Notes on Thoracic Anaesthesia

April 2011

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\[\text{The Anthony Booth Trust}\]
\[\text{An Aplastic Anaemia Support Charity}\]
The single biggest problem we face is that of visualisation.
Richard P. Feynman (1918–1988)\textsuperscript{1}

\textsuperscript{1}The Mathematical Gazette, (1996); 80, 267

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Preface

This introductory booklet is essentially a collection of practical notes on some of the topics relevant to the thoracic anaesthesia training module, and reflects a distinctly personal approach. It is largely a vehicle for useful references, and still represents ‘work in progress’; for example, Chapter 1 is clearly rudimentary—waiting to be distilled further. The main topics are essentially those for which I developed some interest and tried to establish some underlying conceptual structure. Most chapters were motivated by a particular question or line of approach, as follows.

Anatomy — what is the useful functional anatomy for thoracic anaesthetists?
Bronchoscopy — how does the fibreoptic bronchoscope influence our perceived orientation of the anatomy?
Tracheostomy — how can we avoid tracheostomy-related problems?
One-lung anaesthesia — is there an optimum sequence for placing a double-lumen tube?
Drugs — can particular dilutions facilitate administering vasoactive drugs?
Supporting technologies — how did some of the key developments arise?

I would like to thank Dr J James for help with the virtual bronchoscopy part of Figure 5.1, and all those anaesthetists who contributed data to the TEPID database. Finally, I would also like to acknowledge the considerable help and assistance I have received from the Operating Department Practitioners, the Theatre Practitioners, and the staff at the PGMEC library and Department of Medical Illustration.

RWD Nickalls
April 2011

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Index
General topics

1.1 Syllabus

The thoracic anaesthesia syllabus for the CCT in Anaesthesia is detailed in the ‘cardiothoracic’ sections of the Intermediate, Higher and Advanced-level training documents (2nd edition, August 2010) on the Royal College of Anaesthetists website as follows:

Intermediate:  http://www.rcoa.ac.uk/docs/CCTAnnex C.doc
Higher:  http://www.rcoa.ac.uk/docs/CCTAnnex D.doc
Advanced:  http://www.rcoa.ac.uk/docs/CCT in Anaesthetics Annex E.doc

1.2 General resources

Books


[chapters: Anesthesiology national CME program and ASA activities in simulation / Does simulation improve patient safety?: self-efficacy, competence, operational performance, and patient safety / Simulation applications for human factors and systems evaluation / Credentialing and certifying with simulation / Statewide simulation systems: the next step for anesthesiology? / Crew resource management and team training / Simulation: translation to improved team performance / Virtual worlds and team training / Virtual reality simulations / Credentialing and certifying with simulation / Debriefing with good judgment: combining rigorous feedback with genuine inquiry / Integration of standardized patients into simulation ]

[chapters: Evidence-based management of one-lung ventilation / Oxygen toxicity during one-lung ventilation: is it time to re-evaluate our practice? / Anesthetic considerations for airway stenting in adult patients / Perioperative anesthetic management for esophagectomy / Anesthetic considerations for patients with anterior mediastinal masses / The emerging role of minimally invasive surgical techniques for the treatment of lung malignancy in the elderly / Prevention and management of perioperative arrhythmias in the thoracic surgical population / Pulmonary vasodilators—treating the right ventricle / Post thoracotomy pain management problems / Postthoracotomy paravertebral analgesia: will it replace epidural analgesia? / Advances in extracorporeal ventilation ]

• Allman KG, Wilson IH (Eds.) (2006). *Oxford Handbook of Anaesthesia*. 2nd. ed. (Oxford University Press) [good sections on (a) THORACIC ANAESTHESIA (pp. 351–383), and (b) DRUG FORMULARY (pp. 1105–1165).]


**Articles**


Book/journal searches

Good starting points are (a) Google books (http://books.google.com/), and (b) the Wikipedia page on ‘book sources’ (http://en.wikipedia.org/wiki/book_sources/).

For buying books (secondhand and new) Abebooks (http://www.abebooks.com/) is particularly useful, since it is usually possible to find the website & telephone number of the individual booksellers, and then buy from them directly.

- PubMed Central (PMC): http://www.ncbi.nlm.nih.gov/pmc/
  A free archive of life sciences journals. See the ‘journal list’ for list of all available journals.

- Science Direct: http://www.sciencedirect.com/
  A particularly useful interface for viewing the tables of contents (TOC) of journals.

- Unbound Medicine: http://www.unboundmedicine.com/medline/ebm/
  This is a useful free interface to the MEDLINE search engine.

- Priory Medical Journals Online: http://www.priory.com/

- The Cochrane Library: http://www.thecochranelibrary.com/

- Copac: http://copac.ac.uk/
  It is the national, academic and specialist library catalogue. It provides free access to the merged online catalogues of 24 major research libraries in the UK and Ireland, including the British Library, and the national libraries of Scotland, Wales and Ireland.

- British Library (London): http://www.bl.uk/
  The BL integrated catalogue is freely available online.

- Nottingham Univ. library catalogue: http://aleph.nottingham.ac.uk/ALEPH/

- Project Gutenberg: http://www.gutenberg.org/
  Free eBooks on-line.

Anesthesiology Clinics

for TOC see: http://www.sciencedirect.com/science/journal/19322275

- Cardiac anesthesia: today and tomorrow (2008); 26 (September)

- Thoracic anesthesia (2008); 26 (June)

- New vistas in patient safety and simulation (2007); 25 (June)

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Thoracic Surgery Clinics

for TOC see: http://www.sciencedirect.com/science/journal/15474127

- Thymoma (2011); 21 (February)
- Chest wall surgery (2010); 20 (November)
- Air leak after pulmonary resection (2010); 20 (August)
- Technical advances in mediastinal surgery (2010); 20 (May)
- Imaging of thoracic diseases (2010); 20 (February)
- Surgical conditions of the diaphragm (2009); 19 (November)
- Thoracic surgery in the elderly (2009); 19 (August)
- Update on surgical and endoscopic management of emphysema (2009); 19 (May)
- Diseases of the mediastinum (2009); 19 (February)
- Thoracic anatomy: Chest wall, airway, lungs. (2007); 17 (November)

Clinics in Chest Medicine

for TOC see: http://www.sciencedirect.com/science/journal/02725231

- Interventional pulmonology (bronchoscopy) (2010); 31 (March)
- Tuberculosis (2009); 30 (December)
- Obesity and respiratory disease (2009); 30 (September)
- Fungal diseases (2009); 30 (June)
- Nonpulmonary critical care (2009); 30 (March)
- Update in sepsis (2008); 29 (December)
- Controversies in mechanical ventilation (2008); 29 (June)
- Contemporary chest imaging (2008); 29 (March)
- Artificial airways (2003); 24 (September).
- Pulmonary function testing (2001); 22 (December).
- Prolonged critical illness: management of long-term acute care (2001); 22 (March).
- Flexible bronchoscopy update (2001); 22 (June).
• Acute respiratory distress syndrome (2000); 21 (September).
• Intensive care unit complications (1999); 20 (June).
• Flexible bronchoscopy in the 21st century (1999); 20 (March).

History

• Medical History is the journal of the Wellcome Historical Medical Library, London. A useful resource of historical articles—some of which relate to anaesthesia. It is available on-line via PubMed Central at http://www.ncbi.nlm.nih.gov/pmc/journals/228/.
• The Wellcome Collection. http://www.wellcomecollection.org/
• The Wellcome Library. http://library.wellcome.ac.uk/ For a list of free online titles project see http://library.wellcome.ac.uk/backfiles/.

Miscellaneous

• Slinger’s thoracic anaesthesia site: http://www.thoracic-anesthesia.com/
• Chest (see archive, collections and supplements): http://chestjournal.chestpubs.org/

3This excellent edition also includes a commentary, reviews, and copies of the original 1953 papers.
• Medscape’s clinical reference website: http://emedicine.medscape.com/
  There is an extensive database on clinical procedures. See also the sections
  on thoracic_surgery, radiology, critical_care, etc.

• Best evidence topics: http://www.bestbets.org.
  A collection of mini-reviews presenting the evidence base for various topics.

• Virtual Hospital: http://www.uihealthcare.com/vh/
  Many interesting thoracic articles available via their ‘search’ facility.

• Bronchoscopy Atlas: http://www.int-med.uiowa.edu/research/tlirp/
  BronchoscopyAtlas/Home.html

• Clinical Cases and Images website: http://clinicalcases.org/.

• Learning Radiology website: http://www.learningradiology.com/

• Wikipedia is well worth a trawl occasionally, since it has an increasing number of
  surprisingly detailed medical entries with excellent links; for example:
  http://en.wikipedia.org/wiki/Tracheal_intubation

• Anesthesia Patient Safety Foundation (APSF): (http://www.apsf.org/).

• SHEPHERDS FALKINERS, 76 Southampton Row, London, WC1B 4AR. Tel: 020-783-11151 http://store.falkiners.com/ (fine paper & bookbinding supplies)

1.3 Preoperative evaluation

1.3.1 Lung function evaluation

The British Thoracic Society guidelines article (BTS/SCTS Working Group 2001) recom-
mends an \( FEV_1 > 2 \) litres for pneumonectomy, and \( FEV_1 > 1.5 \) litres for a lobectomy.
Patients with values less than these are ‘high risk’ and should be evaluated further (transfer
factor; exercise test). However, some patients with diffuse interstitial lung disease may
well have a low transfer factor in spite of good spirometry, and should be evaluated further.
This document gives details of preoperative respiratory and cardiovascular function tests
for all such patients, and is well worth reading. Good general reviews are Datta and Lahiri

**Role of supine LFTs?** Finally, we should not overlook the possibility that some
supine lung-function testing (and cardiac testing) might be more appropriate for supine
patients under anaesthesia—but I have failed to find any literature on this as yet.


• Powell CA and Caplan CE (2001). Pulmonary function tests in preoperative pulmonary evaluation [92 refs]. In: Chupp GL (Ed), *Clinics in Chest Medicine*; 22 (no. 4, December) [this vol of *Clinics in Chest Medicine* also includes useful articles by Pride NB (Tests of forced expiration and inspiration), and Gibson CJ (Lung volumes and elasticity)]


1.3.2 Cardiac function evaluation


1.3.3 Obesity-related problems

This list is included here since one-lung anaesthesia in obese patients is such a formidable technical exercise. It is sometimes worth considering the use of temporary post-extubation CPAP or BIPAP support in recovery.

- Obesity and respiratory disease. Clinics in Chest Medicine (2009); 30 (September)


1.4 Open lung biopsy

These patients usually have diffuse lung disease and relatively poor lung function, and are generally referred to the surgeons following a failed percutaneous or bronchoscopic lung biopsy. Although a single-lumen tube is often all that is required for a supine procedure (e.g., a mediastinotomy or mediastinoscopy), a double-lumen tube and lateral position is sometimes necessary. Consider selective lobar isolation if lung function is poor (see Mentzelopoulos, Rellos, Tzoufi et al. 2003).

1.5 Tracheal resection


1.6 Differential lung ventilation

This is occasionally indicated in patients where the two lungs have a significantly different compliance. The Dräger ventilators have the facility to be linked in pairs such that one functions as the slave of the other, allowing the phase relations of the two ventilators to be easily controlled.


1.7 Thymectomy and myasthenia gravis

Good articles on the anaesthetic management of myasthenia gravis are few in number; the most useful ones I have found are those by Zielinski (2011), Eisenkraft (1987) and Redfern *et al.* (1987). Note that the February 2011 issue of the *Thoracic Surgery Clinics* is on thymoma.

A patient with myasthenia gravis is typically maintained on oral pyridostigmine tablets. Postoperatively either pyridostigmine (subcutaneously) or neostigmine⁴ is generally used until the patient’s usual oral maintenance dose (pyridostigmine tablets) can be resumed. Note that 60 mg of the oral pyridostigmine preparation is equivalent to 2 mg of the parenteral product.⁵ A typical adult total daily parenteral dose of pyridostigmine is approximately 10–40 mg (1–4 mg given 3–4 hrly).

The exact neuromuscular defect (presynaptic vs. postsynaptic) seems not to be fully resolved as yet. The presynaptic acetylcholine stores and their release are not diminished in myasthenia (Cull-Candy *et al.* 1980).


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⁴Pyridostigmine is the preferred agent owing to its longer action.
⁵CHN Pharmacy factsheet.


1.8 Bilateral pleurectomy via sternal split

An important aspect of this procedure is taking care to avoid having both lungs partially deflated at the same time! This can easily happen once both pleura are open, and the resulting desaturation can be both severe and slow to recover.

The problem tends to occur when the surgeons have almost finished working on the first lung, as then one of the surgeons may start investigating (& collapsing) the other lung before the first lung has been fully re-expanded. Note there is sometimes quite a long time-lag after re-expanding a lung and the saturation improving.

The fact that the patient is supine compounds the problem, as there is no ‘bottom’ lung receiving most of the cardiac output. This is an important difference, since with a normal thoracotomy in the lateral position not only is the bottom lung always fully expanded but it is also receiving the majority of the cardiac output.

The key principles are therefore (a) use a double-lumen tube, (b) use some PEEP, and (c) make sure the first lung is fully re-expanded before allowing the surgeons to start working on the other lung. The same general principles probably also apply for lung-volume reduction surgery, as this is usually a bilateral procedure via a sternal split as well (see below).

1.9 Lung-volume reduction surgery

• Calverley PMA (2003). Closing the NETT on lung volume reduction surgery. Thorax; 58, 651–653. [review of the National Emphysema Treatment Trial (NETT)]


• Cooper JD and Lefrak SS (1999). Lung-reduction surgery: 5 years on. The Lancet; 353 (Suppl. 1[surgery]), 26–27. [At 2 years pulmonary function had improved in the surgical group, but had worsened in the control (no-surgery group). By 3.5–4 yrs mortality during follow up was 30% (surgery) and 52% (no-surgery)]

1.10 Management of flail-chest


  [Internal fixation (IF) is better; with IF mean period of IPPV was 3.9 days versus 15 days with IPPV only].

1.11 Pneumothorax


1.11.1 Radiology


1.11.2 Cavity expansion with N₂O

- Eger EI (1974). Nitrous oxide transfer to closed gas spaces. In: Eger EI *Anaesthetic uptake and action* (Williams & Wilkins Company, Baltimore, USA), Chapter 10, pp. 171–183. [lung cavity expansion is fast: volume doubles with 50% N₂O; vol increases 4-fold with 75% N₂O. Bowel expansion is much slower and less complete; with 75% N₂O bowel gas volume increased 1.8-fold in 2 hrs, 2.5-fold in 4 hrs.]


1.11.3 Chest drains

Chest drains are potentially dangerous, and in May 2008 the National Patient Safety Agency (NPSA) issued an alert following 12 deaths and 15 serious adverse incidents—see the paper by Akram and Hartung (2009).

The sizes of chest drains generally used in thoracic surgery are as follows: ward insertion 28 Fr; theatre insertion 36 Fr.

- Pneumonectomy: 1 drain;

Chest drains are sometimes put on suction postoperatively in order to facilitate lung re-expansion (spontaneously breathing patients only). However, in the presence of a lung leak suction can be the cause of a tension pneumothorax if the total drain air flow is significant (i.e., if the suction cannot handle the total flow). Consequently, if you suspect a tension pneumothorax always disconnect the suction from the under-water seal until the problem has been resolved. Never apply suction to a ventilated patient.

There is also a nice ‘You tube’ internet video on “securing a chest drain” (search google for this).


1.11.4 Chest-drain bottles


---

6If there is a tension pneumothorax, then disconnecting the suction will result in an immediate discharge of air through the under-water seal.
1.11.5 Subcutaneous emphysema


1.12 Empyema


1.13 Sickle cell disease

One-lung anaesthesia in patients with a sickle-cell condition is a formidable problem requiring preparation and coordination with the Haematology department. Unfortunately good references which are useful for thoracic anaesthetists are few in number. The NHS sickle cell and thalassaemia screening programme website ([http://sct.screening.nhs.uk](http://sct.screening.nhs.uk)) is a useful source of information.

1.13.1 Anaesthesia

The main principles are: (a) optimise Hb—aim for [HbA] greater than 70%—and treat anaemia, (b) chest physiotherapy and breathing exercises, (c) good hydration, (d) pre-oxygenation, (e) minimise factors which ‘right-shift’ the Hb dissociation curve, (f) use at

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7See those I have indicated with a triangle △.

8I.e., correct acidosis, and avoid letting ETCO₂ or temperature rise above normal.
least 50% FIO₂, (g) keep the patient warm, (h) no tourniquets (tourniquet deaths have been reported even with sickle-cell trait), (i) consider regional blocks to facilitate vasodilation, (j) avoid pre- and postoperative sedation.

The most useful references I have come across from a purely practical point of view are marked by a triangle △.

- △ Firth PG and Head CA (2004). Sickle cell disease and anesthesia. *Anesthesiology*; 101, 766–785. [excellent review; 180 refs]
- Marchant WA and Wright S (2001). Aortic cross-clamping in sickle cell disease. *Anaesthesia*; 56, 286–287. [letter] [exchanged transfused to Hb 10, HbA 61%, good hydration, warm, mild alkalosis: bypass cross-clamp 9 min; no problems]

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9To facilitate vasodilation.
1.13.2 The transfusion controversy

These articles discuss two pre-operative treatments: (a) aggressive transfusion aimed at decreasing [HbS] to less than 30%, and (b) a more conservative approach designed simply to increase [Hb] to greater than 10 gm/100mls. While both regimens were equally effective in preventing post-operative complications, the ‘conservative’ regimen was associated with half as many transfusion reactions.


1.13.3 Managing sickle-cell crisis


1.13.4 Pathophysiology


1.13.5 Haemoglobin molecular-chemistry

The famous chemist Linus Pauling was instrumental in the discovery that sickle-cell disease was due to a haemoglobin abnormality. Max Perutz and John Kendrew received the Nobel Prize in 1962 for determining the structure of haemoglobin.

• Cooper C and Wilson M (1997). Cold feet. *New Scientist*: The Last Word, (February 1st). [a fascinating note on the mechanism by which the amount of heat (ΔH) associated with the exothermic oxygen-binding to haemoglobin —and its reverse— allows the feet of antarctic penguins to remain non-frozen. Its role in other animals is also explored.]


### 1.13.6 HbS & O$_2$ dissociation curve


VIRTUALLY all thoracic patients at the City Hospital have an epidural block unless this is contraindicated for some reason. Note the recent epidural ‘best practice’ publication (RCoA 2010). For history of the development of the epidural needle see Section 9.2.

- RCoA (2010). Best practice in the management of epidural analgesia in the hospital setting. Faculty of Pain Medicine, Royal College of Anaesthetists, UK (November 2010)


### 2.1 Anatomy

Two excellent books on intercostal and epidural anatomy for anaesthetists are those by Mackintosh and Bryce-Smith (1953) and Mackintosh and Lee (1973). A convenient way of identifying the thoracic levels is to use the surface markings of the scapula; T2 = top border of scapula (*superior angle*); T5 = middle of scapula; T8 = bottom of scapula (*inferior angle*).

Since a typical thoracotomy incision follows the 5th rib, the aim is to locate the tip of the catheter at about T5 (middle of scapula). Inserting the Tuohy needle at the level T7–T8 (bottom of the scapula ± one space) generally works well.

For a thoraco-abdominal incision and also for an Ivor-Lewis operation, the tip of the epidural catheter needs to be at about T7 (middle of the range T4–T10). Consequently, inserting the Tuohy needle two–three spaces further down from the bottom of the scapula (± one space) is generally satisfactory.


2.1.1 The epidural database (TEPID)\(^1\)

Several studies have tried to correlate the depth of the epidural space from the skin with a single parameter (e.g., height, weight or BMI), but none has proved particularly useful (see references in Section 2.1). This suggests that using only a single parameter is probably the wrong approach. Consequently my TEPID database uses three parameters (height, weight and gender) and gives quite accurate predictions (560+ patients in the database). Although the TEPID database was started in order to serve as a guide to the depth of the epidural space, it was John Alfred Lee (1906–1989), who was sufficiently concerned about the depth that he introduced the standard 1 cm markings on the Tuohy needle (Section 9.2) in order to try and reduce the number of inadvertent dural taps (Lee 1960; Maltby 2002).

The midline depth of the epidural space in the region T6–L3 decreases from above downwards, and is, typically, most superficial at the L2/3 space. The TEPID data for an average male and female are shown in Table 2.1. The predictive value of paramedian data was poor, and consequently this is no longer collected or displayed.

