


**Figure 6.** *Prolonged deceleration/bradycardia (A - bracketed) representing fetal distress*

**Table 1.** *Categorisation of CTG*

| | Reassuring | Non-reassuring | Abnormal | | |
|---|---|---|---|---|---|
| **Baseline rate (bpm)** | 110 - 160 | 100 - 109<br>161 - 180 | <100<br>>180 | Comments:- | |
| **Variability (bpm)** | 5bpm or more | <5 for 40 min or more but < 90 min | <5 for 90min or more | Comments:- | |
| **Accelerations** | Present | None | Comments:- | | |
| **Decelerations** | None | Early<br>Variable<br>Single prolonged deceleration up to 3 min | Atypical variable<br>Late<br>Single prolonged deceleration > 3 min | Comments:- | |
| **Opinion** | **Normal CTG**<br>(All four features reassuring) | **Suspicious CTG**<br>(One non-reassuring feature) | **Pathological CTG**<br>(two or more non-reassuring or one or more abnormal features) | | |
| **Dilatation** | | Comments:- | | **Contractions** | **.....:10** |
| **Action** | | | | | |
| Date<br>Time | | Signature<br>Status | | | |

## CTG CATEGORISATION

Following CTG analysis, the findings can be used to categorise the CTG as: normal, suspicious or pathological. Many UK maternity units have adapted recommendations from NICE to produce a sticker (below) that is placed in the records of the labouring woman to assist her on-going management.

## FETAL SCALP ELECTRODE

A fetal scalp electrode is a device that is attached to the skin of the presenting part (most commonly the scalp) of the fetus to assess the fetal heart rate pattern when external monitoring cannot be used or when the quality of external monitoring is poor (e.g. maternal obesity). It provides a more accurate and consistent transmission of the fetal heart rate than external monitoring because external factors such as movement do not affect it.

## FETAL BLOOD SAMPLING

Fetal blood sampling (FBS) involves taking a small sample of blood from the presenting part of the fetus during labour. FBS is advised in the presence of a pathological CTG unless there is clear evidence of severe fetal compromise. If fetal compromise is evident, (e.g. prolonged deceleration of greater than 2 minutes duration), FBS should not be undertaken and the baby should be delivered urgently. Other contraindications to FBS include:

- Maternal infection (such as HIV, hepatitis, herpes simplex virus)
- Fetal bleeding disorders
- Prematurity (<34/40).

## INTRAUTERINE FETAL RESUSCITATION

If CTG monitoring indicates serious fetal compromise, or FBS result is abnormal, a decision to deliver, often by emergency caesarean section, should be made. A number of manoeuvres can be performed to improve fetal oxygenation before delivery.[2] These manoeuvres may be performed with continuous CTG monitoring and if successful may reduce the urgency to deliver allowing time to provide neuraxial anaesthesia. Steps in intrauterine resuscitation can be remembered by the acronym SPOILT:

- **S**yntocinon off
- **P**osition full left lateral
- **O**xygen
- **I**.V. – infusion of crystalloid fluid
- **L**ow blood pressure – if present give i.v. vasopressor
- **T**ocolysis - terbutaline 250mcg sc (a $\beta_2$-agonist) or GTN (2 x 400mcg puffs sublingual)

**Table 2.** *Classification of fetal blood sampling (FBS) results*[1]

| FBS result (pH) | Classification | Recommended subsequent action |
|---|---|---|
| ≥ 7.25 | normal | Repeat within 1 hour if FHR trace remains pathological or sooner if further abnormalities |
| 7.21 – 7.24 | borderline | Repeat within 30 minutes if FHR trace remains pathological or sooner if further abnormalities |
| ≤ 7.2 | abnormal | Seek obstetric advice, delivery is indicated |

## REFERENCES


1. Guideline CG55, National Institute for Health and Clinical Excellence, *September 2007*. Intrapartum care: management and delivery of care to women in labour. Available at: http://guidance.nice.org.uk/CG55
2. Thurlow JA, Kinsella SM. Intrauterine resuscitation: active management of fetal distress. *Int J Obstet Anesth* 2002; **11**: 105-16.

Update in
Anaesthesia

# Pacemakers and implantable cardioverter defibrillators

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**Summary**

Conduct a focused history and full physical examination for all patients with cardiac electronic devices

Gather important information regarding the device: its make and model, basic programming features, battery life, and the underlying rhythm

Involve the cardiology team responsible for management of the device as early as possible in the patient's perioperative care

Confirm the location of the operation in relation to the device and consider deactivating it if exposure to significant electromagnetic interference is expected

Always have an backup plan in case of device malfunction

Magnets should only be used after consultation with the cardiologist responsible for the patient's care.

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## INTRODUCTION

As the number of indications for permanent pacemakers and implantable defibrillators have increased over recent years it has become increasingly common for anaesthetists to care for patients with these devices. Considering the growing range and complexity of these devices, it is not surprising that several surveys have shown that many anaesthetists are not comfortable managing these patients in the perioperative period.[1,2] Placement of a magnet on these devices is rarely an appropriate manoeuvre and a more systematic and structured approach needs to be adopted. This article reviews key recommendations by the American Society of Anesthesiologists Task Force with regards to caring for patients with one of these devices.[3]

## PERMANENT PACEMAKERS

A permanent pacemaker is an electronic device, generally placed subcutaneously, with one or several leads connected to the myocardium. Pacemakers allow both sensing of myocardial electrical activity and delivery of electrical stimulation for pacing functions.

Depending on the patient's clinical need, pacing can be delivered to single, dual, or multiple chambers (in bi-ventricular pacing), using unipolar or bipolar leads. More modern systems use bipolar leads, where the cathode and anode (the positive and negative electrodes) lie in close proximity, to reduce susceptibility to electromagnetic interference (EMI). Newer devices also incorporate a wide spectrum of programmability to allow alterations in pacing rate, energy output, sensitivity, mode of operation, and other functions.

Some pacemakers even utilize piezoelectric crystals to detect physical activity and compensate by increasing the heart rate. Other forms of sensors include: thoracic impedance, minute ventilation, temperature, QT interval, oxygen saturation, and myocardial contractility as means of detecting and adjusting to activity levels.

### Classification

The function and capabilities of a pacemaker are often represented by a simple code. Since 2001, the North American Society of Pacing and Electrophysiology (NASPE)/British Pacing and Electrophysiology Group (BPEG) established a five letter generic code to describe various pacing modes. Devices are usually described using just the first three letters, but some have additional capabilities that are denoted in the final two codes. Familiarisation with this code is essential to understanding the function and management of an individual's pacemaker in the perioperative period.

The following examples illustrate how this code may be used.

#### *AAI mode*

The **A**trium is the chamber that is paced. The **A**trium is the chamber that is sensed and, if an electrical impulse is detected, the pacemaker will be **I**nhibited from delivering an electrical impulse. If no depolarization occurs within a certain period of time, the device will automatically initiate pacing at a pre-set rate. This mode can be used to raise the heart rate of patients with symptomatic sinus bradycardia, but normal AV conduction.

**Table 1.** *The NSAPE/BPEG (NBG) Pacemaker Code*[4]

| I | II | III | IV | V |
|---|---|---|---|---|
| **Pacing** | **Sensing** | **Response** | **Rate modulation** | **Multisite pacing** |
| A = atrium | A = atrium | I = inhibited | R = rate modulating | V = ventricle |
| V = ventricle | V = ventricle | T = triggered | O = none | A = atrium |
| D = dual (A+V) | D = dual (A+V) | D = dual (T+I) | | D = dual (A+V) |
| | O = none | O = none | | O = none |

*VVI mode*

This is similar to AAI mode, but applied to the ventricle. The **V**entricle is the chamber that is paced, the **V**entricle is the chamber sensed for electrical activity and, if a ventricular depolarization is detected, the pacemaker will be **I**nhibited from delivering an electrical impulse. This mode is used to ensure that patients have an adequate ventricular rate in the presence of atrial fibrillation and a slow ventricular response.

*DDD mode*

This mode is used in a variety of clinical scenarios, sensing and triggering or inhibiting pacing of the atrium ventricle or both. **D**ual atria and ventricle are paced (requiring two pacemaker leads), **D**ual chambers are sensed and if electrical activity is detected, then pacing of that chamber is inhibited, or paced if no activity is noted. This is the **D**ual response. This mode can ensure each atrial depolarisation (spontaneous or stimulated) is followed by a ventricular depolarisation at a predetermined rate and maintain atrioventricular synchrony.

*Asynchronous modes*

Asynchronous modes (e.g. AOO, VOO and DOO) do not provide sensing and simply pace the designated heart chambers regardless of the underlying electrical activity. These modes are most often used in emergency situations or when triggered by placement of a magnet over the device.

### Indications

There are three broad indications for cardiac pacemaker insertion: bradycardias (either persistent, intermittent or suspected); resynchronization therapy for cardiac failure; and rare conditions such as long QT syndrome and metabolic disorders. The bradyarrhythmias make up the bulk of these with the most common indications for pacemaker implantation being sinus node dysfunction with AV block. The need for permanent pacing in patients is based upon the severity of symptoms associated with their bradycardia. The most significant symptoms are: syncope, seizures, heart failure, dizziness, and confusion. The American College of Cardiology guidelines for pacemaker implantation grade the indications from Class I (strong evidence or general agreement for indication) to Class III (evidence existing against the procedure).[5]

The Class 1 indications for permanent pacing (American College of Cardiology) are:

- Symptomatic sinus bradycardia
- Symptomatic chronotropic incompetence
- Symptomatic sinus bradycardia resulting from medical drug therapy.

## IMPLANTABLE CARDIOVERTER DEFIBRILLATORS

An implantable cardioverter defibrillator (ICD) resembles a pacemaker in location and lead connection to the myocardium, but differs structurally by including a pulse generator. Defibrillators usually have a sensing lead in the right ventricle to monitor electrical activity and a defibrillation lead to deliver a shock when malignant tachycardias are detected. An additional sensing lead is sometimes placed in the right atrium to help distinguish true ventricular tachycardia (VT) from conducted supraventricular tachycardia, which does not require defibrillation. Apart from the defibrillating function, ICDs may also have bipolar, anti-bradycardia and anti-tachycardia pacing capabilities. As with pacemakers, defibrillators have their own unique coding system to describe their programming and functionality (Table 2).

### Indications

The main indications for insertion of an ICD are:

- secondary prevention in patients with prior sustained ventricular tachycardia (VT)
- ventricular fibrillation (VF)
- resuscitated sudden cardiac death (SCD) due to VT/VF
- primary prevention in patients at increased risk of life-threatening VT/VF.

In 2008, the American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS) issued guidelines for insertion of cardiac defibrillators. The recommendations are graded from Class 1 (strong evidence for ICD therapy) to Class 3 (evidence against the procedure).[5] Table 3 summarizes the Class 1 indications for permanent ICD therapy.

## PERIOPERATIVE MANAGEMENT OF PATIENTS WITH CARDIAC IMPLANTABLE ELECTRONIC DEVICES (CIED)

### Preoperative management

Safe anaesthetic management of patients with these devices should begin with a comprehensive preoperative visit and review of the clinical records. The involvement of the device managing team

**Table 2.** *The NASPE/BPEG (NBD) code for cardiac defibrillators*

| I | II | III | IV |
|---|---|---|---|
| **Shock chamber** | **Anti-tachycardia pacing chamber** | **Tachycardia detection** | **Anti-bradycardia pacing chamber** |
| 0 = none | 0 = none | E = ECG | 0 = none |
| A = atrium | A = atrium | H = haemodynamic | A = atrium |
| V = ventricle | V = ventricle | | V = ventricle |
| D = dual (A+V) | D = dual (A+V) | | D = dual (A+V) |

**Table 3.** *American College of Cardiology Class 1 indications for ICD therapy*

- Left ventricular ejection fraction (LVEF) <35% due to prior myocardial infarction
- Minimally symptomatic VT with LVEF <40%
- VF or SCD survivors
- Nonischaemic dilated cardiomyopathy with LVEF <35%
- Spontaneous sustained VF/VT due to structural heart disease
- VT/VF causing syncope of undetermined origin.

as well as complete information about the device is vital in the perioperative management of these patients and close liaison with the cardiologists and cardiology technicians should be arranged at the earliest opportunity. Numerous case reports have shown how incomplete evaluation of a CIED has resulted in significant intraoperative problems.[6-7]

It is preferable that a pacemaker has been checked *within the last 12 months* and an ICD within the *last 6 months*, however this does not guarantee that the device will not malfunction. A specific history from the patient and a thorough physical examination may provide clues as to their dependence on the device, as well as how well it is functioning.

Preoperative tests should include: a 12-lead ECG, chest Xray, and blood electrolyte levels. ECGs can show the underlying rhythm in the absence of pacing, signs of pacemaker activity and evidence of electrical capture. Examination of the chest radiograph can often provide information about the position, type of device, lead configuration, and the presence of fractured leads.

The choice of anesthetic technique depends on the patient's comorbidities and intended procedures. Most of the commonly used anesthetic agents do not affect EICDs, but some practitioners may avoid suxamethonium due to concerns of over-sensing from muscle fasciculation. A more important perioperative concern is how capture thresholds and impedance of the leads can be altered by anaesthetic management (hypoxaemia, hypercapnia, significant acidosis, marked electrolyte disturbances, volume overload, high concentrations of local anesthetics and myocardial ischaemia).

In addition to standard monitoring, invasive arterial pressure monitoring should be considered in complex cases. If central venous access is being considered, extra care must be taken to avoid dislodging the electrodes. When practical, recommend the use of bipolar diathermy during the operation. Regardless of the type (monopolar versus bipolar), cautery use should be limited to short and irregular bursts at the lowest effective current. If unipolar diathermy is unavoidable, the diathermy and ground plate should be placed far away from the EICD as possible and the current pathway from the forceps to plate should run at a right angle to the leads.

## Magnet use

Most pacemakers implanted after 2000 produce an asynchronous mode of pacing when a magnet is applied. The rate obtained depends on the programming of the device and default settings. Removal of the magnet should result in reversion to the device's baseline programming. *Reprogramming is only recommended for pacemaker-dependent patients who will be exposed to significant EMI*. If no reprogramming is necessary, the rate modulation should still be suspended in the perioperative period.

ICDs should be deactivated preoperatively if diathermy is to be used, to avoid delivery of unnecessary shocks. This is also particularly important in patients undergoing lithotripsy, radiofrequency ablation, and electroconvulsive therapy. Application of a magnet to most modern ICDs disables the arrhythmia detection function and prevents discharge. *However, if significant EMI is expected, it is recommended that the device is reprogrammed and the defibrillation function disabled.*

The defibrillation function of the device should be instantly reactivated following the removal of the magnet, with *no subsequent reprogramming usually needed*. A magnet does not usually alter the pacemaker function of an ICD, as it should deactivate the defibrillating function only. Unfortunately, the magnet response by devices varies considerably between manufacturers. Some ICDs (Boston Scientific and St Jude) can be programmed to ignore the magnet application.[6] With this in mind, it is recommended to contact the manufacturer/cardiologist for more information about how that particular device responds to a magnet if clarification is required.

**In general, the use of a magnet should only be considered after consultation with the patient's cardiologist.**

## Intraoperative

The most common issue arising in the perioperative period is interference with device function from electromagnetic interference (EMI). Table 4 shows a list of common sources of EMI in the hospital setting.

EMI can potentially cause inhibition of pacing due to over-sensing or inadvertent reversion to back up pacing modes in pacemakers. With an ICD, EMI can trigger inappropriate delivery of potentially harmful defibrillatory shocks. Modern devices now incorporate bipolar leads as well as filters and insulating circuit shields to reduce EMI interference, but this is not 100% effective. Current literature advises that the potential for EMI-CIED interactions decreases when the cardiac device gets further away from the EMI source (a critical distance of 15cm has been suggested).[2] It is also theoretically safer if the EMI source is not in a vector parallel to that of the EICD current.

**Table 4.** *Common sources of EMI in the hospital setting*

| |
|---|
| Diathermy (monopolar > bipolar) |
| Magnetic resonance imaging |
| Electroconvulsive therapy |
| Radiofrequency ablation |
| Fasciculation |
| External defibrillation |
| Extracorporeal shock wave lithotripsy |
| Nerve stimulators |

Contingency plans for pacing, defibrillation, or both should always be available in case of EICD malfunctioning. For pacing, this includes inotrope infusions and external transthoracic pacing pads. If these fail, transvenous and transoesophageal pacing should be considered, but both take time and experience to set up. If defibrillation is required or anticipated, patients should be attached to the external defibrillator with an anterior–posterior configuration of the pads in order to minimize exposure to the electrical current. Resuscitative measures should follow the current ACLS algorithms.

**Table 5.** *Recommendations for interrogating CIEDs postoperatively*

- Intraoperative cardiopulmonary resuscitation and cardiac interventions
- Significant EMI exposure during surgery
- Haemodynamically challenging surgeries such as cardiac or high risk vascular surgery
- EICD reprogramed before the procedure
- After insertion of a pulmonary artery catheter, especially in the setting of recently inserted leads (<6 weeks) or after cardiopulmonary bypass/ECMO

### After the operation

Postoperative interrogation of CIEDs is strongly recommended in certain situations (Table 5) to ensure patient safety.

## CONCLUSION

Perioperative management of pacemakers and implantable cardioverter defibrillators can be challenging. All patients should be thoroughly evaluated before surgery and a plan devised for perioperative management of their device in close liaison with the patient's cardiologist. Prior to surgery, the anaesthetist should have a thorough understanding of the patient's underlying cardiac function, the status and function of the device, and its magnet response.

## REFERENCES


1. Allen M. Pacemakers and implantable cardioverter defibrillators. *Anaesthesia* 2006; **61**: 883-90.
2. Stone ME, Salter B, Fischer A. Perioperative management of patients with cardiac implantable electronic devices. *British Journal of Anaesthesia* 2011; **107** (S1): i16-i26.
3. Apfelbaum JL, Belott P, Connis RT, et al., For the American Society of Anesthesiologists Committee on Standards and Practice Parameters. Practice advisory for the perioperative management of patients with cardiac implantable electronic devices: pacemakers and implantable cardioverter-defibrillators. *Anesthesiology* 2011; **114**: 247-61.
4. Rooke, GA, Bowdle, TA et al. Perioperative Management of Pacemakers The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate and multisite pacing. *Pacing Clin Electrophysiol* 2002; **25**: 260-4.
5. Vlay SC. The ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: their relevance to the cardiologist, internist and family physician. *J Invasive Cardiol* 2009; **21**: 234-7.
6. Implantable Cardioverter Defibrillators: Its Not Just About the Magnet. *Anesth Analg*. 2013; **117**: 292-4
7. Schulman PM, Rozner MA. Use Caution When Applying Magnets to Pacemakers or Defibrillators for Surgery. *Anesth Analg* 2013; **117**: 422-27.

Update in
Anaesthesia

# An overview of metabolism

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**Summary**

The metabolic processes described in this article are essential for life and are subject to massive variation depending on the individual's environment, nutritional state and level of exercise or stress. The clinical relevance becomes clear when considering performing major surgery on a fasted patient. Maintaining an adequate nutritional intake preoperatively and oxygen delivery intraoperatively are vitally important in avoiding detrimental catabolic and anaerobic metabolic processes in patients undergoing major surgery and those with critical illness.

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## INTRODUCTION

Metabolism is defined as the chemical processes by which cells produce the substances and energy needed to sustain life. It is subdivided into:

**Anabolism** - the phase of metabolism in which complex molecules, such as the proteins and fats that make up body tissue, are formed from simpler ones.

**Catabolism** - the metabolic breakdown of complex molecules into simpler ones, often resulting in a release of energy.

The process of metabolism within the body changes as a result of surgery or acute illness and so is an important area of knowledge for anaesthetists and intensivists.

A key metabolic process within the body is the liberation of energy by the breakdown of substances ingested as food. The major constituents of a normal diet are **carbohydrates, protein** and **fat**.

## OVERVIEW OF METABOLISM

Figure 1 is an overview of the pathways by which the three major energy sources (fat, carbohydrate and protein) can be used to produce ATP, the main unit of energy within the body. Glucose is predominantly metabolized via glycolysis, free fatty acids via β-oxidation and proteins via deamination. The common endpoint for all three pathways is acetyl coenzyme A (acetyl coA), which can be used as a substrate for very efficient production of ATP via the

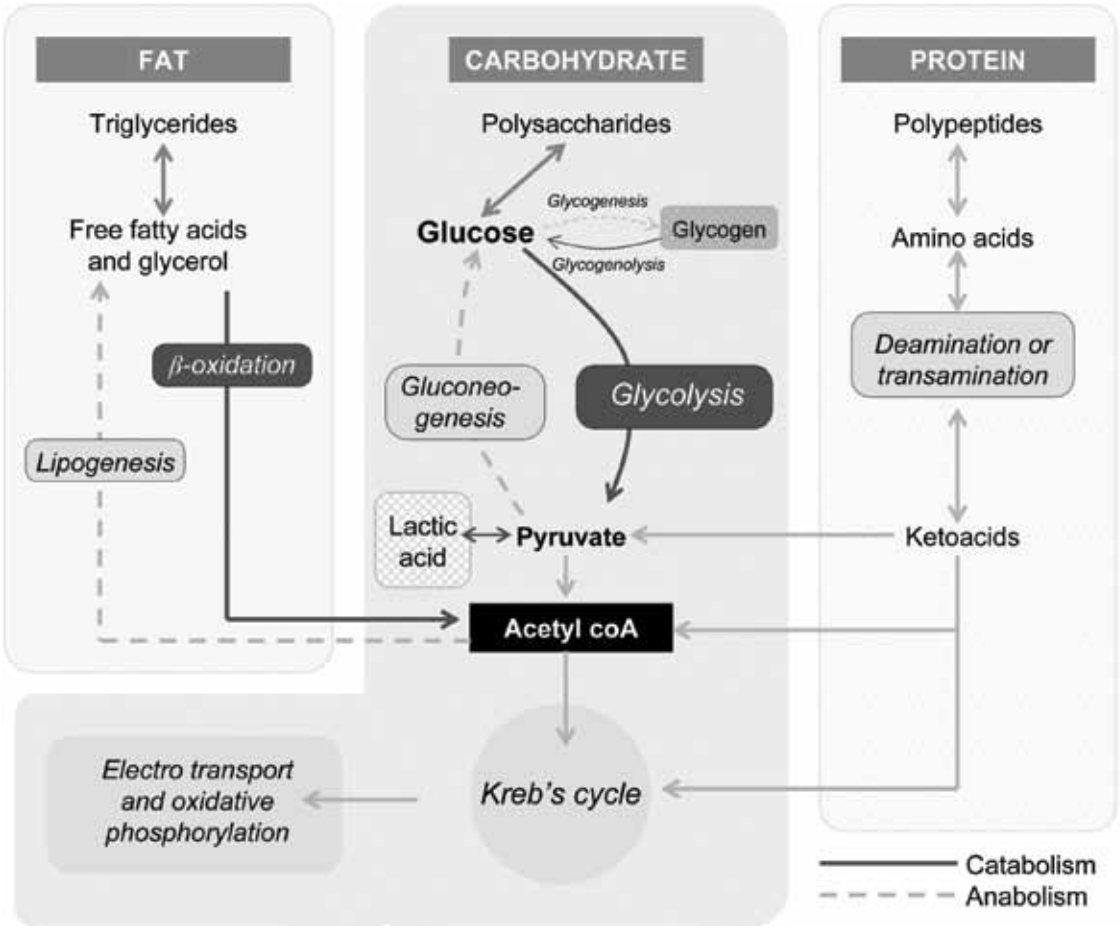


**Figure 1.** *A highly simplified overview of fat, carbohydrate and protein metabolism*

citric acid cycle (also known as the Krebs or tricarboxylic acid cycle), when oxygen is available (aerobic metabolism). Acetyl coA is also the start point by which glycolysis can be reversed (gluconeogensis) - glucose is regenerated and used by specific glucose-requiring organs (the brain and heart) during fasting. All of these processes are explained in more detail below. It is important to understand the concept that different metabolic processes are going on in different organs at different times, dependent on the activities and requirements for energy of those tissues, and those of the body as a whole. For example, the liver may be breaking down glycogen to release glucose for the brain (glycogenolysis), whilst the fat is being broken down by β-oxidation for production of ATP via the Krebs cycle and production of glucose via gluconeogenesis. ATP cannot be transferred around the body, only produced and used within cells, so the body is dependant on efficient transfer of the metabolites and substrates shown in Figure 1 around the body, in order to meet the needs of individual organs under different conditions.

The body has various mechanisms that it can use to adjust to the availability of various substrates:

- They can be transformed into other types of substrate, or
- They can be transported around the body and used as needed by other organs to make ATP, which can then be used within the cells of that organ.

## TYPES OF MOLECULES

### Carbohydrate

Carbohydrate molecules contain carbon, hydrogen and oxygen atoms, usually in the proportion 1:2:1, and must contain both aldehyde and ketone groups.


**Figure 2**. *A - an aldehyde, B - a ketone*

Carbohydrates in their most basic form are called monosaccharides, such as glucose. *Monosaccharide* units may be joined into chains to become polysaccharides, such as starch.

### Fats

Fats are ingested as triglycerides and cholesterol. Triglycerides are esters of three fatty acids chains bonded to a glycerol molecule.

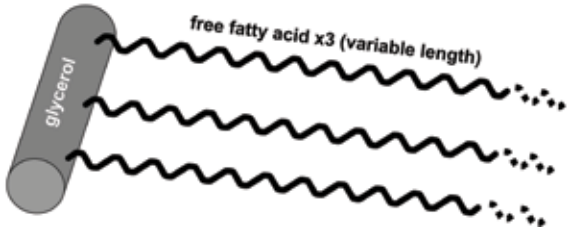


**Figure 3.** *A triglyceride*

### Proteins

Proteins are chains of amino acids, molecules containing an amine group (nitrogen-containing, $-NH_2$), a carboxylic acid group (-COOH) and a specific side chain (R in Figure 4).

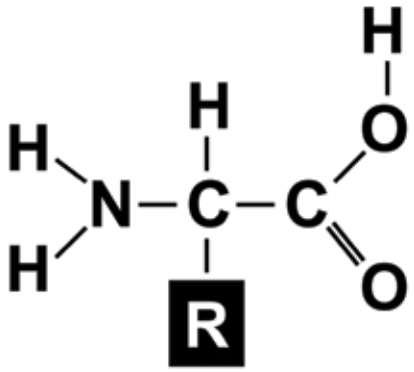


**Figure 4**. *An amino acid*

## ENERGY

Energy is required for the body to perform activities or 'work'. This work can be external (skeletal muscle) or internal (cardiac muscle, biochemical processes). Under normal conditions it is obtained by breakdown of dietary carbohydrate, protein and fat. The average energy requirement for a 70kg man is 2500kcal per day. The basic unit for supply of energy for processes within the body is adenosine triphosphate (ATP), a molecule that has high energy bonds with its attached phosphate groups. The breakdown of adenosine *triphosphate* to adenosine *diphosphate* (ADP) releases energy. ATP can then be regenerated from ADP using either:

- energy generated within the cytoplasm by *anaerobic respiration* (no oxygen is required),
- *aerobic respiration* in mitochondria (oxygen requiring and far more productive of ATP), or
- by direct interaction with creatine phosphate.

Remember that ATP and ADP are intracellular molecules that provide energy *in situ* within a cell, but cannot be transferred around the body from one organ to another. Movement of energy sources around the body is achieved by the pathways for breakdown (catabolism) and build up (anabolism) of the substances shown in Figure 1.

### Acetyl coenzyme A

Acetyl coA is the central converting substance that links together the metabolism of fat, carbohydrate and protein and so is known as the universal intermediate

## CARBOHYDRATE METABOLISM

The major process of ATP production is carbohydrate metabolism within cells. This is done via two main pathways: glycolysis and the citric acid cycle (Kreb's or tricarboxylic acid cycle). Glycolysis is the breakdown of glucose to form pyruvate. At some stages ATP is generated directly, at other stages a proton ($H^+$ ion) is passed to the coenzymes:

- $NAD^+$ (nicotine adenine dinucleotide) to become NADH and
- FAD (flavine adenine dinucleotide) to become $FADH_2$.

The protons are then passed on to a flavoprotein-cytochrome carrier chain within the cell's mitochondria to enable the process by which ATP is regenated. They provide energy for this process by giving up an electron that is passed down the chain by sequential oxidation and reduction (see later).

### Glycolysis

Glucose, a six-carbon molecule ($C_6H_{12}O_6$), is cleaved to eventually form 2 pyruvate molecules each containing 3 carbon atoms (see Figure 1). Initially, glucose is transported into the cell and converted into glucose-6-phosphate (G-6-P) by an enzyme called hexokinase (or glucokinase). G-6-P is unable to move back across the cell membrane and therefore remains within the cell. The enzyme phosphohexose isomerase catalyses the reaction to form fructose-6-phosphate, which is then converted to fructose-1,6-biphosphate. This molecule is then split to form two 3-carbon molecules which then release their phosphate atoms over the next 2 steps of the reaction, releasing enough energy to generate ATP from ADP and phosphate. For each glucose molecule utilized in glycolysis, 4 ATP molecules are produced, however, the process requires 2 ATP molecules, resulting in a net production of 2 ATP molecules per glucose molecule.

Under aerobic conditions (oxygen is available) pyruvate then enters the mitochondria (see box) and is broken down into acetyl coenzyme A which is the main substrate of the citric acid cycle (see Figure 6). The citric acid cycle is a progressive breakdown of citrate, a 6-carbon molecule, into oxaloacetate, a 4-carbon molecule.

**Mitochondrion**

A mitochondrion is a membrane-enclosed organelle found in most eukaryotic cells. Mitochondria are sometimes described as the 'cellular power plants' because they generate most of the cell's ATP.

The citric acid cycle, electron transport, and oxidative phosphorylation take place in the mitochondria.

In addition to supplying cellular energy, mitochondria are involved in a range of other processes, such as signalling, cellular differentiation, cell death, as well as the control of cell growth.