### Table 2.1: TEPID data for midline epidural depths (cm).

<table>
<thead>
<tr>
<th></th>
<th>average male</th>
<th>average female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>wt 76 ± 7.5 kg</td>
<td>wt 67 ± 7.5 kg</td>
</tr>
<tr>
<td></td>
<td>ht 176 ± 7.5 cm</td>
<td>ht 165 ± 7.5 cm</td>
</tr>
<tr>
<td>BS(+0) T7/8</td>
<td>5.7(0.59) [4.5–6.7]</td>
<td>5.5(0.66) [3.7–7.0]</td>
</tr>
<tr>
<td></td>
<td>(20)</td>
<td>(35)</td>
</tr>
<tr>
<td>BS(+1) T8/9</td>
<td>5.3(0.53) [4.5–6.5]</td>
<td>5.0(0.60) [4.0–6.5]</td>
</tr>
<tr>
<td></td>
<td>(12)</td>
<td>(14)</td>
</tr>
<tr>
<td>BS(+2) T9/10</td>
<td>5.0(1.0) [4.0–6.5]</td>
<td>4.9(1.2) [3.5–7.0]</td>
</tr>
<tr>
<td></td>
<td>(6)</td>
<td>(11)</td>
</tr>
<tr>
<td>BS(+3) T10/11</td>
<td>4.4(0.56) [3.5–5.2]</td>
<td>4.0(0.54) [3.0–5.0]</td>
</tr>
<tr>
<td></td>
<td>(8)</td>
<td>(11)</td>
</tr>
<tr>
<td></td>
<td>—</td>
<td>3.7(1.1) [3.0–4.5]</td>
</tr>
<tr>
<td>BS(+4) T11/12</td>
<td>—</td>
<td>(2)</td>
</tr>
<tr>
<td>BS(+5) T12/L1</td>
<td>4 (n=1)</td>
<td>—</td>
</tr>
</tbody>
</table>

The TEPID database, together with a Perl program, is freely available. After entering the patient’s height/weight/gender the program displays both the epidural data and the relevant tube data (single and double-lumen).

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\(^1\)Tube and EPIdural Database (TEPID). This is a collection of thoracic epidural and tube data accumulated over many years. It is freely available from [http://www.nickalls.org/dick/xenon/rwdnXenon.html](http://www.nickalls.org/dick/xenon/rwdnXenon.html)
• Lee JA (1960). Specially marked needle to facilitate epidural block. *Anaesthesia*; 15, 186. [cited from Maltby 2002]


### 2.2 General aspects

#### 2.2.1 Awake or under GA?

There is currently some discussion regarding whether epidurals should be performed under GA or not (following a couple of reported cases from Germany of paraplegia following epidural under GA).


#### 2.2.2 Midline approach

The midline depth of the epidural space appears to decrease from above downwards in the region T6–L3, probably being most superficial at the the L2/3 space. Data from our own thoracic database (TEPID; see section 2.1.1) for an average male and female are shown in Table 2.1.

#### 2.2.3 Paramedian approach

The paramedian depth of the epidural space at T7–T8 is approximately 3–5 cms. Use the depth of the lamina as a guide: the epidural space is usually $\leq 2$ cm deeper than the lamina.
2.2.4 Reducing catheter migration / fallout

At the skin

Catheter fall-out rate is significantly reduced by (a) good strapping, and (b) leaving more catheter in the epidural space. I originally used to leave about 4–5 cm inside the epidural space but this was associated with a significant ‘fall-out’ rate during the first few postoperative days. Some years ago I therefore decided to experiment by having the 15 cm mark at the skin, and since then (a) none has ‘fallen-out’ as far as I am aware, and (b) no adverse effects have been noticed.

At the filter

A recent letter by Picton and Das (2004) described taping the catheter to the filter (including a small redundant loop of catheter) as a good method for reducing catheter disconnection at the filter. The problem of how to proceed if the epidural catheter itself becomes disconnected from the filter is addressed in Section 2.4.1.


2.2.5 Radiographic placement

The following two reports describe a paramedian radiographic technique for locating the tip of the Tuohy needle, and for checking loss of resistance using radio-opaque contrast.


2.2.6 Fibreoptic guided placement


2.3 Drugs

A fairly typical intraoperative epidural dose for a thoracotomy in an adult would be approximately 100–200 µg fentanyl plus 15–20 ml 0.25 % bupivacaine during the course of the operation. One approach is to mix 200 µg fentanyl plus 6 ml 0.25% bupivacaine into the first 10 ml syringe, and give this initial dose over about 15–30 mins depending on the blood pressure. In elderly patients consider reducing the dose of fentanyl.
The usual postoperative regimen at the City Hospital is to use a standardised pre-filled bag (500 mls) containing a mixture of fentanyl 2 mg + bupivicaine 0.125%, starting at about 5 mls/hr (range 0–10 mls/hr). These bags are kept in the recovery room in the DDA cupboard. Typically this standard regimen is started at the beginning of the operation and adjusted accordingly.

An alternative system, suitable for either ITU or HDU, is to use a syringe-driver—in this case a typical regimen for an adult would be to mix 200 µg fentanyl plus 56 mls of 0.25% bupivicaine (total 60 mls), starting at about 5 mls/hr (range 0–10 mls/hr).

The ideal mixture of fentanyl and bupivicaine is somewhat controversial, and practice varies widely. The optimum concentration for epidural fentanyl was stated to be 10 µg/ml fentanyl by Welchew EA (1983), who also noted that the addition of 0.125% bupivicaine can improve the analgesia. A greater strength of bupivicaine is said not to significantly improve analgesia, while a lower concentration of bupivicaine appears to have no advantage over using fentanyl alone (Badner et al. 1994).

A recent study by Tan et al. (2004) compared fentanyl concentrations of 2, 5, and 10 µg/ml in bupivicaine 0.1% (plain) for thoracic epidurals, found that a concentration of fentanyl 5 µg/ml gave the optimum balance between excessive pain and excessive sedation.

Clonidine was used in thoracic epidurals for laparotomy by Curatolo et al. (2000); they suggested that an optimum combination was clonidine (5 µg/hr) plus bupivicaine (9 mg/hr) plus fentanyl (21 µg/hr). The addition of adrenaline to bupivicaine and fentanyl reduced plasma fentanyl concentrations.

**Postoperative pain-relief algorithm:** There is a comprehensive algorithm printed on a poster in the thoracic high-dependency ward. Copies can be obtained from the thoracic surgeons.

- Casati A, Alessandrini P, Nuzzi M, Litti E et al. (2006). A prospective, randomised, blinded comparison between continuous thoracic paravertebral and epidural infusion of 0·2% ropivacaine after lung resection surgery. *European Journal of Anaesthesiology;* 23, 999–1004. [paravertebral is just as effective as epidural for analgesia, and has fewer haemodynamic complications]
- Tan CNH, Guha A, Scawn NDA, Pennefather SH and Russell GN (2004). Optimal concentration of epidural fentanyl in bupivicaine 0·1% after thoracotomy. *Br. J. Anaesth.;* 92, 670–674. [fentanyl 5 µg/ml was optimum]

### 2.4 Complications

#### 2.4.1 Epidural catheter disconnection

The problem of how to proceed if the epidural catheter itself becomes disconnected from the filter is addressed by Grewal, Hocking and Wildsmith (2006), as follows.

A common concern is what to do if the epidural infusion system becomes disconnected somewhere between the bacterial filter and the patient. An interesting laboratory study using deliberately contaminated catheters suggested that reconnection is safe within 8 hrs provided that the fluid inside the catheter is static (or the meniscus has moved < 12.5 cm) and does not move when lifted above the level of the patient. The outside must be soaked in 10% povidone iodine solution, or similar, for 3 mins and allowed to dry thoroughly before up to 20 cm is cut from the end with a sterile instrument. If these conditions are not met, the catheter must be removed.

Grewal, Hocking and Wildsmith (2006)


#### 2.4.2 Abscess

Literature reports of epidural abscess suggest that this complication is not uncommon (about 0.1%). Patients should be examined frequently for fever, local tenderness and neurological deficit. The excellent review by Grewal, Hocking and Wildsmith (2006) and the AAGBI (2002) guidelines should be essential reading for all.


2.4.3 Haematoma & DVT prophylaxis

Low molecular weight heparin (LMWH, enoxaparin, tinzaparin) is currently used in thoracic surgery. It is given once daily in the evening in order to facilitate day-time epidurals. Peak plasma concentrations occur at 4 hours and activity persists up to 24 hours. The PT and APTT are not generally affected by therapeutic doses, so monitoring requires measurement of anti-factor Xa levels (Roberts et al. 2004; Hirsh et al. 2001).

Epidural catheters should not be removed earlier than 8 hours following anticoagulation. See McLeod and Cumming (2004) for an overview. European guidelines recommend (a) once daily dosing with LMWH, (b) 12 hr interval between injection and either epidural catheter insertion or removal (Wheatley et al. 2001).

• McLeod GA and Cumming C (2004). Thoracic epidural anaesthesia and analgesia. Continuing Education in Anaesthesia, Critical Care and Pain; 4 (No. 1), 16–19. [BJA]


2.5 Paravertebral block

Naja, Ziade et al. (2004) suggest that the paravertebral space is divided into a potential anterior (extrapleural) and posterior (sub-endothoracic) compartments by a so-called endothoracic fascia, and that such spaces influence drug spread—see reply letters by Fitzgerald and Harmon (2004) and by Naja and Lönqvist (2004) for more references and discussion.
As regards efficacy, it seems that a paravertebral block compares well with an epidural block (see Casati et al. 2006; Mathews and Govenden 1989).

- Casati A, Alessandrini P, Nuzzi M, Litti E et al. (2006). A prospective, randomised, blinded comparison between continuous thoracic paravertebral and epidural infusion of 0.2% ropivacaine after lung resection surgery. *European Journal of Anaesthesiology*; **23**, 999–1004. [paravertebral is just as effective as epidural for analgesia, and has fewer haemodynamic complications]


- Mathews PJ and Govenden V (1989). Comparison of continuous paravertebral and extradural infusions of bupivacaine for pain relief after thoracotomy. *Br. J. Anaesth.*; **62**, 204–205. [paravertebrals were associated with less hypotension and less urine retention; analgesia was the same in both groups]


- Naja MZ et al. (2005). Distance between the skin and the thoracic paravertebral space. *Anaesthesia*; **60**, 680–684. [median depth was 55 mm; the depth was least in the T4–T8 zone]

• Richardson J, Cheema SPS, Hawkins J and Sabanathan S (1996). Thoracic par-
vertebral space location. *Anaesthesia*; 51, 137–139. [used pressure measurement
during needle advancement; sudden pressure fall as needle traverses the superior
costo-transverse ligament]
Tracheostomy & related airway problems

A tracheostomy is like a snake—it can rear up and bite you when you least expect it.

This is a path littered with unforeseen hazard. Some basic guidelines can therefore be useful since most of us anaesthetise relatively few patients with, or for, a tracheostomy. The key skills to learn are (a) changing a tracheostomy (Section 3.6) and (b) bronchoscopy (Chapter 5).

3.1 Introduction

As a general rule, whenever there is a problem with a tracheostomy (in theatre or on a ward) have a very low threshold for inspecting the position of the tracheostomy with a fibrescope—either with the intubating fibrescope or with the usual ‘large’ fibrescope. Many significant complications arising from a ‘blind’ manoeuvre would have been avoided completely if only a fibrescope had been used initially.

Simple visualisation of the trachea in order to check the position of the tracheostomy does not require local anaesthesia, since this generally can be done by positioning the fibrescope just at the tracheal-end of the tracheostomy, i.e., without needing to touch the tracheal mucosa. However, sometimes it is advantageous to give some local anaesthetic to allow more freedom with the fibrescope. The following simple technique generally works very well.

3.1.1 Local anaesthetic for fibreoptic bronchscopy of the trachea

A reasonably effective adult dose is 80 mg plain lignocaine (2 mls of 4% lignocaine) which can either be blown down the bronchoscope, or down the dilator of a Portex ‘Seldinger’ Mini-Trac (since this has a very useful Luer connector) with the tip positioned well down the tracheostomy.

Take a 20 ml syringe (with a straight Luer connector so it can be pushed into the fibrescope inject port) and pull the plunger out to the 20 ml position; now inject 2 mls of 4% lignocaine into the empty syringe via the nozzle and then hold it vertically (nozzle down); now connect the 20 ml syringe vertically into the inject-port of the fibrescope (tip now positioned at the tracheal-end of the tracheostomy) and inject quickly (ideally at the end of expiration) all 20 mls (2 mls lignocaine + 18 mls air). This will deliver the lignocaine as a fine spray throughout the trachea, and usually gives very effective local analgesia above the carina.

3.2 Tracheostomy tubes

There is a huge range of tracheostomy tubes—cuffed and uncuffed, fenestrated & non-fenestrated; standard forms with and without inner-tubes (e.g., Portex, Tracoe); specialised forms (e.g., Montgomery tube (Section 3.9), Mini-Trac, double-lumen), as well as various attachments (speaking valves, humidifiers). Most of these are well described by Russell and Matta (2004).

When purchasing standard tracheostomy tubes it is useful to consider only those for which the number defining the tube ‘size’ is the same as the internal diameter of the

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1In this case use a 20 ml syringe having a Luer-lock connector (available in ITU), as you generally have to push the syringe plunger in with significant force (since the Portex Mini-Trac dilator has a very narrow channel). Unless you lock the syringe nozzle onto the Luer connector of the dilator it will probably disconnect as you inject, owing to the relatively high resistance to flow.
inner-tube, as is standard with endo-tracheal tubes. The tracheostomy tubes currently used by the City Hospital are Portex and Tracoe, both of which follow this rule.

### 3.2.1 Portex² (Smiths Medical)

(Smiths Medical, Hythe, Kent, UK; tel: +44-(0)-1303-260-551)

The Portex brand (Smiths Medical) includes a percutaneous dilation kit (UniPerc), as well as a range of tracheostomy tubes:

1. Non-fenestrated versions which are intended for short-term use only (usually changed weekly). The internal and external diameter specifications for the standard tracheostomy tube are given in Table 3.1.

2. Adjustable flange versions (e.g., the UniPerc) for use in obese patients.

3. A left double-lumen tracheostomy tube.

### 3.2.2 Tracoe


The Tracoe tracheostomy tubes used at the City Hospital (TRACOE twist MODEL 302) are fenestrated (multiple small holes) polyurethane radio-opaque low-pressure cuffed tubes, intended for medium-term use (up to 31 days). Note that neither the duration nor latex status seems to be specified in their documentation. The twist-lock connection is fully ‘locked’ when the two arrow heads are opposite one another. Each Tracoe box includes

- Outer tube with swivelling neck-plate
- A removable non-fenestrated inner-tube (white 15 mm connector; for suctioning and ventilation).
- A removable fenestrated inner-tube (blue 15 mm connector; for spontaneous respiration weaning)
- Obturator (for insertion) and neck strap

While Tracoe do make decannulation plugs and speaking valves, these are not included in the package (i.e., they are ‘extras’). These speaking-valves and other ‘extras’ are stocked by ITU. The Passey-Muir clinical information pack relating to tracheostomy tubes and speaking-valves is available from Kapitex Healthcare.

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²In 1940 Sydney Leader, a dental surgeon at the Dental Hospital, London, set up a company based in his flat in Great Portland Street. His company, which was called Portland Plastics Ltd., was later renamed Portex Ltd. in 1967 (see: Russell CA (1996). Developments in thermoplastic tracheal tubes. In: Essays on the History of Anaesthesia. (Royal Society of Medicine Press Ltd., London). p. 94–97).
3.2.3 Rösch

(Rösch UK Ltd., High Wycombe, Bucks, HP12 3NB, UK; tel: 01494–532761).

Make a left and right 39 Fr double-lumen tracheostomy tube. Three intratracheal lengths are available (75, 85, 95 mm).

3.2.4 Moore tube


The Moore tracheostomy tube (Boston Medical Products, Westborough, MA, USA) is a soft and flexible long (115 mm) silicone non-cuffed tracheostomy (with a similar inner tube), which is typically used to maintain tracheostomy access (in a spontaneously breathing patient) following removal of a long adjustable-flange tracheostomy. It can be easily cut to the appropriate length. Two sizes are available: 6 (ID 6.6 mm; OD 11 mm) and 8 (ID 7.5 mm; OD 12 mm).

3.3 Tracheostomy—when?

Commonly at 7–10 days or so, depending on likely duration of ITU care. A failed trial of extubation is a common indication for tracheostomy. Patients with concomitant respiratory disease tend to have a tracheostomy performed at an early stage. While a tracheostomy greatly facilitates nursing and respiratory care and shortens ITU stay, it does not generally reduce hospital stay as it tends to lengthen HDU stay, since most hospital wards cannot accommodate tracheostomy patients.


3.4 Percutaneous tracheostomy

Percutaneous tracheostomy owes its popularity to the introduction of the ‘Blue-Rhino’ (William Cook Europe) modification of the dilational technique by the American surgeon Pasquale Ciaglia in 1985 (Eggert and Jerwood 2003). Careful patient selection is important, and there is a significant learning curve.

While a percutaneous tracheostomy tends to have lower rate of long-term complications (e.g., tracheal stenosis) than a surgical tracheostomy, the early ‘percutaneous’ complication rate (e.g., bleeding, tracheal damage, pneumothorax) tends to be higher. Percutaneous complications, when they do occur, tend to be serious. The advantages of prior ultrasound scanning for aberrant vessels is suggested by several authors—see Gwilyn and Cooney
Damage to the posterior tracheal wall is an ever present danger—see Madden et al. (2004) on one approach to this problem using a covered stent.

For a good overview see the handbook by Paw and Bodenham (2004); for details of two meta-analysis studies see the article by Eggert and Jerwood (2003). For experience with the Fantoni Trans-Laryngeal Technique see Lesmo and Ripamonti (2010).


• Walz MK *et al.* (1998). Percutaneous dilational tracheostomy—early results and long-term outcome in 326 critically ill patients. *Intensive Care Medicine*; 24, 685–690. [2 deaths in 326 patients. They also review the literature, revealing a further 7 deaths in 2034 patients (total death rate 1/263 ! Causes of death:- bleeding; obstruction & hypoxia]

• William Cook Europe. *Ciaglia Blue Rhino™*: instructions for use. (Denmark), 2000; 91 pp. [from Eggert and Jerwood 2003]

### 3.5 Surgical tracheostomy

Assuming the patient already has a single-lumen endotracheal tube in place, then the main considerations are as follows (Rogers *et al.* 2001).

• Make sure the cuff is not damaged by the surgeon.

To this end it is recommended that the endo-tracheal tube be first pushed down close to the carina, using a fibreoptic bronchoscope to position the tube safely. Consider changing the endo-tracheal tube if it is too short to reach the carina.

• Be alert to the potential fire risk (see recommendations below).

Although flammable anaesthetic agents are no longer used in the UK, fires in the operating theatre continue to be an occasional hazard, particularly with operations on the airway. Thankfully, most such fires are evanescent and cause no harm, but unfortunately deaths do still occur. For example, in two separate fatalities reported by Stouffer (see Rogers *et al.* 2001), the fires spread uncontrollably with alarming speed, and in one case the operating theatre had to be evacuated. Interestingly, tracheostomy fires tend not to be as catastrophic as other airway fires, possibly because the tracheostomy acts as a vent.

#### 3.5.1 Recommendations

The following recommendations for anaesthesia for surgical tracheostomy are from the article by Rogers *et al.* (2001). The emphasis is on (a) avoiding a fire, and (b) avoiding surgical damage to the ETT cuff. Note the interesting idea of instilling carbon dioxide directly into the tracheostomy wound described by Mani *et al.* (2007).

1. All theatre staff should be aware that an airway fire may occur during tracheostomy.
• Have a fire extinguisher immediately available. It should be mounted inside the operating theatre near the entrance. In practice a carbon-dioxide fire-extinguisher will be the usual choice. Halon fire extinguishers are significantly better for operating theatre fires, but their use is declining owing to environmental concerns.

• Have a bowl of saline and wet drapes available on the surgical instrument trolley at all times.

• Have a self-filling ventilation bag (e.g., Ambu bag) available for ventilating the patient with room air.

• Do not use nitrous oxide or any of the other flammable/explosive anaesthetic agents.

2. Use a single-lumen endotracheal tube which is long enough to allow the tip to be advanced to the carina (the carina is approximately 24–25 cms from the teeth in an average male). If a single-lumen endotracheal tube is in situ and is too short to reach the carina, then change it for one with a suitable length. If a double-lumen endotracheal tube (or a nasotracheal tube) is already in situ then change it for a single-lumen tube before tracheostomy.

3. Use saline to inflate the endotracheal cuff. Make sure there is no leak of anaesthetic gases past the endotracheal cuff.

4. Use the lowest safe $F_{O_2}$ in either nitrogen (air/oxygen mixture) or helium.

5. If the tracheostomy wound is significantly deep (e.g., in an obese patient), use a suction device to clear any build up of diathermy products from within the wound.