### The citric acid cycle

The acetyl coenzyme A produced by glycolysis combines with oxaloacetate to reproduce citrate. This metabolic process is dependant on a supply of acetyl coenzyme A and production of oxaloacetate, as well as the presence of oxygen, without which pyruvate will not cross into the mitochondria.

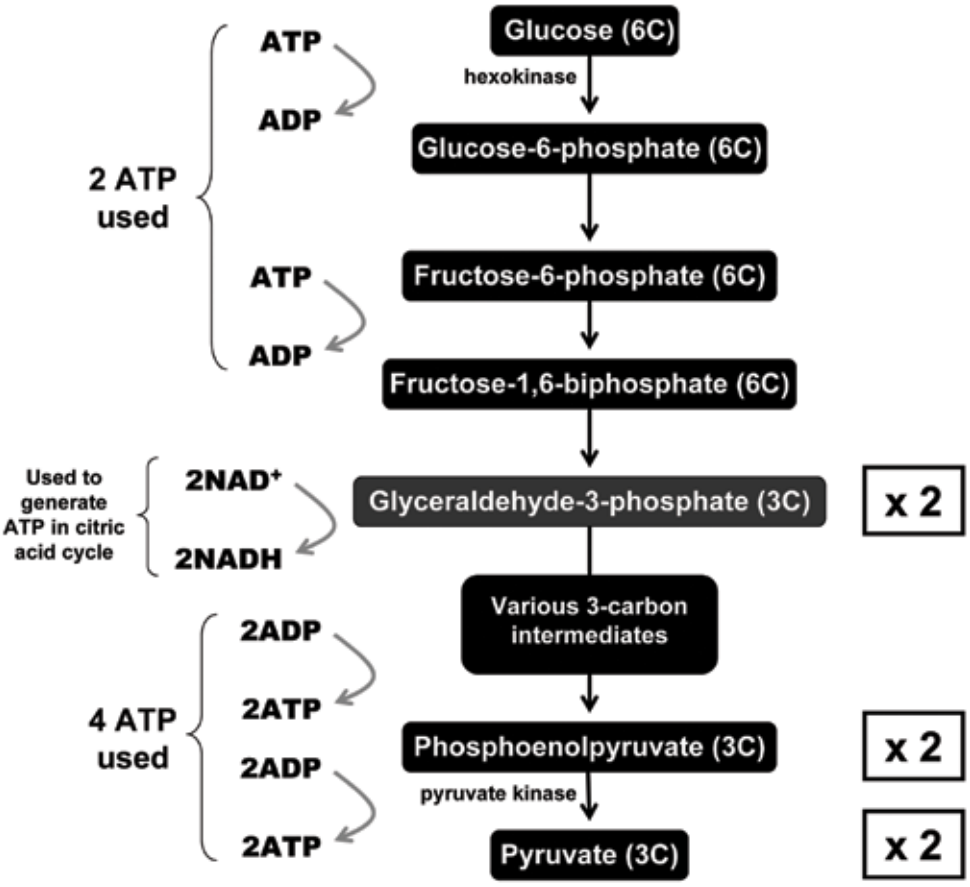


**Figure 5**. *Simplified overview of glycolysis*

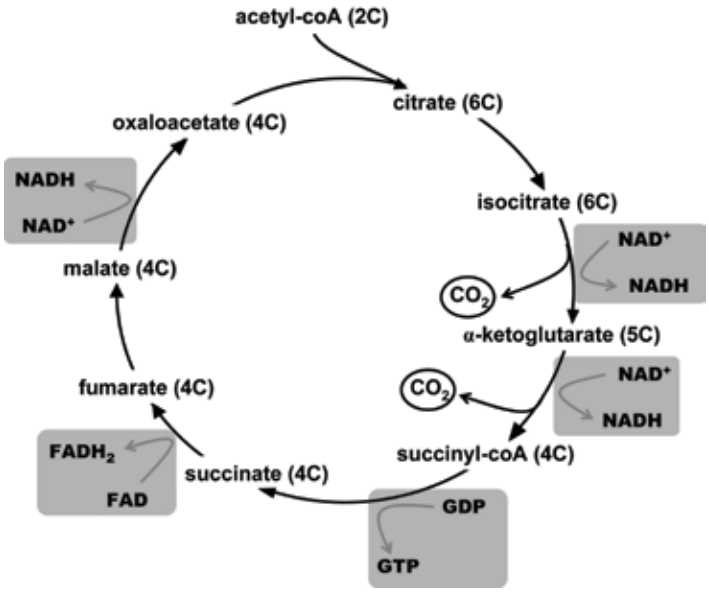


**Figure 6.** *The citric acid cycle (also known as the tricarboxylic acid cycle and the Kreb's cycle)*

For each acetyl coenzyme A molecule that enters the citric acid cycle, 6 nicotinamide adenine dinucleotide molecules and 2 flavin adenine dinucleotide molecules gain a proton to become NADH and $FADH_2$.

These compounds liberate electrons to a series of flavoprotein-cytochrome carriers mounted on the inner mitochondrial membrane (see Figure 7). The electron is passed from carrier to carrier in a series of oxidation/reduction reactions, liberating energy that is used to pump the protons ($H^+$) from NADH and FADH across the inner membrane to the inter-membrane space This creates an $H^+$ concentration gradient across the inner membrane, generating $H^+$ flow across channels containing ATP synthase. $H^+$ flow through the synthase complex provides energy for formation of ATP. Oxygen acts as the final electron acceptor at the end of the electron transport chain ($O_2 + 4e^- + 4H^+ = 2H_2O$). Without oxygen, the electron transport chain cannot function.

### Oxidation and reduction

Oxidation is loss of an electron, reduction is gain of an electron (often donated by hydrogen). As further explanation:

- If you have a carbon atom, it is in its maximally oxidised state when it is just a free carbon atom.
- Each time it forms a covalent bond with a hydrogen atom, it becomes reduced as the hydrogen atom gives (or lends) it an electron; it moves from a high oxidation state (or oxidation number) to a lower one.
- The lowest oxidation state is $CH_4$.

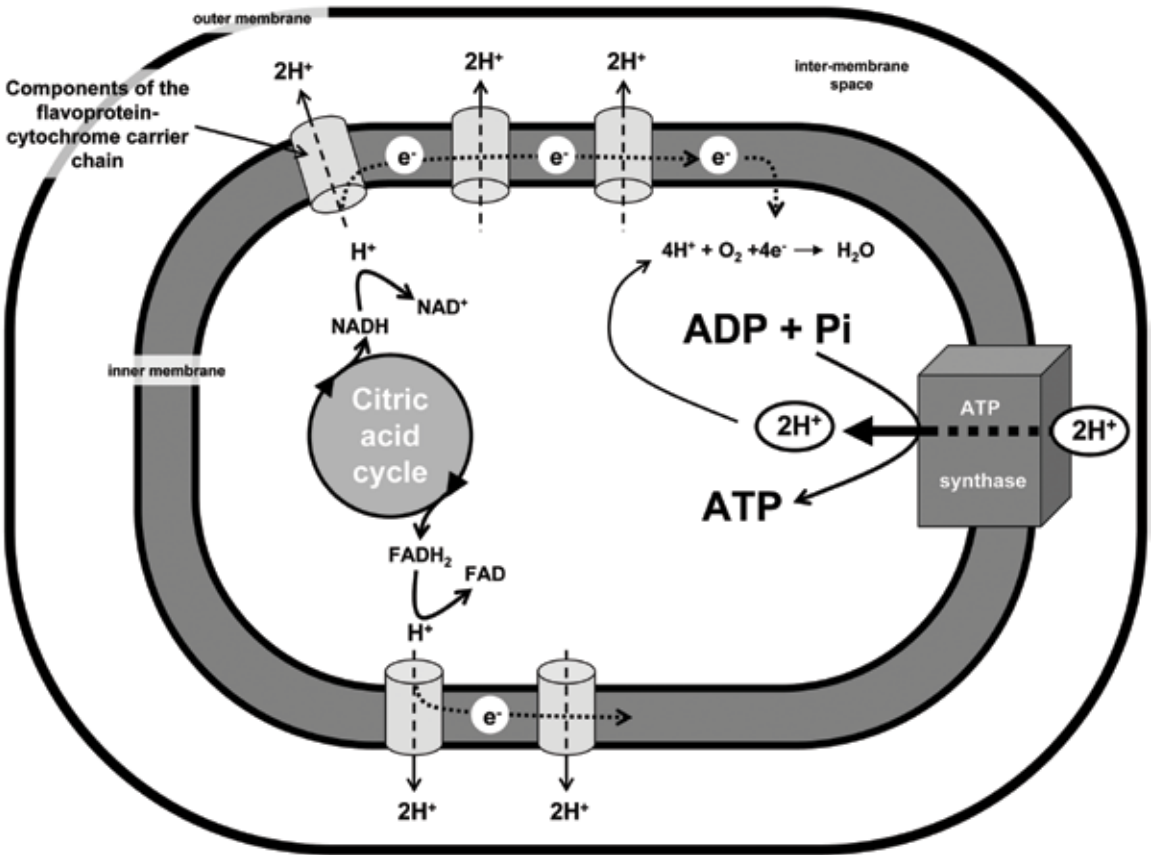


**Figure 7.** *Schematic drawing of generation of ATP using energy derived from NADH and $FADH_2$ in the citric acid cycle*

The production of ATP at each stage can be seen in Table 1. The net result is that for each glucose molecule, metabolised under aerobic conditions, 38 ATP molecules are produced. Acetyl coenzyme A can also be produced by breakdown of fat and protein. Fatty acids are transported into mitochondria where they undergo β-oxidation, combining with coenzyme A to form acetyl coenzyme A. Amino acids undergo oxidative deamination or transamination (the amine group is converted into urea) to form keto acids, which can then be broken down to form acetyl coenzyme A.

Under anaerobic conditions pyruvate is not converted to acetyl coenzyme A, but instead to lactate which is transported out of the cell. The total ATP yield from each glucose molecule under anaerobic conditions is 2 ATP molecules, making it much less efficient than aerobic metabolism.

Carbohydrate that is not immediately needed after absorption into the body, is stored as glycogen in the liver and skeletal muscle. An average 70kg manstores up to 500g of glycogen at any one time. It is formed by *glycogenesis* in which glucose molecules are sequentially added to existing glycogen chains. The process is activated by insulin which is secreted from the pancreas in response to high glucose levels. The major enzyme responsible for glycogenesis is glycogen synthase.

## FAT METABOLISM

Fat is an energy dense substrate capable of yielding 9kcal.g$^{-1}$ (in comparison to 4kcal.g$^{-1}$ from carbohydrate). It is stored as triglyceride in adipose tissue, each triglyceride being composed of three fatty acid chains and a glycerol molecule (Figure 3). In times of adequate nutrition, circulating insulin stimulates lipogenesis. When carbohydrate intake or stores (glycogen) are insufficient for the energy requirements of the body, glucagon causes triglycerides to be broken down into fatty acids and glycerol by the action of the enzyme lipase. Fatty acids are cleaved into 2-carbon molecules (β-oxidation) which are then converted into acetyl coenzyme A which can either feed into the citric acid cycle or be a substrate for gluconeogenesis to generate glucose. Glycerol can also be used in gluconeogenesis.

## PROTEIN METABOLISM

Proteins are large molecules consisting of varying combinations of amino acids linked together. They have numerous important roles in the structure and function of the body such as in muscle, haemoglobin, hormones, fibrin and receptors. They are only used as a source of energy in extreme circumstances. Amino acids consist of carbon, hydrogen, oxygen and an amine group (Figure 4), with some containing sulphur, phosphorous or iron atoms. Branched chain amino acids (such as valine, leucine and isoleucine) are commonly used for energy production. The amino acids undergo oxidative deamination or transamination (the amine group removed from the molecule and converted into urea) to form ketoacids, which can then be broken down to form acetyl coenzyme A. This can be used in the citric acid cycle to produce ATP.

In normal subjects the metabolism of protein is inhibited by the presence of circulating glucose. Only a small glucose intake is required to increase the circulating concentration of insulin to levels which block protein breakdown. Use of high energy glucose drinks on the morning of surgery as part of enhanced recovery programs aims to avoid or delay the catabolic response to surgery.

## STARVATION

Starvation is defined as an absence or inadequacy of exogenous intake and may be partial or complete. The body must survive totally or in part on its internal stores. Starvation initially results in a fall in plasma glucose which triggers a decrease in insulin and an increase in glucagon secretion. This halts glycogenesis and starts the process of glycogenolysis. Glycogen is broken down to glucose-6-phosphate and then to glucose which is distributed to the organs that exclusively metabolise glucose (the brain and heart). Glycogen stores last 24 hours, after which other sources of energy must be utilized.

Gluconeogenesis is the process by which glucose is formed form a number of alternative endogenous sources - it is effectively glycolysis in reverse, and so requires energy to proceed. Glucose production is essential as the brain cannot utilize energy substrates such as lactate

**Table 1.** *Summary of ATP molecules produced at each stage of glucose metabolism*

| **Stage** | | **Net number of ATP produced** |
|---|---|---|
| Glycolysis | **2ATP** (4 produced – 2 consumed) | **2** |
| Conversion of pyruvate to acetyl coA | $2[3(2\ NADH + 2H^+)] =$ **$2 \times 6ATP$** | **12** |
| Citric acid cycle | **$2 \times 1$ ATP**<br>$2 \times 3[3(\ NADH + H^+)] =$ **$2 \times 9ATP$**<br>$2 \times 2(FADH_2) =$ **$2 \times 2ATP$** | **24** |
| **Total ATP** | **2 + 12 + 24** | **38** |

and pyruvate. Potential substrates for gluconeogenesis are pyruvate, lactate, amino acids and glycerol. Fatty acids and ketones cannot be used for gluconeogenesis. The process is stimulated by a decrease in circulating insulin concentration and increased glucagon.

Amino acid use for gluconeogenesis is inefficient with 1.75g of protein being required to produce 1g of glucose. Glycerol, produced by breakdown of triglycerides, and lactate are alternatives. They are transported to the liver to provide substrates for gluconeogensis.

After 48 hours of starvation, or extreme exercise such as running a marathon, ketogenesis becomes a major source of energy. It has been referred to as the 'final adaptive process in starvation'. Free fatty acids are metabolized to form ketone bodies, namely acetoacetate and β-hydroxybutarate. These can be used by all tissue (including the brain) as an alternative energy source to glucose. Ketogenesis occurs in response to low insulin and high glucagons levels. It can be profound in diabetic ketoacidosis when no insulin is present, allowing glucagon to exert its influence unopposed.

The metabolic processes described in this article are essential for life and are subject to massive variation depending on the individual's environment, nutritional state and level of exercise or stress. The clinical relevance becomes clear when considering performing major surgery on a fasted patient. Maintaining an adequate nutritional intake preoperatively and oxygen delivery intraoperatively are vitally important in avoiding detrimental catabolic and anaerobic metabolic processes in patients undergoing major surgery and those with critical illness.

Update in
Anaesthesia

# Apnoea and pre-oxygenation

*This article was originally published as Anaesthesia Tutorial of the Week 297 (2013)*

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**Summary**

Pre-oxygenation is:

safe

simple

cheap

effective

well-tolerated

This article provides a compelling argument in favour of pre-oxygenation prior to all general anaesthesia.

## INTRODUCTION

The purpose of pre-oxygenation is to increase physiological stores of oxygen in order to prolong the time to desaturation during a period of apnoea, a situation that is usual following induction of anaesthesia. This is particularly the case during a rapid sequence induction, when positive pressure ventilation is avoided prior to intubation of the trachea. Pre-oxygenation can also be thought of as *denitrogenation* – highlighting the fact that it is the nitrogen within the lungs that is being displaced by a high inspired oxygen concentration.

The rate of oxygen desaturation is influenced by the balance between oxygen stores and consumption. Oxygen is stored in the body within the lungs, blood and tissues. In the context of pre-oxygenation, the greatest increase in oxygen store is within the lungs; more specifically, the functional residual capacity (FRC), see Figure 1. Lung oxygen reserves are a product of the fractional concentration of oxygen within the alveoli (which we estimate by measuring the oxygen fraction in expired gas – $FeO_2$) and FRC. Ventilation-perfusion (V/Q) mismatch, particularly shunt, is an additional factor that affects the oxygen content of blood. This may be influenced by the relationship between the FRC and the closing capacity (the lung volume at which airway closure first occurs during expiration). Oxygen consumption is influenced by metabolic rate. Shorter time to desaturation occurs in certain clinical scenarios including obesity, sepsis, pregnancy and in children.

## FUNCTIONAL RESIDUAL CAPACITY

The FRC is the volume of gas remaining in the lungs at the end of a normal tidal expiration and reflects the balance between the tendencies for the chest wall to expand outwards (due to tone in the chest wall muscles) and the lungs to collapse inwards. The spirometry diagram in Figure 1 depicts the FRC and other lung volumes. In a healthy adult the FRC amounts to 30ml.kg$^{-1}$, totalling 2100ml in a 70kg adult. However, many patients presenting for surgery have a reduced FRC, which in turn reduces the lung's oxygen store. Reasons for this include obesity, pregnancy, anaesthesia (with or without neuromuscular blockade) and lung disease. Nonetheless, pre-oxygenation is still beneficial in comparison to breathing room air.

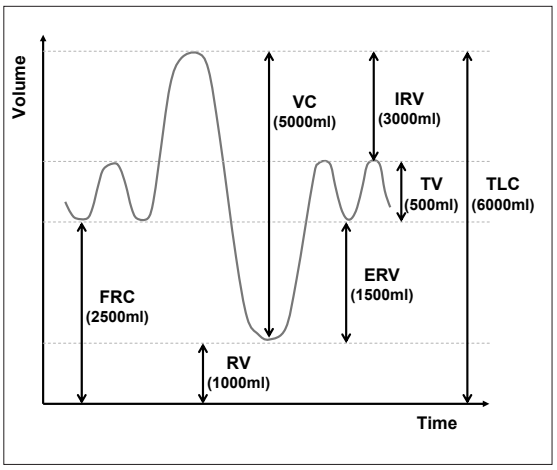


**Figure 1.** *Spirometry trace depicting lung volumes and capacities. IRV = Inspiratory reserve volume, TV = Tidal volume, ERV = Expiratory reserve volume, RC = Residual capacity, VC = Vital capacity, FRC = Functional residual capacity, TLC = Total lung capacity*

### Calculating oxygen reserves

It is possible to calculate oxygen delivery and consumption, to demonstrate the effects of pre-oxygenation:

The alveolar gas equation is used to calculate $P_AO_2$:

$$P_AO_2 = P_IO_2 - [P_ACO_2/R]$$

When breathing air (21% $O_2$):

$$P_AO_2 = 0.21 \times (101.3 - 6.7) - 5.3/0.8 = 13.2kPa$$

This is equivalent to 13% (273ml) of oxygen in an FRC of 2100ml, the remaining contents being 75% nitrogen, and the 7% water vapour and 5% carbon dioxide used in the alveolar gas equation calculation. For the purposes of calculation, oxygen consumption at rest is considered to be 3.5ml.kg$^{-1}$.min$^{-1}$.

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Continuing with the example of a 70 kg adult, approximately 250ml.min$^{-1}$ of oxygen is consumed. Thus, in this model, the FRC provides a reservoir of oxygen equivalent to 70 seconds worth of oxygen consumption. Not all of this oxygen can be extracted from the alveoli; once the $PAO_2$ falls below 6kPa, there will be little concentration gradient to maintain flux of oxygen to haemoglobin. The amount of useable oxygen in this reservoir is therefore likely to be only around 150ml. Actual time to desaturation depends on a complex set of factors as described above.

Pre-oxygenation is a highly efficacious way of extending the time to exhaustion of oxygen reserves and desaturation.

When breathing 100% oxygen*:

$$PAO_2 = (101.3 - 6.7) - [5.3/0.8] = 88kPa*$$

This is equivalent to approximately 88% (1800ml) of oxygen in the FRC – equivalent to more than seven minutes worth of oxygen consumption, or around ten times the amount of useable oxygen compared to breathing room air.

This demonstrates that replacing the nitrogen in the FRC with oxygen greatly increases available reserves.

** Note that this is a theoretical figure. Achieving perfect pre-oxygenation is often not possible; aiming for an $ETO_2$ > 85% is a good goal. This will still give over 1500ml of pulmonary oxygen in this example.*

## SHUNT

As well as providing a smaller reservoir of oxygen, the relevance of a reduced FRC extends to V/Q mismatch. The closing capacity is the lung volume at which small airways will close. If the FRC falls within the closing capacity, airways will close during tidal breathing, resulting in alveoli that are perfused, but not ventilated. This is known as shunt, a phenomenon that is not improved by the administration of higher concentrations of oxygen.

## APNOEA

During the apnoeic period, oxygen continues to be taken up into the blood from the lungs. The uptake of oxygen from the lungs far exceeds the return of carbon dioxide from the blood to the alveoli, due to the body's extensive buffering systems which absorb large quantities of $CO_2$. This net loss of volume leads to development of a negative pressure in the lungs.

If the upper airway is *patent*, gas will be continually drawn via the trachea into the lungs, equalising the pressure gradient. If the gas in the upper airway is 100% oxygen, this pressure gradient can be maintained for a long period - theoretical modelling suggests that this time may exceed 30 minutes. This phenomenon, also referred to as the *oxygen elevator*, can significantly prolong the time until desaturation. It should be noted that carbon dioxide is not transferred out of the lung during this process, so there will be a gradual rise in $PaCO_2$.

If the gas in the upper airway has a low fraction of oxygen (e.g. air), then nitrogen will build up in the lungs, effectively being concentrated, resulting in loss of the pressure gradient and cessation of flow. If the airway is *obstructed* during this time, there will be rapid development of negative intrapulmonary pressure. Not only will this result in missing the benefits of the oxygen elevator, but may cause airway collapse and pulmonary oedema.

This reinforces the benefits of maintenance of a patent airway and application of 100% oxygen as good practice during apnoea at induction of anaesthesia.

## PRACTICALITIES

Various methods have been described to achieve the process of pre-oxygenation. A consistent feature is the requirement for a tightly fitting mask, with the avoidance of any leak which would allow entrainment of room air, and therefore nitrogen. Selection of an appropriate size of mask is important.

Difficulty achieving a good seal may be found with bearded or edentulous patients in particular. In situations where you are unable to prevent leaks or the patient is phobic of the mask, an alternative is to ask the patient to form a seal around the catheter-mount with the mask removed, ensuring that they do not breathe through the nose (consider the use of a nose clip). This can be useful for patients who suffer from claustrophobia (fear of enclosed spaces).

### Timing

The necessary duration of pre-oxygenation has been debated and studied extensively, with options including three minutes of tidal breathing, four vital capacity breaths in 30 seconds or eight vital capacity breaths in 60 seconds. To some extent these fixed regimens are unnecessary in the presence of end tidal oxygen monitoring ($ETO_2$). If this monitoring is available it is possible to observe the rise in $ETO_2$ on a breath-by-breath basis, with an endpoint of achieving an $ETO_2$ > 85% (100% is not achievable due to the presence of $CO_2$ and water vapour). The actual time required varies between patients; it may be achieved more quickly than three minutes, especially if a patient has a relatively small FRC, while in certain circumstances it can take longer. In the absence of $ETO_2$ monitoring, either 3 minutes of tidal breathing or eight vital capacity breaths in 60 seconds are recommended. Use of four vital capacity breaths in 30 seconds has been found to be inferior to the other two methods. With either method, it is advantageous for the patient to exhale completely (down to residual volume) prior to the start of pre-oxygenation.

The filling of the FRC with oxygen can be described by a wash-in curve and the contrasting process of de-nitrogenation is represented by a wash-out curve. Both processes are negatively exponential and allow for an understanding of the methods for pre-oxygenation suggested.

### Time constants

The nitrogen wash-out curve corresponds to the formula:

$$y = a.e^{-kt}$$

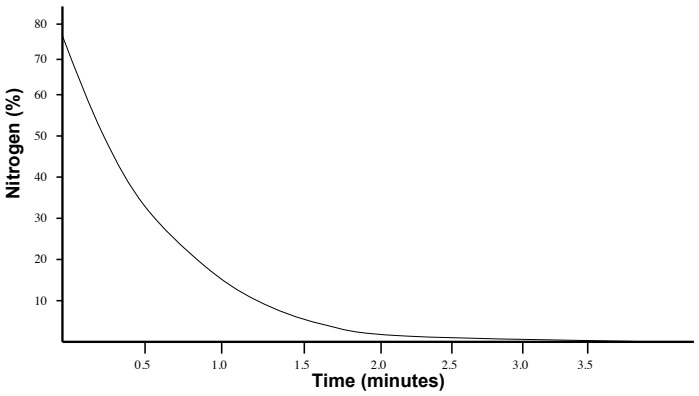


**Figure 2.** *The nitrogen wash-out curve during pre-oxygenation*

The time constant of an exponential process relates to the time taken for the value of the exponential (in this case the amount of nitrogen in the FRC) to fall to 37% of its previous value. As a result, after four time constants the process will be 98% complete. The time constant relates to the ratio of volume and flow, i.e. FRC and alveolar minute ventilation (VA). For a 70kg patient with a tidal volume of 490ml ($7ml.kg^{-1}$), a respiratory rate of 12 breaths per minute and anatomical dead space of 140ml ($2ml.kg^{-1}$), VA would be $4200ml.min^{-1}$. As noted above, the FRC is 2100ml. Consequently, a time constant of 0.5 minutes is obtained.

From the calculations above, it is evident that after two minutes (four time constants), 98% of the nitrogen in the FRC will have been washed out and replaced with oxygen. A three minute period ensures a safe margin to account for inter-patient variability.

### Breathing system

The breathing system employed during pre-oxygenation should be taken into consideration. When using a circle system it is necessary to ensure an oxygen flow greater than minute ventilation (MV); i.e. at least $6L.min^{-1}$ in a 70kg patient, in order to maintain 100% oxygen within the circuit. Higher flows ($15L.min^{-1}$) are required if vital capacity breaths, rather than tidal breathing, are taking place (due to the increased MV). With a Mapleson D breathing system (Bain circuit) high oxygen flows (2-3 x MV) are required to prevent re-breathing of expired nitrogen and carbon dioxide.

**Editor's notes**

Where oxygen concentrators are used, the oxygen flow is generally limited to $10L.min^{-1}$, making effective delivery of 100% oxygen through a circle or drawover system impossible. One alternative is to fill high capacity bin bag with oxygen and use this as a reservoir attached to the open end of the the drawover system.

## OTHER CONSIDERATIONS

### Obesity

As noted above, the balance between oxygen stores and oxygen consumption determines the rate of desaturation of a patient. Obese patients have a reduced FRC (store) and a greater metabolic rate (consumption). Consequently, their rate of desaturation is notably greater than the non-obese patient. Coupled with a higher rate of difficult bag mask ventilation and difficult intubation, there is a key role for pre-oxygenation in the obese patient in order to maximise the $PAO_2$ (store). A further adjustment to the process is to sit such patients up. This improves matters by increasing the FRC compared to the supine position, with a 25 degree elevation having been shown to significantly reduce the rate of desaturation in obese patients.

### Pregnancy

Pre-oxygenation has an important role to play in the anaesthetic management of the pregnant patient. The enlarging uterus causes elevation of the diaphragm and a reduced FRC, within which the closing capacity may fall. Metabolic demand increases due to the growing foetus and placenta. Therefore, desaturation occurs more rapidly. Furthermore, airway management in obstetric patients is known to be difficult more frequently than in the general surgical population. Pre-oxygenation provides an added margin of safety if efforts at establishing an airway become prolonged. Unlike obesity the 25 degree head up position has not been shown to reduce the rate of desaturation of pregnant patients.

### Sepsis

In critically ill and septic patients the time taken to desaturate can be greatly reduced. Factors that influence this are an increase in oxygen demand and cardiac output, and reduced tissue oxygen extraction associated with sepsis. It is likely that V/Q mismatch is increased, further compounding the problem of rapid desaturation. In such patients achieving ideal oxygen saturations approaching 100% can be difficult, even with the administration of 100% oxygen. However, good quality de-nitrogenation of the patient's FRC prior to intubation (and associated apnoea) will still help to delay a precipitous fall in oxygen saturation.

### Paediatrics

Children may be less likely to tolerate the process of pre-oxygenation. However, its use should be carefully considered as children have a higher metabolic rate than adults and de-saturate more quickly as a result. Many children will cooperate with the process when it is explained to them and efforts should be made to do this in individuals at high risk of desaturation.

### Tracheal extubation

Much of the preceding text has referred to the use of pre-oxygenation prior to induction of anaesthesia. It should be noted that the same principles of increasing oxygen stores within the FRC are of use prior to tracheal extubation, providing additional oxygen stores in the event of an airway complication at this time. Correlation with $ETO_2$ is of use to ensure adequate de-nitrogenation.

## CAUTIONS

One deleterious effect of the administration of 100% oxygen is atelectasis. This results from oxygen uptake from poorly-ventilated alveoli leading to alveolar collapse. However, this problem may be easily remedied through the use of recruitment manoeuvres and

should not be seen as a contraindication to the appropriate use of pre-oxygenation. Once a secure airway has been obtained, the $FIO_2$ may be reduced to an appropriate level for that particular patient.

Rare circumstances where the risk-benefit balance may not be in favour of the use of 100% oxygen are in patients receiving bleomycin treatment and those with conditions in which the pulmonary vasculature is sensitive to changes in the $FIO_2$. The latter are a special case where high $FIO_2$ may need to be avoided. Expert advice should be sought. Bleomycin is asssociated with a pneumonitis, which can be potentiated by a high $FIO_2$. The short periods of time for which 100% oxygen is used in the context of pre-oxygenation are deemed to be safe when balanced with a lower maintenance $FIO_2$. Further detail about this is beyond the scope of this article.


## SUMMARY

When properly performed, pre-oxygenation prolongs the time until desaturation when apnoea occurs. Maintenance of a patent airway with continued application of 100% oxygen during apnoea further prolongs the time to desaturation.


## REFERENCES


1. Tanoubi I, Drolet P, Donati F. Optimizing pre-oxygenation in adults. *Can J Anesth* 2009; **56**: 449–66.
2. Hardman JG, Wills JS, Aitkenhead AR. Factors determining the onset and course of hypoxaemia during apnoea: an investigation using physiological modelling. *Anesth Analg* 2000; **90**: 619–24.
3. Wills J, Sirian R. Physiology of apnoea and the benefits of pre-oxygenation. *Continuing Education in Anaesthesia, Critical Care & Pain* (2009) Volume 9 Number 4: 105 -8.

Update in Anaesthesia

# Expanding your anaesthetic technique: an overview on the efficacy of using a lidocaine infusion as part of a balanced anaesthetic

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**Summary**

Lidocaine has many properties that make it useful as an adjunct to anaesthesia. It is used routinely at the University of Virginia and other centers in the US and Europe, and we have successfully used it in the management of neurosurgical cases in Africa.

In this review, we will discuss the clinical use of intraoperative intravenous lidocaine. Additionally, we will describe the method for its use and will identify potential situations where intravenous lidocaine may be of benefit to you in your anaesthetic practice. Lidocaine is relatively inexpensive and safe to administer. We hope this review will educate you about the benefits of using lidocaine beyond its traditional role, and enable you to use lidocaine safely in your anesthetic practice.

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## INTRODUCTION

Lidocaine (lignocaine) is an amide local anaesthetic. Local anaesthetics were first discovered in the 19th century when cocaine was isolated from cocoa leaves. Cocaine was used medically for the first time in 1884 as a topical anesthetic, and was used intrathecally for the first time in 1885. Lidocaine was subsequently developed in 1943. Lidocaine acts primarily via sodium channel blockade, but also has other actions, such as inhibition of G-protein receptors and NMDA receptors. It is metabolized primarily by the liver and is excreted by the kidneys in the form of active metabolites and unchanged drug. It has analgesic, anti-hyperalgesic and anti-inflammatory properties.[1,2]

Lidocaine was used initially in the management of arrhythmias. It subsequently was found to be effective as a topical anesthetic and in the administration of regional and neuraxial anesthesia. But lidocaine has utility beyond these uses and new applications are being studied around the world. Specifically, it can be used as an adjunct to general anesthesia when administered as an intravenous infusion. When used in this manner, it has been shown to reduce the minimum alveolar concentration (MAC) of volatile anesthetics. It can help reduce postoperative pain and opioid consumption in patients undergoing specific types of surgery. It also can help reduce postoperative ileus, in part due to its opioid sparing effects. Finally, the use of intra-operative lidocaine infusions can potentially lead to a reduction in thrombosis, cognitive dysfunction and airway irritability in certain populations of patients.

- Lidocaine infusions reduce minimum alveolar concentration (MAC) of volatile anesthetics
- Lidocaine infusions decrease postoperative pain, opioid consumption, and duration of ileus
- Lidocaine infusions may reduce postoperative risk of thrombosis, cognitive dysfunction and airway irritability in certain populations of patients

## BENEFITS OF INTRAVENOUS LIDOCAINE

### Minimum alveolar concentration

General anaesthesia is usually performed with volatile anesthetics, often supplemented with intravenous opioids, and sometimes with regional anesthesia for the prevention and treatment of pain. Unfortunately, the use of volatile anesthetics is often associated with decreases in blood pressure, which can be particularly pronounced in hypovolemic patients or those with cardiac disease. Hence, approaches that would reduce the requirement for volatile anaesthetics would be helpful.

Intravenous lidocaine, when given as a bolus prior to the start of surgery followed by a continuous intravenous infusion, has been shown to reliably reduce the MAC of volatile anaesthetics. For example, one study demonstrated that the addition of intravenous lidocaine as an adjunct to anesthesia with nitrous oxide and halothane reduced MAC by 10-28 %.[3] We showed that intravenous lidocaine resulted in a 35% reduction in sevoflurane end-tidal concentration required to maintain hemodynamic stability. Animal studies have shown even greater reductions in MAC with the use of intravenous lidocaine.[4] Taken together, these studies indicate that the use of a lidocaine infusion intraoperatively probably reduces MAC by approximately 30%. This effect of intra-operative intravenous lidocaine infusions makes lidocaine useful for nearly all types of surgical procedures if the desired endpoint is a reduction in MAC. This may be particularly useful to avoid the undesired effects of specific anesthetic or to reduce cost when more expensive inhaled anesthetics are used.

- An intravenous lidocaine infusion during surgery can reduce the MAC of volatile anesthetics by approximately 30%

### Postoperative pain, opioid consumption, and ileus

As anesthetists, one of our biggest challenges relates to the prevention and management of postoperative pain in our patients. While opioids are the mainstay of postoperative analgesia, their use can be associated

with a variety of adverse effects, including respiratory depression, sedation, and postoperative nausea and vomiting, ileus, and urinary retention. Furthermore, opioids have been shown to be tumor promoting and can potentially put the patient undergoing surgery for cancer resection at risk for cancer recurrence or metastasis. These side effects can be detrimental to the recovery of the patients we are intending to help and can significantly impact morbidity and mortality. Adjuvant medications can help to reduce the amount of opioids that our patients require.

The use of intravenous lidocaine infusions intraoperatively has been studied in a variety of surgeries including open and laparoscopic abdominal surgery, orthopedic surgery, cardiac surgery, head and neck surgery and ambulatory surgery. Analysis of these data shows a benefit of intravenous lidocaine infusions by reducing postoperative pain and opioid consumption, and reduction of ileus, in patients undergoing abdominal surgery.[2,5]

In a recent review article, McCarthy et al, reviewed 16 randomized controlled trials evaluating the use of perioperative intravenous lidocaine infusions for improving postoperative analgesia and enhancing recovery of bowel function. These trials involved 764 patients, 395 patients receiving intraoperative lidocaine and 369 control patients. Patients undergoing open and laparoscopic abdominal surgery were found to have significant reductions in postoperative pain intensity and opioid consumption. Specifically, pain scores were reduced at rest and with cough and movement up to 48 hours postoperatively. Additionally, opioid consumption was reduced by up to 85% in lidocaine-treated patients when compared to controls. Unfortunately these results were not consistent with all types of surgery. No such benefit was shown in patients undergoing tonsillectomy, total hip arthroplasty, or coronary artery bypass surgery.[2]

At this time, it is unclear why lidocaine was ineffective in the prevention of pain in all types of surgery; however, lidocaine has been shown to alleviate visceral pain in animal models by reducing visceromotor reflexes and evoked and spontaneous activity of neurons excited by colorectal distention.[6] This may explain why lidocaine infusions are more effective at reducing pain in patients undergoing abdominal surgery.

In addition to improvement in postoperative pain and a reduction in postoperative opioid consumption, studies have also shown that intraoperative lidocaine infusions have led to a reduction in postoperative ileus.[2,5,7] In the analysis mentioned above, lidocaine infusions resulted in an earlier return of bowel function, with first flatus occurring up to 23 hours earlier and first bowel movement occurring up to 28 hours earlier.[2] Postoperative ileus has many potential complications. It can lead to nausea, vomiting, increased pain and a prolonged hospital stay. It can also lead to a delay in the time to oral intake, which is vital to the recovery of patients who have undergone surgery.

This beneficial effect of intravenous lidocaine on ileus can be partially explained by the reduction in opioid requirements in patients undergoing surgery. Opioids are known to cause ileus, prolonging intestinal transit time. Additionally, lidocaine has been shown to have a direct excitatory effect on intestinal smooth muscle. Lidocaine prevents several nerve reflexes that are activated upon entering the parietal peritoneum, which leads to inhibition of the gut. Lidocaine has also been shown to block the sympathetic innervation of the bowel leading to increased parasympathetic tone, which promotes gut motility.[8]

- Intravenous lidocaine infusions have been shown to reduce postoperative pain and opioid consumption in patients undergoing abdominal surgery
- Intravenous lidocaine infusions have not been shown to decrease postoperative pain and opioid consumption in patients undergoing head and neck surgery, orthopedic surgery or cardiac surgery
- Intravenous lidocaine infusions can lead to a reduction in ileus postoperatively, specifically in patients undergoing abdominal surgery

### Other potential benefits of IV lidocaine

Aside from the benefits of MAC reduction, decreased postoperative pain, decreased opioid need postoperatively, and quicker recovery of bowel function, intravenous lidocaine infusions have other potential benefits for your patient. These include a reduced risk of developing thrombosis (blood clots), cognitive impairment, and airway irritability.

It has been found that epidural administration of local anesthetics reduces the risk of developing venous thrombosis postoperatively.[9] While this effect is multifactorial, the risk of developing thrombosis is partially reduced by systemic administration of local anesthetics. This is likely due to the fact that local anesthetics, like lidocaine, have a profound anti-inflammatory effect.[10] Intravenous lidocaine has been shown to be highly effective in preventing thrombosis after orthopedic surgery.[11] While lidocaine infusions can help prevent the development of thrombosis, they do not increase the risk of bleeding. Therefore, they are safe to use intraoperatively because they reduce your risk of developing a blood clot without increasing your risk of bleeding after surgery.

Animal studies have demonstrated that lidocaine has neuroprotective properties.[12] Lidocaine has been found to attenuate cognitive dysfunction in rats receiving general anesthesia with isoflurane.[13] Likewise, lidocaine infusions can reduce postoperative opioid requirements.[2,7] Opioids can increase the risk of postoperative cognitive dysfunction, especially in elderly patients.[14] Additionally lidocaine has been found to reduce postoperative pain in patients undergoing abdominal surgery.[2,5,7] Pain is also a risk factor for postoperative cognitive dysfunction. Therefore, it is hypothesized intravenous lidocaine infusions can reduce the risk of postoperative cognitive dysfunction in patients, potentially due to its neuroprotective effects, opioid sparing properties and impact on pain reduction.

Lidocaine has been found to be a potent inhibitor of the cough reflex.[15] Studies have demonstrated that intravenous lidocaine can decrease reflex bronchoconstriction in asthmatics.[16,17] This is likely secondary to neural blockade of vagal reflex pathways. Smokers are

also prone to reflex bronchoconstriction. Endotracheal intubation can be especially stimulating to asthmatics and to smokers placing both populations of patients at risk of airway hyperactivity. At UVA we routinely use intravenous lidocaine infusions to help mitigate airway hyperactivity in both asthmatics and smokers.

- Intravenous lidocaine infusions may reduce the risk of developing a blood clot
- Intravenous lidocaine may infusions reduce the risk of postoperative cognitive dysfunction
- Intravenous lidocaine infusions may reduce airway hyperactivity in smokers and asthmatics

## WHEN SHOULD YOU CONSIDER USING INTRAVENOUS LIDOCAINE?

So far, much has been said about the benefits of using an intravenous lidocaine infusion as an adjunct to general anaesthesia. We have seen that intravenous lidocaine infusions can reduce MAC of Halothane by 30%. They can reduce postoperative pain, opioid consumption and ileus in patients undergoing open or laparoscopic abdominal surgery. They can probably reduce the risk of thrombus formation in nearly all patients due their anti-inflammatory effects. Additionally they may reduce the risk of developing postoperative cognitive dysfunction and they may reduce airway hyperactivity in certain patients such as smokers and asthmatics.

Thus, intravenous lidocaine infusions can have measureable benefit. Given these benefits, when might you consider using one to supplement your anaesthetic?

In our practice, we typically use an intravenous lidocaine infusion as an adjunct to anaesthesia in patients undergoing all open and laparoscopic abdominal surgeries who do not have an epidural. Often, an epidural is not used due to the preference of the surgeon or patient factors that make an epidural contraindicated, like coagulopathy, systemic infection, or patient refusal.

We also use intravenous lidocaine infusions in cases where we want to limit the amount of volatile anesthetics we are providing or in cases where we are providing total intravenous anesthesia and we would like to limit the amount of propofol or opioid that we are administering. Situations where you might want to limit the amount of volatile anesthetics include patients with a cardiomyopathy, or patients who may not tolerate a reduction in systemic vascular resistance caused by volatile anaesthetics, such as those with severe peripheral vascular disease or cerebral ischemia. Additionally, by limiting the amount of volatile anesthetic required, the use of lidocaine may help reduce the cost of safely providing anaesthesia.

## HOW TO USE INTRAVENOUS LIDOCAINE IN YOUR PRACTICE

Typically, a lidocaine infusion that provides 30-40mcg.kg$^{-1}$.min$^{-1}$ or 1.8-2.4mg.kg$^{-1}$.h$^{-1}$ is necessary to reach a serum lidocaine concentration necessary to benefit the patient. As a general rule of thumb, providing an infusion of 3mg.min$^{-1}$ in patients weighing more than 80kg and 2mg.min$^{-1}$ in patients between 50-80kg is appropriate. Often, it is beneficial to provide a 1-2mg.kg$^{-1}$ bolus prior to initiating the infusion. Generally, we run the infusion from the onset of anaesthesia, prior to surgical incision, until the end of the procedure. The infusion can be administered postoperatively (for example in the recovery room), but should only be done so under close supervision.

At our institution we have premixed lidocaine infusion bags, which contain 8% lidocaine. Every milliliter (ml) in these bags contains 80mg of lidocaine. It may be difficult for you to obtain premixed lidocaine infusion bags; however, you can make one with the resources available to you.

For instance, if you have lidocaine 2% available at your institution then you can mix 30ml of lidocaine with 70ml of normal saline to give you a solution containing 6mg.ml$^{-1}$. If your patient is 85kg and you would like to run an infusion at 3mg.min$^{-1}$ then you would run your infusion at 0.5ml.min$^{-1}$ or 30ml.h$^{-1}$. This mixture would provide you with a little over 3 hours of intravenous lidocaine.

If you only have lidocaine 1% at your institution and you want to use a 1 liter bag then you would mix 100ml of lidocaine 1% with 900ml of normal saline to give you a solution containing 1mg.ml$^{-1}$ of lidocaine. If your patient is 65kg and you would like to run an infusion at 2mg.min$^{-1}$ then you would run your infusion at 2ml.min-1 or 120ml.h$^{-1}$. This mixture would provide you with over 8 hours of intravenous lidocaine.

Making a lidocaine infusion is easy and relatively inexpensive compared to many of the drugs that we use in anesthesia. It is important however to know the concentration of lidocaine that you are working with and the amount of medication that you are delivering. This is important because there is a risk of developing local anesthetic toxicity if you make the solution improperly or deliver it inappropriately.

It is important to remember that the toxic dose of lidocaine is considered to be 4.5mg.kg$^{-1}$ and this should not be exceeded in a single dose due to the risk of local anesthetic toxicity. We do not advocate for the use of a lidocaine infusion in patients weighing less than 50kg due to the risk of local anesthetic toxicity. Signs of local anesthetic toxicity include slurred speech, peri-oral numbness, a metallic taste in the mouth, seizures and eventually cardiovascular collapse. This can be difficult to identify in patients under general anesthesia and often it is not recognized until more serious consequences are realized.

It is unlikely that your patients will have local anaesthetic toxicity if you use the doses that we have recommended. However, if local anesthetic toxicity is suspected, discontinue the lidocaine infusion immediately. Notify the surgeon and perform advanced cardiac life support paying special attention to the airway, breathing and circulation. Maintain oxygenation, ventilation and support the blood pressure. Provide chest compressions if necessary. Seizures can be treated with benzodiazepines, barbiturates or even propofol. In addition to advanced cardiac life support, Intralipid 20% has been shown to help in situations where severe lidocaine toxicity, suggested by seizures and cardiovascular collapse, is suspected. If you have Intralipid 20%, a bolus of 1.5ml.kg$^{-1}$ is recommended followed by an infusion of 0.25-0.5ml.kg$^{-1}$.min$^{-1}$ for 60 minutes. Further information can be found about the use of Intralipid at **LipidRescue.org.**

- In patients >80kg we recommend a lidocaine infusion of 3mg.min$^{-1}$
- In patients 50-80kg we recommend a lidocaine infusion of 2mg.min$^{-1}$
- For safety reasons do not use a lidocaine infusion in patients under 50kg
- Prior to the start of the infusion we recommend an intravenous bolus of lidocaine of 1.5-2mg.kg$^{-1}$
- Start the infusion immediately after the start of anesthesia before the surgical incision
- Discontinue the infusion at the end of the procedure
- While a lidocaine infusion will help reduce postoperative pain and opioid consumption, you should still administer opioids for pain

## CONCLUSION

When used appropriately, intravenous lidocaine infusions have been shown to be beneficial to patients undergoing general anaesthesia for surgery. The use of an intravenous lidocaine infusion can be used to reduce the MAC of volatile anaesthetics in an effort to reduce the amount of volatile anaesthesia administered. An intravenous lidocaine infusion can also be used in patients undergoing abdominal surgery to reduce postoperative pain, opioid consumption and ileus in patients who do not have an epidural. Lidocaine infusions may additionally reduce the risk of developing a thrombus, postoperative cognitive dysfunction or airway hyperactivity.

We routinely use lidocaine infusions at our institution to supplement general anaesthesia. We hope that this review has provided you with the knowledge and tools to be able to use intravenous lidocaine infusions in your practice.

## REFERENCES


1. Yardeni IZ, Beilin B, Mayburd E, Levinson Y, Bessler H. The effect of perioperative intravenous lidocaine on postoperative pain and immune function. *Anesth Analg* 2009; **109**: 1464-9.
2. McCarthy GC, Megalla SA, Habib AS. Impact of intravenous lidocaine infusion on postoperative analgesia and recovery from surgery: A systematic review of randomized controlled trials. *Drugs* 2010; **70**: 1149-63.
3. Himes RS, Jr., DiFazio CA, Burney RG. Effects of lidocaine on the anesthetic requirements for nitrous oxide and halothane. *Anesthesiology* 1977; **47**: 437-40.
4. Pypendop BH, Ilkiw JE. The effects of intravenous lidocaine administration on the minimum alveolar concentration of isoflurane in cats. *Anesth Analg* 2005; **100**: 97-101.
5. Marret E, Rolin M, Beaussier M, Bonnet F. Meta-analysis of intravenous lidocaine and postoperative recovery after abdominal surgery. *Br J Surg* 2008; **95**: 1331-8.
6. Ness TJ. Intravenous lidocaine inhibits visceral nociceptive reflexes and spinal neurons in the rat. *Anesthesiology* 2000; **92**: 1685-91.
7. Vigneaut L, Turgeon A, Cote D, Lauzier Fea. Perioperative intravenous lidocaine infusion for postoperative pain control: A meta-analysis of randomized controlled trials. *Canadian Journal of Anaesthesia* 2011; **58**: 22-37.
8. Rimback G, Cassuto J, Tollesson PO. Treatment of postoperative paralytic ileus by intravenous lidocaine infusion. *Anesth Analg* 1990; **70**: 414-9.
9. Rodgers A, Walker N, Schug S, McKee A, Kehlet H, van Zundert A, et al. Reduction of postoperative mortality and morbidity with epidural or spinal anaesthesia: Results from overview of randomised trials. *BMJ* 2000; **321**: 1493.
10. Hollmann MW, Wieczorek KS, Smart M, Durieux ME. Epidural anesthesia prevents hypercoagulation in patients undergoing major orthopedic surgery. *Reg Anesth Pain Med* 2001; **26:** 215-22.
11. Cooke ED, Bowcock SA, Lloyd MJ, Pilcher MF. Intravenous lignocaine in prevention of deep venous thrombosis after elective hip surgery. *Lancet* 1977; **2**: 797-9.
12. Lei B, Cottrell JE, Kass IS. Neuroprotective effect of low-dose lidocaine in a rat model of transient focal cerebral ischemia. *Anesthesiology*. 2001; 95 :445-51.
13. Lin D, Cao L, Wang Z, Li J, Washington JM, Zuo Z. Lidocaine attenuates cognitive impairment after isoflurane anesthesia in old rats. *Behav Brain Res*. 2012; **228**: 319-27.
14. Fong HK, Sands LP, Leung JM. The role of postoperative analgesia in delirium and cognitive decline in elderly patients: A systematic review. *Anesth Analg*. 2006; **102**: 1255-66.
15. Nishino T, Hiraga K, Sugimori K. Effects of IV Lignocaine on airway reflexes elicited by irritation of the tracheal mucosa in humans anaesthetized with enflurane. *Br J Anaesth* 1990; **64**: 682-7.
16. Groeben H, Foster WM, Brown RH. Intravenous lidocaine and oral mexiletine block reflex bronchoconstriction in asthmatic subjects. *Am J Respir Crit Care Med* 1996; **154**: 885-8.
17. Adamzik M, Groeben H, Farahani R, Lehmann N, Peters J. Intravenous lidocaine after tracheal intubation mitigates bronchoconstriction in patients with asthma. *Anesth Analg* 2007; **104**: 168-72.

Update in
Anaesthesia

# Perioperative management of patients with coronary stents for non-cardiac surgery

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**Summary**

This article describes the differences between bare metal and drug eluting stents with the implications for duration of antiplatelet therapy. Up to 5% of patients with coronary stents require non-cardiac surgery within one year of placement and so it is important for anaesthetists to have a full understanding of the balance between stopping and continuing antiplatelet drugs.

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## INTRODUCTION

Percutaneous coronary intervention (PCI) is a common method for management of patients with coronary artery disease. PCI has evolved from balloon angioplasty to insertion of coronary stents. Currently, 90% of all PCIs involve the placement of at least one coronary stent. The popularity of cardiac stent insertion for a patient with coronary artery disease (CAD) implies that an increasing number of patients of CAD with coronary stents in place may present to the anaesthesiologist for non-cardiac surgery. It is estimated that 5% of patients who have undergone PCI require non-cardiac surgery within the first year after stenting.[1]

## TYPES OF CORONARY STENTS

Coronary stents may be:

1. Bare metal stent (BMS)
2. Drug eluting stent (DES)
3. Bioabsorbable stent.

Bare metal stents (BMS) were introduced in 1986 as a development of balloon angioplasty, however 10-30% of patients with BMS develope in-stent re-stenosis, due to neointimal hyperplasia and increased thrombogenicity, thus requiring repeat coronary intervention.[2]

The problem of re-stenosis with BMS led to the introduction of drug eluting stents (DES) in 2003. Their design includes a coating of thin polymer containing an antiproliferative substance that inhibits neointimal hyperplasia and so are associated with a lower incidence of in-stent re-stenosis. DES have reduced the need for repeat coronary intervention to about 2-4%.[3]

Over a period of time endothelialization of the stent causes the device to become incorporated into the artery, like a part of the vessel. While near complete endothelialization of BMS occurs within 4-6 weeks of implantation, this process takes several months for DES. Until adequately covered by a layer of endothelial cells, the exposed metal struts of a newly implanted stent are a potent focus for the formation of platelet rich microthrombi, resulting in stent thrombosis. The drug coating in the DES delays re-endothelialization, thus the precautions against thrombosis (i.e. antiplatelet drugs) have to be observed for a longer period than with the BMS.[4]

Bioabsorbable stents are a new generation of stents which, like metal stents, allow vessel healing and restore blood flow. Their main advantage is that the stent gets gradually reabsorbed within the body and does not require surgical removal.[5]

## ANTIPLATELET THERAPY FOR PATIENTS WITH CORONARY STENTS

Activation of platelets is the primary source of stent thrombosis, hence dual antiplatelet therapy combining **aspirin** (acetylsalicyclic acid) and **clopidogrel** (thienopyridine) is the most commonly used regime for coronary stents. A third-generation thienopyridine, **prasugrel** has been recently introduced into clinical practice, it is more potent than others, while clopidogrel has a more predictable patient response.[2,6]

For BMS insertion, a loading dose of 300-600mg clopidogrel is given followed by continued use of both aspirin 150mg and clopidogrel 75mg for 4-6 weeks after the procedure. For DES, the dual antiplatelet therapy is continued for at least a year until the stent is fully endothelialized. For a bioabsorable stent the duration of dual antiplatelet therapy is 6 months. For all types of stents low dose aspirin must be continued for life.[7]

## PERIOPERATIVE CONCERNS IN PATIENTS WITH CORONARY STENTS

The chief concern is to weigh the risk of perioperative stent thrombosis and myocardial infarction (MI) due to abrupt discontinuation of antiplatelet drugs against the risk of excessive surgical bleeding due to continuation of aspirin and clopidogrel.

Since endothelialization may not be complete at the time of surgery, abrupt discontinuation of antiplatelet drugs along with the prothrombotic state of patient during surgery increases the risk of acute perioperative stent thrombosis (Table 1). Premature cessation of dual antiplatelet therapy during the first six weeks

after angioplasty and stenting, can cause a cardiovascular mortality of up to 71%.[8] This risk is likely to be greatest in patients with recently implanted, poorly endothelialized stents.

**Table 1.** *Pathogenesis of perioperative stent thrombosis*

| |
|---|
| *Premature cessation of dual antiplatelet – clopidogrel + aspirin* |
| ↓ |
| *Rebound phenomenon* |
| ↑ inflammatory prothrombotic state |
| ↑ platelet adhesion |
| ↑ COX1 and thromboxane A2 activity |
| **AND** |
| *Surgical intervention* |
| ↑ prothrombotic and inflammatory state |
| ↑ platelet adhesiveness |
| ↑ release of procoagulant factors |
| ↓ fibrinolysis |
| ↑ cytokines, inflammatory mediator release |

However, both aspirin and clopidogrel may result in 'resistance' which is another predisposing factor to stent thrombosis. The incidence of resistance to aspirin and clopidogrel treatment is 10-20%.[9] Pharmacogenomics play a major role in resistance to clinical effect of these two drugs. Aspirin resistance occurs due to cyclooxygenase-1 polymorphism. Cytochrome P450 gene polymorphism and $P2Y_{12}$ receptor polymorphism are involved in clopidogrel resistance. Other factors involved in variability in response to aspirin and clopidogrel are patient non-compliance, inappropriate dosing and inter-drug interactions.[10] Prasugrel, a new antiplatelet drug, like clopidogrel is an irreversible thienopyridine P2Y12 receptor antagonist, having a faster onset of action and results in more effective inhibition of platelet aggregation.[12] Other antiplatelet drugs undergoing clinical trials are thromboxane $A_2$/prostaglandin $H_2$ receptor antagonist S-18886 (tetratroban) and protease-activated receptor (PAR1) antagonist SCH 530348.[11]

Other risk factors for thrombosis are:

- multiple recently implanted DES stents
- renal failure
- diabetes
- low cardiac ejection fraction, and
- procedures involving bifurcation lesions.

Despite concerns regarding perioperative bleeding, the protective effect of clopidogrel against MI, stroke and death are proven.[1,12,,13,14]

## PERIOPERATIVE MANAGEMENT OF PATIENTS WITH CORONARY STENTS

It is essential to know the indication for stenting, the date of implantation, the type of stent used, the patient's current oral antiplatelet drug therapy and its duration. It is important that the decision regarding perioperative management is taken in consultation with the cardiologist. The patient's haemorrhagic and thrombotic risk should be assessed (Table 2).

**Table 2.** *Assessment of haemorrhagic and thrombotic risk in patients with coronary stents*

**A. Haemorrhagic risk**

- What is the planned operation and anaesthetic technique?
- How necessary is the surgery?
- What is the perceived haemorrhagic risk for the surgical procedure?
- What is the urgency of the surgery?
- Can the surgery be delayed?
- Is there any alternative to surgery available?
- Are there any other haemorrhagic risk factors?
- What will be the consequences of excessive bleeding?

**B. Thrombotic risk**

- When was the stent placed?
- What type of stent has been inserted?
- Number of stents and their location
- Past history of stent thrombosis
- What antiplatelet regime is being followed?
- What is the duration of therapy that has been recommended?
- Are there any associated risk factors e.g. diabetes, renal impairment, low left ventricular ejection fraction?

### Timing of elective surgery after stent placement

Elective non-cardiac surgery should be deferred for at least 6 weeks and ideally 3 months after placement of BMS; and for at least 1 year after DES placement (Table 3). The longer surgery is delayed after stent placement, the lower the risk of **major adverse cardiac events** (MACEs).[14,15]

### Management of antiplatelet therapy

Dual antiplatelet therapy should be continued in all patients with coronary stents presenting for surgery. However, if there is a high risk of surgical bleeding then clopidogrel should be stopped 5-7 days before surgery and monotherapy with aspirin should be continued. Clopidogrel should be restarted as soon as possible post surgery.[14,16,17] Cessation of aspirin therapy may be considered during intracranial surgery and transuretheral resection of prostrate as these procedures

**Table 3.** *Timing of elective surgery after PCI (PCI- percutaneous coronary intervention; BMS- bare metal stents; DES- drug eluting stents)*

| Procedure | Timing of elective surgery after intervention |
|---|---|
| Angioplasty without stenting | 2-4 weeks after placement |
| PCI and BMS | 6 weeks after placement |
| PCI and DES | 12 months after placement |

are associated with an increased risk of bleeding, but only after contemplating the risk-benefit ratio.[18]

In patients presenting for emergency surgery, if the length of dual antiplatelet medication is less than 6 months, then both medications (aspirin and clopidogrel) should be continued. If more than 6 months, discontinue clopidogrel but continue aspirin therapy.

*Bridging therapy* with short-acting platelet-GPIIb/IIIa receptor inhibitors (tirofiban or eptifibatide) may be considered in patients considered at high risk of stent thrombosis, in whom either clopidogrel, or both clopidogrel and aspirin, have been stopped. GPIIb/IIIa inhibitors prevent platelet aggregation and displace fibrinogen from GPIIb/IIIa receptors. Antiplatelet therapy should be restarted as soon as possible after surgery.

Heparin has minimal antiplatelet action and so may not be suitable as a drug for bridging therapy.[2,12,15]

### Anaesthesia technique

Both general and regional anaesthesia technique can be used for surgery in patients with coronary stents. It is desirable to crossmatch blood if excessive surgical bleeding is anticipated.

Central neuraxial blocks are contraindicated in patients on dual antiplatelet therapy. Aspirin monotherapy in a dose up to 300mg per day is not a contraindication to neuraxial block. Clopidogrel therapy is an absolute contraindication for neuraxial blockade and should be stopped 5-7 days preoperatively.[19,20] If a patient on dual antiplatelet therapy is undergoing emergency surgery, which involves a high risk of bleeding or if neuraxial block is essential then it may be necessary to give a platelet transfusion before surgery. A platelet count of 50,000mcL$^{-1}$ is sufficient for subarachnoid block and a count of 80,000mcL$^{-1}$ is sufficient for epidural block.[1,21] However it is worth remembering that an adequate platelet count does not guarantee good platelet function.