6. Before the trachea is opened, advance the endotracheal tube down the trachea so the tip is close to the carina, in order to minimise the likelihood of damage to the cuff when the trachea is incised. Use a fibreoptic bronchoscope to position the tip of the endotracheal tube close to the carina, and mark the tube at the teeth when correctly positioned—this will serve as a useful position guide later if the surgeon fails to place the tracheostomy and you need to push the tube down quickly to get the cuff below the tracheal hole.

7. Incise the trachea using either a scalpel, scissors, or a harmonic knife. Do not use diathermy to cut through the trachea.

8. Once the trachea has been opened and the surgeon is ready to insert the tracheostomy tube, stop ventilating, deflate the endotracheal tube cuff and withdraw the endotracheal tube carefully under direct vision until the tip is just above the tracheal hole (do not withdraw the tube any further at this stage). Be prepared to push the endotracheal tube back down the trachea to secure the airway if there are any difficulties, either while inserting the tracheostomy, or during the initial ventilation
through the tracheostomy. Remember to keep the oral ETT in situ so you can put
the bronchoscope down it (after suctioning) to inspect the tracheostomy.

9. Once the tracheostomy tube is secure in the trachea, inflate the tracheostomy cuff and
suck out the tube using a suction catheter, checking that the suction tube passes easily
through the whole length of the tube. If this is satisfactory then commence ventilation
through the tracheostomy. However, always check that the tracheostomy tube is
correctly located/positioned within the trachea using a fibreoptic bronchoscope—
either now (if in doubt), or at the end of the procedure—remember to keep the oral
ETT in situ so you can put the bronchoscope down it. This is primarily to check that
the new tube is not partially obstructed or in a false passage (note that being able to
pass a suction catheter does not exclude these possibilities), and to check that the
end of the tracheostomy tube is not too close to the carina (this can occasionally be
a problem with a long adjustable-flange tracheostomy tube).

10. If any difficulties arise with the tracheostomy tube, remove the tracheostomy tube
and advance the endotracheal tube down the trachea so that the cuff lies below the
tracheal hole. Have a long endotracheal bougie available (e.g., a gum-elastic bougie),
to facilitate advancing the endotracheal tube in case of difficulties. Sometimes, if the
tube is soft, it may bend in the pharynx and fail to go down into the trachea when
you push it, in which case consider placing a bougie down the tube to stiffen it; a
long gum-elastic bougie can also be useful in this situation.

11. If the endotracheal tube cuff has been damaged and the leak is significant, then
adequate ventilation can usually be maintained by (a) using large tidal volumes,
(b) pushing swabs firmly onto the tracheostomy hole to occlude the leak. Consider
using a wide plastic occlusive dressing (e.g., OpSite or Tegaderm) under the swabs to
reduce the leak further. Control the leak intermittantly as necessary between surgical
attempts to insert the tracheostomy tube. Consider re-intubating if necessary.3

Hazard note: If a significant leak occurs during a tracheostomy when using a
pressure-cycled ventilator (e.g., an ITU ventilator in BIPAB mode), the ventilator
may fail to cycle and not ventilate the patient. Have a low threshold for switching to
manual bag-ventilation whenever there is a leak in this particular setting.

12. In the event of fire, immediately disconnect the patient from the anaesthetic machine,
switch off the anaesthetic gas flow, disconnect the gas pipelines, and ventilate with
air using a self-inflating bag. Use an airway filter if there is smoke in the theatre.
Consider flushing saline down the endotracheal tube to extinguish any intraluminal
fire. Consider removing or changing the tube to minimise the inhalation of toxic
products of combustion and spread of fire into the tracheobronchial tree. However,

3The neck will likely be well extended and without a pillow at this stage, and so you may need to insert a
pillow to get good visualisation of the larynx. Also, use an uncut tube in order to make sure you will be able to
get the cuff below the tracheostomy hole, and check the position relative to the carina with a fibrescope.
changing the tube may be more risky than leaving it in if the patient was previously
difficult to intubate, or the airway has become oedematous.

13. **Finally**: It is important to check the position of a fenestrated tracheostomy with
the fibreoptic-bronchoscope by viewing it from above—from the larynx—in order
to check whether the whole of the fenestration, or fenestration holes, of the outer
tube are **within** the trachea. In practice this is most easily done via the original
oral ETT—which should still be in situ for just this purpose. Note that the Tracoe
tracheostomies have a total of nine small fenestrations symmetrically arranged in a
circle on the larynx side of the tube; the three holes forming the vertical diameter
should all be visible through the fibreoptic bronchoscope.

In obese patients the distance between the skin and the anterior tracheal wall is
often too big for the tracheostomy, resulting in some of the fenestration(s) being
outside the trachea. The potential consequence of this, especially for ventilated
patients, is that air may track back up the tracheostomy (between the inner and
outer tubes) and out through the fenestration and into the tissue spaces of the neck,
resulting in surgical emphysema. If the neck is too big, then use an adjustable flange
tracheostomy instead.

**References**

- **ASA Taskforce (2008).** Practice advisory for the prevention and management of
  operating room fires. [a report by the American Society of Anesthesiologists Task

- **ECRI (2003).** A clinician’s guide to surgical fires: how they occur, how to prevent
  them, and how to put them out [guidance article]. *Health Devices*; **32**(1), 5–24. From:
  Sentinel Event Alert, Issue 29: June 24, 2003. (Joint Commission on Accreditation
  of Healthcare Organisations) [http://www.jointcommission.org/sentinel_event_alert_issue_29_preventing_surgical_fires/]

  & Analg.*; **101**, 1563–1564. [letter]

- **Hamza M and Loeb RG (2000).** Fire in the operating room. *Journal of Clinical

- **Mani N, Malik V, Brewis C and Gray R (2007).** Prevention of airway fire during a
  tracheostomy — a further precaution. *Annals of the Royal College of Surgeons of
  England*; **89**, 818. [describe using an NG-tube via the larynx to pass carbon dioxide
  into the trachea close to the tracheostomy site]

- **Molodecka J and Long TMW (1992).** Difficult tracheostomy and the ‘adjustable
  flange tracheostomy tube’. *Today’s Anaesthetist*; **7**, 100.


• Yardley IE and Donaldson LJ (2010). Surgical fires, a clear and present danger. *The Surgeon*; 8, 87–92. [review]

A useful list of references relating to ‘Fire prevention and safety during surgical procedures’ can be found at: [http://www.valleymbeducation.org/fire/pages/fire-read.html](http://www.valleymbeducation.org/fire/pages/fire-read.html)

### 3.6 Changing a tracheostomy tube

See also Section 3.11

Typically a tracheostomy is changed because it is either time-expired, \(^4\) damaged (hole in the cuff), or because a different size or format is now required. Occasionally a tracheostomy has to be changed as an emergency procedure owing to malposition, in which case particular care has to be taken as regards bronchoscopic visualisation and railroading over a suitable guide (bougie, oxygenating catheter, bronchoscope etc.).

The main practical considerations are (a) whether the tracheostomy was percutaneous or surgical, (b) the external diameter of the existing tracheostomy tube, (c) whether the patient is ventilated or breathing spontaneously, (d) whether the patient is awake or anaesthetised, (e) how long since tracheostomy formation or last tube change, (f) whether malposition is suspected.

**Percutaneous:** Since a percutaneous tracheostomy tube is often tightly held by the skin it may be quite difficult to remove and replace. In view of this it is important to use only tracheostomies having an inner-tube, in order to avoid the necessity of having to change it soon after when the patient goes to the ward, or to facilitate weaning.

It is important to be aware of the external diameter of the existing tracheostomy and aim to replace it with one having the same or slightly smaller external diameter if the skin is tight around it.

**Surgical:** Since a surgically placed tracheostomy tube is usually only loosely held by the skin, changing the tracheostomy tube rarely presents a problem with regard to the size of the new tube, and it can usually be replaced (even with a slightly larger tube) without difficulty.

\(^4\)A tracheostomy without an inner-tube (e.g., Portex) should be changed weekly; a tracheostomy with an inner-tube (e.g., Tracoe) should be changed monthly.
Table 3.1: Portex and Tracoe tracheostomy tubes

<table>
<thead>
<tr>
<th>Size</th>
<th>PORTEX ID mm</th>
<th>OD mm</th>
<th>TRACOEt twist (fenestrated) ID mm</th>
<th>OD mm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(inner-tube)</td>
<td>(outer-tube)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>4·0</td>
<td>4·0</td>
<td>7·2</td>
<td></td>
</tr>
<tr>
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<td>5·0</td>
<td>8·6</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>6·0</td>
<td>8·3</td>
<td>6·0</td>
<td>9·2</td>
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<td>7·0</td>
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<td>8·0</td>
<td>11·9</td>
<td>8·0</td>
<td>11·4</td>
</tr>
<tr>
<td>8 Long</td>
<td>8·0</td>
<td>11·0</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>9</td>
<td>9·0</td>
<td>13·3</td>
<td>9·0</td>
<td>12·5</td>
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<td>10</td>
<td>10·0</td>
<td>14·0</td>
<td>10·0</td>
<td>13·8</td>
</tr>
<tr>
<td>10 Long</td>
<td>10·0</td>
<td>13·8</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

3.6.1 Preparation

- Always check the chest x-ray, listen to the chest, check pulse & blood pressure, oxygen saturation, inspired oxygen, and make sure there is venous access (flush an existing cannula to make sure it is working). Check the notes to see (a) if the patient was difficult to intubate through the mouth, (b) if previous tracheostomy changes were problematic, and (c) if there is any drug allergy. Have drugs and intubation equipment available.

- Check whether the tracheostomy was percutaneous or surgical.

- Check the size and make of the existing tracheostomy tube. Check its external diameter—if this is smaller than that of the one you propose to replace it with then you may have difficulties, particularly if the tracheostomy was made percutaneously (usually this is not a problem if the tracheostomy was a ‘surgical’ one). The external diameters of tracheostomy tubes are given in Table 3.1.

- Check if the tracheostomy has a cuff (look for the pilot balloon). If there is no cuff then the patient may be breathing via the tracheostomy and via the nose/mouth (this will have implications for pre-oxygenation—see next Section).

- Check the status of the inner-tube. If a fenestrated inner-tube is in situ (Tracoe blue connector) first change it for the non-fenestrated version (Tracoe white connector),
since this (a) allows you to ventilate the patient if you need to, and (b) allows easy passage for a suction catheter/bougie since there is no hole for it to get stuck against.

3.6.2 Changing the tube

- If the patient is awake, explain exactly what you are going to do.
- Preoxygenate the patient. If there is no cuff (or it is damaged) then remember to preoxygenate via both the mouth (with a mask) and the tube. If there is a cuff then preoxygenate via a catheter mount at the tracheostomy, and check that you can gently ventilate the patient by hand, inflating the cuff as necessary. If there is no cuff, then preoxygenate both via a catheter mount at the tracheostomy and with a face mask, as the patient may be breathing via both routes.
- Suck out the tracheostomy checking that the suction catheter passes easily into the trachea. If there is a lot of secretions, then continue suctioning until it is as dry as possible before changing the tube.
- Have a large (orange tipped) suction catheter available for railroading the new tube.
- Extend the patient’s neck using a supporting pillow under the shoulders for maximum access.
- Prepare the new tracheostomy tube by first checking the cuff is intact, and then deflate the cuff fully and lubricate the cuff with some KY-jelly. Make sure that you have the non-fenestrated inner-tube inside, and that the suction catheter (for railroading) passes easily through the middle (use KY-jelly as necessary). Use the largest diameter suction catheter you can (orange is usually best) which will pass through the non-fenestrated inner-tube, and remember to cut the connector off the suction catheter. Have a smaller size tracheostomy available just in case.
- Remove the neck ties holding the tracheostomy in place.
- Have an assistant hold the patient’s head as a precaution. Have an assistant ready with a suction device for sucking out the stoma if necessary once the tracheostomy is out, since removing the tracheostomy often releases a lot of secretions into the stoma.
- Insert the railroading catheter through the existing tracheostomy and pass it a good way into the trachea.
- Remove the existing tracheostomy (remember to let the cuff down), suction the stoma as necessary, and railroad the new one into position. Be prepared to use a smaller tracheostomy if necessary.
• Once the new tracheostomy is in place inflate the cuff and check (a) you can pass a suction catheter easily into the trachea, (b) that you can ventilate the patient easily and that there is no cuff leak, (c) if the patient is breathing spontaneously check that the bag moves easily, and (d) listen carefully to the chest.

• If there are difficulties consider (a) removing the new tracheostomy and trying again after suctioning, (b) railroading the tube over a bronchoscope, (c) intubating through the mouth if necessary.

3.6.3 Check the position bronchoscopically

• Always check at the end of the procedure that the new tracheostomy tube is correctly located/positioned within the trachea using a bronchoscope—check you can see the carina. This is the only reliable way of confirming that the new tube is not partially obstructed or in a false passage (note that being able to pass a suction catheter does not exclude these possibilities). Note the distance between the end of the tracheostomy and the carina.

• Finally, do a chest X-ray, and make an entry in the medical notes (size, method, problems, and bronchoscopy findings).

3.7 Anaesthetising a patient with a laryngectomy

Patients with a well established laryngectomy will of course be familiar with tracheostomy use and its management. Consequently, I find the most convenient approach is to insert a cuffed tracheostomy on the ward preoperatively on the day of the operation, thus allowing plenty of time for the patient to get used to the tracheostomy before arriving in the anaesthetic room. This greatly facilitates induction and reduces the incidence of coughing.

3.8 Anaesthetising a patient with a tracheostomy in situ

A few basic precautions are worth bearing in mind. Be prepared to change the tracheostomy if necessary (Section 3.6), and always have a bronchoscope handy so you can check position/location if necessary. Before inducing the patient make sure you can actually ventilate the patient through the tracheostomy; i.e., check whether (a) the tracheostomy has a working cuff, and (b) if there is an inner-tube, make sure it is the non-fenestrated one.

• Check the notes to see if the patient was difficult to intubate through the mouth.

• Check the notes to see whether the tracheostomy was surgical (usually easy to replace with the same or larger size), or percutaneous (usually more difficult to change; may need a smaller size available).
• Check the tracheostomy itself carefully to determine (a) the manufacturer, (b) the size, (c) whether it has a cuff (look for the pilot balloon), (d) is the inner-tube fenestrated or not? (remove it and see if there is a posterior hole in it), (e) is the inner-tube ‘locked’ in position? (check that the two small arrows (Tracoe) are aligned; Shiley tracheostomy tubes have two small dots which need to be aligned). Turn the inner tube clockwise to lock it.

• Check you have a new (unopened) same-size same-make tracheostomy tube available. For tracheostomy tubes having an inner-tube this precaution will also guarantee that you have to hand the all-important non-fenestrated inner-tube (white connector for both Shiley and Tracoe tubes). If the tracheostomy is a percutaneous one, then have the next size smaller also available.

• Since a spontaneously-breathing patient may come to theatre with a fenestrated inner-tube in situ (Shiley green connector; Tracoe blue connector) remember to check before induction the status of the inner-tube (remove it and see if it has a posterior hole), and change to the non-fenestrated version (white connector for both Shiley and Tracoe) if necessary—this is why it is important to have a new unopened tracheostomy tube of the same size immediately available in theatre. Note that if you induce a patient with a fenestrated inner-tube in situ you may well not be able to ventilate the patient adequately owing to the huge leak into the pharynx which will become apparent as soon as you try to ventilate the patient.

• If the patient comes to theatre with a fenestrated inner-tube in situ, do any suctioning after first changing to a non-fenestrated inner-tube (white connector), as otherwise the suction catheter may get held up by the fenestration in the inner-tube. In the case of a Shiley tracheostomy tube the catheter may even pass through the large single fenestration in the outer tube and damage the posterior wall of the trachea. Note that Tracoe tracheostomy tubes have multiple small holes in the outer tube which prevent this problem.

• Check that the patient’s tracheostomy is a cuffed one (i.e., look for the pilot balloon) and that the cuff is intact (check with a bag that there is no leak, and that you can (gently) ventilate the patient by hand). If the cuff leaks consider replacing the tracheostomy before induction (but remember, a fenestrated inner-tube will also cause a leak—see above).

• Have some large (orange tipped) suction catheters available (for railroading) in case you need to change the tracheostomy tube.

• **Disconnection hazard:** Since the anaesthesia circuit connects directly on to the inner-tube of the tracheostomy, then if the alignment arrows (or dots) at the connection become misaligned during the operation (e.g., if the patient is turned laterally),

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5Check that the plastic bag inside the box is unopened—it is not uncommon (unfortunately) for someone to have opened it and taken the non-fenestrated inner-tube!
the anaesthesia circuit may rotate (anticlockwise) sufficiently to unlock the inner-tube, and may cause the inner-tube to fall out resulting in a disconnection. It is therefore extremely important to make sure that the tracheostomy and the alignment arrows (or dots) are clearly visible at all times, and that no anticlockwise torque is exerted by the circuit on the inner-tube.

3.8.1 Postoperative management

While patients with a tracheostomy generally wake up very smoothly, they do present an unusual airway risk with regard to aspiration should they vomit. Consequently, the recovery staff need to be familiar with tracheostomy care, and be aware of the aspiration risk. A useful approach is to protect the airway using a catheter mount (on the tracheostomy), and have it directed to one side.

3.9 Montgomery T-tube placement

These T-tubes are used to stent a collapsing trachea. Surgical placement is via the trachea, and is usually extremely difficult (may take up to 1 hour). Anaesthesia ideally requires two anaesthetists—one to manage the airway and jet-ventilation, and one to control TIVA and relaxation (see Section 5.7.2). Note that once the tube is in place it is then not possible to use an endotracheal tube for ventilation, and so the options are either an LMA, bag & mask, or perhaps jet-ventilation via a catheter.


3.10 Difficult airway & trans-tracheal needle ventilation

See also Section 5.7.4 for references regarding Sanders jet ventilation via endotracheal tubes and rigid bronchoscopes.

- Debenham TR (1985). Emergency transtracheal ventilation in anaesthesia or casualty department. Anaesthesia; 40, 599-560. [describes use of a standard IV fluid giving-set—cut off the drip chamber and insert the sharp point (which is usually inserted into the infusion bag) into the trachea, and then connect the oxygen to the drip chamber using some connector]
CHAPTER 3. TRACHEOSTOMY & RELATED AIRWAY PROBLEMS


- Layman P (1996). Difficult intubation. *Today’s Anaesthetist*; April, p. 52 [letter describing his technique of elective trans-tracheal (crico-thyroid) needle placement under local anaesthesia prior to a gas induction or difficult intubation. This letter cites the following three references.]


3.11 Miscellaneous problems

You may sometimes be called to see a patient whose tracheostomy tube has developed some ‘problem’. In general, the problem is either obstruction (partial or total), an air leak, or the tube has come out slightly and cannot be re-sited. If in doubt bronchoscope down the tracheostomy to check alignment and position.

If the patient is paralysed & ventilated and the problem cannot be fixed quickly, then consider reintubating through the mouth using an uncut tube and advance the tube so it is just below the stoma (taking care not to inadvertently intubate a main bronchus),

\[\text{Uncut—since the tube must be long enough to get the cuff below the stoma.}\]

\[\text{This is a very common error, and all too easy to make when using a long (uncut) tube in an emergency situation. Unfortunately, if you are intubating orally in order to overcome an obstructed tracheostomy (and are already wondering whether the tracheostomy tube has made a false passage), and then you discover the new oral tube seems to be obstructed as well, it is easy to wrongly assume that the cause is somehow related to the original tracheostomy problem and fail to appreciate that this new obstruction is simply due to the uncut oral tube being too far down. Checking the tube distance at the teeth is the best clue, since even a quick bronchoscopy at this stage may well be confusing (for example, showing strange and unfamiliar anatomy of small lower-lobe basal bronchi) unless you are already considering the possibility of the tube being too far down.}\]
and then bronchoscope to check position in relation to the carina. If you have to bag the patient on a mask after the tracheostomy has been removed, then cover the hole with a wide plastic occlusive dressing (e.g., OpSite, Tegaderm) and ask someone to press on it to keep it in place and make it air-tight. If oral intubation is difficult, consider intubating through the tracheostomy stoma, railroading over a bougie if necessary—stop as soon as the cuff is in the trachea, since you will be very close to the carina—and bronchoscope to check position.

If the patient is breathing spontaneously and the problem cannot be fixed easily, then consider removing the tracheostomy and railroading a new one. Consider oxygenating using both a face mask and a tracheostomy circuit, since the patient may be breathing through both routes.