For patients requiring emergency postoperative PCI for perioperative MI or coronary stent thrombosis, presence of an indwelling epidural catheter may increase the risk of neuraxial haematoma, as these patients must receive antiplatelet and thrombolytic drugs during the PCI. For this reason continuous neuraxial anaesthesia via an indwelling catheter is generally avoided in patients with coronary stents.[1,8,12]

### Perioperative monitoring

Besides standard monitoring, it is important to diagnose any myocardial insult as quickly as possible. New onset irregular cardiac rhythm, or pulmonary oedema suggests myocardial ischaemia. Postoperatively, where available, patients should be monitored in a high dependency area. Serial 12-lead electrocardiogram (ECG) is the most cost effective means of detecting ischaemia. Although most perioperative myocardial infarctions are 'silent', onset of angina and other objective signs of ischaemia in patients with coronary stents warrants an urgent cardiology opinion. Stent thrombosis most often presents as an ST elevation MI (STEMI), for which early reperfusion with PCI is recommended. Systemic thrombolytic therapy is not possible since it increases the risk of excessive bleeding and also is less effective than primary PCI.[1]

## CONCLUSION

The number of patients with coronary stents presenting for noncardiac surgery is increasing in many parts of the world. It is essential that the anaesthesiologists are aware of balance of risks and benefits to be considered in patients on antiplatelet therapy, particularly as our surgical colleagues are understandably swayed in favour of avoiding surgical bleeding. These management decisions should involve the patient, surgeon, cardiologist, anaesthesiologist and the haematologist. Surgery for patients with coronary stents should ideally be performed at centers where interventional cardiology and cardiac surgery facilities are available.

## REFERENCES


1. Howard-Alpe GM, J. de Bono, L. Hudsmith W.P. Orr, Foex P, Sear JW. Coronary artery stents and non-cardiac surgery. *Br J Anaesth* 2007; **98**: 560-74.
2. Barash P, Akhtar S. Coronary stents: factors contributing to perioperative major adverse cardiovascular events. *Br J Anaesth* 2010; **105**(S1): i3-i15.
3. Dehmed GJ, Smith KJ. Drug- Eluting Coronary Artery Stents. *Am Fam Physician* 2009; **80**: 1245-51.
4. Chassot P-G, Delabays A, Spahn DR. Perioperative antiplatelet therapy: the case for continuing therapy in patients at risk of myocardial infarction. *Br J Anaesth* 2007; **99**: 316-28.
5. Ormiston JA, Serruys PWS. Contemporary Reviews in Interventional Cardiology: Bioabsorbable coronary stents. *Circ Cardiovasc Interv* 2009; **2**: 255-60.
6. Pothula S, Sanchala VT, Nagappala B, et al. The effect of preoperative antiplatelet/ anticoagulant prophylaxis on postoperative blood loss in cardiac surgery. *Anesth Analg* 2004; **98**: 4-10.
7. Stone GW, Aronow HD. Long term care after PCI: focus on the role of antiplatelet therapy. *Mayo Clin Proc* 2006; **81**: 641-52.
8. Ghassot PG, Marcucci C, Delabays A. Perioperative antiplatelet therapy. *Am Fam Physician* 2009; **82**:1484-89.
9. Alexander KP, Peterson ED. Minimizing the risks of anticoagulants and platelet inhibitors. *Circulation* 2010; **121**:1960-70.

10. Bode C, Zahender M. The use of antiplatelet agents following percutaneous coronary intervention: focus on late stent thrombosis. *Eur Heart J* 2007;9(S D);D10-D19.

11. Husted S. New developments in oral antiplatelet therapy. *Eur Heart J* 2007 ; **9** (S D);D20-D27

12. Newsome LT, Robert SW, Gerancher JC, et al. Coronary artery stents II: Perioperative complications and management. *Anesth Analg* 2008; **107**: 57-90.

13. Iakovou I, Schmidt T, Bonizzani E et al. Incidence, predictors, and outcome of thrombosis after successful implantation of DES. *JAMA* 2005; **293**: 2126-30.

14. Fleisher LA, Beckman JA, Brown KA et al. Guidelines on perioperative cardiovascular evaluation and care for non cardiac surgery: Executive summary: A report of the American College of Cardiology / American Heart Association Task Force on practice guidelines. *J Am Coll Cardiol* 2007; **50**: 1707-32.

15. CSNAZ Guidelines for the use of antiplatelet therapy in patients with coronary stents undergoing non cardiac surgery. [updated 2009 August 12]. Available at: http://www.csanz.edu.au

16. Kovesi T, Royston D. Is there a bleeding problem with platelet-active drug? *Br J Anaesth* 2002; **88:** 159-63.

17. Bertrand ME. When and how to discontinue antiplatelet therapy. *Eur Heart J* 2008; **10**(SA); A35-41.

18. Burger W, Chemnitius JM, Kneissl GD et al. Low-dose aspirin for secondary cardiovascular prevention- cardiovascular risks after its perioperative withdrawl versus bleeding risks with its continuation- review and meta-analysis. *J Intern Med*.2005; **257**: 399-414.

19. Horlocker TT, Wedel DJ,Schroeder DR et al. Preoperative antiplatelet therapy does not increase the risk of spinal hematoma associated with regional anesthesia. *Anesth Analg* 1995; **80**: 303-9.

20. Horlocker TT, Wedel Dj, Rowlingson JC et al. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy. American Society of Regional Anesthesia and Pain Medicine. Evidence – based Guidelines (third edition). *Reg Anesth Pain Med* 2010; **35**: 64-101.

21. Samama CM, Djoudi R, Lecompte T, et al. Perioperative platelet transfusion. Recommendations of the French Health Products Safety Agency. (AFSSAPS). *Minerva Anestesiol* 2006; **72**: 447-52.

Update in Anaesthesia

# Statistics for anaesthetists

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**Summary**

Statistics are used to package raw data (often a large amount) into a form that is concise and has meaning to the reader. An understanding of statistics is important to medical practitioners to update clinical practice by analysis of experimental data and conclusions presented to us in medical journals or by company representatives. Summary conclusions should not be accepted at face value and, unless a study has been conducted rigorously, the results may be inaccurate and open to misinterpretation.

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## INTRODUCTION

"*Facts are stubborn things, statistics are pliable*" (Mark Twain)

"*There are three types of lies... Lies, damn lies and statistics*" (often attributed to Benjamin Disraeli).

## DATA COLLECTION

The basic premise of clinical research is to answer a question, for example, "Is treatment A more effective than treatment B?" In order to answer this question a researcher must identify a sample of individuals that is representative of the target population and use an appropriate study design to collect accurate and reliable data. If this is not done correctly, then the quality of statistical analysis is irrelevant - the findings of the study will be unreliable and potentially false, and so misleading and clinically harmful conclusions will be drawn.

## SAMPLING

A target population is an entire group that a researcher is interested in, but the group is often too large to be accessed by the researcher (e.g. all patients undergoing elective total hip replacements in a country). To overcome the problem of accessibility, a sample is selected whose individual's characteristics are representative of the characteristics of the target population. The most representative sample is a simple random sample, in which every member of the target population has an equal chance of being selected. Statistical assessment of how well a sample's results match the entire target population's true value (sample error) is discussed later.

## STUDY DESIGN

Study design falls into two broad categories:

1. Observational
2. Experimental (also called intervention or treatment).

### Observational studies

Observational studies collect data from a sample group, but they do not introduce any intervention. Examples of observational studies include cross-section studies, cohort studies and case control studies.

*Cross-sectional study*

A cross-sectional study is a snap-shot of a situation at a certain point in time, where measured variables are compared and conclusions made (see Study 1, below).

*Cohort study*

Cohort studies (also called prospective or longitudinal studies) involve following a sample group over a period of time, with measurements taken at intervals. An example is follow up of patients after colonic resection using a novel technique, to define the incidence of cancer recurrence.

*Case control study*

Case control studies (also referred to as longitudinal or retrospective studies) involve selecting a sample group with a known outcome and comparing the subjects to a sample group who do not have the same outcome, but in other ways are similar. Inferences are then made from differences between the groups.

## WORKED EXAMPLES

**Study 1: Cross-sectional study into the risk factors for postoperative nausea and vomiting**

If we wanted to investigate the causes of postoperative nausea and vomiting (PONV), an observational method is appropriate.

*Sampling*

- Defining our target population - this could be all patients undergoing general anaesthetic or be more specific to a certain type of surgery or anaesthetic technique.
- Sampling technique - random sample from the population, e.g. alternate patients.

*Study design*

- Identify an outcome measure - in this case a score of over 5 on the simplified PONV impact scale, would be used to define clinically significant PONV.
- A cross-sectional study could be used appropriately to identify likely risk factors. All possible risk factors should be predefined and that data collected (e.g. age, gender, weight, surgical procedure, past history of PONV).

The major drawback with observational studies is that confounding variables made lead us to draw false conclusion about the relationship between risk factors and outcome (see Box).

*Experimental studies*

In experimental studies the researcher measures the effect of an intervention they expose the sample to. For comparison, a proportion of the randomly selected sample becomes a control group, who do not receive the intervention. Selected subjects are entered into the treatment or control group in a random fashion; this process is called *randomization*, and reduces selection bias.

**Confounding variables**

These are unmeasured variables that influence the measured outcome. The confounding variable may be a third variable that correlates with both measured variables and so falsely leads us to conclude that there is a correlation between the two variables we have measured. For example, consider a study designed to identify a link between surgery performed as a neonate, with subsequent developmental delay. A confounding variable would be low birth weight, as this is likely to correlate with both the need for neonatal surgery and developmental delay, without itself being a causative factor.

Bias can also be reduced by blinding. In this process, the patient or the assessor is blinded from knowing which group a subject is/was allocated to. If both patient and assessor are kept unaware of allocation, this is called double blinding. Double blinded randomized controlled trials (RCTs) generally produce the most reliable results and are viewed as the gold standard. A practical issue associated with RCTs occurs when subjects drop out of either the intervention or control group - when this occurs it is viewed as good practice to analyse the data as if the lost subjects were still within the study - this is known as *intention-to-treat analysis.*

**Bias** is defined as an un-random influence which causes results to deviate from the true value. For example, if a study designed to assess recovery after knee surgery only enrols patients who are willing to come back to the hospital for assessment on day 3 post-operatively, this will introduce a bias towards enrolling fitter, more active patients. **Randomization** and **blinding** are techniques used to reduce both intentional and unintentional bias.

## DESCRIPTIVE STATISTICS

Descriptive statistics are used to present large amounts of data in an interpretable form, using numerical or graphical methods.

### Data types and their representation

Data can come in a number of forms that fit into different categories (Figure 1). The implication of the different categories is that data requires different methods of analysis and presentation depending on its form.

The basic division of categories is between whether the data is qualitative (also referred to as categorical) or quantitative (also referred to as numeric/metric).

*Qualitative data*

This is descriptive in nature and can be further sub-grouped into nominal and ordinal categories. Ordinal data is qualitative data that has a sequential order; examples include American Society of Anaethesiology (ASA) grades, Mallampati score and Visual Analogue

## WORKED EXAMPLES

### Study 2: Difference in speed of onset between two antiemetic drugs

This is a simple example of a study designed to answer the question - Does antiemetic A or antiemetic B have a faster onset?

*Sampling*

- Target population - all patients receiving a general anaesthetic who suffer PONV in recovery and require administration of an antiemetic.
- Random sample or all patients, depending on numbers.

*Study design*

- Randomised double blind study comparing the two groups. A control group is inappropriate in this scenario as it is unethical not to treat PONV. Random allocation of patients into group A (receive drug A) or group B (receive drug B).
- Blinding of assessor to patient group allocation. It is also possible to blind the person administering the drug if they are prepared in pharmacy and labelled as drug A and drug B. Complete blinding may not be possible - if drug A is prochlorperazine, it will cause tachycardia and so the administering nurse will be able to guess that drug A is indeed prochlorperazine.

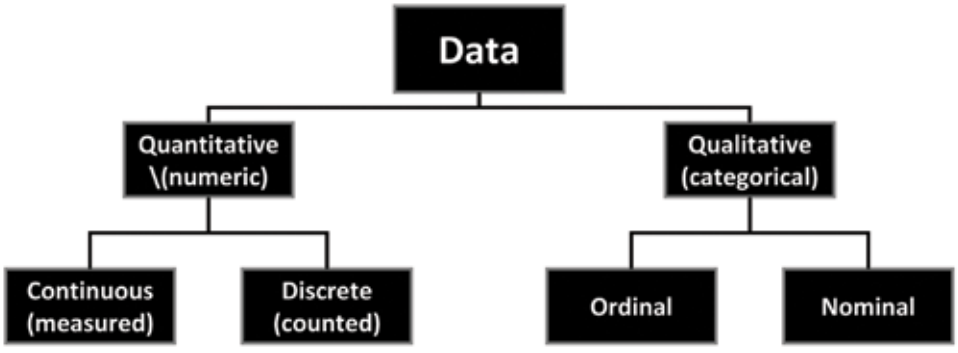



**Figure 1**. *Types of data*

(VAS) scores (e.g. for pain assessment). Nominal data is qualitative data that has no particular order; examples include type of operation, blood group and gender.

*Quantitative data*

This is numeric data that may be placed on a scale and that has units of measurement. Quantitative data can be further sub-grouped into continuous and discrete data. Continuous (also called measured) data can be any number including fractions of a whole numbers; for example height, weight, age. Discrete (also called counted) data can only be a whole number; for example heart rate, number of hospital admissions.

Table 1 shows the appropriate graphical forms of data presentation and display according to the data type.

| Nominal | Ordinal | Discrete | Continuous |
|---|---|---|---|
| Pie Chart | Bar Chart | Bar Chart | Dot Plot |
| Bar Chart | Dot Plot | Dot Plot | Histogram |
| | | Line Chart | |
| | | Histogram | |

**Table 1**. *Data presentation according to data type*

All types of data can be summarized in a tabulated format such as a frequency table (Tables 2 and 3 under 'Worked examples'). When tabulating continuous data there are often too many measured variables for practical use, therefore authors will often present the data in a grouped format (Table 3). The risk in this process is that, if there are too few groups, the level of detail in the data is lost.

## Measures of central tendency, variability and distribution

As well as display in graphical and tabulated format, data can be summarized numerically. It is necessary to summarise any presented data in a way that enables it to be evaluated or compared to other data. In some instances this can be achieved by using percentages or proportions. Data may also be summarized by providing a measure of central tendency (mean, mode, median) along with a measure of the spread of data around it and the type of distribution.

Central tendency is defined as 'the tendency for the values of a variable to cluster around its mean, mode or median'. The type of data being summarized again dictates whether the mean, mode or median are used.

*Mean*

The mean is the sum of all values divided by the number of observations and is only used to summarise quantitative data. Qualitative data is not appropriately summarized with a mean as, although the data may be numeric (e.g. VAS pain score), there is a lack of 'objective proportionality' to it (i.e. a score of eight does not mean the pain is twice as bad as a score of four).

*Mode*

The mode is the value that occurs most frequently and is the only measure of central tendency that can be used with nominal data.

*Median*

The median is the middle value that separates the lower half and upper half of a data set (it will have a decimal place in an even numbered data set). The median is used to summarise ordinal and quantitative data.

There are advantages and disadvantages to using either the mean or median as a summary of quantitative data. The mean includes every value from the data set, which intuitively suggests it offers a more complete summary, however it will also be influenced by extreme outlier observations which may produce a misleading summary. The median is unaffected by extreme outliers. An example of this is length of stay after surgery, where one patient who has a stay of 4 months, amongst many who stay for 4-7 days, will greatly affect the mean but not the median. By convention it is usual to use the mean to summarise normally distributed data and the median for skewed (non-normally distributed) data (see below).

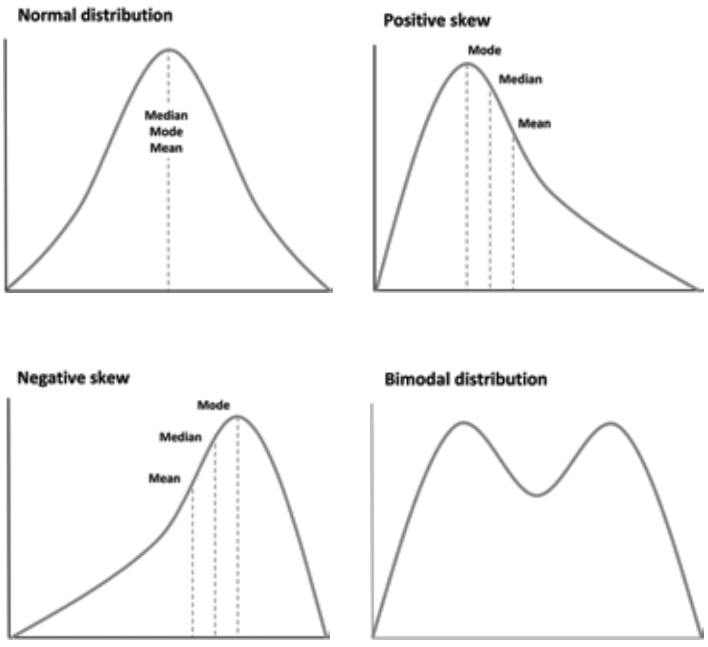
## Spread

Spread refers to how tightly the measured variables of a dataset cluster around the value of central tendency - the tighter the cluster of variables around the central point, the narrower the spread. Spread is used as an indicator of how likely it is that the sample mean represents the true mean of the target population, i.e. a narrower spread corresponds with an increased likelihood of the sample mean representing the target population mean.

The choice of method of measuring spread of data in a sample is influenced by the type of data. As nominal data has no order, spread cannot be used. However spread is used with ordinal and quantitative data, where there is an order to data.

**Figure 2.** *Graphical representation of normal, positively skewed, negatively skewed and bimodal distribution*

When using the median as the description of central tendency i.e. for ordinal or quantitative data, then the measure of spread is the inter-quartile range. The *inter-quartile range* is defined as the range of values between the 75th centile and the 25th centile of the values



measured. In other words it marks out the middle 50% of values within the set of data (with the median being the centre point). The inter-quartile range is used rather than the whole range to eliminate extreme outliers.

When using the mean to indicate central tendency, then the measure of spread is the *standard deviation* (SD). This represents the average distance of all the data values from the mean value, the smaller that value the narrower the spread. Calculating the standard deviation is relatively laborious and involves subtracting the mean from each individual value in the sample and squaring the difference (to eliminate the negative values of those data below the mean), all the squared values are then added with the total being divided by the number of values in the sample minus 1 (n-1) - this figure is called the *variance*. The square route of the variance is the standard deviation.

The type of data distribution influences the choice of the most appropriate method of analysis. Data may be distributed as (see Figure 2):

- normal (parametrically) where the mean, median and mode are all equal,
- positively skewed where the mean is greater than the median,
- negatively skewed where the mean is less than the median,
- bimodal where there are two peaks.

The distributive pattern of data can be assessed visually from graphical data or with statistical tests, such as the Shapiro-Wilkes test.

In normally distributed data, 67% of a sample's values will lie within one standard deviation either side of the mean, 95% lie within two standard deviations either side of the mean and 99% lie within three standard deviations either side of the mean.

## DEDUCTION AND INFERENTIAL STATISTICS

Inferential statistics are used to assess the relevance of the findings of a study to a target population. This includes evaluating the element of chance for different findings between groups, evaluating the difference in baseline characteristics between groups and estimating how closely the sample represents the target population.

### Standard error of the mean and confidence intervals

It is possible to use descriptive statistics (such as the mean) to make informed estimations of how accurate a representation a sample is of the target population. In other words, we can assess sample error using the mean. The sample error is quantified by calculating the standard error of the mean (SEM). The standard error of the mean is inversely proportional to the number of subjects within a sample and is calculated by dividing the standard deviation by the square route of the number of subjects in the sample.

A confidence interval is another method of estimating how similar the results of a sample are likely to be to the true population. The confidence interval is a range of values with a percentage estimation of how likely it is that values of the true population are to be found within this range. For example, if the average diastolic blood pressure in a sample of healthy pregnant women at term was analysed and was

## WORKED EXAMPLES

### Study 1: Cross-sectional study into the risk factors for postoperative nausea and vomiting

A sample of 100 subjects was identified as suffering PONV in the cross-sectional study. To present the relationship of PONV with the type of surgery we could include a frequency table (Table 2) and/or a pie chart (Figure 3). There were twenty-five subjects in each of the four groups. Due to the nominal nature of the data, markers of central tendency and spread are not indicated.

**Table 2.** *Frequency table of PONV according to type of surgery*

| PONV | Orthopaedics | ENT | Plastics | General |
|---|---|---|---|---|
| Yes | 2 | 15 | 1 | 5 |
| No | 23 | 10 | 24 | 20 |
| **Total** | **25** | **25** | **25** | **25** |

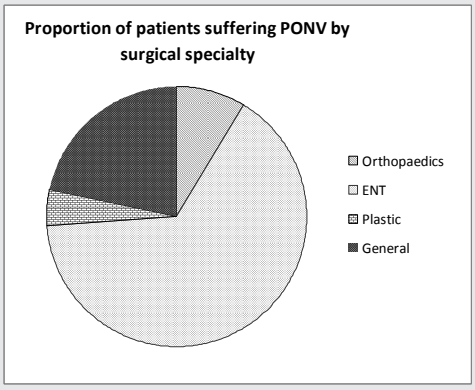



**Table 3.** *Pie chart of PONV according to type of surgery*

**WORKED EXAMPLES**
**Study 2: Difference in speed of onset between two antiemetic drugs**

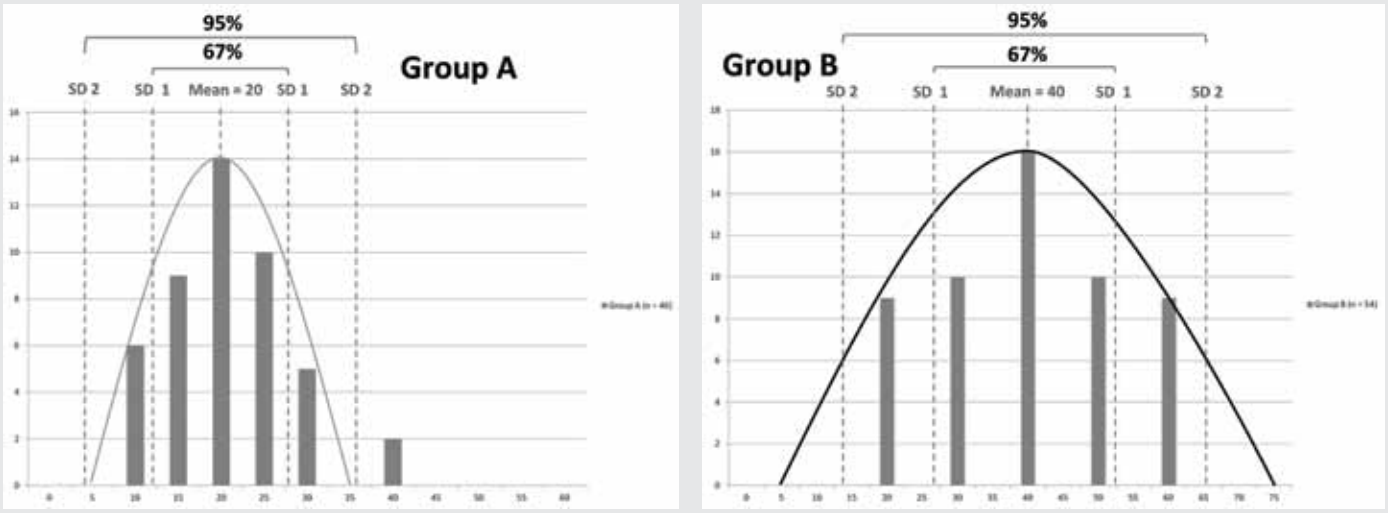
The measured variable is time, which is quantitative continuous data. However the presence of PONV was only measured at five minute intervals, effectively making the data discrete. Visual representation could be using a frequency table (Table 3), a histogram (Figure 4) or frequency curves. Summarising the data with a measure of spread and central tendency is influenced by the distribution of the data. The results (Table 3 and Figure 4) demonstrate a normal distribution, so the mean and standard deviation are used to summarise both groups.

**Table 3.** *Frequency table of time to resolution of PONV after antiemetic administration*

| Minutes | 0 | 5 | 10 | 15 | 20 | 25 | 30 | 35 | 40 | 45 | 50 | 55 | 60 | 65 | 70 | 75 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Group A (n = 46) | 0 | 0 | 6 | 9 | 14 | 10 | 5 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Group B (n = 54) | 0 | 0 | 0 | 0 | 9 | 0 | 10 | 0 | 16 | 0 | 10 | 0 | 9 | 0 | 0 | 0 |

| | Median | Mode | Mean | Variance | SD | SEM |
|---|---|---|---|---|---|---|
| Group A (n = 46) | 20 | 20 | 20 | 60.9 | 7.8 | 1.1 |
| Group B (n = 54) | 40 | 40 | 40 | 170.4 | 13.1 | 1.8 |

**Figure 4.** *Bar chart (histogram) of time to resolution of PONV after antiemetic administration*




normally distributed we could infer, with 95% confidence, that the mean diastolic blood pressure for all healthy women in the population at term fell within two standard deviations either side of the sample mean (and be 99% confident that it fell within three standard deviations).

Confidence intervals can also be calculated from the median, a process that usually requires a computer program, which bases its calculation on the Wilcoxon signed-rank procedure.

**Probability theory and statistical tests**

Fundamental to the usefulness of a study is how confident we are that results are valid; in other words, how likely is it that any difference between groups is the result of the intervention or just down to chance? Inferential statistics rely on the use of probability (the p-value) to analyse the relationships between different variables. The p-value is defined as a measure of the chance of having a particular outcome in a given set of circumstances. If the outcome is certain then the probability is 1, if it is impossible then the probability is 0. The convention in clinical research is to accept that any differences with a p-value of less than 0.05 (i.e. 1 in 20) are likely due to the intervention rather than by chance. The smaller the p-value the more confident a researcher will be that any differences are not due to chance. So, if a study suggests that antiemetic A is better than antiemetic B, with a p-value of 0.05, then we can be 95% certain that this study reflects a true finding in the target population. Put another way, if we repeated the study 20 times, on average one of the studies would give us an untrue result (not representative of the true effect of the anti-emetics on the target population). So, if a journal publishes 20 studies, all with p values of 0.05, then one of these studies is likely to be misleading.

The p-value is calculated from data using statistical tests. The appropriate choice of test depends on a number of factors such as;

the type of data, distribution of the data, the number of groups and whether the data is paired or unpaired. Figure 5 identifies which statistical test is appropriate depending on the characteristics of the data.

Two common statistical tests are the chi square test and the student t test (Table 4). The chi square test is used on qualitative (categorical) data and can be used to test the equality of population properties in two or more independent groups. The student t test is used on quantitative data. Both the chi square and student t test use the sample data to calculate a figure which is used in conjunction with the degrees of freedom (Table 4) from the individual study to calculate a p-value from statistical tables (available on line).

### The 'null hypothesis'

Traditionally all studies should propose a null hypothesis - an assumption that there is no difference between experimental interventions. Therefore if a significant difference in the measured out-come between groups is found then the null hypothesis is rejected.

### Error and power

Error occurs when the wrong conclusions are drawn from a study

**Table 4**. *Statistical equations*

| Term | Definition |
|---|---|
| Chi square test | $\sum (O-E)^2 / E$<br>0 = observed frequency<br>E = expected frequency<br>$\sum$ = sum of |
| Student t test | Difference in sample means / estimated standard error of the difference |
| Standard error of the difference | (SD of group $A^2$ / n group A) +<br>(SD of group $B^2$ / n group B) |
| Degrees of freedom (df) | The number of means which are free to vary.<br>1. For student t test, df = n-1<br>2. For chi square,<br>df = (number of possible outcomes -1) + (number of groups -1) |

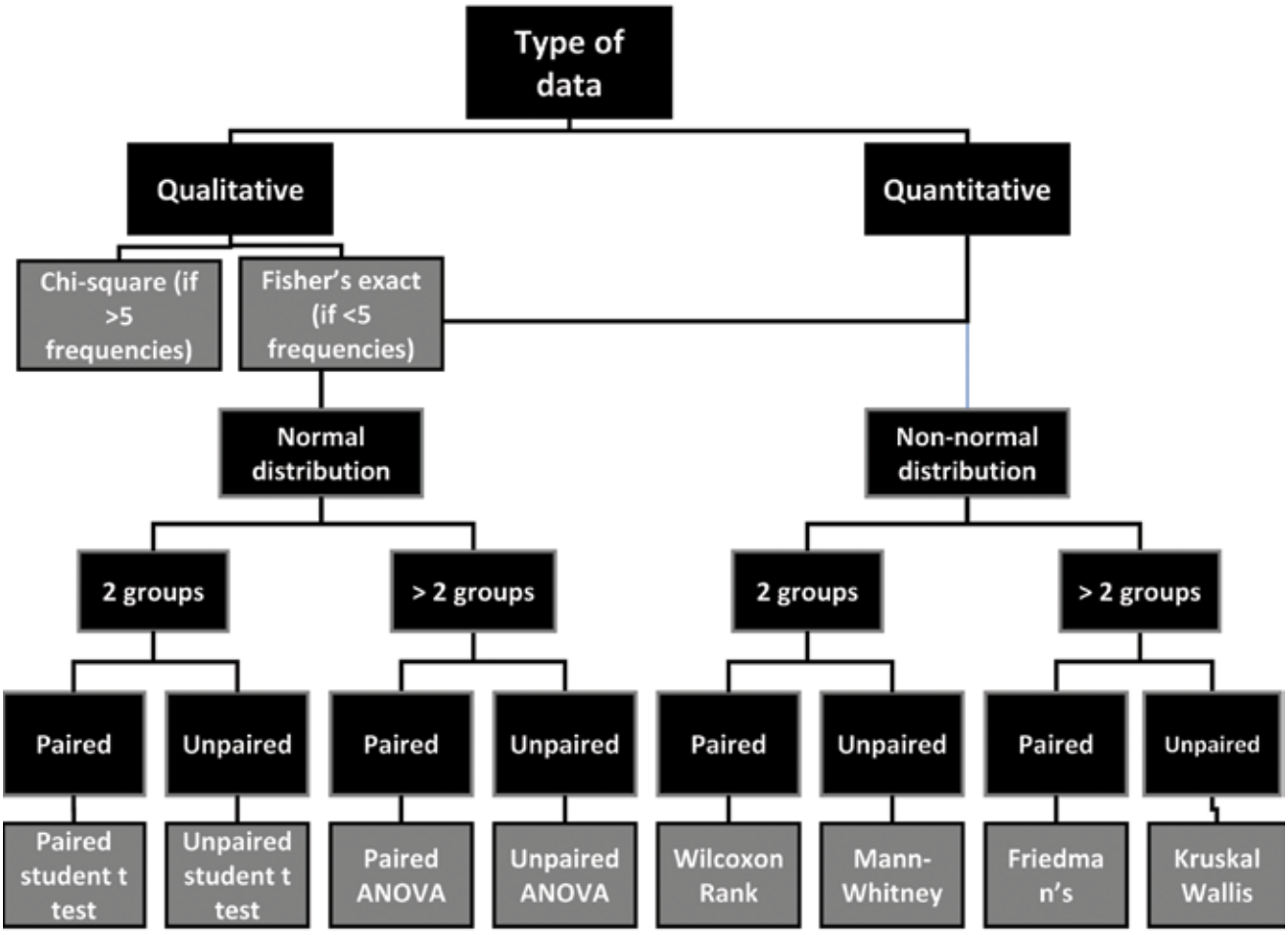



**Figure 5**. *Factors influencing choice of statistical test*

and are classified as type 1 and type 2 errors.