3.11.1 Obstruction

The commonest presenting problem is difficulty to pass a suction catheter. Any suggestion from the nursing staff that there are difficulties with suctioning must be taken seriously and investigated urgently. It is essential to use a bronchoscope in such cases in order to exclude partial (or even total) obstruction due either to malposition (e.g., where the tip of the tracheostomy may be only partially within the tracheal lumen), or to dried secretions.

Remove any valve attachment (e.g. speaking valve etc) connected to the tracheostomy. Remove the inner-tube and check the lumen is patent. Check whether a suction catheter can be passed easily; check air movement with a bag (note that one can often pass a suction catheter and see bag movement even in cases of severe partial obstruction). Consider the cuff—this may be overinflated and obstructing the end (unlikely though as the cuff will be made of plastic and not rubber)—and see if letting the cuff down makes any difference. Consider malposition, especially if recent tracheostomy, recent tracheostomy change, or if the tracheostomy flanges do not lie flush with the skin. Sometimes simply releasing the retaining straps completely and observing whether the tracheostomy flanges sit nicely on the skin or not (sometimes this manoeuvre reveals a malpositioned tracheostomy being forced into an apparently normal position by the straps). Finally, consider removing the tracheostomy tube altogether if necessary. Always inspect with a bronchoscope.

- Kazi ST, Ali MA and Donohoe BO (2006). Accidental oro-endtracheostomy intubation. *Anaesthesia, 61*, 918. [death following tracheal obstruction, owing to failure of oral intubation—the oral ETT made an undetected anterior false passage at the tracheostomy stoma: importance of inspecting and then railroading over a fibroscope even with oral intubation if difficulties occur; may have overcome the problem if had fibrescope down ETT for guidance, or had railroaded the tracheostomy over a fibroscope initially]

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8It is quite alarming in ITU how often ETTs are found to be partially obstructed, even after quite short periods of intubation. Even moderate secretions (and especially with thick secretions) should alert you to the possibility of impending obstruction, and to consider pre-emptive check-bronchoscopy and tube change if necessary.

9Note that ability to pass a suction catheter does not exclude significant obstruction due to dried secretions.


**3.11.2 Difficulty inserting the inner tube**

Just occasionally, in a long term tracheostomy patient on a ward, the inner tube becomes difficult to insert. The most likely causes are (a) wrong size inner tube, (b) deformed inner tube etc. Try a new inner tube from a new tracheostomy package. Otherwise, consider inspecting the tube fenestrations with a fibreoptic bronchoscope (from the inside without the inner tube in) and also checking whether the tracheostomy is seated correctly on the skin. If the outer tube is not in sufficiently far, it may be that some of the more superficial holes are outside the trachea, in which case inspecting these from the inside with the bronchoscope reveals that the superficial holes appear pink (outside the trachea), while the deep ones appear black (inside the trachea). Tissue growing in through the holes may be enough to make it difficult to insert the inner tube. The solution is either to push the tracheostomy in fully, or possibly, just change the tracheostomy.

This sort of problem arises because the tracheostomy has come out slightly on the ward, and remained not properly seated on the skin for several days or more. Although the bronchoscope and light-source can be taken to the ward, it is usually more practical to bring the patient to the ITU for investigation.

**3.11.3 Air leak**

Consider a damaged cuff, poor position of the tracheostomy, wrong inner-tube (e.g., trying to ventilate a patient with a *fenestrated* inner-tube), or tracheostomy being too small (cuff leaks even when fully inflated). Finally, consider untied the straps and looking at the natural position of the tube and its flange in relation to the neck. Occasionally this reveals an obviously malpositioned tube, with the flange not sitting on the skin; the tracheostomy being forced into an apparently normal looking position by tight straps.
3.11.4 Tracheostomy recently removed

It is not uncommon for a patient whose tracheostomy has recently been removed (decannulated) to experience renewed secretion or airway problems, and require a new tracheostomy to facilitate suction, bronchoscopy and airway management. If the interval since decannulation is less than about one week then it is generally a simple matter to insert a new tracheostomy since there is usually still a hole, albeit small, at this stage.

Since the aim is typically to facilitate suctioning, then occasionally just a Portex Mini-Trac may suffice, but generally a small tracheostomy (say, size 7) is the minimum requirement,\(^{10}\) at least initially. Note that for ward use, the tracheostomy must have an inner-tube.\(^{11}\) Both the Portex ‘Seldinger’ Mini-Trac dilator,\(^{12}\) and also the blue rhino dilator,\(^{13}\) are extremely useful tools to have handy when dealing with this problem.

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\(^{10}\)Note that while a size 7 will allow tracheal inspection/visualisation with the small ‘intubating’ fibroscope (available from theatres), a size 8 tracheostomy is the smallest which will allow you to use the full-size suctioning fibroscope.

\(^{11}\)A patient with a tracheostomy which does not have an inner-tube must be managed on HDU/ITU since this form of tracheostomy can easily get blocked by dried secretions.

\(^{12}\)This has a very useful Luer connector and hence allows you to inject local anaesthetic down it directly into the trachea via even the smallest tracheal orifice. I have even used it in this way down a malpositioned tracheostomy tube (see Section 3.1.1).

\(^{13}\)This is in the percutaneous tracheostomy pack, and can be used to further dilate the tracheal orifice if necessary.
Lung anatomy

The thoracic anaesthetists’ interest in anatomy relates mainly to thoracic epidurals, bronchoscopy and tube positioning. While epidural anatomy is well catered for (see Section 2.1), copies of the primary texts on the relevant lung anatomy (Brock 1942–1944, Brock 1954, Boyden 1955, Hollinger and Johnston 1957, Kavuru and Mehta 2004) are difficult to find. I am pleased, therefore, to be able to include (with permission) some diagrams and plates from Brock 1 (1942–1944, 1954), which contain quite the best lung anatomy diagrams for thoracic anaesthetists that I have found so far, not withstanding the excellent diagrams in Kavuru and Mehta (2004). See also references to the anatomy of the epidural space (Section 2.1), radial artery (Section 9.5.2), central veins (Section 9.6.4), and bronchoscopic anatomy (Chapter 5). Other useful texts are Burwell and Jones (1996), Ellis et al. (2004), Itoh et al. (2004), Minnich and Mathisen (2007), Deslauriers (2007).

4.1 Anatomical terms

Alveolus (Gk): Diminutive of alveus (cavity) → alveolus (small cavity).

Azygos (Gk): a (without) + zugon (yoke) → azygos (not yoked), i.e., not a paired structure. A non-paired body part, especially a vein.

Azygos vein: A vein of the right superior thorax draining into the superior vena cava.

Azygos lobe: A lung zone separated by an indentation (typically from above down) formed by an azygos vein and its superior mesentery (‘meso-azygos’). The so-called ‘azygos lobe’ is not a true lobe (it does not have a constant bronchus and vessels). First described by HA Wrisberg (1737–1808) in 1778—hence the ‘lobule of Wrisberg’ (Brock 1954, p. 216).

1Copies are available in the British Library, London.
2Lord RC Brock; thoracic surgeon at Guy’s Hospital, London.
Bougie (Fr): bougie (candle).
Bronchus (Gk): brogkhos, bronchos (windpipe).
Bulla (L): bulla (bubble-like).
Carina (L): carina (keel of a boat); carinatus (keel-like). The last ring of the trachea has a keel-like inferior projection carried back in the fork between the major bronchi.
Chyle (Gk): chyl (juice).
Clavicle (Gk): kleis (a key). (L): clavis; dim. clavicular (a bar for closing a window). Some suggest it is named from the Greek owing to its fancied resemblance to a key. However, it is most likely derived from the Latin because it resembles a curved window fastener, and joins or “locks the shoulder girdle to the body.” The Roman clavis was also an S-shaped metal bar used to strike bells.
Costal (L): costa (rib).
Cricoid (Gk): krikos (a ring). The shape of the cricoid cartilage is like a signet-ring.
Diaphragm (Gk): dia (through, across) + phragma (fence).
Effusion (L): effundere (pour out); effusus is past participle of effundere.
Empyema (Gk): empyesis (suppuration), empyematos (abscess).
Fissure (L): fissus (split, cloven).
Hiatus (L): hiatus (a gap).
Hilum (L): hilum (a trifle, a small thing). Point of attachment; point where an organ is attached by vessels & nerves.
Lingula (L): lingere (to lick); lingula (tongue-like). Part of the left upper lobe (equivalent to the right middle lobe), consisting of superior and inferior segments supplied via the lingula bronchus. It is occasionally separated by a partial fissure from the rest of the upper lobe. The following is from Brock (1954, p. 82).

The term “lingula” really refers to the tip or tongue-like projection of the lowest and most anterior part of the left upper lobe, but it is justifiable to make use of the name to describe the whole portion of which the lingula is really but a part. . . . Its chief practical importance lies in the frequency of which it is involved by bronchiectasis in common with the left lower lobe.

Lung (Anglo-Saxon): lungen (light). The lungs were originally known as ‘lights’ because they were so light in weight.
Manubrium (L): manubrium (a handle). The manubrium sterni is shaped like the handle of a sword.
Mediastinum (L): Probably from per medium tensum (that which is tight down the middle); hence between the two lungs.
Oesophagus (L): -phagos (eating) → ysophagus. From the Greek oisophagos (gullet).
Pharynx, pharyngeal (Gk): pharyngos (throat).
Phrenic (Gk): phren (the mind). Taken to mean the seat of emotion around the heart, hence associated with the diaphragm.
Pleura (Gk): pleura (side, rib).
Sternum (Gk): sternon (the male breast).
Stomach (Fr): estomac, stomaque; (L) stomachus; (Gk) stomakhos.
Thorax (Gk): *thorax*, *thorakos* (the chest).

Thymus (Gk): *thymos* (thyme; an aromatic herb). The thymus gland is so named owing to a resemblance to a bunch of thyme.

Throid (Gk): *thyra* (a door) → *thyreos* (shield with a notch for the chin). Apparently the name ‘thyroid’ was introduced in 1646 by Thomas Wharton (1616–1673), English anatomist and physician at St. Thomas’ Hospital, London.

Trachea (L): *trachia* (rough). (Gk): *trachys*. Also: Aspera arteria (air conduit).

Vertebra (L): *vertebra* (a joint). From *vertto* (I turn).

4.2 History of lung anatomy

Lung anatomy was initially investigated using the process of injecting coloured wax into the bronchi and vessels—a technique largely developed by Jan Swammerdam (1637–1680). Two types of wax models were developed: the so-called ‘wax corrosion’ cast, and the later wax-injected dried dissections; details of these techniques were often included in early anatomy books. A fine example of an early wax corrosion cast in a glass bell-jar is shown in the portrait of William Hunter (1718–1783) at the Royal College of Surgeons, London (Tompsett, 1965).

The process of establishing the true basic anatomy of the lung was very slow, the first serious attempt to describe the anatomy in any useful detail being that by the famous Swiss anatomist Christoph Theodor Aeby (1835–1885). However, the models he worked from were poor and he made significant errors—based on comparative anatomy—which were perpetuated through his writings (Aeby 1880). Fortunately, these errors were soon revealed by the pathologist William Ewart, whose careful work led to the modern concept of the bronchopulmonary segment.

4.2.1 Bronchopulmonary segment

William Ewart (1848–1929) was a pathologist at the Brompton Hospital, London, and is generally regarded as the ‘father of segmental anatomy’ (Tompsett, 1965). Ewart was uneasy about the somewhat casual approach to lung anatomy, as he tells us in his treatise (Ewart 1889):

Moreover, a suspicion had arisen in my mind that the present deficiencies in our anatomical knowledge . . . might perhaps be held responsible for the halting . . . in the development of Pulmonary Surgery, contrasting with the steady progress made in the surgery of other organs. Ewart 1889 (from Tompsett 1965)

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4For overviews see Tompsett (1965), Boyden (1955), Sealy et al. (1993), Fell and Pearson (2007).

5Tompsett (1965).

6Pole (1790) was one of the first to include details of the injection process for making anatomical models.

7This is highlighted in the title of Ewart’s book (Ewart, 1889) in which he includes the following: ‘… with a criticism of Professor Aebby’s views on the bronchial tree . . . ’
Ewart’s magnificent achievement was the realisation that the lung consists of a number of functionally and anatomically separate components, which he termed ‘respiratory units’. He summarises his key findings regarding the bronchi as follows:

(3) **The bronchial tubes do not anastomose.** Each lobe therefore receives from the main bronchus a distinct air-supply. The same principle of separate supply extends, within the lobe, as far as the infundibula. This absence of anastomosis is physiologically of great moment. …

**Respiratory districts** — At the root of the lung the conditions are very different, since the primary, secondary, and tertiary branches from the main bronchus radiate towards the periphery for considerable distances, without bearing any lobules. Within each lobe, large groups of lobules being served by separate bronchi are thus kept in practical isolation from each other as regards their air-supply. Each of these sublobar groups may be considered as forming a separate respiratory district, within which the tidal air, or the bronchial contents in general, may, perhaps, be capable of interchange from lobule to lobule. …

A knowledge of the situation, within each lobe of the respiratory districts of which it is composed, is likely to be valuable to the clinical physician. But an attempt to define their anatomical boundaries would with advantage be postponed until a full description of the bronchial tree had supplied a sound basis for the subdivision of each lobe into its lobular groups.


Ewart died in 1929, only a few years before the surgical significance of his ‘respiratory districts’ became widely appreciated. Just three years later Kramer and Glass (1932) extended Ewart’s concept, viewing his respiratory districts as ‘pathological units’. Glass (1933) then coined the term *bronchopulmonary segment*, and soon after Churchill and Belsey (1939) demonstrated the surgical resectability of bronchopulmonary segments—establishing that they were effectively also ‘surgical’ units. A new era of thoracic surgery was being ushered in by developments in anatomy, as Boyden (1955) describes:

The modern period begins with the suggestive studies of Kramer and Glass (1932), the one a bronchoscopist, the other a surgical resident at the Mount Sinai Hospital in New York. Pressed by surgical colleagues for a better localization of lung abscesses, including a knowledge of where to enter the chest for drainage, Glass proposed “to establish a smaller and more accurate unit of localization than the lobe.” Unaware of Ewart’s pioneer work, he named the *bronchopulmonary segment* and stated that it represented “not only an anatomic but a pathologic unit;” this, by virtue of the fact that its orifice occupies a prominent position in the lobar bronchus and therefore is vulnerable to aspiration of infected material. Besides giving us the now generally accepted name for this unit, Glass injected the main divisions of each lobar bronchus with different “colored fluid dyes,” thereby providing the first diagrams of surface distribution. Relationship to the thoracic cage was determined from bronchograms of

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9Dr Rudolph Kramer: see also Kirschner (2003).
the lung. Altogether, eleven segments were recognized. In general these corresponded
to Ewart’s districts, except that the two subsegments of the pectoral district and two
subdivisions of the middle lobe were given the status of segments.

Boyden 1955, p. 14

These were important and exciting times for thoracic surgery. Kirschner (2003) puts these
great achievements in context as follows:

. . . Recognized today as a momentous advance in the surgical anatomy of the lung,
this laid the groundwork for the localization and precise drainage of lung abscess, and
the anatomical basis for precise pulmonary resection.


4.3 Lung development & embryology

The recent advent of molecular biology and gene expression promises a seriously detailed
understanding of the development of the lung, as indicated by a recent paper by Metzger
et al. (2008). Using a mouse model they reveal that—to use a computer programming
analogy—lung development appears to be largely under the control of a molecular master
branching-program coordinating three slaves (branching-subroutines).

The development of lung branching is therefore perceived as proceeding according
to a relatively fixed sequence of repeating functional molecular branching-instructions,
controlled by a relatively small number of genes operating at branch tips. The editorial
relating to this paper summarises the interest in this area, as follows:

Whether a master branch generator controlling a select few slave subroutines represents
a general developmental strategy that has been reused over evolutionary time in
different branched organs, remains an intriguing possibility. Also, solving the specific
problem of gas diffusion as a limit on size, and discovering how simplified, genetically
controlled branching routines interact with physical and biological factors to direct
complex yet reproducible patterns of development, will be matters of great interest.
To quote Charles Darwin as interpreted by biologist Sean Carroll,\(^{10}\) they will aid our
understanding of how “endless forms most beautiful”\(^ {11}\) have evolved from a relatively
simple tool-box of genetic modules.


\(^{10}\)SB Carroll (2007), *Endless forms most beautiful: the new science of Evo Devo and the making of the animal

\(^{11}\)This is a quote from the last paragraph of Darwin’s *The origin of species* (1859): ‘There is grandeur in this
view of life, with its several powers, having been originally breathed into a few forms or into one; and that, whilst
this planet has gone cycling on according to the fixed law of gravity, from so simple a beginning endless forms
most beautiful and most wonderful have been, and are being, evolved’ (Nickalls, 2009).
4.4 Nomenclature

In May 1949 the nomenclature of the bronchi and segments was standardised by the Thoracic Society (BTS, 1950). For discussion see Boyden (1953).

4.4.1 Right lung

The bronchus to the right upper lobe should no longer be called the ‘eparterial’ bronchus—use ‘upper lobe bronchus’ instead. The part of the bronchus between the upper lobe bronchus and the middle lobe bronchus should be called the ‘upper part of the right main bronchus’, and the lower part should be called the ‘lower part of the right main bronchus’.

**Upper lobe**
1. Apical bronchus and segment
2. Posterior bronchus and segment
3. Anterior bronchus and segment

**Middle lobe**
4. Lateral bronchus and segment
5. Medial bronchus and segment

**Lower lobe**
6. Apical bronchus and segment
7. Medial basal (cardiac) bronchus and segment
8. Anterior basal bronchus and segment
9. Lateral basal bronchus and segment
10. Posterior basal bronchus and segment

4.4.2 Left lung

(a) No segment 7, (b) the upper lobe has an upper division and a lower (lingula) division, (c) use the term ‘lingula’ in preference to ‘lower division’

**Upper lobe**

— **upper division bronchus**
1. Apical bronchus and segment
2. Posterior bronchus and segment
3. Anterior bronchus and segment

— **lingula (lower division) bronchus**
4. Superior bronchus and segment
5. Inferior bronchus and segment

**Lower lobe**
6. Apical bronchus and segment
8. Anterior basal bronchus and segment
9. Lateral basal bronchus and segment
10. Posterior basal bronchus and segment
4.5 Carina

The safe positioning of an endotracheal tube relies not only on an understanding of the applied anatomy and an awareness of the likely position of the carina, but also on an appreciation of those factors which can move the carina relative to the end of the tube. In particular, the distance of the tip of a tube from the teeth is a very useful guide for recognising when a tube is likely to be close to the carina, or even beyond it. The surface marking of the carina in a supine patient is the manubrio-sternal angle (Burwell and Jones 1996, Ellis et al. 2004, Minnich and Mathisen 2007).

Table 4.1:
Approximate distances in an average supine adult male.

<table>
<thead>
<tr>
<th>Distance</th>
<th>Distance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teeth → vocal cords</td>
<td>15 cm</td>
</tr>
<tr>
<td>Trachea</td>
<td>10 cm</td>
</tr>
<tr>
<td>Carina → left-subcarina</td>
<td>5 cm</td>
</tr>
<tr>
<td>Carina → right-upper lobe orifice</td>
<td>2.5 cm</td>
</tr>
<tr>
<td>Teeth → carina</td>
<td>25 cm</td>
</tr>
<tr>
<td>Teeth → left-subcarina</td>
<td>30 cm</td>
</tr>
</tbody>
</table>

4.5.1 Factors moving the carina

The distance of the carina from the teeth varies quite markedly with (a) neck position (±2 cm with flexion/extension), (b) body position (supine, lateral, lithotomy, Trendelenberg) particularly in obese patients or those with a large or distended abdomen, and (c) body height/weight. The length of the trachea (and hence position of the carina) is greatly influenced by the position of the diaphragm.12

While the carina is typically about 24 cms from the teeth in a lean supine adult male of normal height, it can be as little as 18 cms in an equivalent obese patient in lithotomy. The recent letter by Greenland (2004) gives some useful references regarding anatomy and how to position an endotracheal tube safely.

With a single-lumen tube it is not uncommon for the tip to be inadvertently positioned at or below the carina in short and/or obese patients; the risk increasing further when such patients are head-down and/or in lithotomy. The risk is even more significant if there is also raised intra-abdominal pressure, as in laparoscopy (Nishikawa et al. 2004). Furthermore, in short patients the larynx-intubation guide-marks on standard endotracheal tubes may locate the tip of the tube too close to the carina (Chong et al. 2006; Cherng et al. 2002).

Appreciating the factors which influence the position of the carina is especially important when positioning double-lumen tubes, since in this setting even small movements of the carina may not only jeopardise the adequacy of lung isolation and ventilation (see

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12Hence: ‘what moves the diaphragm also moves the carina’ (see Section 7.4).
Section 7.4), but can equally well lead to obstruction and lobar collapse. The TEPID database (see Section 6.4.3) is a good predictor of the distance to the carina in supine patients, and has proved to be a useful practical guide in this regard. It is important to have a low threshold for using a fibreoptic bronchoscope to check the tube position following any change in the patient’s position.

Note that tube/carina problems are particularly likely to arise in the ITU, owing to the fact that patients ventilated in the ITU are subject to the combination of (a) PEEP, and (b) being nursed in a 45 deg semi-sitting position. This combination of factors typically results in the carina being significantly further away from its usual supine location. For example, when trainees bronchoscope such patients they often express surprise at how far away the carina is. One must take care to avoid inadvertently positioning the ETT close to the carina if the patient is in a semi-sitting position, since later, when the patient is supine (for nursing or other reasons), the carina may rise too close to the end of the tube. The expected number of cms from the teeth is usually the best initial guide to ETT position in this setting.

4.6 Right-upper lobe orifice

Typically the right-upper lobe (RUL) orifice is 2–2.5 cm below the carina, and is often positioned very slightly antero-lateral (i.e., a right-sided endo-bronchial tube often needs to be rotated slightly anticlockwise in order to align the side hole with the RUL orifice).

The location of the RUL orifice relative to the carina is somewhat variable, and can even originate from the trachea directly (see below).

4.7 Aberrant bronchus

**Supernumerary bronchus**: A bronchus supplying a (supernumerary) segment which is additional to the usual bronchus (supplying its usual lung zone or segments). It is most frequently associated with the right upper lobe, and typically arises laterally from the right side of the trachea, about 2 cm above the normal right upper-lobe bronchus (Brock 1954, p. 201). For clinical implications see Section 7.9.3.

**Displaced bronchus**: A bronchus which is otherwise normal (i.e., supplies its usual lung zone or segment), but is displaced (up or down) from its usual location. Displaced bronchi are much more common than supernumerary bronchi. The commonest displaced bronchus is an upward displacement of the apical bronchus of the right upper lobe. On the left side, a not uncommon displaced bronchus is that of the medial basal (‘cardiac’) bronchus, arising medially from the left lower-lobe bronchus above the origin of the anterior basal bronchus—exactly analogous to the ‘cardiac’ bronchus of the right lower lobe (Brock 1954, p. 207). For clinical implications see Section 7.9.3.
Figure 4.1:
Drawing of a dissection to show the relation of the fissures of the right lung, and to demonstrate the depth from the skin surface of the termination of the fissure (From Brock (1942–1944), with permission)
Figure 4.2:
Similar drawing of the left lung to show the fissure (From Brock (1942–1944), with permission)
Figure 4.3:

**Top**: Chest diagrams to show the level of the lobes and interlobar fissures of the lungs as seen from the front and back.

**Bottom**: Similar diagrams to show the bronchopulmonary segments.

The lateral subsegments of the right upper lobe are indicated by broken lines

*(From Brock (1942–1944), with permission)*
Figure 4.4:

**Top:** Chest diagrams to show the bronchopulmonary segments as seen in the lateral view. The lateral subsegments of the right upper lobe are indicated by broken lines.

**Bottom:** Similar diagrams to show the bronchi supplying the bronchopulmonary segments (*From Brock (1942–1944), with permission*)
Figure 4.5: Right lung. Lateral and medial views in which the individual segments have been injected with coloured gelatin. (From Brock, 1942–1944, with permission)
Figure 4.6: **Left lung**: Lateral and medial views in which the individual segments have been injected with coloured gelatin. *(From* Brock (1942–1944), with permission)*
Figure 4.7:
Drawings of normal bronchi as seen at bronchoscopy in a supine patient. However, in my experience the left upper bronchial orifice in the left lower bronchi drawing is generally seen much closer to the 10–11 o’clock position—compare with Figures 5.1 and 5.2. See Section 4.4 for the more modern nomenclature. (From Brock (1942–1944), with permission).
4.8 References

- Anatomy Atlases: see the link from the Virtual Hospital website: [http://www.uihealthcare.com/vh/](http://www.uihealthcare.com/vh/)


- Brock RC (1942). The level of the interlobar fissures of the lungs. *Guy’s Hospital Reports;* 91, 140–146;


- Brock RC, Hodgkiss F and Jones HO (1942). Bronchial embolism and posture in relation to lung abscess. *Guy’s Hospital Reports;* 91, 131–139;


• Deslauriers J [Ed.] (2007). Thoracic anatomy, Part I —Chest wall, airway, lungs. *Thoracic Surgery Clinics*; 17 (November), 443–666 (Elsevier, Inc) [chapters: Historical perspectives of thoracic anatomy / Surface anatomy and surface landmarks for thoracic surgery / Muscles of the chest wall / The anatomy of the ribs and the sternum and their relationship to chest wall structure and function / The intercostal space / The costovertebral angle / Anatomy of the thoracic outlet / Correlative anatomy for the sternum and ribs, costovertebral angle, chest wall muscles and intercostal spaces, thoracic outlet / Anatomy of the neck and cervicothoracic junction / The glottis and subglottis: an otolaryngologist’s perspective / Glottis and subglottis: a thoracic surgeon’s perspective / Anatomy of the trachea, carina, and bronchi / Lobes, fissures, and bronchopulmonary segments / Pulmonary vascular system and pulmonary hilum / Bronchial arteries and lymphatics of the lung / Correlative anatomy for thoracic inlet; glottis and subglottis; trachea, carina, and main bronchi; lobes, fissures, and segments; hilum and pulmonary vascular system; bronchial arteries and lymphatics]


• Netter Images. This is a commercial site (Elsevier) giving high quality medical illustrations. For their excellent figure of bronchopulmonary segments see http://www.netterimages.com/image/4426.htm

• Nickalls RWD (2009). Evolution of the end of ORIGIN. Science; 326, 801.


• Pole T (1790). The anatomical instructor, (W Darton & Co., London); (2nd ed 1824).


• Wang Ko-Pen, Mehta AC and Turner JF (2004). Flexible bronchoscopy. 2nd ed. (Blackwell Publishing, UK)

Fibreoptic bronchoscopy

Fibreoptic bronchoscopy is an essential tool for viewing bronchial anatomy, and for facilitating correct placement of single and double-lumen endotracheal tubes, bronchial blockers and tracheostomies. In the Intensive Care Unit it is also used for bronchoalveolar lavage (BAL), secretion control and to facilitate lung re-expansion. Facility with a fibreoptic bronchoscope and familiarity with the endobronchial anatomy should be essential for all anaesthetists. Note the recent bronchoscopy issue of Clinics in Chest Medicine edited by Mehta (2010).

Fibreoptic brochoscopy should, in my view, be a much more routine procedure in the general operating room, since in my experience there are many general surgery patients who stand to benefit from a quick peri-operative bronchoscopy while they are intubated for surgery. For example, all those patients with pulmonary secretions, recent chest infection, COPD, smokers etc. Bronchoscoping such patients for secretion control may well improve their oxygenation during anaesthesia, and will greatly reduce the likelihood of their suffering postoperative lobar collapse—a common cause for ITU admission postoperatively. Obese patients will often benefit from having the position of the ETT checked using a fibrescope, especially if in Trendelenberg.


1 Although it is common practice to use a size 8 mm ETT for females in ITU, I find 8.5 mm a better size with regard to fibreoptic bronchoscopy. Once an 8 mm ETT has been in place for more than about 24hrs, even a small amount of tube secretions is often sufficient to make passing the regular (large) fibrescope difficult, sometimes requiring the ETT to be changed to 8.5 mm.

2 If the ETT requires suctioning, then a quick bronchoscopy will probably be of greater perioperative benefit.

3 Remember to send off a sputum or BAL sample for Gram stain, microscopy and culture.


• Wang Ko-Pen, Mehta AC and Turner JF (2004). Flexible bronchoscopy. 2nd ed. (Blackwell Publishing, UK). [see the excellent cardio-thoracic anatomy diagrams in chapter 5, showing how the vessels are related to the bronchial tree (Applied anatomy of the airways) by Kavuru MS and Mehta AC (2004), pp. 36–38]


### 5.1 History

Although the Englishman John Tyndall described the optical properties of flexible glass fibres in 1870, it was not until 1957 that the first ‘gastro-fibrescope’ was developed by B. Hirschowitz in the USA. An improved version was subsequently developed in Japan by the Machida Endoscope Co. Ltd in 1962. In 1964 the Japanese physician Shigeto Ikeda, in collaboration with the Machida Endoscope Co. Ltd, started developing a fibreoptic bronchoscope which was eventually manufactured in 1967 (Ikeda *et al*. 1968; Ikeda 1974). Ikeda’s conference presentation of an early prototype in 1966 is remembered by Dr Ono as follows:

It was at this transitional period of decreasing pulmonary tuberculosis to increasing lung cancer that a flexible bronchofibrescope came to be recognized. The credit for the first to report on the subject must go to Dr Shigeto Ikeda. He demonstrated it with motion pictures . . . in Copenhagen in August 1966. Also, Dr Ikeda was first to publish

Ono J (Foreword; In: Ikeda, 1974).


5.2 Bronchoscopy simulator

There is a useful online simulator for demonstrating endobronchial anatomy on Peter Slinger’s thoracic anaesthesia website ([http://www.thoracic-anesthesia.com/](http://www.thoracic-anesthesia.com/)). You first have to take a brief test on double-lumen tube placement, giving a username and password, after which you can access the simulator. Importantly, you are then free to log-in and use the simulator anytime thereafter. Unfortunately some of the video images in the test are poor and unclear, but the simulator is generally good value and quite realistic.

5.3 Carina

The position of the carina is surprisingly variable (see Section 4.5), and depends on body shape, size, posture, operation (e.g., laparoscopy). Factors which alter the position of the diaphragm generally move the carina in a similar direction. Consequently, one should have a low threshold for using the fibrescope to check the position of the tube—when in doubt—and especially in the various cases described in Section 4.5. The TEPID database predicts the distance to the carina in supine patients reasonably well (see Section 6.4.3).

To measure the distance between the end of the ETT and the carina, first place the tip of the fibrescope on the carina and then grip the fibrescope at the ETT swivel-connector. Now, while maintaining the same grip on the fibrescope, slowly withdraw the fibrescope until the end of the ETT just comes into view. Now the distance between your grip on the fibrescope and the ETT swivel-connector is the same as the distance between the end of the ETT and the carina.
5.4 Left subcarina & beyond

Figures 5.1 and 5.2 show the anatomy as seen down the fibrescope by an anaesthetist positioned at the head end of a supine patient (without the camera attachment).

The key features to note are (a) the orifices of the second-order bronchi either side of the left subcarina lie on a line running from top left to bottom right (see dashed line in Figure 5.1), (b) the first part of the left lower-lobe bronchus is characterised by the orifice of the bronchus to the apical segment of the left lower lobe at the 6–7 o’clock position, and (c) the orifice of the lingula bronchus (lower division of the left upper lobe bronchus) is the first division (on the RHS) of the left upper lobe bronchus (see Section 4.4 for nomenclature).

Figure 5.1:

Left: The left subcarina viewed from the carina, constructed from a CT-scan (so-called ‘virtual bronchoscopy’), showing the typical orientation of the left upper and lower second-order bronchi when viewed from the head end in a supine patient. Copyright © RWD Nickalls & J James 2005

Right: Schematic of the left picture, showing how the left upper-lobe bronchus divides into the lingula bronchus (Li) and the left upper division bronchus (LUL). In addition we see the characteristic position of the orifice of the apical bronchus (A) of the apical segment of the left lower lobe (LLL) just inside the entrance of the left lower-lobe bronchus, typically at the 6–7-o’clock position. Note the typical orientation (straight dashed line) of the second-order bronchi either side of the subcarina. The schematic shows the view associated with the closest safe approach of the end of the double-lumen tube (dashed circle) with respect to the left subcarina and second-order bronchi. Copyright © RWD Nickalls 2005

See Section 5.6.3
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Figure 5.2:

Top left: Left lung medial supine view.  Top right: Supine view of left lower-lobe bronchus.  Bottom: Close up view of the left supine hilum.

Note that the lung is shown in the supine position being viewed medially (cf. Figure 4.6), and hence the orientation of the left subcarina here is consistent with the images in Figure 5.1. Since this view also has the same orientation as that seen down the bronchoscope (viewed from the head end), it makes the anatomy much easier to understand. For example, we can now see clearly how in the supine position the bronchus to the apical segment (yellow) of the left lower-lobe descends almost vertically down from the first part of the lower lobe bronchus (see also Figure 5.1 opposite). (From Brock (1942–1944), with permission).
CHAPTER 5. FIBREOPTIC BRONCHOSCOPY

5.5 Right subcarina & beyond

Figure 5.3 shows the right upper lobe and the typical arrangement of the three bronchopulmonary segments.

Figure 5.3:
Left: The RUL viewed from the right main bronchus in the supine position, showing the typical orientation of the three bronchopulmonary segments; apical (blue), posterior (red), anterior (green).
Right: Lateral prone view—cf. Figures 4.5 and 5.4 (From Brock (1942–1944), with permission).

Figure 5.4 shows the anatomy as seen down the bronchoscope by an anaesthetist positioned at the head end of a supine patient (without the camera attachment.) While the entrance to the right upper lobe is straightforward to recognise, its exact distance from the carina is fairly variable. The part between the right upper lobe and the middle lobe bronchus is known as the lower part of the right main bronchus.

The key bronchoscopic features to note are (a) the entrance to the right upper lobe, and the configuration of its immediate subdivisions, (b) the orifice of the bronchus to the apical segment (yellow) of the lower lobe typically at the 5–6 o’clock position, and (c) the orifice of the middle lobe bronchus typically at the 12–2 o’clock position.

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See Section 5.6.3

Historically known as the bronchus intermedius—see Section 4.4 for correct nomenclature.

Typically three symmetrical sub-bronchi as shown in Figure 4.7—but quite variable.

Sometimes known (incorrectly) as the superior segment—see Section 4.4.
Figure 5.4:
Top left: Right lung medial supine view.  Top right: Supine view of right lower lobe bronchus.
Bottom: Close up view of the right supine hilum.

Note that the lung in these views is shown in the supine position (cf. Figure 4.5). The orifice of the bronchus to the apical segment (yellow) of the lower lobe is typically in the 5–6 o’clock position. The orifice of the middle lobe bronchus is at the same level but in the 12–2 o’clock position. (From Brock (1942–1944), with permission).
5.6 Image orientation

A significant but seemingly neglected aspect of the fibreoptic bronchoscope (fibrescope) is the influence of the 3-D geometry of the combined set of optical fibres on the fidelity of the perceived orientation of the viewed image associated with (a) axial rotation, and (b) bending of the fibrescope. This is an interesting, if somewhat non-intuitive, feature of fibreoptics which has a significant bearing on the interpretation of the viewed images. Surprisingly, I have not as yet found any texts which discuss this.

It is important to be aware of this aspect of fibreoptic geometry, since appreciation of position within the essentially fractal structure of the bronchial tree is largely a matter of orientation and knowledge of asymmetric anatomical features. In my experience axial rotation (Section 5.6.1) can cause gross distortion of image orientation, whereas that associated with bending (Section 5.6.2) is generally minimal in a clinical setting.

To further complicate matters, these effects vary depending on whether the fibrescope is being used normally (monocular-mode) or with the camera attachment (camera-mode). We will address camera-mode at the end, but in the meantime unless specified, we will assume that we are dealing with normal monocular-mode.

5.6.1 Axial rotation

Under normal circumstances (monocular-mode) when a fibrescope is rotated axially (and able to freely rotate throughout its length), the visual image remains fixed (on the retina)—providing the observer’s head is fixed—and hence the image does not rotate. However, if, during manual proximal axial rotation, the fibrescope is gripped distally (i.e., fails to rotate synchronously with the proximal end) then the observer (fixed) will see the image rotate with, and in the same sense as, the proximal end of the fibrescope.

Consequently, I routinely use the following simple manoeuvre to determine whether a given image reflects the true orientation of the object, namely:- manually rotate the fibrescope (axially) back and forth slightly and observe whether the image rotates accordingly or not. If the image fails to rotate (i.e., the fibrescope is not gripped or restricted distally) we can be confident that the image shows the true orientation of the object, in which case the observed orientation can be safely used to guide the observer regarding true location within the bronchial tree.

If the image does rotate with the fibrescope (i.e., the fibrescope is gripped or restricted distally), then a situation of false orientation can be said to exist, and hence the orientation must be assumed to be false unless proven otherwise, in which case the user should not place any reliance on the perceived orientation of the image when determining location. In this case, only those anatomical landmarks having a known asymmetry can be relied upon for determining location within the bronchial tree.

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9Because no rotation implies that the fibrescope is not gripped, and therefore we know the view is ‘true’.

10In the same way that although a ‘stopped’ watch will occasionally be correct (twice a day if it is an analogue watch), in practice the time shown must be assumed to be false until proven otherwise.
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For example, the left upper- and lower-lobe orifices are orientated either side of the left subcarina typically on a line running from top-left to bottom-right when viewed in a supine patient from the head end (see Figure 5.1). If the fibrescope is gripped sufficiently so that ‘false orientation’ exists, then the apparent orientation of the left subcarina will vary with the rotation of the fibrescope. Consequently, the anaesthetist may be misled by the perceived orientation unless the existence of ‘false orientation’ is checked for (see above) and recognised. If ‘false orientation’ is confirmed, then the anaesthetist will need to check for known asymmetries (e.g., the location of the bronchus to the apical segment of the left lower lobe) in order to confirm that the object in question is actually the left subcarina.

Naturally, in the context of thoracic surgery, one must be alert to the possibility that local pathology may alter the expected orientation of structures.

5.6.2 Bending

As the fibrescope passes further into the bronchial tree, it is necessarily bent in various directions. For example, in order to look at the left subcarina the fibrescope must pass down the trachea (inclined approximately 15 degrees below the horizontal) and then down the left main bronchus (deviated about 45 degrees towards the left). Bending the fibrescope successively through these two directions results in the tip of the fibrescope being rotated axially in a clockwise direction (compared with a straight fibrescope held horizontally in the direction of the trachea), resulting in a small ‘false’ anti-clockwise rotation of the image of the left subcarina. The magnitude of the image rotation is the product of the first angle multiplied by the sine of the second angle, and in this particular example would be a barely noticeable 10 degree anticlockwise rotation,\(^{11}\) namely \(15^\circ \times \sin 45^\circ = 10.6^\circ\).

This represents another interesting, if somewhat even more non-intuitive, example of orientation distortion arising from the 3-D geometry of the fibrescope. If you removed the left main bronchus, mediastinum and right lung so as to be able to look directly at the left subcarina in the supine position (see Figure 5.2) you would see the true orientation, as shown by a CAT scan (see Figure 5.1).

5.6.3 Camera-mode

A camera attachment is often used with the fibrescope for teaching purposes, and also to facilitate visualisation during percutaneous dilational tracheostomy.

**Hazard for the unwary:** It is very important to appreciate that camera-mode introduces two significant differences with respect to image orientation compared with monocular-mode, and hence the orientation must be assumed to be false unless proven otherwise. Firstly, the camera introduces a systematic and arbitrary image rotation—since

\(^{11}\)Note that assigning a negative sign to the directions Lower and Left (and conversely)—when viewed monocularly from the head-end of a supine patient—associates anticlockwise with +ve, and clockwise with −ve image rotation. Thus, in the above example of the left subcarina, we have \((-15^\circ) \times (\sin -45^\circ) = +10.6^\circ\), i.e., there is anticlockwise image rotation.
it can be attached to the eye-piece in any position. Second, the camera reverses the axial rotation effect on the screen image compared to monocular-mode.

For orientation to be correct during camera-mode, the camera position relative to the bronchoscope needs to be ‘calibrated’ against reality; i.e., when attaching the camera one must first rotate it relative to the bronchoscope in order to align the screen image appropriately with the patient before locking it in position. For example, when using camera-mode while performing a tracheostomy, we first ‘calibrate’ by rotating the camera relative to the bronchoscope until anterior movement on the trachea corresponds with vertical motion on the monitor/screen. Failure to calibrate accurately can result in a true anterior indentation of the trachea appearing instead to be from one side.

As before, there are two scenarios to consider: (1) fibrescope free to rotate, and (2) fibrescope gripped distally.

1. **Fibrescope free to rotate**: When the fibrescope-camera unit is rotated axially the screen image rotates in the opposite direction, since the image mapping from the camera to the monitor screen is fixed [in monocular-mode, assuming the viewer’s head is fixed, there is no such rotation]

   This is typically the situation when surgeons use the camera attachment on a fibrescope passed down the lumen of a rigid bronchoscope, since in this setting the fibrescope is always free to rotate as there is nothing to grip it.