A type 1 error (alpha error or false positive) is the rejection of the null hypothesis inappropriately - assuming a significant effect from an intervention where one does not truly exist. Type 1 errors are related to the p-value. There is a 5% (1 in 20) chance of a type 1 error with a p-value of 0.05.

A type 2 error (beta error, false negative) is the acceptance of the null hypothesis when it is false - concluding there is no effect when there actually is. The chance of a type 2 error is related to sample size. Convention dictates that it is acceptable to allow a 20% chance of a type 2 error. The power of a study is the chance of avoiding a type 2 error and is defined as 1-beta (ie 0.2). Statisticians can calculate the number of patients required to ensure a study has sufficient power prior to the start of the study by using a combination of equations.

## CONCLUSION

Statistical analysis is relevant to all medical practitioners. This article has covered some of the basic principles and enables the reader to have an understanding of the processes data is been put through and which processes are appropriate for different scenarios.

## FURTHER READING


1. Medical statistics from scratch. David Bower. 2002. Wiley, UK.
2. Statistics at square 1. 10th ed. Swinscow TD, Campbell MJ. 2002. BMJ books.


## WORKED EXAMPLES

### Study 1: Cross-sectional study into the risk factors for postoperative nausea and vomiting

We can evaluate the nominal data collected from our observational study with the chi square test. This enables us to infer causation between the type of surgery and the likelihood of PONV by evaluating the element of chance in the results.

To calculate the expected frequency we divide the number of patients defined as suffering PONV by the total number of subjects.

- in this case 23/100 gives us a figure of 0.23;

To find the expected number of patients with PONV according to each surgical specialty we multiply the number of subjects in each group by 0.23;

- 25 x 0.23 = 5.75 as the expected number of patient s to suffer PONV in each group (as there are 25 subjects in each group.

We will also need to calculate the expected frequency of patients to not suffer PONV by following the same process;

- 77/100 = 0.77.
- 25 x 0.77 = 19.5

**Table 5.** *Chi square analysis of data*

| PONV | Orthopaedics | ENT | Plastics | General | Expected Cases |
|---|---|---|---|---|---|
| Yes | 2 | 15 | 1 | 5 | 5.75 |
| No | 23 | 10 | 24 | 20 | 19.25 |
| Chi square score | 3.2 | 29.1 | 5 | 5.5 | - |
| p-value | >0.1 | <0.001 | >0.1 | >0.1 | - |

We can now input the figures relevant to each group into the Chi square equation to calculate if the incidence of PONV is statistically significant compared to what would be expected. To calculate the chi square equation we subtract expected frequency from the observed frequency, square this number and divide the result by the expected frequency. The sum of the values gives the chi square score which is used (with the number of degrees of freedom) to read off a p-value from an appropriate statistical table.

- For the orthopaedic surgery group; ((2-5.75)squared/5.75) + ((23-19.5)squared/19.5) = 3.18

The chi square scores for each group are displayed in Table 5 along with the p-value indicating if observed frequency was statistically different to the observed. The results from this (fictitious) study suggest that, with all else being equal, patients undergoing ENT surgery are significantly more likely to suffer PONV compared to those undergoing general, orthopaedics and plastic surgery.

**WORKED EXAMPLES**
**Study 2: Difference in speed of onset between two antiemetic drugs**

- Null hypothesis: That there is no difference in the speed of onset of antiemetic A when compared to antiemetic B.
- Assess baseline variability between groups. Chi square can be used on multiple variables (age categories, ASA grade, type of surgery, group numbers etc) to assess if there is significant baseline variability between the two sample groups.
- Evaluate the difference in outcome measure between groups using the student t test to calculate a p-value.

Figure 4 and Table 3 demonstrate a difference in findings between the two (fictitious) groups. We use the student t test to evaluate the element of chance as the cause for this difference rather than the intervention.

The student t test formula (difference between sample means/estimated standard error of the difference) gives a "t value" which in conjunction with the number of degrees of freedom can be used to arrive at a p-value from appropriate statistical tables.

The standard error of the difference is calculated by: dividing the square of the standard deviation of group A by the number of subjects in group A then adding this number to the square of the standard deviation in group B divide by the number of subjects in group B

- ((7.8x7.8)/46)+((13.1x13.1)/54 = t value of 4.4

A t value of 4.4 with 98 degrees of freedom (df: n46 - 1 + n54 - 1) produces a p-value of <0.01. Therefore the null hypothesis could be rejected and antiemetic A claimed to have significantly faster onset than antiemetic B.

The standard error of the mean result suggests the results of group A are more likely to represent the mean of the target population than group B.

Update in
Anaesthesia

# Aeromedical transfer for the critically ill patient - an introduction and best practice clinical overview

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**Summary**

This article provides an overview of aeromedical transfer and some of the considerations required in the aviation environment, in terms of equipment, patient care and team safety. The overarching principles of transfer of a critically ill patient also apply and are not discussed in detail here. Aeromedical transfer is an emerging sub-specialty and there are specific considerations that must be addressed in order to make pre-hospital medicine and retrieval of patients effective, safe and (therefore) a viable alternative to road ambulance services.

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## INTRODUCTION

Aeromedical transfer may be used in the management of critically ill or injured patients from point of wounding (POW) to definitive care, or between hospitals as part of the evacuation pathway through the levels of secondary care. It comprises advanced resuscitation techniques and allows life, limb and eyesight saving procedures to be performed en route and in a timely fashion. In many parts of the world road transfers are neither suitable nor time effective, due to geographical remoteness, arduous terrain or hostile human factors. Aeromedical retrieval may provide the optimum solution. There is also increasing evidence to suggest that patients who receive pre-hospital stabilisation have better outcomes.[1,2]

Options for aeromedical transfer can be broadly divided into fixed wing (FW) and rotary wing (RW), each having advantages and disadvantages compared with the other.[3] FW retrieval offers casualty transfer over great distances with rapidity. It often allows an optimised clinical environment, lower ambient noise, easier communication (with patient, colleagues and flight crew) and may operate in a greater range of weather conditions. Despite this, FW retrieval flights are always bound by access to commercial airports, private runways or temporary landing zones (TLZ) and therefore incur an additional road transfer burden at source and destination.

RW retrieval offers robust flexibility and typically allows casualty collection from the point of wounding (POW) or source hospital, and direct disposal at the destination facility. Helipads are increasingly common features of regional trauma centres in developed countries and this strongly supports the RW option. Helicopter platforms incur minimal road transfers as the asset is commonly able to 'land-on'.

In addition, RW platforms tend to have faster response times (i.e. from notification of retrieval requirement to being airborne) and therefore are ideal for enabling 'Golden Hour' pre-hospital trauma care and evacuation timelines. Clinical conditions and working environments vary greatly however, from compact, bespoke platforms with excellent lighting and noise isolation, to large military-type helicopters effective communication is problematic, even with dedicated headsets. The latter range of RW platforms is often seen in civil emergencies or natural disaster evacuations, and therefore the author recommends all retrieval clinicians have an understanding of military RW platforms used in MEDEVAC (e.g. CH-47, Lynx, Merlin, Sea King and Puma UK assets).

In order to ensure safe, auditable and defensible pre-hospital medical retrieval, organisations must have Quality Assurance (QA) and Healthcare Governance (HG) frameworks in place.[3] A named clinical lead is responsible for on-call acute advice and the ownership of appraisal, QA and audit. Equipment and drug supplies must be serviced and maintained to the same standards as the parent secondary care trust (as a minimum).

## PHYSIOLOGY

An understanding of the changing effects of altitude on both casualty and crew is fundamental to the safe aeromedical transfer of patients.[5]

### Boyle's Law

Boyle's law states that, at a constant temperature, the pressure of a gas is inversely proportional to its volume. This is important for any gas filled space. Pathological examples include pneumothorax, pneumocephalus, small bowel obstruction and decompression illness. It also can have effects on equipment, for example cuffed endotracheal tubes. The cuff pressures should be measured prior to take off and during flight and adjusted accordingly.

### Dalton's Law

Dalton's law states that the total pressure of a gas mixture equals the sum of the partial pressures of each gas, as if they existed on their own. As altitude increases, atmospheric pressure decreases and a resultant partial pressure of oxygen falls. Patients requiring a high $FIO_2$ at sea level, may well require intubation and ventilation for aeromedical transfer due to the reduction of oxygen delivery at sub-atmospheric pressures. It is paramount to try and anticipate if patients require intubation and ventilation prior to take off, as space, staffing and resources in the aircraft may be limited.

### Temperature

The partial pressure of water and temperature both fall with increasing altitude, resulting in a significant reduction in the humidity of clinical gases. This can affect patients with respiratory pathology and patients with burns. The ability to heat the clinical cabin area of the aircraft (and road ambulances) should be utilised whenever possible. Fluids will be cooler at altitude and efforts to warm them prior to administration should be made where possible. Solutions include the portable blood product warmers (such as Enflow and Buddy Lite systems). Conversely, hot climates can equally cause problems in some areas of the world for pre-hospital patients. Unwell patients, in a hot environment, can become even further dehydrated through occult huge insensible losses, dressings do not adhere effectively and patients are more likely become agitated and distressed. Efforts should be made to shade the patient on the scene, maintain euvolaemia and use external cooling methods (fans, stripping and spray cooling).

## CLINICAL APPROACH

The medical team normally comprises a critical care physician and a flight nurse or paramedic. Basic retrieval and transfer tasks will be activated with a flight paramedic or nurse only. For critical care retrievals, the staff must be competent in advanced airway and resuscitation skills, with a minimum of two staff for a single patient transfer.[6,7]

The clinical assessment begins as soon as the referral has been activated. A thorough triage history can give important information to both the aviation staff and the clinical team. This will encompass scene safety, weather forecast, journey length, as well as the referring and receiving facilities. A synopsis of the transfer referral will highlight any specific considerations such as multiple casualties, the type of trauma or medical condition, and whether any additional equipment of drugs is required, for example anti-venom, blood products, or vehicle extrication kit.

On arrival at the patient, scene safety should first be addressed and, once this is secured, the patient can be approached. A combination of rapid assessment and resuscitation should take place in a systematic fashion, using CABCDE (C-spine, ABCDE) or an alternative approved method. In the pre-hospital environment, only life, limb or eyesight saving procedures should take place at the scene. The transport of patients who require primary surgical stabilisation should not have their evacuation delayed while clinical teams provide advanced adjunctive medical care. On-going reassessment of the primary survey and initial secondary survey, as well as advanced resuscitation including drugs and blood products can take place en route.

### Personal protective equipment (PPE)

Appropriate clothing must be chosen prior to flight, although this is more important for RW transfer than FW flights. Lessons learnt from PHEM specialists should be acknowledged and combined with clothing to mitigate the problems associated with aeromedical work. Specific considerations include hearing protection (commonly integrated into a flight helmet with internal radio communications), eye protection (from rotor blade particulate debris), robust gloves (to operate helicopter equipment and extraction machinery - especially in cold environments) and layered undergarments to cope with changeable weather conditions and altitude. PPE must also be provided for the patient(s), including consideration of closed-circuit radio comms for the conscious and communicative patient.

### Pre-hospital rapid sequence induction (RSI)

There is increasing evidence to suggest that complex trauma patients (especially traumatic brain injuries) who receive a secured, definitive airway early in their care have increased survival rates and better functional outcomes. Indications for aeromedical intubation include airway compromise, ventilatory failure, unconsciousness, anticipated clinical cause, agitated head injury patients and injured or medical patients who cannot be managed safely without a definitive airway. Patients with multi-system trauma, undergoing RSI, with manual inline stabilisation of the cervical spine, have an increased incidence of difficult, or failed, tracheal intubation. RSI should only be performed by practitioners who have the appropriate skills and competence to do so.

At all times, pre-hospital anaesthesia should adhere to the same principles as emergency in-patient anaesthesia.[1,4]

Four staff members are optimal for an RSI: the operator performing the intubation, an airway assessment, person performing cricoid pressure and someone to perform manual inline stabilisation. As discussed above, there are rarely four members of an aeromedical clinical team (the normal number is two) and this reinforces the mantra of 'prior preparation and forward thinking' - performing certain procedures on the ground, prior to flight, with additional staff and resources (normally road ambulance crews or referring hospital staff). Preparation of the patient, equipment and drugs will decrease complications. Hard collar and blocks should be replaced by manual inline stabilisation for the procedure and then replaced afterwards.

A 'kit dump', in which drugs, oxygen, airway equipment, including difficult airway rescue adjuncts, supraglottic airways and surgical cricothyroidotomy kits, should be placed with easy access by the clinician and assistant. Drugs must be labelled clearly. The patient should be positioned with easy access from all angles, 360° if possible, and in the best possible position for intubation. A checklist and briefing should take place with all team members, including a failed intubation plan. Induction agents and neuromuscular blocking drugs used should chosen according to patient physiology and clinician's experience. Two suction units should be checked and readily available. Pre-oxygenation should take place, and there is some emerging evidence for apnoeic ventilation. Care should be taken if using a bag-valve-mask not to increase intra-gastric pressures. PHEM organisations should have guidelines for failed intubation, again mirroring hospital medicine best practice. The recommended Difficult Airway Society guidelines (DAS, UK - www.das.uk.com/guidelines) are easy to follow and effective. After tracheal intubation, anaesthesia should be maintained with intravenous agents, titrated to patient physiology.

### Blood products

The administration of blood products in the pre-hospital environment can be life saving and reduce morbidity. Packed red cells (and fresh

frozen plasma) may be stored according to MHRA requirements for blood banks and in partnership with the local secondary care trust or UK National Blood Service authority. Local protocols, written to mirror national guidelines, for the safe administration of blood products should be formulated and adhered to.[8]

Tranexamic acid (TXA) is an anti-fibrinolytic agent useful in the management of haemorrhage secondary to trauma, but also should be considered in other causes of haemorrhage. Current evidence recommends use within 180 minutes of injury (CRASH2 and MATTERs studies) however further research is on going to refine best practice recommendations in the trauma setting.[9,10]

Finger thoracostomy is a safe and effective procedure in the treatment of pneumothorax, in patients who are intubated and ventilated. It is arguably more effective for pre-hospital patients as thoracostomy sites can be re-opened with ease using a 'finger sweep'. Chest tubes require more time to insert and are susceptible to blockage and kinking. This is difficult to address in a small space. There is no evidence that thoracostomy increases the risk of empyema over that of a chest drain inserted in the field.[11]

Pre-hospital thoracotomy remains a hotly debated PHEM procedure, indicated only in cardiac arrest from suspected cardiac tamponade arising from penetrating trauma to the thorax. There are several case studies in the literature that report successful outcome if only attempted for the above traumatic aetiology. It is far more likely to achieve success than needle pericardiocentesis.[12]

## OTHER PATIENT CONSIDERATIONS

On FW and RW aircraft, the patient's hearing must be protected, even if he/she is sedated and paralysed (see PPE section above). Anti-emetics should be considered for patients routinely if travelling by air, as log rolling and moving patients in flight is risky and difficult.

## MONITORING

All critical care patients in the pre-hospital arena should have a minimum standard of monitoring, similar to patients undergoing elective hospital anaesthesia:[6,7]

- A minimum of 2 appropriately trained staff, skilled in advanced airway management and resuscitation.
- Resuscitation and emergency airway kit readily available (oxygen supply, oral suction, self inflating bag and mask, airway adjuncts, advanced airway equipment, defibrillation, labeled sedative agents, emergency drugs to support cardiorespiratory depression available, reversal agents available).
- Continuous pulse oximetry and 3 lead ECG monitoring, non invasive blood pressure measurement, respiratory rate or ventilator monitoring.
- End tidal wave capnography.
- Two reliable sites of peripheral venous access should be placed and secured.

## EQUIPMENT

Equipment should be light, portable yet robust. Battery life of the monitoring equipment should be recorded and maintained in between transfers, and spare battery packs should be available at all times.

Oxygen tanks should be full. Oxygen and battery life of monitoring should be calculated in accordance to the anticipated task time, with back-up in the event of delays or re-tasking.

Suction equipment, with a back up facility, should be easily available on the scene and during transport. Venturi suctioning consumes large volumes of oxygen and used only when necessary. An electrical or hand held suction device avoids this problem, but are often not as effective.

Transport ventilators need to be small and light, have clear visual alarms, with standard alarms for ventilator parameters. Ideally the ventilator needs to work from an external power source, rather than oxygen, which consumes larger quantities of gas. Oxygen flow may need to be adjusted to prolong the duration of time for which oxygen is available. Non-invasive ventilation is not usually suitable for aeromedicine as the oxygen consumption is unpredictable and high. Patients needing NIV should usually be intubated, to mitigate unplanned delays such as airframe diversion for fuel, or to transit to a secondary destination facility.

Vibration and movement of the aircraft affect most monitoring equipment. Bespoke fit is an option, but is costly and requires a dedicated platform to permanently mount the devices. Clinical monitoring is therefore essential in addition to the above, however, elements of clinical examination (e.g. auscultation) are somewhat inhibited during flight (especially RW transfer). It is vital to site and secure monitoring equipment optimally prior to receiving the patient to ensure the screens are visible to both members of the medical team throughout flight. During FW take-off and landing, medical crew will need to be secured in their seats and access to the patient is particularly restricted at this time. Non-invasive blood pressure monitoring is acceptable for most transfers, unless arterial monitoring is already in situ from the referring facility. Insertion of an arterial line should not compromise the evacuation of a patient who needs time critical definitive management.[13]

## INTRAVENOUS INFUSIONS

Rationalisation of infusions should be strongly considered to minimise the number of pumps for transfer. The more lines, equipment and pumps that are attached to the patient during ingress onto and egress from the platform, the more likely something will be dislodged or dislocated. This applies to most transfers, however, in order to keep weight, and thus fuel costs down, a limited number of pumps (two or three usually) will normally be available. Many drugs that in hospital would be routinely infused can be mixed or given in bolus dose. For example, muscle relaxants can be given as boluses and sedative drugs can often be combined. Syringe pumps should be compatible with multiple sizes of syringes and have visual alarms to identify occlusion or an empty syringe. For administering fluids, pressure bags will be required to give fast infusion rates, as height of the cabin is limited and gravity should not be relied upon.[3,5]

All equipment should be checked daily and restocked as per a hospital department. A checklist should be followed before and after each retrieval task and kit bags should be secured and dated where possible

to make sure equipment stays present and stocks are up to date.

If drugs have a stated temperature window for storage and are carried in personal kit, ambient cabin temperatures should be monitored and recorded. Awareness of decreased drug life spans outside normal temperature ranges should be understood and protocols of how long drugs can remain out the fridge should be developed in accordance with manufacturing guidelines.

## OTHER SPECIAL CONSIDERATIONS

### Sea level cabin

A pressurised cabin is standard in most FW aircraft. It is also possible to provide a sea level cabin, however this will compromise on fuel economy and range of the aircraft. Small decreases in ambient air pressure can worsen conditions such as acute decompression illness. Transport for fixed wing aircraft at sea level cabin pressure is recommended. RW platforms usually need a minimum of 1000 feet altitude and a route will need to be planned in order to provide a flight as near to sea level as possible.[3,5]

### Obstetric and neonatal patients

Transport of labouring women is suboptimal and where possible, women should deliver prior to aeromedical transfer.[3] Delivery on an airframe poses several problems including increased risk of maternal complications, and difficult neonatal resuscitation, lack of specialty care in transit and the doubling of acute patients from one to two! The need for aeromedical transfer for labouring women should be quantified against these risks as part of an encompassing medical estimate. Indications for rapid maternal transfer by air may include preterm labour, eclampsia, post partum haemorrhage, and sepsis. It may be prudent to slow the onset or progression of labour with salbutamol or nitrate. Neonatal transport is best achieved with dedicated neonatal teams, using specific neonatal equipment. An incubator, monitors, specific infusers and medical air are needed, which requires different weight and set up considerations. Once again, this returns us to the mantra of proper prior planning and preparation before embarking on the transfer.

### Psychiatric patients

Psychiatric patient may pose many challenges to safe aeromedical transfer. The issues around aviation safety as well as staff and patient safety must be carefully considered. Any patient who requires transportation to inpatient psychiatric care and is deemed a risk to themselves or others, is by definition, unfit to travel on an aircraft. It is the responsibility of all team members to assess the risk of the patient, but ultimately the decision as to whether to fly is with the pilot. The medical team's goal is to enhance safety and facilitate definitive care of the patient; safe and effective transfer may require sedation or general anaesthesia. Patient consent should be sought for transport. If consent is not achievable, the appropriate section of the national Mental Health Act should be consulted.[14]

### Bariatric retrieval

There is an increasing incidence in obesity worldwide and this affects problem for all areas of health care. Obese patients will often have a range of associated comorbidities, in addition to difficulties with intravenous access, airway, ventilation, monitoring equipment and procedural techniques. In the pre-hospital arena, bariatric ambulances and different aircraft can be used with specialized lifting devices, stretchers and monitoring. Fuel consumption is greater due to the extra weight of this equipment, and journey planning has to be adjusted accordingly.[5]

## CONCLUSIONS

Critical aeromedical retrieval involves bringing the mobile intensive care unit to the patient for facilitation of transfer to definitive care. It can significantly improve the outcome of patients, as long as appropriate pre-flight estimates are done in terms of patient selection, manning and equipment. Consideration of the patient's physiology, safety and intimate communication between facilities are required to improve morbidity and mortality.

## REFERENCES


1. Greaves I, Porter K. Oxford Handbook of Pre-Hospital care. Oxford University Press 2011.
2. Galvagno SM Jr, Haut ER, Zafar SN et al. Association between helicopter vs. ground emergency medical services and survival for adults with major trauma. *JAMA* 2012; **307**: 1602.
3. Everest E, Munford B. Transportation of the critically ill. Chapter 4. Oh's Intensive Care Manual. 6th ed. Philadelphia: Butterworth-Heinemann; 2009.
4. AAGBI safety Guideline: Pre-hospital Anaesthesia. The Association of Anaesthetists of Great Britain and Ireland 2008.
5. Martin T, Glanfield M. Aeromedical Transportation: A Clinical Guide. 2nd ed. Ashgate Publishing Limited. 2006.
6. Recommendations on Monitoring during Anaesthesia. Australian and New Zealand College of Anaesthetists 2008.
7. Recommendations for standards of Monitoring during Anaesthesia and Recovery. The Association of Anaesthetists of Great Britain and Ireland. 4th ed. 2007.
8. MHRA Haemovigilance Team: Report on the UK Regulation of Blood Safety and Quality 2005 – 2010. Medicines and Healthcare products Regulatory Agency 2012.
9. The CRASH-2 Collaborators. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant haemorrhage (CRASH-2): a randomised, placebo-controlled trial. *Lancet 2010;* **376:** *23-32.*
10. Morrison JJ, Dubose JJ, Rasmussen TE, Midwinter MJ. Military Application of Tranexamic Acid in Trauma Emergency Resuscitation (MATTERs) Study. *Arch Surg* 2012; **147:** 113-9.
11. Massarutti D, Trillo G, Berlot G et al. Simple thoracostomy in prehospital trauma management is safe and effective: a 2-year experience by helicopter emergency medical crews. ***European Journal of Emergency Medicine*** 2006; **13:** 276-80.
12. Davies GE, Lockey DJ. Thirteen survivors of prehospital thoracotomy for penetrating trauma: a prehopsital physician-performed resuscitation procedure that can yield good results. *J Trauma* 2011; **70:** 75-8.
13. N. McMahon, L. A. Hogg, A. R. Corfield, A. D. Exton. Comparison of non-invasive and invasive blood pressure in aeromedical care. *Anaesthesia* 2012; **67:** 1343–7.
14. M Le Cong. Retrieval Sedation for the acutely disturbed patient. RFDS Queensland Clinical Practice Guidelines 2010.
15. Collopy K, Kivlehan S, Snyder S. How Obesity Impacts Patient Health and EMS. EMSWORLD 01 April 2012. Web. 09 Sept 2013

Update in
Anaesthesia

# Low cost simulation training in anaesthesia

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**Summary**

Simulation is increasingly used in medical education, particularly in anaesthesia and critical care. In the UK many centres focus on use of high fidelity mannequins, however evidence suggests that many of the benefits of teaching through simulation can be gained using basic, low technology equipment. This is particularly true of novice learners. Simulation techniques should be chosen carefully as most modalities are well-suited to particular aspects of learning.

**QUESTIONS**

Consider the following questions. The answers are contained within the text.

1. What is the definition of fidelity?
2. List the different forms of simulation available.
3. Answer True or False for the following statements:
   a. The use of high tech computer-controlled manikins has been proven to be the most effective way of providing simulation teaching.
   b. Cognitive overload results in a decline in learning.
   c. An instructor providing video assisted feedback is the most effective way of providing feedback.

## INTRODUCTION

Simulation is defined as a set of techniques to replace or amplify real experiences with planned experiences. The experiences are often immersive in nature and should evoke or replicate substantial aspects of the real world in a fully interactive fashion. (*Immersive* means having the perception you are physically present in a situation that is not real, and also used in relation to 'virtual reality' technologies). Simulation training in medicine has been shown to be effective and to improve performance; it is associated with moderate to large improvements in educational outcomes when compared to no intervention, and small to moderate improvements when compared to instructional approaches, such as lectures. This potential has been widely accepted by the anaesthetic community, although we have not yet fully established how and when the use of simulation is most effective.

Medical simulation training in the UK is generally associated with use of high-tech, computer-controlled manikins that are used in scenario based training. This type of simulation is traditionally known as *high fidelity*. However simulation may take many diverse forms and some of the most useful and productive training can be achieved without the need for expensive equipment.

## SIMULATION IN ALL ITS FORMS

Simulation takes many forms from home made part-task trainers, to high-tech computer-controlled manikins in simulation suites.

There are several proposed classification systems for simulators used in anaesthesia, though none are widely accepted (Table 1).

**Table 1.** *Classification of simulators*

**Type of interaction**

- Hardware based – using physical interaction
- Screen based – using a mouse and keyboard
- Virtual reality based – using headsets or haptic devices that provide physical feedback
- Human based – using actors

**Response of the simulator**

- No physiology – no physiological response
- Physiology – the simulator responses are either controlled by a script or use a mathematical model to determine response

**Type of teaching**

- Knowledge
- Psychomotor skills
- Drills
- Performance

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## THE SCOPE OF SIMULATION USED IN ANAESTHESIA

### Hardware based simulators

- *Part task trainers* (commercial) e.g. epidural models, peripheral nerve block models, model heads for airway skill and IV access models.
- *Part task trainers* (home made) e.g. Fruit models for epidural training, ultrasound phantoms made from gelatin or meat, cricothyroidotomy models from tape and tubing and animal specimens.

### Screen based simulators

- *Computer based simulators*. There are many of these available.

### Virtual reality based simulators

- These exist for IV access training and are in development for ultrasound guided regional anaesthesia.

### Human based

- *Role-play scenarios*, which may be performed using members of staff as actors, or by using standardised patients for greater psychological fidelity. Standardised patients are actors who may have the physical characteristics of the patient they are portraying, e.g. amputees with theatrical make-up for trauma scenarios.

### Other types of simulator

- *Hardware and human based*. High-tech, computer-controlled simulators, which are traditionally known as high fidelity.
- *Hybrid simulators* which are devised using part task trainers and actors.

## WHAT IS FIDELITY?

Fidelity is defined as the degree of exactness with which something is copied or reproduced. Simulation training has traditionally been described as high or low fidelity, with high fidelity meaning immersive computer-controlled manikin based simulation, and low fidelity describing other types of simulation training. There are different forms of fidelity, and the use of high and low fidelity may be misleading. The nomenclature has changed to reflect this:

- Physical fidelity (it looks real)
- Functional fidelity (it works)
- Psychological fidelity (is has the same effect on the user as the real thing).

For example, using a banana to teach the loss of resistance technique for epidural placement is low cost and low tech, but has a high degree of functional fidelity.

It is not necessary for all aspects of fidelity to be high in order to provide effective simulation training. However it is essential to provide fidelity appropriate to the desired learning objectives.