2. **Fibrescope gripped**: If the fibrescope is gripped distally while the proximal end is manually rotated, then the screen image does not rotate. [in monocular-mode there is rotation in the same direction]

   This situation often arises when the fibrescope is passed down an endotracheal tube, since the fibrescope is usually gripped to some extent by the rubber air-tight seal at the entrance of the endotracheal tube.

### 5.7 Anaesthesia for bronchoscopy

#### 5.7.1 Short duration

Intermittent boluses of propofol, suxamethonium and remifentanil\(^\text{12}\) increments.

#### 5.7.2 Long duration

Propofol TIVA is particularly useful for prolonged bronchoscopy (e.g., for reboring tracheal tumours, multiple biopsies, insertion of stents\(^\text{13}\)) with the Alaris pump use the Schnider

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\(^{12}\)See Section 8.4.

\(^{13}\)e.g., Montgomery tubes (see Section 3.9)
algorithm for propofol TCI, and the Minto algorithm for remifentanil TCI (Absalom and Struys 2007), since both of these use the Lean Body Mass (LBM) calculated from the entered total body weight and height. An arterial line is worth considering, especially with frail patients and difficult cases.

An induction plasma concentration ($C_p$) target for adults of 7 µg/ml followed (once the rigid bronchoscope has been inserted) by a maintenance target of about 5–6 µg/ml plus a narcotic (e.g., remifentanil) generally works well (reduce these somewhat if the patient is elderly and/or frail). Consider an initial remifentanil bolus of about 100 µg (for a 70 kg patient) followed by about 250 µg/hr. Sometimes it is more convenient to give the remifentanil as intermittent boluses rather than run a second pump. A long acting relaxant is generally best, but sometimes it is worth starting with intermittent suxamethonium and converting later if necessary.

Historically, a suxamethonium infusion would often have been used in this setting; it can still be useful on occasions. The technique for adults is to use 500 mg in 500 mls saline—run at about 3 mg/min (a normal blood giving-set has 25 drops ≡ 1 ml, so 3 mg/min is 75 drops/min). Avoid using the infusion for more than about 30 mins (in order to keep the total suxamethonium dose less than about $3 \times kg$), and always use a nerve stimulator to help minimise the total dose and avoid a type-II block (always take care to label the infusion very clearly).


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14Note that there is a potential problem when using TCI with lean body mass (LBM) algorithms in obese patients. For any given height the calculated LBM rises to a maximum and then falls as weight increases, and so in obese patients you need to use that body weight which generates the maximum LBM—see Absalom and Struys (2007), pp. 30–33 for details and useful charts.
5.7.3 Local anaesthesia & sedation


5.7.4 Venturi jet ventilation

The first practical venturi jet system for ventilating down a rigid bronchoscope was developed in 1967 by Richard Douglas Sanders (Buckley 1992; Sanders 1967; Aikens and Bancroft 1977; Maltby 2002). See article by Baraka *et al.* (2001) for details of its use for ventilating down an endotracheal tube (above a tracheal stenosis). Robinson (1997) describes the use of a Hunsaker jet ventilation tube. The potential dangers (e.g., pneumothorax) associated with jet ventilation via exchange catheters is addressed by Benumof (1991). See also Section 3.10 on difficult airways.

5.8 References


• Patel C and Diba A (2004). Measuring tracheal airway pressures during transtracheal jet ventilation: an observational study. *Anaesthesia*; 59, 248–251. [carinal pressure changes were small; approximately 13 mm Hg only]


5.8.1 Complications


5.8.2 Fibreoptic intubation

• Hawkins N (2000). *Fibreoptic intubation*. (Greenwich Medical Media Ltd, Lond.)

• Murphy PA (1967). Fibre-optic endoscope used for nasal intubation. *Anaesthesia*; 22, 489.


Tubes and bronchus blockers

Two recent articles are worth noting.


6.1 The Univent tube, 1984

The Univent tube is a combined blocker and ETT, and made in Japan. The literature suggests that they are not as good as a well positioned double-lumen tube, but may well be useful in unusual situations.

- Campos JH, Reasoner DK and Moyers JR (1996). Comparison of a modified double-lumen endotracheal tube with a single-lumen tube with enclosed bronchial blocker [Univent tube]. *Anesthesia and Analgesia*; 83, 1268–1272. [conclusion:- DLT better than Univent tube (more malpositions with the Univent tube)]


6.2 The Hunsaker jet ventilation tube


6.3 Bronchus blockers

Although Fogarty catheters have been used as bronchial blockers in adults (Ginsberg 1981) and in children (Tan and Tan-Kendrick 2002), special bronchus-blocker kits are now available. The one we generally use at the City Hospital is the *Arndt endobronchial blocker set* manufactured by Cook.\(^1\) This consists of a 9 Fr-gauge bronchus blocker (78 cm) having a blue balloon at the end, together with a special tube-adaptor having three channels one each for anaesthesia gases, the bronchus blocker, and the fibrescope. A new form of blocker has recently been described by Mungroop *et al.* (2010).

The Arndt bronchus blocker has a small loop at its tip, and is guided into position by first sliding it along the fibrescope, and then positioning it under direct vision. The art seems to be to place the tip of the fibrescope just inside the required main bronchus (i.e., not right down to the subcarina)\(^2\) and then push the blocker down. Once the blocker falls off the end of the fibrescope it will be visible and can then be manipulated into position under direct vision. It is important to make sure that both the end loop and balloon remain within the main bronchus (i.e., do not migrate further down into a second-order bronchus where they might stray into surgical territory).

Note that two slightly different shaped balloons are supplied; one is long and thin (for the left main bronchus), and the other is fatter in the middle (for the right main bronchus). The length of each is about 2–2.5 cm (i.e., about half the length of the left main bronchus in an average adult man).


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\(^1\)William Cook Europe A/S, Sandet 6, DK-4632 Bjaeverskov, Denmark; http://www.cookmedical.com/cc/products.do/

\(^2\)Described to me by Dr K Alagesan.


### 6.4 Double-lumen tubes

Several modern well engineered versions are available (e.g., Mallinckrodt, Portex, Rüsch). While none is perfect—each has its own advantages and disadvantages—all are certainly far better than the old red-rubber versions. The Mallinckrodt *BronchoCath* would seem to be the best of those currently available in the UK as regards ease of placement, bronchoscopic access, and having good robust connectors. Both Portex and Rüsch also make double-lumen tracheostomy tubes (see Section 3.2).

#### 6.4.1 History

Initial progress in thoracic anaesthesia seems to have been held up largely by trying to figure out how to overcome the problems associated with pneumothorax in a spontaneously breathing patient. Although physiologists have been ventilating the lungs of animals using bellows via a tube in the trachea for centuries, there seems to have been a mental-block about this in the medical community until relatively recently.

The double-lumen tube was originally developed by the physiologist Henry Head (1889). He was interested in separating gas entering and leaving each of the two lungs in animals. His tube was widely used for determining differential lung function well before it was used in thoracic anaesthesia (Comroe 1977, p. 15).

In 1949 Carlens (1908–1990) designed a double-lumen endobronchial tube for use in broncho-spirometry (Carlens 1949), and subsequently for one-lung anaesthesia (Björk and Carlens 1950). A series of different modifications by Bryce-Smith (1959), Bryce-Smith and Salt (1960), White (1960a) and Robertshaw (1962) followed. Note that the routine use of one-lung ventilation for lung resection was first advocated only in 1957 (see Slinger 1990).

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3The physicist Robert Hooke showed that a dog could be kept alive indefinitely by IPPV via the trachea using a set of bellows (Hooke, 1666). Importantly, he also indicated in this paper that after making numerous holes in the lungs, the dog could be maintained just as easily using a continuous through flow of air. Hence he proved that, contrary to contemporary thinking, the physical movement of the lungs did not of itself benefit an animal other than by simply ensuring the necessary movement of air in and out of the lungs, and that the lungs were simply a device for oxygenating the blood flowing through them. Note that the book *De Motu Cordis* by William Harvey (1578–1657) describing the circulation of blood through the lungs was published in 1632.

See also articles on the general history of endobronchial anaesthesia (White 1960b), and on the history of the various tubes (Pappin 1979) and instruments (Hillard and Thompson 1963). See also the cardiothoracic section in Rushman, Davies and Atkinson (1996) for various historical references.

**The Robertshaw tube (1962)**

A red-rubber tube with D-shaped lumina which has a lower resistance to gas flow than either the Carlens or White double-lumen tubes (Robertshaw 1962). Angular deviation of the endobronchial part from the midline is \(20^\circ\) on the right and \(45^\circ\) on the left. Three sizes only; large, medium, small.

**References**

- Hooke R (1666). An account of an experiment made by Mr. Hook, of preserving animals alive by blowing through their lungs with bellows. *Philosophical Transactions of the Royal Society*; 1665–1678; vol 2, vol 2 1666-1667. [http://www.journals.royalsoc.ac.uk/]
6.4.2 The BronchoCath

[Mallinckrodt Medical (UK) Ltd, 11 North Portway Close, Round Spinney, Northampton, NN3-8RQ, UK.]

The BronchoCath (Mallinckrodt Medical, 1983) is a disposable polyvinylchloride (PVC) low resistance double-lumen endobronchial tube. Angular deviation of the endobronchial part from the midline is 15° on the right and 30° on the left. Five sizes for the left, four sizes for the right (see Table 6.1).

<table>
<thead>
<tr>
<th>BronchoCath</th>
<th>Body of tube OD mm</th>
<th>L endobronchial OD mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>41 Fr</td>
<td>14–15</td>
<td>10.6</td>
</tr>
<tr>
<td>39 Fr</td>
<td>13–14</td>
<td>10.1</td>
</tr>
<tr>
<td>37 Fr</td>
<td>13–14</td>
<td>10.0</td>
</tr>
<tr>
<td>35 Fr</td>
<td>12–13</td>
<td>9.5</td>
</tr>
<tr>
<td>32 Fr</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>28 Fr</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

The Mallinckrodt bronchial cuff was originally clear and transparent (just like the tracheal cuff). It was subsequently changed to the current blue colour following complaints that the cuff was difficult to visualise during fibreoptic bronchoscopy.
### Table 6.2:

<table>
<thead>
<tr>
<th>BronchoCath</th>
<th>Size</th>
<th>Length to teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large</td>
<td>41 Fr (L/R)</td>
<td>30 ± 1 cm</td>
</tr>
<tr>
<td>Medium</td>
<td>39 Fr (L/R)</td>
<td>28 ± 1 cm</td>
</tr>
<tr>
<td>Intermediate</td>
<td>37 Fr (L/R)</td>
<td>27 ± 1 cm</td>
</tr>
<tr>
<td>Small</td>
<td>35 Fr (L/R)</td>
<td>26 ± 1 cm</td>
</tr>
<tr>
<td>V. Small</td>
<td>32 Fr (L only)</td>
<td>—</td>
</tr>
<tr>
<td>Child</td>
<td>28 Fr (L only)</td>
<td>—</td>
</tr>
</tbody>
</table>

### Table 6.3:

<table>
<thead>
<tr>
<th>Measured tracheal width on chest x-ray</th>
<th>Predicted L bronchus width</th>
<th>Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 18 mm</td>
<td>≥ 12.2 mm</td>
<td>41 Fr</td>
</tr>
<tr>
<td>≥ 16 mm</td>
<td>≥ 10.9 mm</td>
<td>39 Fr</td>
</tr>
<tr>
<td>≥ 15 mm</td>
<td>≥ 10.2 mm</td>
<td>37 Fr</td>
</tr>
<tr>
<td>≤ 14 mm</td>
<td>≤ 9.5 mm</td>
<td>35 Fr</td>
</tr>
</tbody>
</table>

Although Mallinckrodt recently made some improvements to their left-sided tube, in my view these tubes still have serious design faults; for example (a) the left endobronchial tip is bevelled medially (should be bevelled slightly laterally to face the subcarina), (b) the right endobronchial tube should have a much larger right upper-lobe orifice.


### 6.4.3 The tube database (TEPID)

Several studies have tried to correlate both tube size and depth of tube insertion with a single body parameter (e.g., height, weight or BMI), but none has proved particularly useful (see references below). This suggests that using only a single parameter is probably not a useful approach—not unexpected since tube distance from the teeth is markedly influenced by both diaphragm position (& hence with the degree of abdominal obesity) and height. Consequently my TEPID database uses three parameters (height, weight and gender), and gives quite accurate predictions (560+ patients in the database).

The TEPID database of double-lumen tubes (DLT) can be useful for predicting both tube size and length for a given patient. For example, the predicted double-lumen tube lengths for an average supine male and female are given in Table 6.4.

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5 Tube and Epidural Database (TEPID). This is a collection of thoracic epidural and tube data accumulated over many years. It is freely available from http://www.nickalls.org/dick/xenon/rwdnXenon.html
The TEPID program also gives the position of the DLT’s tracheal orifice, since this can be used to estimate the likely carina position (by adding approximately 1.5 cm, as the tracheal orifice of the double-lumen tube is typically 1–2 finger-breadths⁶ from the carina).

The TEPID database, together with a Perl program, is freely available. After entering the patient’s height/weight/gender the program displays the relevant tube sizes and lengths (single and double-lumen).

### Table 6.4:

TEPID data for length of double-lumen tube and tracheal orifice position (cm) in an average supine male and female. In the UK the average male and female heights are approximately 5ft 9in (176 cms) and 5ft 4in (164 cms) respectively. See text regarding the value of the tracheal orifice measurement. The results are given as: mean [range] (n)

<table>
<thead>
<tr>
<th></th>
<th>average male</th>
<th>average female</th>
</tr>
</thead>
<tbody>
<tr>
<td>wt</td>
<td>76 ± 7.5 kg</td>
<td>67 ± 7.5 kg</td>
</tr>
<tr>
<td>ht</td>
<td>176 ± 7.5 cm</td>
<td>165 ± 7.5 cm</td>
</tr>
<tr>
<td>41L</td>
<td>30 [27.5–32] (n=56)</td>
<td>—</td>
</tr>
<tr>
<td>39L</td>
<td>29 (n=1)</td>
<td>28.1 [26–30] (n=15)</td>
</tr>
<tr>
<td>37L</td>
<td>—</td>
<td>27.3 [26–29] (n=10)</td>
</tr>
<tr>
<td>35L</td>
<td>—</td>
<td>27.1 [26.5–28] (n=8)</td>
</tr>
<tr>
<td>41R</td>
<td>29.1 [28–30] (n=6)</td>
<td>—</td>
</tr>
<tr>
<td>39R</td>
<td>28 [27.5–29.5] (n=4)</td>
<td>—</td>
</tr>
<tr>
<td>37R</td>
<td>—</td>
<td>26.6 [25–29] (n=4)</td>
</tr>
<tr>
<td>35R</td>
<td>—</td>
<td>25.7 [25–27] (n=4)</td>
</tr>
</tbody>
</table>

The TEPID database, together with a Perl program, is freely available. After entering the patient’s height/weight/gender the program displays the relevant tube sizes and lengths (single and double-lumen).

### References


⁶Measured using a fibrescope.


• Dyer RA, Heijke SAM, Russell WJ, Bloch MB and James MFM (2000). Can insertion length for a double-lumen endobronchial tube be predicted? Anaesthesia and Intensive Care; 28, 666–668. [they compared (separately) height, weight, and the external surface distance (sternal-angle to ear-lobe via mouth) and found no useful correlation]


One-lung anaesthesia

We now focus on the practical details of positioning double-lumen tubes and the subsequent management of one-lung anaesthesia. The core skills are (a) knowledge of the relevant anatomy, (b) some facility with the fibroscope, and (c) a clear idea of what defines the ‘optimum’ position of the tube or blocker. For anaesthetic details regarding the standard range of thoracic operations/procedures see the overviews by Sanders (2006) and by Pearce & Gould (2005). Thoracic anaesthesia was the subject of an excellent recent issue of Anesthesiology Clinics (Slinger 2008).

Preoperatively it is important to look at both the chest X-ray and CT-scan for potential problems not only with the operative side, but also with the dependent side and the trachea—for example, always check for a tracheal bronchus (see Ho et al., 2004). Listen to the chest to identify, among other things, any silent areas; after intubation such areas might otherwise mistakenly suggest tube malposition. In particular, note whether the upper lobes sound clear or not. Always use a CVP line for a thoracotomy.

7.1 Right double-lumen tube

Owing to the hazard of obstructing the right upper-lobe bronchus, relatively few right-sided tubes are used—probably only when a left-sided tube or bronchus blocker cannot be used (e.g., a sleeve resection of the left main bronchus).

7.2 Left double-lumen tube

For most left-lung surgery and all other thoracic surgery it is possible to use a left-sided tube or a bronchus blocker. However, when planning to use a left-sided tube or blocker for left lung surgery, it is a good idea to discuss this with the surgeons, just in case they are
expecting a right-sided tube to be used. For example, they might anticipate having to do a sleeve resection for which you may not be able to use a left-sided tube.

Since a left sided tube is used for both left and right thoracic surgery the strategy while on one-lung varies slightly depending on whether the endobronchial part of the tube is in the top lung (operative side) or the bottom lung (dependent side). The position of the patient (supine or lateral) also influences the strategy.

**Top-side (left endobronchial tube):** In this case the primary danger is that the bronchial cuff may migrate back into the trachea and obstruct ventilation to the bottom lung. Consequently the strategy when on one-lung is to try and keep the bronchial cuff well below the carina, and not to worry unduly if the tip of the tube gets fairly close to the left subcarina.

If when on one-lung, ventilation suddenly becomes obstructed, then this is almost always because the bronchial cuff is obstructing the bottom lung, and so the solution is (a) deflate the bronchial cuff temporarily, (b) push the tube further down the left main bronchus, (c) re-inflate the cuff, and finally (d) check the cuff position with a fibrescope. While you are on one-lung it is of no matter if the tip of the tube now obstructs the left upper lobe—the tube can be pulled back as necessary when returning to two-lungs, or when the surgeon needs to staple a lobar bronchus.

Note that once the patient is in the lateral position, then the bulk of the cardiac output is going to the bottom lung, and so inadvertently obstructing the upper-lobe prior to going on to one-lung is not too problematic—the top lung will need to be collapsed anyway once the surgeons open the chest. However, this is not the case while the patient is supine (e.g., in the anaesthetic room) and significant desaturation may occur if the tube is pushed too far down at this stage.

**Bottom-side (left endobronchial tube):** In this case the primary danger is that the tube may migrate further down and obstruct ventilation to the left upper lobe. Consequently the strategy when on one-lung is to try and keep the bronchial cuff close to the carina. Sometimes the endobronchial part of the tube is slightly too long for the patient, such that the tip is dangerously close to the left subcarina even when the cuff is at the carina, in which case a smaller tube is necessary.

### 7.3 Placing double-lumen tubes

Is there an optimum method for placing a given double-lumen tube? If by ‘optimum’ we mean the most efficient sequence in the sense that information arising from each manoeuvre builds logically on previous manoeuvres, then the answer would seem to be ‘yes’. Prior to intubation it is important to exclude a supernumerary or displaced ‘tracheal’ bronchus (see Section 4.7) during bronchoscopy. Once intubated, it makes sense to start bronchoscopy with the tracheal side (view the carina via the tracheal lumen),

Typically this is performed by the surgeons; it is important to (a) check for a tracheal bronchus, (b) check the position of the right upper lobe bronchus, and (c) suction any secretions, particularly in the ‘bottom’ lung.
and then progress to the endobronchial side, since information gathered by looking at 
the tracheal side influences how one interprets findings on the endobronchial side. An 
optimum sequence for positioning a double-lumen tube would seem to be something along 
the following lines.

- Listen to the chest preoperatively.
- Bronchoscopy (to detect a tracheal bronchus, visualise the position of the RUL 
orifice and configuration of its bronchopulmonary segments, suction secretions).
- Insert double-lumen tube.
- Stethoscope check (to detect upper-lobe obstruction by the tube),
- Bronchoscope check:
  - tracheal side first (to see if the tube is down the correct side, and if there is any 
    bronchial cuff above the carina).
  - bronchial side second (fine-tune position of the end of the tube).
  - tracheal side again (check and fine-tune position of bronchial cuff).

Note that numerous studies have shown that double-lumen tubes are rarely optimumly 
positioned without bronchoscopic fine tuning.

It is useful to consider the process of placing a double-lumen tube in several stages as 
follows: (a) preparation, (b) intubation, (c) stethoscope check, (d) bronchoscopy check, 
(e) final volume and pressure check, (g) turning the patient laterally. We now consider 
these in order.

7.3.1 Preparation

Consider a ‘secretion drying’ premed (e.g., hyoscine)

Hyoscine hydrobromide ² (0.2–0.4 mg IM 1 hour preop).