## OPTIONS WHERE RESOURCES ARE LIMITED

There is evidence that the use of high tech simulators does not always add educational benefit and that less expensive technologies (animal models, simple manikins, wooden box trainers) can be used without sacrificing effectiveness.

When learners compared a commercially available part task trainer for cricothyroidotomy with a home made model, similar evaluation scores were found in both groups. The use a banana for epidural insertion training has been shown to provide an effective model for demonstrating loss of resistance to novice trainees. Comparison of fibreoptic intubation training on low or high fidelity models, found no correlation between the training method used and time to visualization of the carina. Regional anaesthesia teaching models can be constructed using Gelatin, processed meat, or animal models, and communication skills may be taught effectively without any expensive equipment.

A recent review found that anatomically correct simulators do not always add value to education, and that trainees are satisfied and learn well from simulators consisting of realistic tissues (animal and fruit models).[2]

When comparing learning outcomes of simulation with high or low fidelity, a further review found that, in nearly all of the studies, there was no significant advantage of high fidelity simulation over low fidelity simulation.

## EFFICIENCY AND EFFCTIVENESS OF SIMULATION TRAINING

In order to delivery efficient simulation training, both in terms of time and money, the chosen simulation modality should be suited to the learning outcomes required.

Using immersive computer controlled manikin based simulation to teach the management of a 'can't intubate, can't ventilate' (CICV) event, in learners without the necessary knowledge, skills and drills, is likely to be inefficient and less effective. Immersive simulation is expensive both in terms of time and money, and should be used when the maximal benefit can be gained.

Training to perform a complex procedure, such as CICV, can be broken down into sequential steps:

- **Knowledge** – the information required to perform the procedure (tracheal anatomy)
- **Skills** – technical skills (how to perform crico-thyroid puncture)
- **Drills** – practical ability to perform a drill (the steps in the CICV algorithm)
- **Performance** – The ability to integrate all of the above in to practice, both real and simulated (integrating CICV into a scenario with distractions).[11]

These different components are taught most effectively by different modes of teaching, some simulated, some not (Table 2)

We know from studies in psychology that working memory has limited capacity, and learning is impaired when this capacity is overloaded. Simulation training in learners with limited clinical experience is associated with a high cognitive load, and they are therefore at risk of cognitive overload, and a resultant decline in learning. When an individual is in the early stages of learning, information should be presented in smaller units to allow efficient processing and assimilation.

**Table 2**. *Components of a procedure and how they may be taught*

| Domain | Modality taught by: |
|---|---|
| Knowledge | Lectures<br>Tutorials<br>Demonstrations<br>e-learning |
| Skills | Home made or commercial part task trainers |
| Drills | Part task trainers, or full manikins, and may involve some role play. Training in skills and drills allows opportunity for repetitive blocked practice with frequent feedback. This is the most efficient method of learning for novice learners. |
| Performance | Immersive computer controlled manikin based simulation, when the necessary knowledge, skills and drills are in place. Immersive simulation allows random practice, increased stress and delayed feedback with opportunity for self reflection. This method is most efficient for proficient learners. |

This idea is supported by a learning model known as the challenge point framework. As task difficulty increases, learning also increases. This continues until the optimal challenge point is reached. At this point practice performance begins to decrease, immediate performance is negatively affected, but long term learning is enhanced. If the difficulty of the task is increased beyond the optimal challenge point, then both practice performance and learning are impaired. Therefore the aim is to target the learning to the challenge point for each individual learner, on the continuum from novice to expert.

## DE-BRIEFING AND FEEDBACK

Debriefing and feedback are about the realisation of a performance gap, and have been shown to increase the efficacy of simulation training. However, in a recent systematic review, the combination of two or more information sources during feedback does not add benefit, compared with a single information source. An instructor providing video assisted feedback was no better than an instructor providing verbal feedback alone. This may be due to the stress of public evaluation limiting the effectiveness of the feedback, and should encourage those running simulator sessions without the capacity to provide visual feedback.[2]

## CONCLUSION

Simulation training has been shown to be beneficial in medical education, and encompasses a wide range of teaching methods, of which the cheaper, simpler options may be more efficacious, especially for novices.

By matching the simulation modality used to the learning outcomes required, simulation training can be included in a training program with maximal cost and time efficiency. In situations where financial constraints limit the availability of equipment, high tech simulation may be reserved for advanced training and can reasonably constitute only a small part of a simulation education programme.

## REFERENCES


1. Gaba DM. The future vision of simulation in health care. Qual Saf Health Care 2004; 13(suppl 1): i2-i10.
2. Lorello GR, Cook DA, Johnson RL, Brydges R. Simulation-based training in anaesthesiology: a systematic review and meta-analysis. *British Journal of Anaesthesia* 2014; **112:** 231-45.
3. Cumin D, Merry AF. Simulators for use in anaesthesia. *Anaesthesia* 2007; **62:** 151-62.
4. Byrne A. What is Simulation for? ***Anaesthesia*** 2012; **67:** 213-5.
5. Raj D, Williamson RM, Young D, Russell D.A simple epidural simulator: a blinded study assessing the 'feel' of loss of resistance in four fruits. ***European*** *Journal of Anaesthesiology* 2013; **30:** 405-8.
6. Varaday SS, Yentis SM, Clarke M.A homemade model for training in cricothyrotomy. *Anaesthesia* 2004; **59:** 1012–5.
7. Raj D, Williamson RM, Young D, Russell D.A simple epidural simulator: a blinded study assessing the 'feel' of loss of resistance in four fruits. *European Journal of Anaesthesiology* 2013; **30:** 405-8.
8. Crabtree NA, Chandra DB, Weiss ID, Joo HS, Naik VN. Fibreoptic airway training: correlation of simulator performance and clinical skill. *Canadian Journal of Anaesthesia* 2008; **55:** 100-4.
9. Norman G, Dore K, Grierson L. The minimal relationship between simulation fidelity and transfer of learning. *Medical Education* 2012: **46:** 636-47.
10. K Fraser et al. Emotion, cognitive load and learning outcomes during simulation training. *Medical Education* 2012: **46:** 1055-62.
11. Anaesthetists as Educators, RCoA. Simulation Un-Plugged,
12. Guadagnoli M, Morin M-P, Dubrowski A. The application of the challenge point framework in medical education. *Medical Education* 2012: **46**: 447-53.

Update in
Anaesthesia



## BRIEF COMMUNICATION

# Management of infantile hypertrophic pyloric stenosis

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**Summary**

**Objective.** To collect details of common UK management practices for infantile hypertrophic pyloric stenosis (IHPS) and compare this to our practice at the Royal Cornwall Hospital Trust (RCHT).

**Aim.** To compare local with national practice and published evidence, leading to a local guideline for the perioperative management of IHPS.

**Results.** Although there was inevitable variability in some of the above areas, our audit at RCHT was generally consistent with practices identified in the survey.

**Conclusions.** Our perioperative management of IHPS at RCHT is reasonably concordant with national practice and published evidence.

### INTRODUCTION

Infantile hypertrophic pyloric stenosis (IHPS) is a common disease with an incidence of between two and five per thousand live births. It characteristically presents between the third and eighth week after birth and there is a male to female preponderance of four to five times.[1] The aetiology is unclear, although abnormalities in both the innervation of, and nitric oxide synthesis in, pyloric musculature have been implicated.[2] Hypertrophy and hyperplasia creates a pyloric channel which gradually becomes elongated and narrowed.

Patients typically present with non-bilious vomiting after feeding, which can be difficult to distinguish from feed intolerance and gastro-oesophageal reflux. High serum bicarbonate may be helpful, although sensitivity is low.[3] There may be inadequate weight gain or weight loss since birth. Although the late signs of projectile vomiting, visible peristalsis and a palpable 'olive-sized mass' in the right upper quadrant (RUQ) of the abdomen are fairly specific for IHPS (up to 0.9), overall clinical examination has a specificity of less than 0.5.[4] Ultrasonography is now established as the imaging modality of choice and is both highly sensitive and specific for IHPS.[5] Once considered the gold standard, the upper gastrointestinal series involves exposure to ionising radiation and is now reserved for equivocal cases.[6]

Ongoing vomiting causes dehydration and a hypochloraemic, hypokalaemic metabolic alkalosis.[7] There may be a deficit of sodium and occasionally there is renal impairment or an unconjugated hyperbilirubinaemia. Urine is paradoxically acidic due to secondary hyperaldosteronism, which further exacerbates the metabolic alkalosis. Acid-base balance should be restored prior to surgery, as untreated alkalaemia may result in apnoea during the recovery period.[7] The degree of dehydration is estimated and appropriate intravenous fluids are given for resuscitation, maintenance and replacement of ongoing nasogastric (NG) losses.

Pyloromyotomy is curative, well established and survival approaches 100%.[8] It should be carried out during the normal working day once the biochemical abnormalities and dehydration have been corrected. It can either be performed as an open (OP) or laparoscopic (LP) procedure and involves making a small cut in the musculature of the pylorus whilst leaving the mucosa intact. There are two possible OP approaches, umbilical and RUQ.[8]

Guidelines produced by the Association of Paediatric Anaesthetists of Great Britain and Ireland (APAGBI) include recommendations for the perioperative fluid management and correction of biochemistry.[9] However, until recently, there were no agreed approved guidelines at the Royal Cornwall Hospital Trust (RCHT), nor indeed in the South-Western region of England, encompassing the perioperative management of these cases.

We aimed to collect details of common practice in the UK, compare this to our practice and to publish local guidelines for the perioperative management of IHPS at RCHT.

### METHODS

#### Local audit

We completed a retrospective notes-based audit of our practice at RCHT between December 2006 and September 2008, specifically focusing on the factors listed in Table 1.

#### Survey

We then conducted an email survey of 32 anaesthetic departments across England, Wales and Scotland. We directed our questionnaire either to the designated lead paediatric anaesthetist, or to those who most frequently anaesthetise patients of this age group within each department. We asked them to provide answers they felt to be representative of their department and concentrated on key areas to maximise compliance. We specifically focused on the factors listed in Table 1.

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**Table 1**. *Specific areas of focus in the local audit and survey*

| | Local audit | Survey |
|---|---|---|
| **Demographics** | Gender<br>Gestational age<br>Age at presentation<br>Birth weight<br>Presentation weight | |
| **Biochemistry** | Admission bicarbonate<br>Preoperative bicarbonate | Preoperative bicarbonate |
| **Fluid** | Maintenance<br>NG losses<br>Postoperative | Preoperative<br>Intraoperative<br>Postoperative |
| **Anaesthetic Technique** | Premedication<br>NG tube suck out<br>Pre-oxygenation<br>Airway<br>Circuit<br>Induction<br>Maintenance<br>Muscle relaxants<br>Reversal of muscle relaxants | Premedication<br>NG tube suck out<br>Pre-oxygenation<br>Airway<br>Circuit<br>Induction<br>Maintenance<br>Muscle relaxants<br>Reversal of muscle relaxants |
| **Analgesia** | Local anaesthetic infiltration<br>Paracetamol<br>Opiates<br>NSAIDs | Local anaesthetic infiltration<br>Paracetamol<br>Opiates<br>NSAIDs |
| **Surgical Factors** | Operative approach<br>Length of stay<br>Surgical timing | Operative Approach<br>Estimated usual length of stay |
| **Numbers** | Number of cases over study period<br>Number of anaesthetists<br>Number of surgeons | Estimated number of cases per year<br>Number of anaesthetists<br>Number of surgeons |

### Comparison and implementation of guideline

The survey was analysed to identify common aspects of perioperative care. The management of our cases was then compared to the most common national practices identified in the survey. A guideline was published within RCHT in April 2009 (see Appendix 1 for summary) and also circulated to those centres that responded to the survey.

## RESULTS

### Surveyed centres

Of the 32 centres surveyed, 17 replied (53%). Five of these were District General Hospitals (DGHs) that either never, or very rarely, operated on patients with IHPS and were therefore excluded. 12 provided answers to our questions (37.5%). Of these four were DGHs and eight were tertiary referral regional centres.

### Patient demographics

Our audited case patients had a mean gestation of 39.9 weeks and a mean age at presentation of 4.7 weeks. The mean birth weight (BW) was 3.78kg and the mean weight gain from BW to presentation was 0.19kg (range -0.23 to +0.95kg).

There were 12 males and two females.

### Biochemistry

In our audit, the mean pre-operative serum bicarbonate concentration was 25.3mmol.$L^{-1}$ and the highest instance was 30mmol.$L^{-1}$.

Our national survey revealed a range of 26-34mmol.$L^{-1}$ as a prerequisite for anaesthesia, but the mode of 28mmol.$L^{-1}$ was quoted by 50% of respondents (Figure 1).

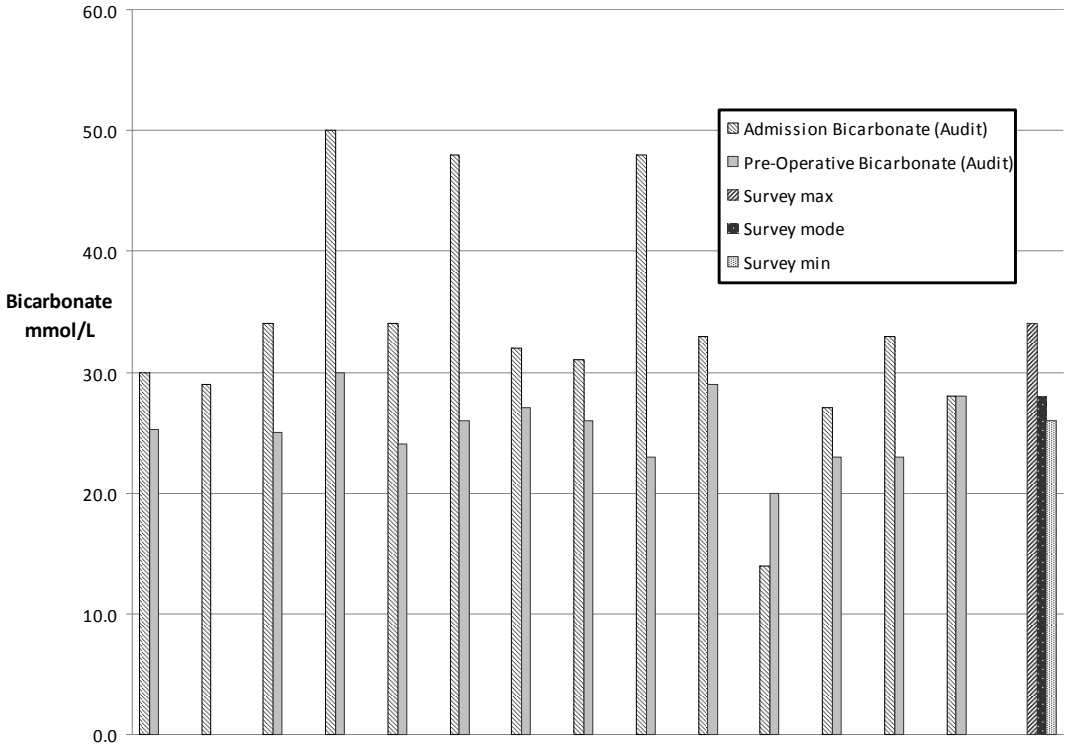


**Figure 1.** *Admission and pre-operative serum bicarbonate (Audit), with survey minimum, mode and maximum values also shown*

### Fluid

In our Trust we used two different maintenance fluid regimes; both consisted of 0.45% saline in 5% glucose with 10mmol potassium chloride added to each 500ml bag. In seven cases this was infused at $100ml.kg^{-1}.day^{-1}$ and in the other seven at $150ml.kg^{-1}.day^{-1}$. Replacement of NG losses with an equal volume of 0.9% saline occurred in 12 cases. There was variability from our survey, but the most common (six centres) fluid used was 0.45% saline in 5% dextrose, with or without additional potassium. All but one centre stated that they continue preoperative or 'maintenance' fluids until tolerating feeds. All of our audited cases were normoglycaemic.

### Anaesthetic technique

None of the surveyed centres or our patients received premedication. All of our cases and all centres surveyed used nasogastric tubes (NGTs), all of which were aspirated prior to anaesthesia. In two surveyed centres but none of our audited cases, a 3600 suck-out technique was employed. All of our cases and all but one centre reported performing preoxygenation. Trachea intubation was routine in all cases in our audit and those surveyed. All of our audited cases and 3/12 surveyed centres used an Ayre's T-piece. Of the remaining nine centres, five used a circle system and four used a T-Piece for induction in the anaesthetic room, changing to a circle once in theatre. Both the survey (5/12 inhalational, 5/12 variable) and audit (11/14 inhalational) favoured inhalational inductions. The remaining inductions were either intravenous (IV) or rapid sequence inductions (RSI) (Table 2).

Anaesthesia was maintained with sevoflurane in all of our cases, in addition to nitrous oxide ($N_20$) in 5/14. Of those surveyed all used volatile, with additional $N_20$ in 4/12 and remifentanil in 1/12

**Table 2.** *Methods of induction of anaesthesia*

| Induction technique | Our practice (14 cases) | Survey (12 centres) |
|---|---|---|
| Inhalational | 11 (79%) | 5 (42%) |
| IV | 2 (14%) | 2 (16%) |
| RSI / Modified RSI | 1 (7%) | 0 |
| Variable (Inhalational, IV or RSI) | 0 | 5 (42%) |

**Table 3.** *Technique for maintenance of anaesthesia*

| | Our practice (14 cases) | Survey (12 centres) |
|---|---|---|
| Sevoflurane | 14 (100%) | 2 (17%) |
| Isoflurane | 0 | 1 (8%) |
| Desflurane | 0 | 1 (8%) |
| Volatile (not specified) | 0 | 8 (67%) |
| TIVA | 0 | 1 (8%) (occasionally) |
| + Nitrous Oxide | 5 (36%) | 4 (33%) |
| + Remifentanil | 0 | 1 (8%) |

centres. One centre reported 'occasionally' using total intravenous anaesthesia (TIVA) (Table 3). Excluding suxamethonium (which was used in all of our RSIs), non-depolarising neuromuscular blockers (NDNBs) were given in all of our cases; 8/14 received atracurium and 6/14 vecuronium. 11/12 centres surveyed used atracurium but one centre did not routinely give NDNBs at all. Regarding reversal

of NDNB with neostigmine and glycopyrrolate, seven centres always, three sometimes and two never did. From our audit, none of our cases appear to have received reversal (Table 4).

**Table 4.** *Reversal of neuromuscular blockade*

| | Our practice (14 cases) | Survey (12 centres) |
|---|---|---|
| Atracurium | 8 (57%) | 11 (92%) |
| Vecuronium | 6 (43%) | 0 |
| Reversal | None or N/S | 7 (58%)<br>3 (25%) 'sometimes'<br>2 (17%) 'not' |

### Analgesia

All of our cases and those surveyed use local anaesthetic infiltration by the surgeons and paracetamol. Additional analgesic requirements (usually codeine) were minimal, both in our audit and the survey. Only 1/12 centres and 3/14 of our cases reported prescribing non-steroidal anti-inflammatory drugs (NSAIDs) post-operatively. One centre 'sometimes' inserted rectus sheath blocks (Table 5).

**Table 5.** *Analgesia*

| | Our practice (14 cases) | Survey (12 centres) |
|---|---|---|
| Paracetamol | 14 (100%) | 12 (100%) |
| Codeine | 2 (14%) | 2 (17%) |
| NSAIDS | 3 (21%) | 1 (8%) |
| Local Anaesthetic Infiltration | 14 (100%) | 12 (100%) |
| Rectus Sheath Block | | 1 (8%) |

### Surgical factors

All of our audited cases had an OP technique. Of our two surgeons, one performed all umbilical and the other all RUQ. Only two of the centres surveyed used the RUQ approach, both DGHs. The remaining centres (two DGHs and all the regional centres) used either the umbilical approach or a combination of umbilical and LP.

The median post operative stay from our audit was three days, as compared to estimates of 24 - 48 hours from the survey.

After diagnosis the maximum time to surgery in all of our cases was 48 hours, reflecting time for adequate resuscitation. There were three cases with a delayed diagnosis, resulting in four, seven and 12 day delays from presentation to surgery respectively.

### Incidence

14 cases of IHPS were identified during the defined study period of 22 months at RCHT, a DGH serving a population of nearly 500 000. This compares to survey means of 22.0 estimated cases per year (range 3-40) for the 4 DGHs and 61.7 (range 50-80) for the regional centres that gave estimates.

Our workload was evenly spread between two consultant surgeons and 5 consultant anaesthetists. This compares with survey means of 2.8 and 5.3 for the four DGHs, and 5.4 and 9.4 for the eight regional centres respectively.

## DISCUSSION

### Patient demographics

Although a well recognised feature of IHPS is a failure to gain weight, this is not always the case as demonstrated by our figures. The relationship between incidence of IHPS and low birth weight is also unclear, with studies reporting both positive[10] and negative associations.[11]

### Biochemistry

Both our audit and survey demonstrated that anaesthetists generally ensure that metabolic alkalosis has normalised before pyloromyotomy, although there is variability in the absolute threshold used. The most frequently quoted figure was a serum bicarbonate of <28mmol.L$^{-1}$ (50% of respondents). This is consistent with the above-mentioned APAGBI guidelines which suggest the following as a prerequisite for anaesthesia: bicarbonate <28mmol.L$^{-1}$, chloride >100mmol.L$^{-1}$ and BE <+2.9

### Fluid

Our audited maintenance rates were evenly split between 100ml.kg$^{-1}$.day$^{-1}$ and 150ml.kg$^{-1}$.day$^{-1}$. The APAGBI guideline, which was published during our audit period, suggests that in the specific case of IHPS maintenance fluids should be given at 1.5 times the normal rate, calculated using the formula as described by Holliday and Segar.[12] Although several of our cases fell into the neonatal category, there is no clear relationship between which regime was followed and post-conceptual age, admission weight, or date of admission.

The constitutions of our intravenous fluid regimes for maintenance (0.45% saline in 5% dextrose) and replacement of NG losses (0.9% saline) were consistent with the APAGBI guideline for infants (>44 weeks post-conception age). No consensus was reached within this document regarding neonates (≤44 weeks post-conception age), although 0.18% saline in 10% dextrose was suggested. Our survey found that the most frequently (6/12 centres) used maintenance fluid was 0.45% saline in 5% dextrose. Only one (a large tertiary referral centre) reported using 0.18% saline. This may be in part due to a National Patient Safety Agency (NPSA) safety alert regarding the use of 0.18% saline in children,13 also published during the study period.

### Anaesthetic technique

Premedication was universally not felt to be necessary. Aspirated NGTs were used in all cases, although a 360$^0$ suck-out technique was infrequently used. There was a broad consensus about pre-oxygenating and all agreed upon tracheal intubation.

All of our audited cases used a T-Piece circuit throughout. This contrasts to the survey, in which most centres reported using a circle, either throughout or after transfer into theatre for maintenence. A recently published postal survey of APAGBI members[14] found that despite improvements to paediatric circle systems the Ayre T-Piece remains popular, but also that the practice of switching to a circle system is gaining in popularity due to its practical advantages.

Analysis of the type of induction for pyloromyotomy reveals interesting changes over time; in the late 1970s inhalational induction was more common than intravenous (IV) (85% and 15% respectively), but by 1984 this trend had completely reversed (15% and 85% respectively).[15] In 1994 another postal survey of APAGBI members[16] found that 60% of anaesthetists use a RSI or modified RSI, but both our survey and audit suggested that inhalational inductions are once again gaining in popularity.

The near universal use of volatile for maintenance of anaesthesia and NDNBs was as expected. Most centres reported the routine use of neostigmine with glycopyrrolate for the reversal of neuromuscular blockade. This does not appear to have been given to any of our audited cases, however this does not mean that reversal was not administered and may represent a failure of documentation.

### Analgesia

The use of paracetamol and local anaesthetic infiltration was universal, and analgesic requirements in addition to this were minimal in both our audit and survey. This is consistent with previously published series.[17]

### Surgical factors

The commonest approaches from our survey were umbilical OP and LP. LP confers shorter recovery times, but may result in higher complication rates and lower efficacy when compared to OP.[8] Both LP and umbilical OP are associated with a superior cosmetic result when compared with RUQ OP.[18]

After diagnosis, our maximum delay to surgery of 48 hours is reasonable, given the need for pre-operative optimisation in these cases as discussed above. Our median post-operative stay of three days is longer than that from the survey, in which all quoted less than 48 hours as standard. This could however represent aspiration rather than reality, as a survey of anaesthetists is perhaps not the best way to investigate post-operative length of stay.

### Incidence

We performed fewer than average when compared to other DGHs (14 in 22 months, versus the survey estimated average of 22 per year), although we were not the least frequent operators in the group. We have a comparable number of consultant surgeons and anesthetists performing pyloromyotomies to the four other DGHs surveyed. This reflects an appropriate concentration of expertise given the fewer numbers performed.

### Study limitations

The number of audited cases was relatively small; we are currently undertaking a re-audit of further cases after implementation of the guideline. There are potential problems in comparing the results of a local audit to a survey and then extrapolating to the wider population. The survey is potentially biased; for practical reasons we targeted one representative per department and many of the responses were estimates; there is a tendency to report good more than bad practice; what people report they do is not necessarily what they actually do, or what they document they do. This is highlighted by the example of reversal of NDNBs; in our audit it was not documented for any case, but in the survey most respondents said they use it.

## CONCLUSION

Our practice at RCHT demonstrates reasonable concordance with national practice. Despite the trend towards centralizing services, there is a balance between the need to protect capacity for local care in remote areas such as Truro and the need for sufficient exposure to maintain skills, as set out by the Care Quality Commission.[19] There is, as always, a range of anaesthetic practice but the most important factors are probably experience and the use of familiar techniques.


### Acknowledgements

Mr W Faux, Paediatric Surgeon and Dr Sian Harris, Consultant Paediatrician, Dr S Thorogood Consultant Radiologist, Royal Cornwall Hospital, Truro, Cornwall

The South West Paediatric Surgical Network

The South West Association of Children's Anaesthetists

*Competing interests*

No external funding and no competing interests declared.


## REFERENCES


1. MacMahon B. The continuing enigma of pyloric stenosis of infancy: a review. *Epidemiology* 2006; **17:** 195-201.
2. Letton RW Jr. Pyloric stenosis. *Pediatric Annals* 2001; **30**: 745-50.
3. Smith GA, Mihalov L, Shields BJ. Diagnostic aids in the differentiation of pyloric stenosis from severe gastroesophageal reflux during early infancy: the utility of serum bicarbonate and serum chloride. *American Journal of Emergency Medicine* 1999; **17**: 28-31.
4. White MC, Langer JC, Don S et al. Sensitivity and cost minimization analysis of radiology versus olive palpation for the diagnosis of hypertrophic pyloric stenosis. *Pediatric Surgery* 1998; **33**: 913-7.
5. Hernanz-Schulman M, Sells LL, Ambrosino MM et al. Hypertrophic pyloric stenosis in the infant without a palpable olive: accuracy of sonographic diagnosis. *Radiology* 1994; **193**: 771-6.
6. Hernanz-Schulman M. Infantile hypertrophic pyloric stenosis. *Radiology* 2003; **227**: 319-31.
7. Goh DW, Hall SK, Gornall P et al. Plasma chloride and alkalaemia in pyloric stenosis. *British Journal of Surgery* 1990; **77**: 922-3.
8. Hall NJ, Van Der Zee J, Tan HI et al. Meta-analysis of laparoscopic versus open pyloromyotomy. *Annals of Surgery* 2004; **240**: 774-8.
9. The Association of Paediatric Anaesthetists of Great Britain and Ireland (APAGBI). APA Consensus Guideline on Perioperative Fluid Management in Children v1.1. London: APAGBI; 2007.
10. Hitchcock NE, Gilmour AI, Gracey M et al. Pyloric stenosis in Western Australia, 1971-84. *Archives of Disease in Childhood* 1987; **62**: 512-3.
11. Applegate MS, Druschel CM. The epidemiology of infantile hypertrophic pyloric stenosis in New York State, 1983 to 1990. *Archives of Pediatric and Adolescent Medicine* 1995; **149**: 1123-9.

12. Holliday MA, Segar WE. The maintenance need for water in parenteral fluid therapy. *Paediatrics* 1957; **19**: 823-32.

13. National Patient Safety Agency (NPSA). Patient Safety Alert: Reducing the risk of hyponatraemia when administering intravenous infusions to children. London: NPSA; 2007.

14. Macdonald NJ, Fitzpatrick GJ, Moore KP et al. Anaesthesia for Congenital Hypertrophic Pyloric Stenosis: A Review of 350 Patients. *British Journal of Anaesthesia* 1987; **59**: 672-7.

15. Peutrell JM, Wilkins DG. Pyloric stenosis in full term babies: a postal survey of the management by paediatric anaesthetists. *Pediatric Anesthesia* 1994; **4**: 93-7.

16. Habre W, Schwab C, Gollow I et al. An audit of postoperative analgesia after pyloromyotomy. *Pediatric Anesthesia* 1999; **9**: 253-6.

17. Marsh DF, Mackie P. National survey of pediatric breathing systems use in the UK. *Pediatric Anesthesia* 2009; **19**: 477-80.

18. Mullassery D, Perry D, Goyal A et al. Surgical practice for infantile hypertrophic pyloric stenosis in the United Kingdom and Ireland: a survey of members of the British Association of Paediatric Surgeons. *Journal of Pediatric Surgery* 2008; **43**:1227-9.

19. Care Quality Commision (formerly Healthcare Commision). Improving Services for Children in Hospital; Report of the follow-up to the 2005/06 review. London: Commission for Healthcare Audit and Inspection; 2009.

Update in
Anaesthesia



# Cerebral challenge 1

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## Case 1

A 72-year-old male with known ischaemic heart disease is admitted to recovery after cystectomy. He wakes up from the anaesthetic complaining of chest pain. A 12-lead electrocardiogram (ECG) is recorded.

1. What does this ECG show?
2. How would you manage this patient given his recent major surgery?

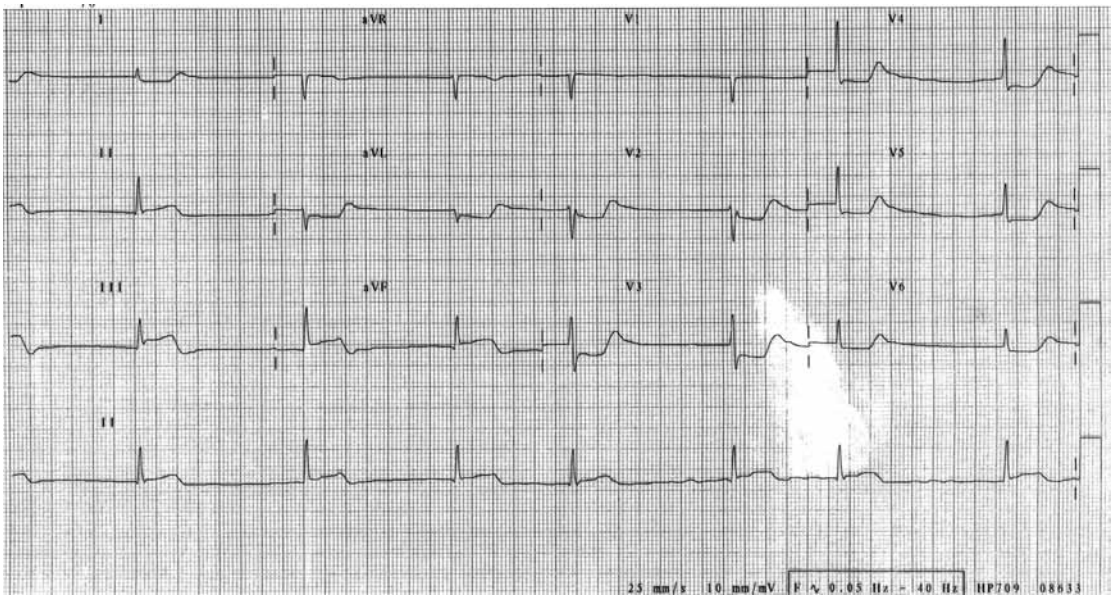


**Figure 1.** *ECG of patient 1*

## Case 2

A 19-year-old male motorcyclist is brought to the Emergency Department following a road traffic accident. On examination, his oxygen saturations are

95% on high flow oxygen with a patent airway, blood pressure is 90/70mmHg, heart rate is 118 bpm, and GCS is 7 (E2, V2, M3). You note that his GCS was 14 (E3 V5 M6) at the scene. His temperature is 34.2ºC. Tracheal intubation is performed. He is then taken to the CT scanner.

1. What does the CT head scan show?
2. What are the critical care priorities for this patient?

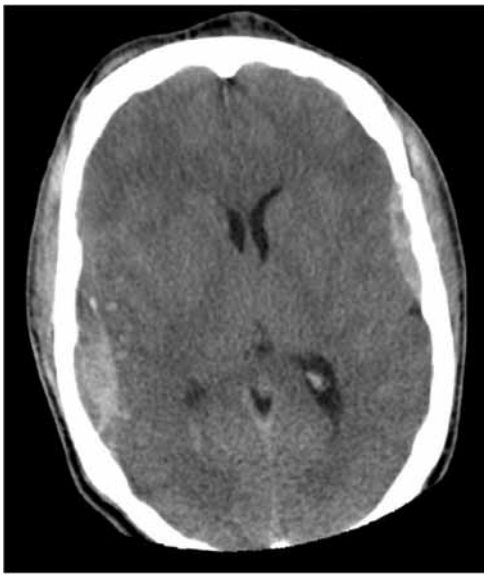

**Figure 2.** *CT head scan of patient 2*

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### Case 3

You are called to the Emergency Department, where a 20-year-old male has collapsed following an overdose. He was intubated at the scene with a GCS of 6. His $SpO_2$ is 84%, heart rate 96bpm, blood pressure 105/62, with equal reactive pupils. The paramedics report he has been 'difficult to bag' and on examination you note poor air entry over the left chest wall. A chest Xray has been performed.

1. What does the chest radiograph show?
2. What are the differential diagnoses of this appearance on chest Xray?
3. What is the immediate management of this situation?
4. What are the risk factors that can predispose to these findings?

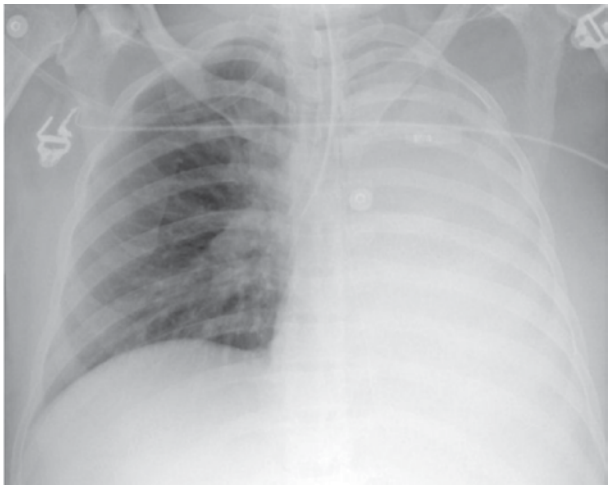

**Figure 3.** *Chest Xray of patient 3*

## DISCUSSION

### Case 1

The ECG in Figure 1 shows ST elevation in the inferior leads (leads II, III and aVF) indicating inferior ST elevation myocardial infarction (STEMI). There are with reciprocal anterior-lateral ST changes and slow atrial fibrillation.

STEMI is diagnosed by a history of characteristic chest pain and an ECG showing one of:

- ST-segment elevation of >1mm (1mV) in two or more adjacent limb leads (i.e. leads I, II or III).
- Elevation of >2mm (2mV) in two or more adjacent precordial leads (V1-V6).
- New onset left bundle branch block (LBBB).

These diagnostic criteria are important, as they are generally required to persuade a cardiologist to undertake PCI (percutaneous coronary intervention, i.e. angioplasty and stent insertion) in settings where this is available.

The term 'nodal rhythm' is used in situations where the atrioventricular (AV) node rather than the sinoatrial (SA) node sets the rate of contraction of the heart. This may be caused by a variety of pathologies affecting the SA node, however right coronary artery occlusion is a relatively common cause since the SA node is supplied by the right coronary artery (RCA) in 90% of individuals. In the remainder it is supplied by the circumflex artery. All myocardial cells have their own inherent rhythmicity - i.e. they set their own rate of contraction if left unstimulated. In health the natural faster rate of the SA node predominates, stimulating cells further down the conducting system of the heart at a faster rate than their own inherent rate. Unstimulated ventricular cells will generally contract at around 30-40bpm and so this is the rate seen in complete heart block. In this patient the cells with the next fastest inherent rate have taken over the pacing of the heart (as so-called 'escape pacemaker'). As narrow QRS complexes have been generated, this escape pacemaker lies within the usual conducting system and may be nodal (i.e. the AV node, sometimes called junctional) or infra-nodal.

Figure 4 illustrates that the RCA supplies the right atrium, right ventricle, and also, in the majority of patients, the atrio-ventricular (AV) node (in addition to the SA node).

Therefore patients with RCA ischaemia may present with:

- bradyarrhythmias (atrial fibrillation, heart block) due to AV node ischaemia, and
- right ventricular failure.

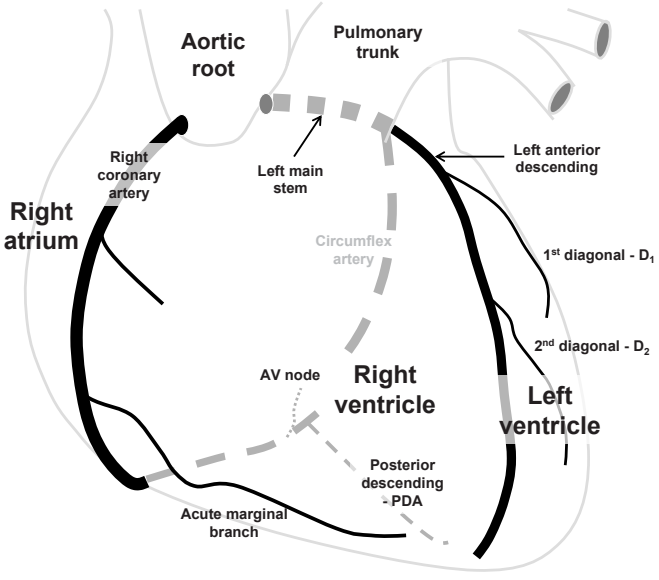


**Figure 4.** *Anatomy of the coronary arteries*

In most cases, the posterior descending artery that supplies the inferior left ventricular wall is also a branch of the RCA. Therefore some RCA infarcts, affecting predominantly the inferior part of the heart, also have posterior extension. Standard leads do not overlie the posterior aspect of the heart, however 'reciprocal' or opposite changes may be seen in the leads that lie over the anterior wall of the heart. So posterior

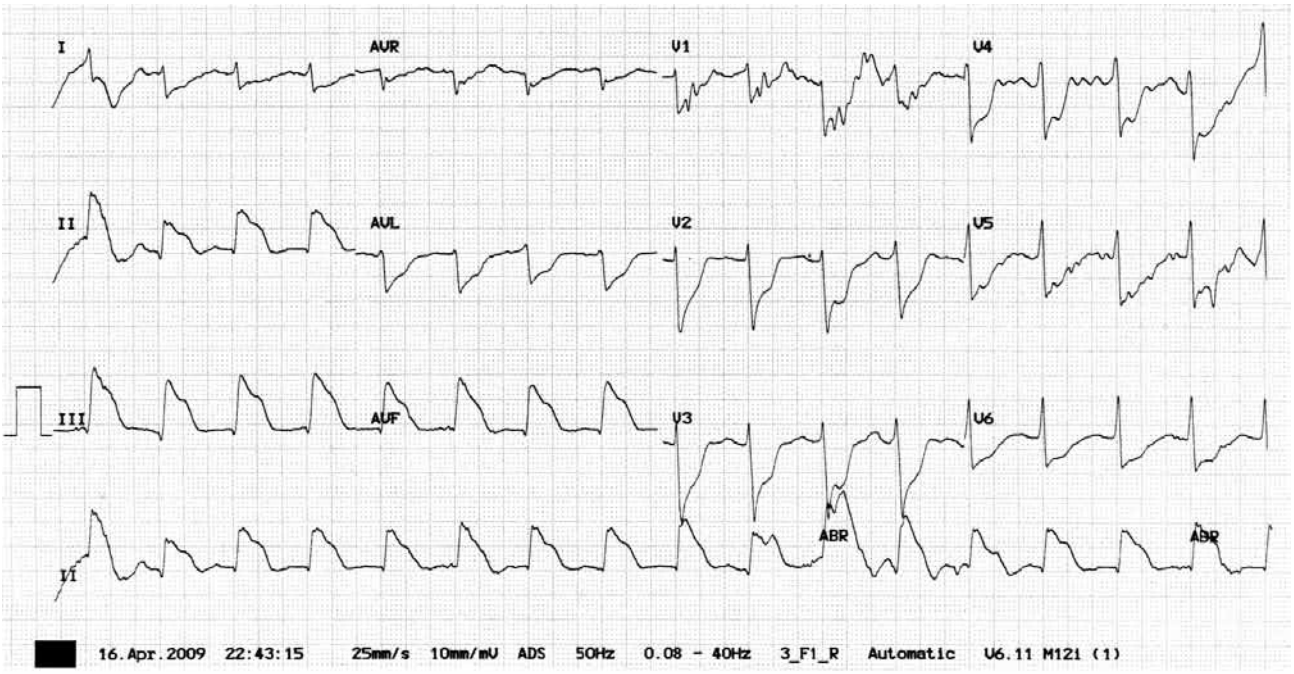


**Figure 5.** *'Tombstone' ST elevation in the inferior leads*

wall infarction may be seen as ST depression (the reciprocal of ST elevation) and R waves (the reciprocal of Q waves) in leads V1 and V2. This also explains why people describe the ST depression seen in leads V2-6 in Figure 1 as 'reciprocal' - these leads are opposite those directly over the affected inferior wall of the heart and so show the opposite changes.

Figure 5 is another illustration of an acute inferior MI, with so-called 'tombstone' ST elevation in the inferior leads. The 'tombstone' effect is due to markedly prolonged R waves secondary to a build up of potassium and extracellular metabolites in ischaemia. This accumulation depolarises the myocardium but then inhibits sodium channels, causing delayed transmural action potential transmission. The name of this ECG finding is related to the associated mortality.

The main treatment aims are of physiological optimisation and the reduction of further myocardial ischemic damage. The initial assessment and general early management should be rapid. For this patient, who has just undergone major body cavity surgery, all available therapies should be considered, but consideration of the risk and benefit may preclude them.

**Management of acute MI in the perioperative setting:**

*Early therapies*

- Oxygen therapy at 2-5L.min$^{-1}$
- Analgesia (morphine 2-10mg or diamorphine 2.5-10mg intravenously with accompanying antiemetic).
- Sublingual/intravenous nitrates if blood pressure allows. Be cautious in inferior MI if there are signs of right heart failure (high JVP, hepatic engorgement, peripheral oedema) - venodilation will reduce right ventricular filling and result in profound hypotension.
- Consider antiplatelet therapy (ideally aspirin 300mg and clopidogrel 300mg). This must be a joint decision between surgeon, cardiologist and anaesthetist, in order to weight the relative benefits of limiting myocardial damage, against the risk of postoperative bleeding.
- Immediate discussion with a senior cardiologist regarding patient suitability for reperfusion therapy (thrombolysis or PCI). Cystectomy will preclude this therapy in this patient, with the risk of catastrophic bleeding outweighing any potential benefit until the patient is outside the window for such therapy. Of note, coronary artery stent placement also has an essential component of antiplatelet therapy to prevent stent thrombosis, limiting its use in this setting.
- Beta-blockade (e.g. metoprolol 1-2mg up to 15-20mg) should be considered in the setting of tachycardia. Ensure that hypovolaemia and pain are not contributing factors. Aim for a heart rate 60 90bpm and a systolic blood pressure >100mmHg. Some experts recommend waiting 48-72hours before commencing beta blockade and this would be sensible in view of his major surgery. If already taking a beta-blocker this should be continued preoperatively.
- Intensive care unit admission for monitoring for 24-72 hours.

*Later therapies*

- Introduction of a statin (for cholesterol lowering and coronary artery plaque stability) and an ACE inhibitor to enhance ventricular function.
- Formal echocardiography to assess left and right ventricular performance.
- Formal assessment of coronary disease (angiography) once patient stability achieved.

## Case 2

The CT head scan shows bilateral extra-dural haemorrhages (EDH) with mild ventricular compression indicating raised intracranial pressure, and a small volume of blood in the left ventricle. The larger haemorrhage on the right is confined posteriorly by the lambdoid suture and by the coronal suture anteriorly. No bony fractures are evident, but the scan should be viewed on 'bone windows' to exclude this. The mid-line is not shifted, but this may be a false reassurance, as bilateral injuries disguise this.

An extra-dural haemorrhage most commonly occurs following tempero-parietal trauma and is often secondary to laceration of either the middle meningeal artery and/or accompanying venous vessels. Less commonly, a dural venous sinus tear is responsible.

A history of decreasing consciousness is a 'red flag' warning sign of rising intracranial pressure (ICP) and EDH should be suspected. Classically, there will be a lucid interval shortly after the injury, prior to a fall in consciousness. Other symptoms are nausea, vomiting, worsening headache, confusion and seizures. On examination, neurological signs such as worsening GCS, unilateral or bilateral up-going planters, brisk reflexes, hemiparesis, and ipsilateral pupil dilation may be present. Other external signs of possible intracranial haemorrhage are scalp lacerations and depressed skull fractures. 'Coning' due to raised ICP manifests clinically as bradycardia and hypertension (the Cushings reflex) & are pre-terminal signs. Deep irregular breathing with bilateral spastic paresis occurs shortly before respiratory arrest.

EDH is a neurosurgical emergency. Management of EDH is focussed on stabilisation and resuscitation before urgent transfer to the closest neurosurgical unit for clot evacuation.

Whilst awaiting transfer to theatre or another centre, there are several aspects of his care that can be optimised to limit secondary brain injury, by maintaining cerebral perfusion pressure above 70mmHg.

**REMEMBER: CPP = MAP – ICP**

(CPP = Cerebral perfusion pressure, MAP = Mean arterial pressure, ICP = Intracranial pressure).

- Normal ICP is 5-12mmHg. In a head injured patient it is reasonable to assume it is 20mmHg and so a mean arterial pressure of 90mmHg is needed to achieve a CPP of 70mmHg.
- Blood pressure should be monitored regularly, and invasively if possible.
- If thoracic and lumbar spine have been cleared then patient should be 30° head up to improve cranial venous drainage.
- Endotracheal tubes should be taped in place rather than tied to prevent ties obstructing venous drainage.
- $PaCO_2$ should be maintained in the low normal range (4.5-5kPa).
- Maintain normoglyacemia.
- Maintain oxygen saturations >95%.
- Any seizures should be controlled (e.g.18mg.kg-1 phenytoin).
- A brief period of hyperventilation (to lower the $PaCO_2$) and/or mannitol 0.5g.kg$^{-1}$ IV can be used to control acute surges in ICP. Hyperventilation should not be sustained as cerebral vasoconstriction may lead to further ischaemia.

An appropriate target is that all patients requiring emergency neurosurgery have a CT head with 1 hour of arrival, and transfer to a neurological centre is complete within 4 hours. This can be practically difficult to achieve. Adequate resuscitation must first be implemented to optimise the patient pre-transfer. However, transfer to a neurosurgical unit should occur as soon as possible to enable potentially definitive and life-saving treatment. Prolonged delay is inappropriate.

During the transfer cervical spine protection must be maintained. The patient should also receive the same standard of ICU physiological monitoring, and pupillary responses must be assessed every 15 minutes.

## Case 3

The chest Xray shows right endobronchial intubation and complete atelectasis of the left lung.

The differential diagnoses of a complete hemithorax 'white-out' depend on the position of the trachea:

| Position of trachea relative to opacification | Causes |
|---|---|
| Central | Consolidation<br>Chest wall mass<br>Pleural mass (e.g. mesothelioma)<br>Pleural effusion/haemothorax (small to moderate) - may be a 'veiling opacity' if film taken supine |
| Towards opacification | Total lung collapse (e.g. endobronchial intubation)<br>Pneumonectomy<br>Pulmonary hypoplasia |
| Away from opacification | Pleural effusion/haemothorax (large) |

Endobronchial intubation is most likely immediately prior to intubation, but may occur at any time during surgery or an ICU stay. It should be suspected in any case with low or falling arterial oxygen saturations, particularly when inflation pressures are high and expansion or air entry is asymmetrical. It is more likely to occur in children, where positioning is more critical due to the short length of the trachea. Where inubation has been difficult or prolonged there is a tendency to over insert the endotracheal tube and this is particularly likely when insertion has been blind, for example over a bougie.

Check that the endotracheal tube (ETT) is at an appropriate expected length for the patient. Deflate the cuff and slowly withdraw the ETT until breath sounds are auscultated on the anterior chest wall of the non-inflating side. This can be done under direct laryngoscopy or using a fiberoptic scope to check the position above the carina, and minimises the chance of inadvertent extubation. If you do not have chest Xray evidence of endobronchial intubation and the above measures do not improve bilateral lung expansion, consider alternative diagnoses. Once you are satisfied that the tube is a suitable length, is patent and you can auscultate equal air entry bilaterally, reinflate the cuff, re-secure the tube. In those with pulmonary disease a lung recruitment manoeuvre (e.g. 40cm$H_2O$ for 40 seconds) followed by PEEP will help to reinflate the affected lung.

Other risk factors for bronchial intubation include:

- intubation by non-anaesthetists in emergency situations,
- use of uncut endotracheal tubes
- long insertion depths (>21cm at the teeth in women and >23cm in men),
- prone positioning,
- shared airway surgery,
- operations involving a pneumoperitoneum with steep head down positioning.

## FURTHER READING


- Ratib K. Bhatia G. Uren N. Nolan J. Emergency Cardiology, 2nd Ed. Hodder Arnold, UK. 2011. p6-26.
- Ramrakha P. Hill J. Oxford Handbook of Cardiology. Oxford University Press, UK. 2006. p155-73.
- Recommendations for the Safe Transfer of Patients with Brain Injury. 2006. Association of Anaesthetists of Great Britain and Ireland.

Update in
Anaesthesia

# Cerebral challenge 2

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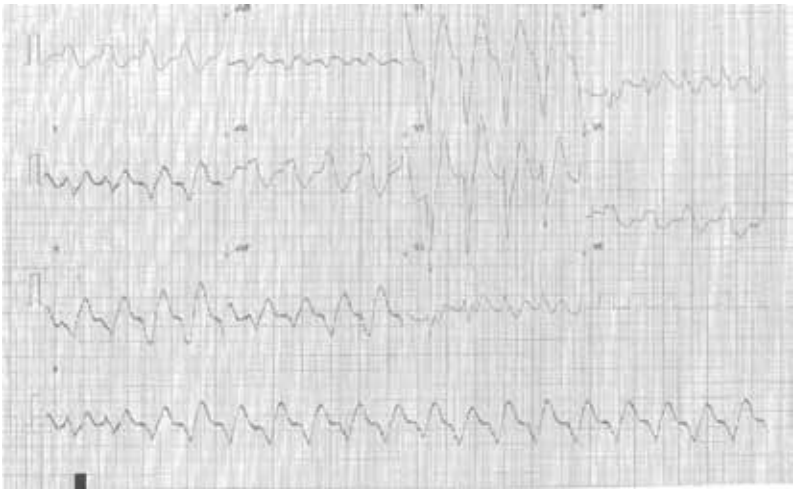
## Case 1

You are called to the emergency department, where a 43-year-old female with no medical history has been brought in by ambulance after taking an intentional overdose. She was found with a Glasgow Coma Score of 3/15 and was intubated by the paramedics at the scene. Two empty packets of amitriptyline were found beside her. In the ambulance she had a palpable brachial pulse but the ECG monitor showed intermittent irregular broad complexes. A 12-lead ECG (Figure 1) was recorded in ED shortly after her arrival.



1. What does the ECG show?
2. What is the likely cause of these abnormalities?
3. How would you manage this patient?
4. What new drug is gaining credibility in the treatment of such an overdose?

**Figure 1.** *ECG of patient 1*

## Case 2

A previously well 52-year-old woman is brought to the emergency department having been trampled by her horse. She walked briefly after the injury, but complained of severe chest and abdominal pain when the paramedics arrived. While on her way to ED, she became hypotensive, tachycardic and drowsy. Examination findings on arrival were: BP 70/40, HR 130min$^{-1}$, cool peripheries, tense abdomen and unresponsive to painful stimulus. The admitting doctor began resuscitation, including assisted ventilation with a bag and mask. You have been called to secure the airway, and the trauma team has been summoned. The surgeon suspects a ruptured spleen. As part of the primary survey, a chest Xray was taken following intubation (Figure 2).

1. What are the main abnormalities that this chest Xray demonstrates?
2. How should the team investigate and manage the suspected splenic rupture?

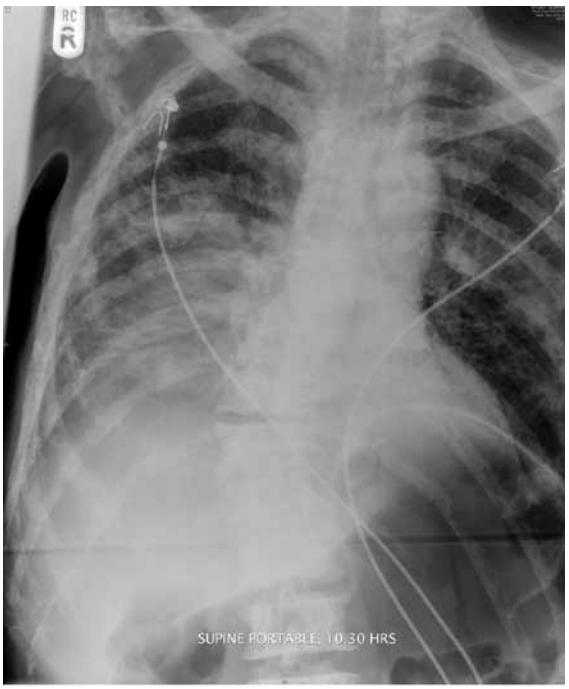



**Figure 2.** *Chest Xray of patient 2 after tracheal intubation*

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### Case 3

A 14-year-old old boy presents into ED with a history of general malaise and reduced exercise tolerance over the last 10 days. His parents are concerned by his progressive lethargy and shortness of breath and have brought him in to ED today after he complained of abdominal pain. He is normally fit and well. On examination he looks unwell and is lethargic. He is afebrile, tachycardic and has a blood pressure of 76/40. Heart sounds are normal but quiet. He has cool peripheries, a central capillary refill time of 3 seconds and a weak pulse. Examination of his abdomen reveals diffuse discomfort to palpation with a palpable liver edge but no evidence of peritonism.

You order a chest Xray and an ECG.

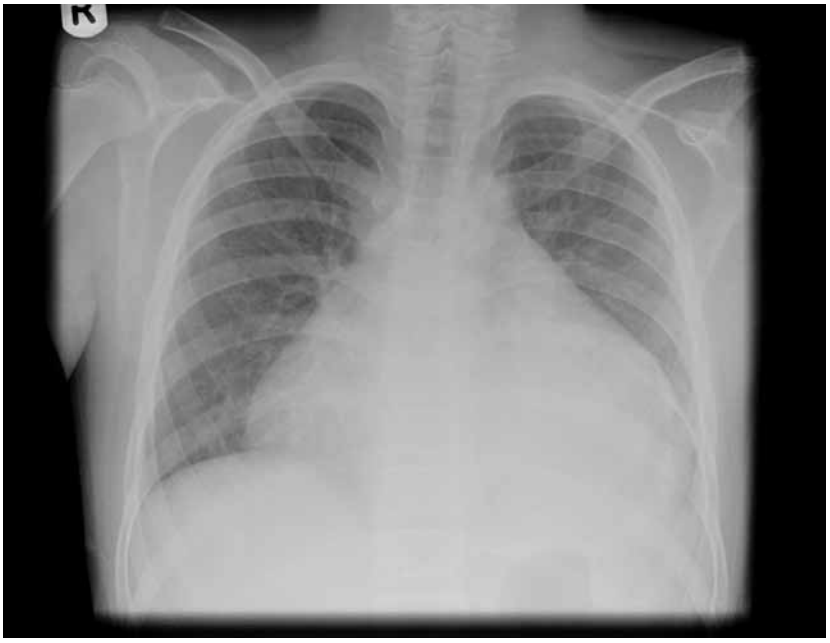


**Figure 3.** *Chest Xray of patient 3*

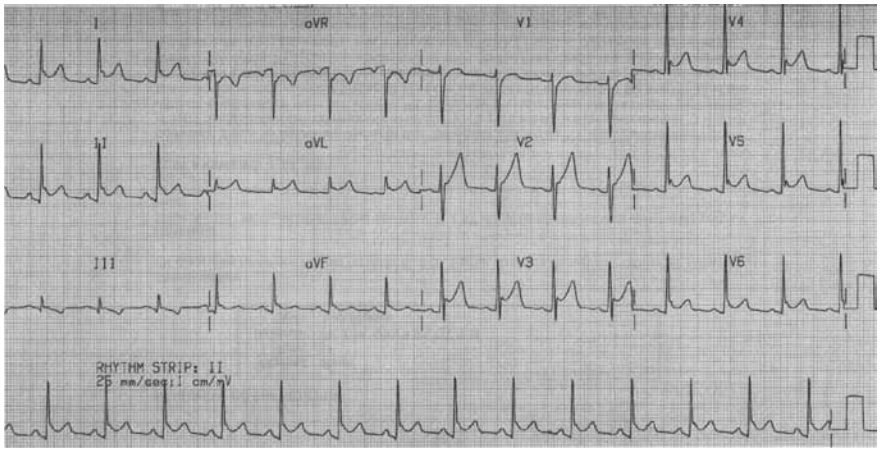



**Figure 4.** *ECG of patient 3*

1. What abnormality does the Xray show?
2. What does the ECG show?
3. Given the history and the findings of the Xray and ECG, what is the likely diagnosis?
4. What other possible findings on examination are consistent with this diagnosis?
5. What are the main causes of the most likely diagnosis?
6. What are the anaesthetic implications of caring for a child like this?

## DISCUSSION

### Case 1

The ECG shows broad QRS complexes (lasting 0.19 seconds or 190ms); the normal QRS duration is less than 0.12 seconds (3 small squares with a standard ECG recording).

There is also a prolonged QT interval (0.55 seconds, 550ms). The QT interval is measured from the beginning of the Q wave to the end of the T wave (Figure 5) and, as it varies with heart rate, it should be corrected for this. The normal corrected QT interval (QTc) is less than 0.45 seconds in males and less than 0.47 seconds in females. The QTc may be calculated using the formula QTc = QT/√R-R interval.

Prolonged QTc is associated with an increased risk of developing life-threatening arrhythmias such ventricular tachycardia and ventricular fibrillation.

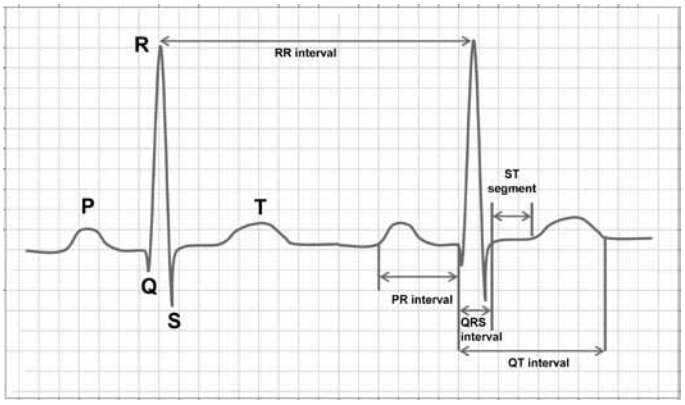



**Figure 5.** *A typical ECG complex with normal morphology showing standard intervals. Normal values are: PR interval, 0.12s (3 squares) - 0.2s (5 squares); QRS interval, <0.12s (3 squares); QT interval, <0.45s (males), <0.47s (females)*

Given the history, the likely cause of these abnormalities is an amitriptyline overdose. Amitriptyline is a tricyclic antidepressant (TCA) whose main mechanism of action is serotonin and norepinephrine reuptake inhibition at the postganglioninic membrane. In addition, it inhibits sodium channels, L-type calcium channels, certain voltage-gated potassium channels and has anticholinergic effects. This broad spectrum of actions accounts for its central and cardiovascular toxicity.