This greatly helps visualisation of the bronchial anatomy, and facilitates double-lumen 
tube placement. I therefore routinely use a hyoscine premed when I intend to place 
either a right-sided double-lumen tube or a bronchial blocker, as in these cases it helps 
to have particularly good viewing conditions since correct placement is sometimes not 
straight-forward.

Getting the timing/dose of hyoscine right is quite important, since the action of hyoscine 
rarely lasts longer than about 90 mins in my experience.³ The doses I use are: average 
adult female (0.2–0.3 mg IM); average adult male (0.3–0.4 mg IM).

²Hyoscine hydrobromide (ampoules of 0.4 mg in 1 ml) Hyoscine is an anti-muscarinic agent; slightly sedating; 
and sometimes results in a slowish heart rate. Important to use the full name on the prescription form to avoid 
possible confusion with Buscopan (hyoscine butylbromide; 20 mg in 2 mls.)

³It may therefore be better to use a longer acting agent like glycopyrollate instead, but I have not used it in 
this setting.
Check the tubes, cuffs and suction catheters

- Double-lumen tubes.
  Make sure you are familiar with the particular variety of double-lumen tube to be used, and that all the appropriate sizes are available. Make sure the correct connectors are available and that they all fit together. Check that the pilot balloons connect to the correct cuffs (see Nystrom 2003) and that the cuffs are intact; tracheal cuff (approx. 8 mls); bronchial cuff (approx. 3–4 mls).

- Check the tube accommodates the ‘intubating’ fibrescope.
  If you are likely to need the ‘small’ double-lumen tube (35 Fr), then check that the fibrescope will pass through the tube. The Mallinckrodt BronchoCath sizes 41–37 Fr will always accept the 4·5 mm diam intubating fibrescope. However, sometimes the 35 Fr (‘small’) tube will not accept the 4·5 mm fibrescope, so it is as well to check this before (i.e., find a 35 Fr which will accept the fibrescope). As regards lubrication, consider using Xylocaine spray as this seems to be much better than KY-jelly.

- Check the suction catheters are long enough (165 cms)
  The suction catheters should be about 5 cm longer than the double-lumen tube.

Consider starting with a single-lumen tube

In potentially difficult patients, and those likely to be in the anaesthetic room for a long time (e.g., oesophagectomy and other major cases), consider starting with a single-lumen tube, and placing the double-lumen tube as the final anaesthetic-room procedure once all the lines are in. This has the advantage of allowing you to place the CVP, arterial line, NG-tube, bladder catheter etc. without worrying whether the double-lumen tube may have moved in the meantime, possibly resulting in the upper-lobe becoming obstructed (see Section 7.3.3). I therefore routinely adopt this approach in all cases needing a CVP line.

Anticipate the insertion distance of the double-lumen tube

The TEPID database of tubes is useful for predicting both tube size and length for a given patient (see Section 6.4.3). In general the required size for an average male and female is 41 Fr and 39 Fr respectively. The distances are roughly as follows: 41 Fr (30–31 cm), 39 Fr (29 cm), 37 Fr (27–28 cm), 35 Fr (25–26 cm). A rough rule for the number of centimetres is the French gauge minus 10.

7.3.2 Intubation

Apply gel lubrication to the tracheal cuff as it reduces leakage past the cuff (Sanjay et al. 2006). Use the stylette to give a suitable bend on the endobronchial part of the tube, and insert with the curve facing upwards. Once the bronchial cuff is through the cords rotate
the tube back into its anatomical position, and push *gently* into position. Since the trachea has only a thin layer of muscle posteriorly, it is easily perforated. If there is any resistance then consider trying a smaller tube.

Both cuffs are extremely delicate and are easily torn. Consequently take great care not to let the cuffs touch the teeth; consider using a protective gauze/drape if necessary. Always check that the cuffs are still intact following intubation—it will be difficult to change the tube once the patient is on the table.

Once the tube is in place, then inflate the tracheal cuff so you can ventilate both lungs satisfactorily. Then inflate the bronchial cuff with a small amount of air (say, 2–3 mls) just so that you will be able to see the cuff when viewing with the fibroscope. Its not important to have the bronchial cuff fully inflated at this stage.

**Railroading the tube**

Sometimes intubation can be extremely difficult, and the only way is to ‘railroad’ the tube over a bougie. Check that the bougie is long enough—at least 60 cm long (i.e., 35 + 25 cm).

Railroading a double-lumen tube usually requires a fair bit of force, and so some care is necessary in order not to damage the trachea or lung. The aim is to keep the end of the bougie above the carina at all times.

Since the carina is approximately 24–25 cms from the teeth (adult male), then in order to keep the end of the bougie above the carina (to avoid damage from inadvertently pushing it in too far) it is useful to make a clear mark on the bougie at about 24 cms, and maintain this at or above the teeth when railroading the tube. Since all tubes are now transparent, this mark will be easily visible through the tube wall.

**If encountering problems, consider placing a single-lumen tube and checking the anatomy with a fibroscope**

If there are problems positioning the double-lumen tube then it is sometimes a good idea simply to replace it with a single-lumen tube and check the anatomy carefully with the fibroscope, rather than run the risk of making things worse (e.g., obstructing an upper lobe and collapsing it).

Sometimes just checking you can identify the anatomy, or even measuring the distance to the carina or left subcarina, will greatly help during the next attempt at placing the double-lumen tube. Consider railroading the double-lumen tube over the fibroscope. Consider using a single-lumen tube and a bronchus blocker. The TEPID database can be a useful guide for tube size and length, especially in short patients (see Section 6.4.3).

The commonest initial problems are (a) the tube keeps going down the wrong side, (b) the endobronchial part of the tube is too big and fails to go down the main bronchus, (c) the tube is in too far, (d) sometimes the endobronchial part of a left-sided tube is slightly too long for the patient, and a smaller tube is needed.
7.3.3 Stethoscope check

• Once the double-lumen tube is in place first check that there is good air entry (i.e., with no added sounds) to both upper lobes by listening with a stethoscope just inferior to (and touching) the clavicles. Air-entry here should sound exactly the same as for the lower lobes (see Chapter 4 for surface markings). If there are any added sounds in the upper-lobe zone (or if it is quiet) then gently withdraw the tube about 1 cm at a time while listening until the air entry improves.

For example, if after placing a left double-lumen tube we hear added sounds over the right upper lobe, then we can assume (providing it was clear before) that the tube has gone down the wrong side. Conversely, if the left upper lobe is now quiet, then we know the tube is down the correct side, and all we need to do is pull it back until we have good air entry in the left upper lobe.

It is important to auscultate the upper lobes immediately and carefully after intubation because an undetected obstructed upper-lobe will very quickly collapse (within about 5 minutes); the speed being hastened partly by the high inspired oxygen typically used at this stage, and partly by the increased pressures generated by the ventilator. Once the lobe has collapsed, auscultation over the lobe may well seem quite normal, since the remaining lung expands to fill this space.

If the collapsed lung is on the operation side (‘top’ side) then this will be revealed at thoracotomy. However, if this occurs on what will be the dependent side (‘bottom’ side) and goes un-noticed, then this could be disastrous—most probably resulting in severe hypoxia when on one lung.

• Next, check air-entry to the lower lobes by listening on the mid-axillary line below the level of the nipples (see Figures 4.1 and 4.2 for surface markings).

• Now check lung isolation. Clamp each side in turn and check (a) that there is no air-entry on the clamped side (add air to the bronchial cuff as necessary), and (b) that there is good air-entry on the opposite side. It should be easy to squeeze the bag when on one lung—any significant resistance at this stage is usually a sign that the tube is not correctly positioned. If in doubt, let the bronchial cuff down and then check further with the fibrescope—the bronchial cuff can be inflated later after confirming that the tube is correctly positioned.

• Mark the tube at the teeth (with a felt-tip pen) and then tie the tube in place using a rolling-hitch, as described recently by Frank Aldridge (Aldridge 2006), making sure the mark is visible above the knot and from the ‘top’ side (so you can see it once the patient is in the lateral position). This mark will be useful during the operation.

Note that the pressures generated by the ventilator tend to keep a partially obstructed lobe open. Once a lobe is totally obstructed it will experience a net compression from the lung surrounding it. This, plus a high FiO2, results in rapid collapse of the lobe.

This may be associated with some desaturation, especially while supine—see Section 7.2.
(to indicate if the tube has moved), and also at the end of the operation when the
patient is supine again (the tube can be pulled back to its original anaesthetic-room
position—see Section 7.6).

Make sure that the two pilot balloons are inside the tie—if they remain outside there
is the danger that they may be damaged if the tube position has to be adjusted and
the knot moved along the tube.

• Finally, suck out audible secretions at this stage (i.e., before using the fibrescope).

7.3.4 Bronchoscopy — left-sided tube

The strategy underlying placement of a left-sided tube has two aims as follows.

1. To position the bronchial cuff at or below the carina.
2. To position the tip of the tube proximal to the left subcarina.

Bronchoscope check—tracheal side

We proceed by asking three questions.

1. Is the tube down the correct side?
   If not, then reposition the tube. If simple measures to reposition the tube fail
   (withdrawing the tube, turning the head to the right, advancing the tube again), then
   consider railroading the tube into the left main bronchus under direct vision over the
   fibrescope (place the fibrescope down the endobronchial (left) side; withdraw the
   tube so the end is just above the carina—usually at about 23–24 cms at the teeth in
   an average man; advance the fibrescope down the left main bronchus close to the
   left subcarina, and railroad the tube down to a suitable position).

2. Is there any bronchial cuff above the carina?
   If there is cuff above the carina, then push the tube down a bit further so the cuff is
   just below the carina. If you can’t see the cuff at all, then the tube may well be too
   far down (we check this later when looking down the endobronchial side). However,
   in view of the earlier stethoscope-check we should be confident at this stage that the
   tube is not so far down that it is compromising the left upper-lobe.

3. Is the tracheal lumen of the tube very close to the carina?
   If it is too close (less than one finger width) then the tube may well be too far down
   (see Section 5.3 on how to measure this distance).

Armed with all this information, we now bronchoscope the endobronchial side.

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6Improves the alignment of the trachea with the left main bronchus.
Fig. 7.1: Left lung: medial supine view of the hilum—enlarged from Figure 4.6 and rotated into the supine position. Notice how in the supine position the bronchus to the (yellow) apical segment of the lower lobe descends vertically down from the first part of the left lower-lobe bronchus—compare with Figure 7.2 where the orifice is denoted by the letter A (From Brock (1942-1944), with permission).

Bronchoscope check—endobronchial side

There are two stages.

1. Observe the carina and right main bronchus (dark circular shadow) through the plastic wall of the tube (i.e., looking medially). Notice the position of the bronchial cuff (blue) in relation to the carina—i.e., check to see that the top of the bronchial cuff is at or below the carina.

2. Position the end of the double-lumen tube optimumly in relation to the left subcarina, by noting the position of the upper and lower-lobe bronchi in relation to the end of the tube (see Figures 7.1 and 5.1). The closest the tube should be to the subcarina is when the orifices of the two second-order bronchi either side of the left subcarina, together just fit into the diameter of the end of the tube, as shown in the schematic diagram in Figure 7.2. The tube can of course be further out than this, providing that the bronchial cuff remains at or below the carina.
Figure 7.2:

**Left:** This is a schematic showing the position of the orifice of the lingula bronchus (Li) and the origin of the bronchus to the apical segment (A) of the left lower lobe in relation to the second-order bronchi and the end of the double-lumen tube (dashed circle), as viewed down the fibrescope from the head end in a supine patient.

This schematic depicts the closest safe approach of the end of the tube to the left subcarina. We can define this as being when the two diameters of the orifices of the second-order bronchi bordering the left subcarina just fit into the diameter of the end of the tube. Compare this with the bronchoscopic image on the right which shows the view seen when the tube is slightly too far down.

**Right:** View of the entrance of the left lower-lobe bronchus (same orientation as in the schematic on the left). If you see this view bordered by the end of the tube, then the tube is slightly too far down. The shadow at the 11 o’clock position is the orifice of the left upper-lobe bronchus (partially hidden by the end of the tube which is almost touching the left subcarina).

Since the endobronchial end of the left double-lumen tube is unfortunately usually bevelled slightly medially a tube in this position tends to look down into the left lower lobe, as illustrated here. The orifice at the 6 o’clock position is the entrance of the bronchus to the apical segment of the left lower lobe; the remaining orifices are two basal bronchi b of the left lower lobe—compare with Figures 7.1, 4.6 and 5.2 (from the website of P Slinger with permission).

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a Sometimes (wrongly) described as the superior segment.
b The tube should really be bevelled slightly laterally (i.e., to be facing the subcarina).
c Two of the three basal bronchi are visible at the 3 o’clock position.
7.3.5 Bronchoscopy — right-sided tube

The strategy underlying placement of a right-sided tube has two aims as follows.\(^7\)

1. To align the side hole with the right upper-lobe bronchus.

2. To ensure there is an adequate gap between the endobronchial part of tube and the wall of the upper part of the right main bronchus\(^8\) (i.e., distal to the right upper-lobe bronchus). This is a useful precaution, since if the side hole later becomes mis-aligned (often the case once the patient has been turned into the lateral position) then the gases can still get in and out of the right upper-lobe by passing between the tube and the bronchial wall.

The commonest initial problem associated with placing a right double-lumen tube is to inadvertently position it too far down; the top of the medial part of the bronchial cuff needs to be extremely close to the carina—at least initially anyway.

Bronchoscopy—tracheal side

Since it is generally necessary to have the bronchial cuff very close to the carina in order to align the side hole with the right-upper lobe bronchus, start by positioning the tube so that the medial side of the top of the bronchial cuff lies at or just below the carina (i.e., as close to the carina as possible). Note that the Mallinckrodt right-sided tube has a large amount of blue plastic cuff-material stuck to the tube above the bronchial cuff on the medial side, so look carefully to distinguish between the blue ‘real’ cuff and the reflected blue plastic on the tube proximal to the cuff.

Having positioned the cuff close to the carina, then mark the tube on the top side at the teeth (with a felt-tip pen) making sure the mark is just visible above the knot.

Bronchoscopy—endobronchial side

There are two stages.

1. Pass the fibrescope to the end of the tube and withdraw slowly looking towards the right-hand side of the tube until the side hole comes into view. Advance the fibrescope through the side hole and try and locate the right upper-lobe bronchus. If the bronchus or edge of the orifice is visible, then move the tube slightly (rotating it if necessary) in order to get maximum alignment. If the bronchus is not visible, then move the tube in and out slightly, repeating after a small rotation if necessary, until the right upper-lobe bronchus is found.

\(^7\)Note that the algorithm for placing a right-sided double-lumen tube is significantly different from that for placing a left-sided tube.

\(^8\)This used to be called the bronchus intermedius—see Section 4.4 for correct nomenclature.
2. Now proceed to determine whether there is an adequate gap (distal to the side hole) between the endobronchial part of the tube and the bronchial wall. Advance the fibroscope through the side hole and try to look distally at the space between the tube and the bronchial wall. If there is a gap then it is usually fairly easy to see. Even if it seems quite small, this is usually acceptable since the gap will increase slightly when on ‘one-lung’ ventilation as the mean airway pressure will be increased then. If there is no gap at all, then consider using a smaller tube, or perhaps a bronchus blocker, unless you are confident you can maintain good alignment in the lateral position. For a given patient, I therefore use a smaller right-sided tube (one size smaller) than I would for a left-sided tube.

7.3.6 Final tidal volume and pressure check

Just before turning the patient into the lateral position it is useful to check while the patient is still supine that the ventilator pressures and tidal volume generated by the theatre ventilator are appropriate. Strange pressures at this stage may suggest that all is not well with the tube position.

As a general rule, a normal tidal volume (TV) and respiratory rate (RR) will generate a pressure on two lungs of about 20 cm H$_2$O (average male using a 41 Fr double-lumen tube). Clamping the ‘top’ side should then result in the peak pressure rising to no more than 30–35 cm H$_2$O, with a plateau of about 25 cm H$_2$O. If the pressures are much different from these, then now is a good time to adjust the TV, flow and RR accordingly—the fine tuning can be done later. Aim to use the same TV and RR for one-lung as for two-lungs.

7.4 Turning the patient laterally

The initial bronchoscopy after turning the patient into the lateral position commonly shows some degree of tube malposition—sometimes major malposition—the bronchial cuff typically now appearing to be positioned slightly above the carina, even though it had been perfectly positioned when supine. The tube position, however, is generally unchanged relative to the teeth, since the tube is usually well tied in. The cause is therefore, not tube movement, but movement of the carina.

The reality, therefore, is that turning patients from supine to the lateral position is often associated with significant caudal movement of the carina. This is especially the case in obese patients, and those with a pronounced abdominal paunch. Conversely, there is rarely much movement of the carina in thin patients.

This caudal movement of the carina is most likely due the fact that the diaphragm descends further once the patient is in the lateral position since the abdominal contents will move caudally and outwards. Consequently the lungs also descend, resulting in the carina being pulled downwards towards the bronchial cuff, with the effect that the bronchial cuff may then come to lie in the trachea. If turning is also associated with some outward
movement of the tube at the teeth, then catastrophic tube malposition can easily occur, with the end of the tube coming to lie in the trachea, or even in the wrong bronchus.

- Before turning first check that the patient is on two-lung ventilation. With a top-side tube consider temporarily deflating the bronchial cuff (leave the 5-ml syringe attached so you can easily inflate the cuff with the same volume afterwards).

- In patients in whom the tube has been difficult to position, consider checking the ventilation pressures and volumes just prior to turning; these can serve as a useful guide after the patient has been turned (see Section 7.3.6).

- Hold the tube at the mouth during turning, taking care to not to let the tube come out any distance at all. In an obese patient push the tube in slightly during turning to the lateral position (perhaps 1 cm) to compensate for the anticipated caudal movement of the diaphragm and hence of the carina also.

- Once the patient is in the lateral position, check that you have easy access to the two pilot-cuff balloons. Reinflate the bronchial cuff if you deflated it prior to turning (keeping the syringe attached serves as a useful reminder).

- Finally, check the tube position bronchoscopically and adjust accordingly. Try to do this before starting one-lung ventilation.

If the cuff is now in the trachea, then check first whether or not the tip is still in the correct bronchus before pushing it further down. If already on one-lung ventilation, then consider returning to two-lung ventilation before repositioning the tube, since you can then deflate the bronchial cuff and see more clearly. You may be forced to deflate the bronchial cuff anyway if ventilation becomes obstructed while on one-lung. Deflate both cuffs before moving the tube in order to avoid damaging the trachea and bronchus. After repositioning the tube you may well have to tie the tube in again.

7.5 One-lung anaesthesia

It is convenient to consider the following stages: (a) preparation, (b) going on to one-lung, (c) management of one-lung anaesthesia, (d) returning to two-lungs, (e) turning supine, (f) extubation.

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9A useful technique is to first attach an empty 5 and 10 ml syringe to the bronchial and tracheal cuffs respectively. When you are ready to advance the tube under bronchoscopic control, ask someone to deflate the cuffs—but to keep the syringes firmly attached. Once the tube is in the new position, the cuffs can be quickly inflated with the same volume which was withdrawn originally.
CHAPTER 7. ONE-LUNG ANAESTHESIA

7.5.1 Preparation

- Check the position of the tube with the fibrescope.
- Check the tidal volume (TV) and pressures.

An average-sized man with a 41 Fr double-lumen tube and normal tidal volume and respiratory rate (say, 10/min) will have a peak inspiratory pressure with two-lung ventilation of approximately 20 cm H₂O. With one-lung ventilation the peak inspiratory pressure typically increases to about 30 cm H₂O.

**Two lungs:** Check that the TV is reasonable and that the inspiratory pressure is ≤ 20 cm H₂O. Use a relatively slow rate (say, 10/min).

**One-lung:** Clamp the top lung (at the end of expiration) just for a few breaths to see what TV and pressures are generated. Check that the inspiratory pressure rises smoothly and uniformly. Aim for a peak one-lung pressure of ≤ 30 cm H₂O, with a plateau pressure of ≤ 27 cm H₂O (Slinger 2003). If the peak pressure remains high, then exclude causes of increased resistance (e.g., secretions, bronchospasm, tube too far in etc). If the plateau pressure remains high with a reasonable TV, then check the tube position. If the tube position is fine, then consider reducing the TV slightly, and adjusting the respiratory rate as necessary.

- Check the end-tidal P\textsubscript{CO₂}.
  - If this is high, then try to bring this down to normal values before the surgeons need one-lung ventilation.
- Have a Jackson-Rees paediatric T-piece circuit available on a separate oxygen flowmeter for use later with the top lung.