**Table 1.** *Causes of a prolonged QT interval*

| | |
|---|---|
| Genetic | long QT syndrome eg. Romano-Ward, Jervell-Lange-Nielson |
| Drug induced | amiodarone, quinidine, sotalol, haloperidol, clarythromycin, tricyclic antidepressants, alcohol and many others |
| Metabolic | hypothyroidism |

**Table 2.** *Symptoms and signs of TCA toxicity*

| | |
|---|---|
| Peripheral | dry skin, urinary retention |
| Central | drowsiness, hyper-reflexia, respiratory depression, seizures |
| Cardiovascular | intraventricular conduction delay (QRS, PR & QT prolongation), hypotension, ventricular tachycardia/fibrillation |

Your management of this patient should follow an 'ABCD' approach. In this case, particular attention should be paid to:

1. Correcting hypoxia caused by a depressed conscious level or seizures.
2. Treating arrhythmias by correcting any acid-base disturbance. Adults with a tachycardia >100bpm and/or ECG changes should be treated with 50mmol of 8.4% sodium bicarbonate solution even in the absence of acidosis. This interferes with the binding of TCAs to the myocardium.
3. Amiodarone should be avoided when treating arrhythmias as it prolongs the QT interval, and may exacerbate hypotension.
4. Seizures should be controlled with benzodiazepines. Phenytoin should be avoided as it blocks sodium channels and exacerbates TCA mediated conduction delay.
5. Resuscitation may be prolonged, due to the duration of action of amitriptyline.

There is increasing evidence for the use of Intralipid® (lipid emulsion) in the management of a variety of drug toxidromes. First used in 2006, this lipid emulsion is now the first line treatment for local anaesthetic toxicity and its use has been successfully extended to treat TCA, calcium channel antagonist, beta-blocker and antipsychotic overdose. Its mechanism of action remains uncertain, but may be via the absorption of lipid-soluble drugs into fat droplets, effectively removing them from the circulation. Intralipid® may be considered in severe cases of TCA overdose which are refractory to conventional treatment.[1]

*Intralipid® dose guide*

There is no robust evidence based regimen for the use of intralipid in TCA overdose, but a protocol guiding the use of intralipid in local anaesthetic toxicity may be used. This guideline, endorsed by both the American Society for Regional Anaesthesia and the Association of Anaesthetists of Great Britain and Ireland, is shown below.[2]

1.5ml.kg$^{-1}$ bolus of 20% lipid emulsion over 1 min

followed by

15ml.kg$^{-1}$.h$^{-1}$ infusion of 20% lipid emulsion

If cardiovascular instability persists after 5 min or a previously stable circulation deteriorates:

A maximum of 2 repeat boluses of **1.5 ml.kg$^{-1}$** may be given at 5 min intervals

The infusion rate may be doubled to **30ml.kg$^{-1}$.h$^{-1}$**

Continue the infusion until cardiovascular stability is restored or the maximum dose of **12 ml.kg$^{-1}$** 20% lipid emulsion is reached


**REFERENCES**

1. www.lipidrescue.org
2. www.aagbi.org/sites/default/files/la_toxicity_2010_0.pdf


## Case 2

The Xray shows a number of abnormalities. Most prominent is the significant bilateral air space shadowing, predominantly in the right mid-zone and base; this is most likely to be due to significant pulmonary contusion. There is a very enlarged gastric bubble and a coincidental finding of a marked thoracic scoliosis.

Gastric insufflation can often occur during resuscitation as a result of hand ventilation through a partially obstructed airway. Meticulous attention to preserving airway patency will decrease gastric inflation. Useful measures to achieve this include:

1. Head tilt/chin lift or jaw thrust with in-line stabilisation where cervical injury is suspected.

2. Early use of airway adjuncts (e.g. oropharyngeal or nasopharyngeal airway device, noting that the latter is contraindicated in suspected base of skull fracture).

3. Synchronisation of bag-squeezing with any patient respiratory effort.

4. Avoidance of excessive inflation pressures and tidal volumes.

If bag and mask ventilation has been necessary for a significant period, gastric decompression via a nasogastric or orogastric tube should be performed as soon as possible if there are no contraindications. The tube position must be confirmed on Xray or trauma CT scan.

There is nothing on the chest Xray to explain the degree of haemodynamic compromise in this patient. The surgeon is probably correct to suspect abdominal injury and bleeding. Despite its position under the left ribcage, the spleen is the organ most commonly injured in blunt abdominal trauma. The adult spleen weighs 150-200g and receives approximately 5% of cardiac output.

Splenic injuries should be treated non-operatively where possible. Conservative management is currently favoured for those patients who are haemodynamically stable, have no other significant injuries, are aged under 55 years and maintain a steady haemoglobin level for 12 to 48 hours. A CT scan is the investigation of choice for these patients. The American Association for the Surgery of Trauma (AAST) have the following injury grading system.

Those with significant compromise require urgent intervention. Your patient falls into this category. In many hospitals, an urgent laparotomy and splenectomy would be the intervention of choice. Recently splenic angio-embolization by an interventional radiologist has gained popularity in centres where this facility is available at short notice. This approach avoids the surgical risks associated with a laparotomy, but the patient is still likely to require an anaesthetic. A surgeon and operating room facilities should remain on standby when unstable patients are managed in this way.

The role of CT scanning for unstable patients with suspected high grade splenic injuries has also evolved alongside the development of splenic angio-embolisation (SAE). It was previously accepted that delaying transfer to theatre in order to perform a CT scan would cost valuable time and lead to increased morbidity and mortality. Now, with the advent of much faster scanners and in hospitals with suitable facilities, it can be possible to perform a rapid trauma CT scan followed by urgent SAE. In these centres, imaging may be considered even in those patients presenting with cardiovascular instability.

**Tranexamic acid in trauma**

| | |
|---|---|
| Adult regime: | loading dose 1000mg in 100ml 0.9% saline over 10 min |
| | infusion 1000mg in 100ml 0.9% saline over 8hrs |

**Grade I**

- subcapsular haematoma < 10% of surface area
- capsular laceration < 1cm depth

**Grade II**

- subcapsular haematoma 10 - 50% of surface area
- intraparenchymal haematoma <5cm in diameter
- laceration 1 - 3cm depth not involving trabecular vessels

**Grade III**

- subcapsular haematoma >50% of surface area or expanding
- intraparenchymal haematoma >5cm or expanding
- laceration >3cm depth or involving trabecular vessels
- ruptured subcapsular or parenchymal haematoma

**Grade IV**

- laceration involving segmental or hilar vessels with major devascularization (>25% of spleen)

**Grade V**

- shattered spleen
- hilar vascular injury with devascularised spleen

A recent study of the value of tranexamic acid in trauma (CRASH-2), showed that administration of this drug within 4 hours of injury is associated with decreased mortality.[4] Tranexamic acid is an antifibrinolytic agent whose mechanism of action is to inhibit the conversion of plasminogen to plasmin, the molecule responsible for fibrin degradation.

The spleen is the main site of production of IgM antibodies required for the body's immune response to encapsulated micro-organisms. Patients who undergo splenectomy following trauma have an increased susceptibility to infection by *H. influenza, S. pneumonia* and *meningococcus*, and should be vaccinated against these bacteria at least 14 days post-operatively. Earlier immunisation has been associated with lower subsequent antibody function and is not recommended.[5] Prophylactic daily penicillin is recommended in asplenic children and should be considered in otherwise immunocompromised adults although regional guidelines vary.

## REFERENCES


1. Tinkoff G, Esposito TJ, Reed J et al. American Association for the Surgery of Trauma Organ Injury Scale I: spleen, liver and kidney, validation based on the National Trauma Data Bank. *J Am Coll Surg* 2008; **207**: 646.

2. The CRASH-2 collaborators. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. *Lancet* 2010; **376**: 23-32.

3. Shatz DV, Schinsky MF, Pais LB et al. Immune rsponses of splenectomised trauma patients to the 23-valent pneumococcal polysaccharide vaccine at 1 vs 7 vs 14 days after splenectomy. *J Trauma* 1998; **44**: 760.

## Case 3

The Xray shows the patient's heart is large and globular with sharp outlines. Given the history, the diagnosis is likely to be pericardial effusion with cardiac tamponade. Pericardial collections of greater than 250ml cause the heart shadow to enlarge and become globular or pear shaped in appearance.

The typical ECG findings associated with a pericardial effusion are low-voltage QRS complexes and a sinus tachycardia. Large effusions can allow the heart to change position within the pericardium from beat to beat (known as a 'swinging heart'). This can occasionally be seen as beat to beat changes in the appearance of the QRS complex, a phenomenon known as 'electrical alternans' (Figure 6).

Pericarditis is the most common cause of a pericardial effusion seen in the UK and an ECG is diagnostic (although it may be normal in 10% of cases). Acutely, raised ST segments with a 'saddle shape' are often seen, as shown in Figure 4. The ST segment is always depressed in aVR. PR segment depression is sometimes seen and whilst it is highly diagnostic it has to be specifically sought.

ST elevation caused by pericarditis is often misdiagnosed as an ST elevation myocardial infarction in leads V2, V3 & V4. Infarct related ST elevation is seen in territorial leads, e.g. anterior or inferior and rarely in multiple territories, while ST elevation seen in pericarditis should be global. Thrombolysing a patient with acute pericarditis, misdiagnosed as acute MI, can cause a catastrophic haemopericardium.

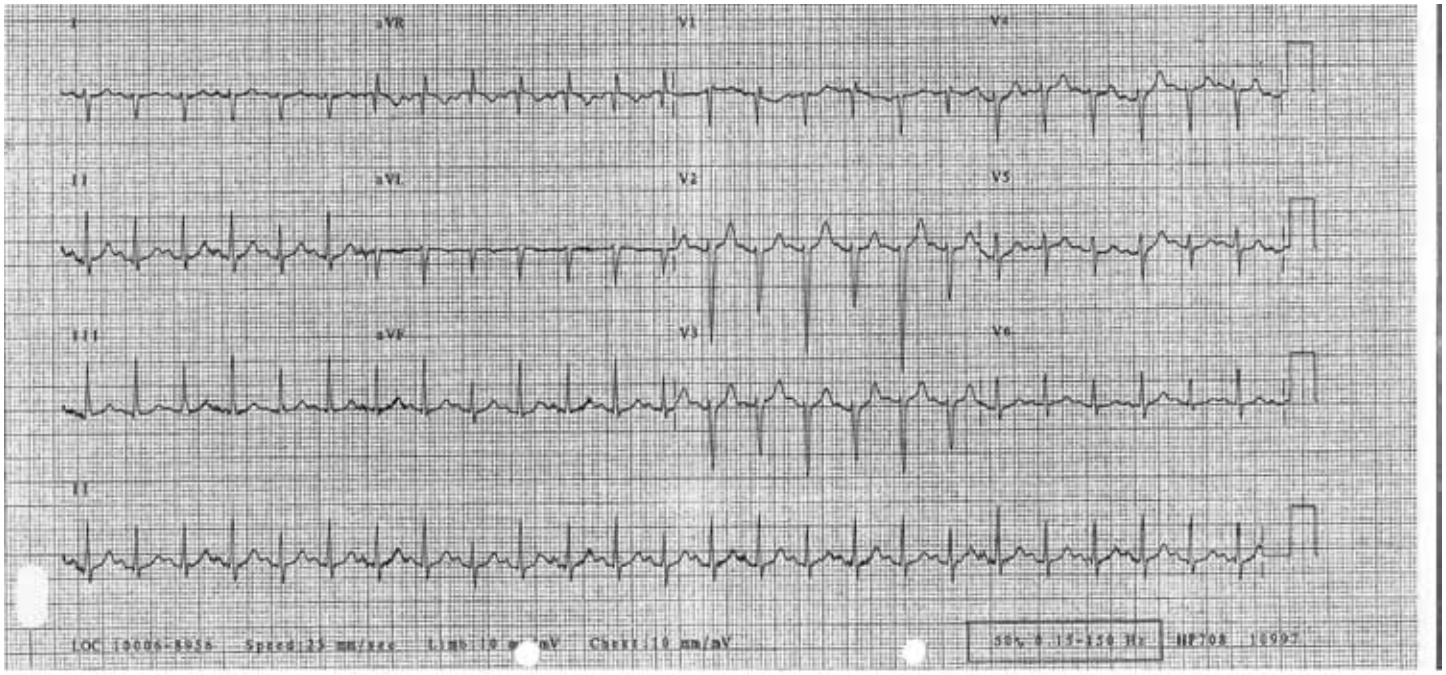
Cardiac tamponade occurs when the volume of the effusion between the pericardial sac and the heart muscle is sufficient to cause compression of the cardiac chambers. The effects of compression are greatest on the right atrium and ventricle, as these form a lower pressure system, therefore the clinical effects of compression are those of right ventricular failure. The main variable that contributes to the magnitude of these haemodynamics effects is the speed of accumulation of the effusion. Acute tamponade in an adult may occur when little as 100-200ml of fluid accumulates in the pericardial sac, whilst chronic effusions can reach volumes of 1000ml without clinical signs of tamponade. The speed of onset is often dependent on age, cardiovascular 'fitness', the cause of the effusion and the response to any treatment. The size of the effusion itself does not correlate particularly well with the degree of haemodynamic compromise.

**Presenting symptoms of cardiac tamponade**

| | |
|---|---|
| Acute | cardiac arrest (pulseless electrical activity)<br>hypotension<br>shock<br>confusion |
| Sub-acute | shortness of breath (cardiac failure)<br>chest discomfort<br>symptoms of complications of compromised circulation<br>e.g. renal failure, liver/mesenteric ischaemia compression of adjacent structures e.g. hoarse voice |

A pericardial effusion has a number of associated clinical features. Individually they are not diagnostic, but together they are suggestive. Beck's Triad of hypotension, distended neck veins and muffled heart

**Figure 6.** *An ECG (from a different patient), showing electrical alternans. This patient suffered acute pericardial tamponade due to bleeding, on the first postoperative night following coronary artery bypass surgery*



sounds is considered pathognomic of cardiac tamponade, but is only present in a minority of cases.

| Signs of a pericardial effusion | |
|---|---|
| Heart sounds soft and distant | The effusion effectively dampens sound transmission through the thorax |
| Apex beat obscured | Dampening of the palpable beat by the effusion. |
| Pericardial friction rub | Often auscultated best over the apex<br>May be present if effusion caused by pericarditis but rub subsides as effusion develops. |

| Signs of cardiac tamponade | |
|---|---|
| Raised jugular venous pressure | Caused by high right heart pressures |
| Hypotension | Secondary to reduced stroke volume (reduced right ventricular output, dictates low left ventricular output) |
| Pulsus paradoxus | Represents a drop in inspiratory systolic pressure of >10mmHg with respiration |
| Confusion | Secondary to low blood pressure |

*Causes of pericardial effusion*

There are a large number of causes of pericardial effusion. They may be considered as pathologies that cause 'acute' or 'sub-acute' effusion or tamponade.

This child is haemodynamically unstable and, importantly, he has a fixed cardiac output due to compressive limitation on his right ventricular output. General anaesthetic will abolish any compensatory increase in his systemic vascular resistance and may reduce his heart rate and cardiac contractility. This will further compromise his circulation and possibly precipitate cardiac arrest and death. A general anaesthetic must be avoided if at all possible.

Needle aspiration of the effusion and drain insertion may be achieved under local anaesthesia. Aseptic technique is essential and ultrasound guidance should ideally be used. If time permits, application of local anaesthetic cream to the chest can achieve effective anaesthesia. This can then be supplemented with local anaesthetic infiltration if required. Parental assistance may be very helpful to try to keep the child relaxed and compliant with the treatment.

Monitoring of BP, HR and oxygen saturation is essential throughout any intervention. Poor patient tolerance may threaten the safety and efficacy of the procedure. Ketamine sedation may be considered in such cases with extreme caution. If this is absolutely necessary very small bolus doses should be used, starting with perhaps 0.125mg. $kg^{-1}$ IV.

If a general anaesthetic is unavoidable then the following points should be considered.

1. The risks should be clearly explained and documented.
2. A preoperative echocardiogram to establish the degree of tamponade and impairment of cardiac function is helpful.
3. Central venous pressure and invasive arterial pressure monitoring should be achieved.
4. Pericardial drainage should be sought prior to surgery if possible.
5. Drugs that cause a reduction in systemic vascular resistance (SVR) or cardiac contractility should be avoided and the patient kept normotensive. Consider induction and maintenance of anaesthesia with ketamine, as it increases heart rate, contractility and SVR.
6. As positive pressure ventilation reduces venous return spontaneous breathing should be maintained if possible.
7. Hypovolaemia can precipitate tamponade in an otherwise haemodynamically asymptomatic effusion.
8. IV diuretics are contraindicated and may be fatal.

**Acute tamponade/effusion**

Myocardial infarction leading to cardiac tissue rupture

Trauma

Aortic dissection

Spontaneous bleeding, may be seen in;

- uraemia (inhibits platelet function)
- thrombocytopenia
- anti-coagulation

Cardiac surgery or pacing (iatrogenic trauma to cardiac tissue)

**Sub-acute tamponade/effusion**

Malignant disease

Ionising radiation

Systemic lupus erythematosis

Hypothyroidism

Idiopathic pericarditis

Infections

- TB
- bacterial

Update in
Anaesthesia

# From the journals

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## Critical Care - Hypothermia after cardiac arrest

**Targeted temperature management at 33°C versus 36°C after cardiac arrest.**
Nielsen N, Wetterslev J, Cronberg T et al for the TTM Trial Investigators. *New England Journal of Medicine* 2013; **369**: 2197-206. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24237006.

Therapeutic hypothermia is a established treatment approach for unconscious survivors following out-of-hospital cardiac arrests. This has been included in international resuscitation guidelines based on the improved mortality and neurological outcomes demonstrated in previous studies. However, there has been some debate surrounding the potential limitations of these studies and more specifically the optimum target temperature.

This multicentre international trial set out to compare two target temperatures – the standard 33ºC versus 36ºC ('normothermia') in unconscious patients following cardiac arrest. 950 patients were randomly assigned to one of these target temperatures for a period of 28 hours then gradually rewarmed. This was achieved using either intravascular or surface cooling devices. The end points included all-cause mortality and poor neurological function.

The results showed no significant difference in all-cause mortality in the 33ºC group compared to the 36ºC group (50% vs 48%, p=0.51). Follow-up at 180 days showed the proportion of patients with poor neurological function to be similar between the two groups (54% vs 52%, p=0.78). In addition, a standardised protocol was used in order to guide withdrawal of life-sustaining treatments in study participants.

Overall, the authors state that cooling patients to the traditional 33ºC confers no additional benefit when compared to maintaining a target temperature of 36ºC. They suggest that the active prevention of fever may have more importance than active cooling in this particular patient group. Although targeted temperature control remains important, this study has lead to adoption of a more conservative approach to cooling in most units.

## Critical Care - Target blood pressure in septic shock

**High versus low blood-pressure target in patients with septic shock.**
Asfar P, Meziani F, Hamel JF et al for the SEPSISPAM Investigators. *New England Journal of Medicine* 2014; **370**: 1583-93.

In the initial resuscitation of patients with septic shock, a mean arterial blood pressure (MAP) of at least 65 mmHg has traditionally been targeted as recommended by the Surviving Sepsis Campaign. This study set out to determine whether targeting a higher MAP would result in better patient outcomes.

This multicentre trial was conducted across 29 centres throughout France. 776 patients with septic shock were randomly assigned to the high target group (MAP 80 to 85 mmHg) or the low target group (MAP 65 to 70 mmHg). Following initial fluid resuscitation, vasopressor therapy was titrated according to patient group for a maximum of 5 days. Endpoints included 28-day mortality, 90-day mortality and serious adverse events.

The results showed no significant difference in either 28-day or 90-day mortality between the two groups. Incidence of new onset atrial fibrillation was increased in the high target group compared to the low target group (6.7% vs 2.8%, p=0.02). In addition, patients with chronic hypertension were more likely to require renal replacement therapy when a lower target MAP was used. In both groups, the measured MAP was often higher than the desired target range although

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the authors report that the 'between-group difference' was well maintained.

In conclusion, the authors state that targeting a higher MAP (80 to 85 mmHg) in patients with septic shock does not improve mortality when compared to the standard target MAP (65 to 70 mmHg).

However, in patients with chronic hypertension they argue that targeting a higher MAP may provide additional benefits in terms of renal protection.

## Critical Care - Protective ventilation strategy in theatre

### A trial of intraoperative low-tidal-volume ventilation in abdominal surgery.

Futier MD, Constantin JM, Paugam-Burtz C et al for the IMPROVE Study Group. *New England Journal of Medicine* 2013; **369:** 428-437.

Use of low tidal volumes for ventilation is an established therapy for management of patients with ARDS in critical care. However, patients undergoing general anaesthesia have traditionally been ventilated using much higher tidal volumes. The role of lung-protective ventilation strategies on patient outcomes following elective surgery remains unclear. This study set out to compare the two approaches in patients undergoing elective abdominal surgery.

This multicentre double-blind trial recruited 400 patients deemed at higher risk of pulmonary complications following abdominal surgery. These patients were then randomised into the lung-protective ventilation group (tidal volume 6-8ml.kg$^{-1}$ ideal body weight, PEEP and recruitment manoeuvres) or the non-protective mechanical ventilation group (tidal volume 10-12ml.kg$^{-1}$). The primary end point was the occurrence of a major complication in the first 7 postoperative days – these included pneumonia, acute respiratory failure requiring ventilatory support, sepsis and death.

Overall, major postoperative complications were significantly increased in the non-protective group compared to the lung-protective group (27.5% vs 10.5%, p=0.001). The non-protective group also had higher rates of acute respiratory failure requiring non-invasive ventilation (14.5% vs 4.5%, p=0.002). Although hospital stay was slightly reduced in the lung protective group, overall mortality rates were approximately 3% in both groups.

The authors state that intra-operative lung-protective strategies for mechanical ventilation improve outcomes in patients in undergoing abdominal surgery when compared to more traditional approaches. Adoption of lung-protective strategies beyond the realms of critical care may lead to significant improvements in post-operative outcomes in elective surgical patients. Considering the numbers of patients who may potentially benefit from this approach, reduced length of stay and overall complication rate may lead to significant reductions in healthcare utilisation.

## Critical Care - Prone ventilation in severe ARDS

### Prone positioning in severe acute respiratory distress syndrome.

Guerin C, Reignier J, Richard JC et al. *New England Journal of Medicine* 2013; **368**: 2159-68.

Although previous studies on prone positioning in Acute Respiratory Distress Syndrome (ARDS) have showed improved physiological parameters, no benefit in patient survival has been demonstrated. This trial examined the effect of early prone positioning in patients with severe ARDS on mortality.

Inclusion criteria were:

- ARDS as defined according to the American–European Consensus Conference criteria
- severe ARDS (defined as a $PaO_2$:$FIO_2$ ratio of <150 mmHg, with an $FIO_2$ of ≥0.6, a PEEP of ≥5 cm of water
- intubated and ventilated for ARDS for less than 36 hours.

466 patients were randomly assigned to either prone (intervention) or supine positioning (control). The intervention group were positioned prone for at least 16 consecutive hours per day on standard ICU beds. Prone positioning was stopped once patients could achieve predetermined oxygenation targets whilst supine for at least four hours. The primary outcome was 28-day all-cause mortality.

Prone positioning significantly reduced 28-day all-cause mortality when compared to supine positioning (16.0% vs 32.8%, p<0.001). The 90-day all-cause mortality was also examined and again prone positioning showed significantly improved outcomes (23.6% vs 41.0%, p<0.001). Although the patients in the supine group had slightly higher SOFA scores at baseline, mortality rates remained significantly lower in the prone group following adjustment. There was no significant difference in incidence of complications between the two study groups. It should be noted that the units taking part in the study had significant experience in using this particular prone positioning protocol.

The authors conclude that early prone positioning in prolonged sessions for patients with severe ARDS may significantly improve survival. This relatively large reduction in mortality strongly supports the adoption of this intervention in the management of severe ARDS.

## Paediatric Anaesthesia - Supraglottic airway devices in children with known difficult airway

### Elective use of supraglottic airway devices for primary airway management in children with difficult airways.

Jagannathan N, Sequera-Ramos L, Sohn L et al. *British Journal of Anaesthesia* 2014; **112**: 742-8.

Supraglottic airways (SGAs) are regularly used in the management of both routine and emergent difficult airways. Although there are many reports of these being used to facilitate fibreoptic-guided tracheal intubation, limited data exists on the use of SGAs are the primary means of managing a difficult airway. The authors of this study set out examine the success rates and adverse events associated with the use of these devices as the primary airway in children with difficult airways.

This study was conducted as a retrospective analysis of data from a single paediatric hospital in the United States. Over a four-year period, 77 272 children received general anaesthesia for a wide range of procedures. 459 patients were reported as having a difficult airway – defined as difficult direct laryngoscopy (Cormack and Lehane grade 3 or above), difficult mask ventilation or both.

The SGA was used as the primary airway in 109 patients with a success rate of 96% (105/109 cases). Two patients required an alternative SGA and two required tracheal intubation using a fibreoptic bronchoscope through the SGA. There were no reported cases of regurgitation of gastric contents, bronchospasm or death. However, there are a number of confounding factors which may have influenced the clinician to opt for tracheal intubation as the primary airway thus excluding these patients from this group. In addition, the retrospective nature of the study limits the reliability of the findings.

The authors propose that SGAs provide an effective option for primary airway management in the paediatric difficult airway population. The relative success of these devices in this patient group may be attributable to the high proportion of upper airway conditions which can successfully be bypassed with SGAs. In addition to being less invasive than tracheal intubation, it provides a useful adjunct to fibreoptic intubation should the need arise (Plan B). Further prospective work in this area is required to support these findings.

## Obstetric Anaesthesia - Failed intubation in parturients

### Failed tracheal intubation in obstetric anaesthesia: 2 yr national case-control study in the UK.

Quinn AC, Milne D, Columb M, Gorton H, Knight M. *British Journal of Anaesthesia* 2013; **110**: 74-80.

Although the incidence of failed intubation is significantly higher in the obstetric population, the figures quoted are highly variable; often in the region of 1 per 250 cases. At present, there is no national reporting of these events within the UK. The aim of this study was to estimate national incidence and identify risk factors by establishing a surveillance system.

This prospective case-control study obtained data from all consultant-led obstetric units in the UK over a two-year period. For each reported failed intubation (index case), these units provided additional data on the two preceding successful intubations to allow comparison (controls). There were 57 reports of failed intubation which could be extrapolated to estimate incidence in the obstetric population at 1:224 cases (95% CI 179-281).

Multivariate analysis of these cases found that increased age and raised BMI were significant independent predictors of failed intubation. The authors report that for every unit increase in BMI, the risk of failed intubation increased by 7%. Maternal age may reflect the increased incidence of co-morbidities. Airway assessment was often incomplete and the documentation of a Mallampati score was in itself a predictor of increased risk. There was a 2.5 fold increase in rates of failed intubation when trainee anaesthetists were present when compared to consultant colleagues. The standard LMA was used as a rescue airway in the majority of cases (39/57) with one patient requiring a surgical airway. Although there were four reported cases of gastric aspiration in the failed intubation group there were no maternal deaths during this study period.

The study effectively confirms the estimated incidence of failed intubations within the UK obstetric population. The authors highlight the impact of increasing obesity within this group and are keen to support the development of failed intubation drills specific to this specialty.

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Update in Anaesthesia

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

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- Some readers' first language may not be English. Please keep your text straightforward and avoid long sentences and complex terminology. Explain words and abbreviations that may not be universally standardised. Aim to include the full range of therapies available worldwide, but provide most detailed descriptions of those therapies available in resource-poor settings (see 'Management of sepsis with limited resources' in *Update 23* – www.worldanaesthesia org/component/option,com_docmantask,cat_view gid,67 Itemid,49/). Discuss older drugs as well as newer ones; halothane, thiopentone, ketamine and ether are widely used around the world.
- The article should be long enough to cover the topic in reasonable detail. Many readers will not have access to texts or journals to supplement their reading. Include text boxes and teaching points to make the layout interesting. Avoid long numbered lists with complex subdivisions. Check that your text is correct, particularly drug doses, as many readers will not be able to verify them.

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

### Drug doses

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

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

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

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### Tables

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

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

### References

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

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

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

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

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

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

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

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

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

## UPDATE SHORT REPORTS

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

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

### Legal considerations

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- Avoid use of identifiable names, initials and hospital numbers of patients.

- Human subjects of case reports, research or audits should not be identifiable. Manuscripts should not disclose patients' names, initials, hospital numbers (or other data that might identify the patient(s)).

- Guides for use of tables, figures and illustrations are as described above for Clinical Overview articles.

### Brief Communications

- Original investigative articles or audits of patient outcome or clinical techniques.

- Up to 1500 words (approximately 2 pages of *Update in Anaesthesia).*

- Subdivided into:
  - Summary (maximum five sentences) and key words
  - Introduction
  - Patients and methods
  - Results
  - Discussion
  - Acknowledgements
  - References – maximum 15
  - Tables and/or figures - limited to two per article.

### Case Reports

- Suitable for presenting descriptive studies (a series of cases), personal experience or individual case reports of particular interest.

- Up to 800 words. Three tables or figures is allowed in addition to text.

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- Up to seven references may be given.

### Correspondence

- Welcomed on any subject, including editorials or articles that have appeared in *Update in Anaesthesia*.

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### Proofs

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

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

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# Update in Anaesthesia

Education for anaesthetists worldwide

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