7.5.2 One-lung ventilation

For an excellent overview regarding the possible options for managing hypoxaemia during one-lung ventilation see the recent article by Rozé, Lafargue and Ouattara (2011)

- Before going on to one-lung ventilation (a) note the tidal volume, end-tidal P\textsubscript{CO₂}, saturation and airway pressures (peak and plateau), and (b) increase the F\textsubscript{I\textsubscript{O}_\textsubscript{2}} to at least 50%.
  - If using nitrous oxide and a volatile remember to increase the volatile concentration when reducing the nitrous oxide concentration in order to maintain the same MAC—see Figures 9.1, 9.2, 9.3.
- Go on to one-lung ventilation by clamping the top lung (proximal to the suction orifice) at the end of expiration, and open the suction orifice to air (to allow the top lung to collapse).
• Check that the tidal volume and airway pressures are appropriate on one-lung.

If there is an air-leak check that the bronchial cuff is adequately inflated. If the airway pressures are too high coupled with a small TV consider going back on to two lungs and reviewing the tube position.

• Check to see if the top lung is deflating (ask the surgeons if necessary).

• Check for auto-PEEP.

Listen to the bottom tube with a stethoscope (use the diaphragm) while watching the airway-pressure dial, and check that (a) the inspiratory and expiratory breath sounds are clear and uniform, and (b) no auto-PEEP is being generated (i.e., check there is an end-expiratory pause).

If there is no end-expiratory pause then lengthen the expiratory phase by first reducing the respiratory rate (see the excellent paper by Szegedi et al. 2002), and adjust the TV as necessary to minimise rise in end-tidal PCO₂. Suck out any audible secretions.

• If everything is fine (and the lungs are visible) then connect the Jackson-Rees T-piece (with 100% oxygen at about 1–2 L/min) to the top lung. It seems that even when the lung has collapsed down there is significant entrainment of gas by the lung (see articles by Pfitzner et al. 1999; 2001), and so the idea of the T-piece is to allow oxygen to be entrained by the lung instead of air. Have the oxygen enter the T-piece as close to the double-lumen tube as possible.

Check that the T-piece bag has a hole in it (note that some disposable T-piece circuits use a valve and a closed reservoir bag).

• Check that the pulse, blood pressure and end-tidal PCO₂ are satisfactory.

• Start monitoring the saturation more closely as this will usually begin to fall over the next few minutes.

7.5.3 Management of one-lung anaesthesia

There are some general concepts worth bearing in mind.

• Keep the saturation ≥ 90% by increasing the FIO₂ as necessary.

In particular, avoid the combination of low saturation plus low cardiac output (i.e., low oxygen delivery to the coronaries). To this end, avoid giving very much down the epidural until the saturation and BP have stabilised at a reasonable level.

If necessary periodically squeeze oxygen into the top lung manually in order to

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10 This precaution lessens the risk of barotrauma should the hole in the T-piece bag become obstructed—providing the surgeons can see the lung they will alert you if the lung starts expanding.

11 E.g., using a paediatric T-piece circuit.
maintain an adequate oxygenation (avoid applying PEEP to the bottom lung—unless the patient is obese—as this generally makes matters worse by shunting more blood to the top lung). Once you know you can control the saturation at a reasonable level, then gradually ‘top-up’ the epidural as necessary.

• Avoid excessive ventilator pressures.
  Slinger (2003) highlights the association of postoperative acute lung injury with plateau ventilatory pressures on one lung $> 27$ cm H$_2$O.

• Try to keep the end-tidal PCO$_2$ below 5.5 kPa.
  The end-tidal to arterial PCO$_2$ difference can be quite large on one-lung (Ip-Yam PC et al. 1994), so it is best to try and keep the end-tidal level reasonably low to avoid a respiratory acidosis. Do a blood-gas to check the PaCO$_2$ if necessary. As a general rule the end-tidal PCO$_2$ is a poor guide to the arterial PCO$_2$ if it does not plateau out, in which case a blood gas is more useful.

• Suction out the top lung periodically.

• Periodically check the tube position with the fibroscope.
  Avoid looking down the ‘bottom’ lung unnecessarily at this stage in order to keep the bottom lung well inflated, since there is always a leak when using the fibroscope. Once you are happy the tube is correctly positioned, then top lung bronchoscopy will serve as a guide to tube movement.

With a right-sided double-lumen tube check (a) the alignment of the side hole with the right-upper lobe orifice, and (b) the position of the bronchial cuff in relation to the carina.

When a left-sided tube is used in the bottom lung avoid having the end of the tube too close to the subcarina (danger of the tube obstructing the upper-lobe of the bottom lung). Conversely, when a left-sided tube is used in the top lung avoid having the bronchial cuff too close to the carina (danger of the bronchial cuff herniating above the carina and obstructing gas flow to the bottom lung).

• Control hypotension with fluids and/or vasoconstrictors as necessary.
  A thoracic epidural will usually make the hands vasodilate significantly, and this, when it occurs, is a helpful sign. Metaraminol (10 mg in 20 mls saline; dose 1–2 mls) seems to be better and longer lasting than phenylephrine (1 mg in 20 mls saline; dose 1–2 mls). If having to give frequent boluses, then consider a noradrenaline infusion (2 mg in 50 mls saline; rate 2–8 mls/hr)—see also Section 8.1. Consider dopamine (200 mg in 50 mls; rate 2–8 mls/hr) for bowel surgery (e.g., oesophagectomy).

12Make a note of the arterial–end-tidal difference for use as a guide later.
• Avoid excessive IV fluids, especially crystalloids.

Slinger (2003) highlights the association of postoperative acute lung injury with excessive IV fluids in the first 24 hours. Limit crystalloids to about 1 L, and then use only Gelofusin or blood depending on the haematocrit. If the patient is clearly vasodilated (perhaps from the epidural) then consider using a noradrenaline infusion (Section 8.1). Aim to be more conservative with fluids for a pneumonectomy.

7.5.4 Returning to two-lungs

• Always suction out the top lung before inflating it.

• Following a lobectomy or pneumonectomy the surgeons will usually wish to test the stump under water using inflation pressures of approximately 30–40 cms water, so remember to remove the tube clamp and close the suction orifice. If there is a lung leak then you may need to return to one-lung for a while before testing the stump again. Once the surgeons are happy with the stump then return to two-lung ventilation (with smaller tidal volumes perhaps while the chest drains are inserted). Sometimes going back on to two-lungs is associated with a fall in blood pressure, so be prepared to infuse fluids or give vasoconstrictors at this stage.

• Once the saturation is adequate, then the \( \text{F}_{\text{I}_2} \) can be reduced as necessary. Remember to reduce the volatile concentration (if adding nitrous oxide) in order to avoid an unduly high total-MAC.

• Check all parts of the lung are adequately inflated before the surgeons close the chest completely.

• Adjust the epidural rate/dose as necessary so as to have given an adequate amount before the end of the operation (i.e., at least 15–20 mls 0.25% bupivicaine before the end).

• Make sure the chest drains are attached to the under-water seal as soon as the chest is air-tight.

  Check that the chest-drain bottles are attached correctly (i.e., chest drains are attached directly to the under-water seal tube) and that the under-water seal is working. Check also that the air-exit hole is not obstructed—remove the bung completely if in doubt. Inflate the lungs manually a few times to remove as much of the residual air as possible.

7.6 Turning the patient supine

Turning the patient from the lateral position (on the table) to supine onto the HDU bed is a particularly hazardous moment (see Pearce and Gould 2005) with respect to cardiac arrest
(the heart can get trapped in a pericardial window—cardiac herniation), exsanguination (a vascular tie can slip or work loose) and tension pneumothorax (a chest drain can become occluded).

- Keep the saturation probe and the arterial line attached and running (monitoring) until after extubation to safeguard against overlooking significant haemodynamic or respiratory changes.
- Increase the F\textsubscript{I\textsubscript{O\textsubscript{2}}} to 100%.
- The effect of changing posture back to the supine position will cause the carina to move cranially (see Section 7.4), with the effect that the endobronchial part of the tube may inadvertently occlude the upper lobe bronchus. Following a pneumonectomy, hypoxia at this stage may indicate an obstructed upper lobe. Such problems can be avoided by (a) pulling the tube back out to the position it had in the anaesthetic room (i.e. look for the mark on the tube—see Section 7.3.3), and (b) sitting the patient up somewhat.
- Check the chest drains for any significant blood loss or air leak.
- If the patient is to be electively ventilated in ITU then change the tube to a single lumen tube before going to ITU. Suction out both lungs before changing the tube.

### 7.7 Extubation

- Position the patient sitting up at about 45 degrees.
- Suction out both lungs before extubation.
- If the operation is a pneumonectomy, then just prior to extubation remember to inflate the lung well and clamp the chest drain (two clamps) while the pressure is maintained (usually only one drain following a pneumonectomy).

### 7.8 Complications

#### 7.8.1 Intraoperative

Most intraoperative complications are tube & airway related (e.g., damage to the trachea or bronchus), obstruction from tracheal compression (thyroid surgery, big mediastinal tumours), lung collapse secondary to failure to position the tube correctly. Other significant problems are pneumothorax of the dependent lung (often difficult to diagnose for certain), hypotension, major blood loss, bradycardia/cardiac arrest (carcinoid tumours—see Figure 8.1).
An unusual but dramatic complication which is easily treated, is an acute unilateral pulmonary oedema which arises from the operative side as a result of an inadvertent acute incarceration or twisting of the exposed lung causing an obstruction of venous outflow of a section of lung. Typically occurring after a period of one-lung anaesthesia, the oedema arises from a piece of lung which the surgeon has pushed to one side away from the operative field. Bronchoscopy demonstrates the oedema is associated with the operative side. The surgeon should immediately and carefully check all the collapsed lung for any twisting, which once straightened, will resolve the problem quite quickly.


### 7.8.2 Postoperative

Pulmonary complications are the leading cause of morbidity in the postoperative thoracic patient; for example atelectasis, secretions, lower respiratory infection and ventilator insufficiency. Postoperative bronchoscopy therefore has a major role in managing these common and potentially life-threatening complications. All too often bronchoscopy is unduly delayed; it should be seen as an adjunct to physiotherapy and other more routine elements of postoperative care, and not viewed as a treatment of last resort. See the excellent review and pointer to recent literature by Keith and Kernstein (2005). This is on the CD.
Sometimes a small/moderate bronchial tear is found at routine postoperative bronchoscopy; this will usually heal well if left alone (see Skobel et al. 2004 above).


### Acute lung injury/pulmonary oedema

An excellent editorial by Slinger (2003) comments on the findings of Licker et al. (2003), and highlights four factors thought to be significant independent risk factors for ‘acute lung injury’ following one-lung anaesthesia, namely (a) excessive intravascular volume, (b) high intraoperative ventilatory pressures (> 27 cm H$_2$O), (c) pneumonectomy, (d) preoperative alcohol abuse. Suggested guidelines for the management of one-lung anaesthesia are as follows.

1. Avoid over inflation of the dependent lung. Consider using smaller (more physiological) tidal volumes if possible (say, 5 mls/kg) plus oxygen insufflation, and with PEEP if necessary. Limit plateau inspiratory pressures to < 25 cm H$_2$O.

2. Minimise pulmonary intravascular pressures. This is primarily by fluid restriction, but also by minimising hypercarbia and hypoxia.

To this end a plot of the Datex AS/3 monitor’s plateau ventilatory pressure ($P_{\text{plateau}}$) was added to our computer-generated anaesthetic record sheet, in order to have a record of this parameter in the notes, as shown in Figure 8.1 (page 135).


Pneumonectomy
The recent UK pneumonectomy outcome study (Powell et al. 2009) showed that

The major complication incidence was: 30-day mortality 5.4%; treated cardiac arrhythmia 19.9%; unplanned intensive care unit admission 9.3%; further surgery 4.8%; inotrope usage 3.5%. Age, ASA physical status ≥ P3, pre-operative diffusing capacity for carbon monoxide (DLCO) and epidural analgesia were collectively the strongest risk factors for major complications.

7.9 General references


• Cheong KF and Koh KF (1999). Placement of left-sided double-lumen endobronchial tubes: comparison of clinical and fibreoptic-guided placement. *Br. J. Anaesth.*, 82, 920–921. [compared blind intubation with intubation with the the fibroscope already in the tracheal lumen watching the tip and bronchial cuff as you approach the carina and left main bronchus. They preferred the second method as easy and more reliable in awkward cases.]


and Intensive Care; 28, 666–668. [they compared (separately) height, weight, and the external surface distance (sternal-angle to ear-lobe via mouth) and found no useful correlation]


• Sanjay PS, Miller SA, Corry PR, Russell GN and Pennefather SH (2006). The effect of gel lubrication on cuff leakage of double lumen tubes during thoracic surgery. Anaesthesia, 61, 133-137. [gel lubrication of the tracheal cuff significantly reduced leakage past the cuff]


7.9.1 Tracheostomy and one-lung ventilation


7.9.2 One-lung anaesthesia


7.9.3 Tracheal bronchus

See article by Ho et al. (2004) for a brief review of the recent literature


• Ho AM-H, Karmakar MK, Lam WW, Lam FO, Lee TW, Ng SK and Chung DC (2004). Does the presence of a tracheal bronchus affect the margin of safety of double-lumen tube placement? *Anesthesia and Analgesia*; 99, 293–295. [reviews the recent literature on tracheal bronchus — nearly all are on the right-hand side and within 2 cm of the carina; 10 refs]


7.9.4 Physiology & pathology

• Abe K et al. (1998). The effects of propofol, isoflurane and sevoflurane on oxygenation and shunt fraction during one-lung anaesthesia. *Anaesthesia & Analgesia*, 87, 1164–1169. [propofol was best]


• Eisenkraft JB (1994). Anaesthesia and hypoxic pulmonary vasoconstriction. In: *Recent advances in anaesthesia and analgesia* 18; Chapter 7, pp. 103–122. [86 refs]


### 7.9.5 Flow/pressure/volume loops


Drugs

8.1 Cardiovascular drugs

The key to using these drugs effectively is knowing the likely side effects, appropriate dilutions and safe bolus doses. Learn how to bolus the drugs listed in Table 8.2 (see Section 8.1.2) using a 1 ml syringe—very useful when the pumps/power fail.

Use dedicated CVP ports—one drug per port. Avoid piggy-backing several drugs together, especially when titrating a new drug infusion. Always reduce infusions slowly and review the effect—avoid stopping these infusions suddenly as blood pressure may fall rapidly. Check infusions will not run out during transfer to and from ITU.

Table 8.1:
Commonly used drugs for rapid bolus control of hypotension in adults (70 kg).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dilution (in saline)</th>
<th>Bolus dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ephedrine</td>
<td>30 mg/5 ml</td>
<td>0.5–1 ml</td>
</tr>
<tr>
<td>Metaraminol (\text{(Aramine)})</td>
<td>10 mg/20 mls</td>
<td>0.5–1 ml</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>1 mg/20 mls</td>
<td>0.5–1 ml</td>
</tr>
<tr>
<td>Methoxamine (\text{(Vasoxine)})</td>
<td>20 mg/20 mls</td>
<td>0.5–1 ml</td>
</tr>
</tbody>
</table>
• **Adrenaline** has both α and β effects.

• **Dobutamine** has a predominant β₁ effect (inotropic & chronotropic), with minimal effect on peripheral vascular resistance (due to its modest β₂ and α effects). Dobutamine is the only catecholamine administered as a racemic mixture. It is this mixture which results in its characteristic β₁ selectivity, since the −ve isomer is an α agonist, while the +ve isomer is an α antagonist (Zaritsky and Chernow 1988). Dobutamine may cause hypokalaemia.

• **Ephedrine** is a so-called ‘indirect’ agonist, as it causes release of catecholamines (α + β) at the terminal, and hence tachyphylaxis occurs with frequent use. It causes a moderate increase in BP and HR. If ephedrine fails to have much effect,¹ then consider using a pure α-agonist (e.g., metaraminol, phenylephrine, noradrenaline). Avoid using ephedrine if HR > 100. For useful references regarding ephedrine and phenylephrine see Cooper (2005), Gambling DR and McLaughin KR (2010). See editorial by Smiley (2009) regarding use in Cesarean section.

• **Isoprenaline** has almost pure β effects. Used almost exclusively to increase the heart rate in patients with acute complete heart block.

• **Metaraminol** is a pure α-agonist. An IV bolus lasts about 5–10 minutes and commonly elicits a small reflex bradycardia (therefore avoid using if bradycardia exists). Metaraminol is slightly longer acting than phenylephrine, and is therefore better for bolus use. If you need to give multiple metaraminol boluses then consider using a noradrenaline infusion via a CVP line (see below). If no CVP line then metaraminol can be used as a 5 mg IM dose, or as a temporary slow infusion (5–10 mg in 1 litre).

• **Noradrenaline** is an almost pure α-agonist. It does actually have a very small β effect on heart rate, and, unlike metaraminol, does not cause a reflex bradycardia with bolus doses. Contrary to popular opinion, a noradrenaline infusion for supporting a normal blood pressure is generally beneficial to the kidney and renal function in sepsis (Lee et al. 2004; Bellomo and Giantomasso 2001). Noradrenaline is often of value both operatively and postoperatively in patients with a history of hypertension, to counteract the vasodilatory effects of anaesthesia (general and epidural) and maintain their normal preoperative blood pressure and renal function.

• **Phenylephrine** is a pure α-agonist. It seems to have a slightly shorter duration of action compared to metaraminol, but otherwise its action is essentially similar to metaraminol. See related references cited for Ephedrine above.

• **Terlipressin** (triglycyl-lysine-vasopressin) is a pro-drug which breaks down to the active lysine-vasopressin (Kam, Williams and Yoong 2004). Terlipressin is often

¹If initial doses of ephedrine fail to have much effect, then this is usually a sign of sepsis and that noradrenaline should be used instead.
used in ITU to supplement a noradrenaline infusion in septic shock (better blood pressure control, less tachycardia, better renal and gut perfusion). The adult dose is 1–2 mg IV (repeat 4-hourly initially); effect half-life is 6 hrs.

- **Vasopressin (ADH)** is increasingly being used in septic shock to supplement a noradrenaline infusion (see Roth 2006; Kam, Williams and Yoong 2004, Sun *et al.* 2003). Vasopressin (American Pharmaceuticals) is available in ampoules of 20 U/ml.

### 8.1.1 Infusions: dilutions and use

An important but commonly overlooked aspect of practical drug delivery is the dilution. In my view the hallmark of a well chosen dilution is that, in addition to satisfying the necessary pharmaceutical requirements, it facilitates both the mental calculation and the means of infusion and safe effective bolus delivery, particularly in urgent/emergency situations.

The dilutions listed in Table 8.2 approach this ideal; they are practical and easy to remember, dosage is easy to determine without reliance on calculators or charts. Indeed, an awareness of Table 8.2 would easily have avoided the calculation error described recently by de Wildt *et al.* (2007), associated with setting up a noradrenaline infusion.

**Table 8.2:**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dilution (in saline)</th>
<th>Bolus</th>
<th>Infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>3 × kg 1 mg in 50 mls</td>
<td>0.1–0.2 ml</td>
<td>1–10 mls/hr</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>3 × kg 10 mg in 50 mls</td>
<td>0.1–0.2 ml</td>
<td>(1–10 µg/kg/min)</td>
</tr>
<tr>
<td>GTN</td>
<td>3 × kg 10 mg in 50 mls</td>
<td>0.1–0.2 ml</td>
<td>1–10 mls/hr</td>
</tr>
<tr>
<td>SNP</td>
<td>3 × kg 100 mg in 50 mls</td>
<td>0.1–1 ml</td>
<td>(0.1–1 µg/kg/min)</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>3 × kg 100 mg in 50 mls</td>
<td>0.1–1 ml</td>
<td>1–40 mls/hr</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td></td>
<td></td>
<td>(0.01–0.4 µg/kg/min)</td>
</tr>
<tr>
<td>Isoprenaline</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The entries in Table 8.2 are based on the following rule, which is a simplified and more practical form of that described by Sellick (1985), namely: for a patient weighing $W$ kg, then diluting $3 \times W$ mg of any drug in 50 mls yields a solution for which 1 ml/hr is equivalent to 1 µg/kg/min.

When initiating an infusion, use the ‘single strength’ concentration listed in Table 8.2 (adults), and start at a rate of approximately 5 ml/hr, using boluses (see Section 8.1.2) as necessary. If the patient’s condition later requires increasingly large infusion rates,
then higher concentrations (e.g., double strength, quadruple strength etc) can be used instead to avoid volume overload. If the patient is ‘obviously’ septic then start with a ‘double-strength’ dilution. A useful rule-of-thumb is to increase the infusion rate in steps of about 20%, but to decrease it in smaller steps (e.g., 10%).

Although 50 ml syringe-drivers are generally used in the operating theatres, in an ITU however, where the infusions often run for long periods of time, it is generally more convenient to use larger volumes (e.g., 100 ml or 250 ml bags) in conjunction with the usual ITU infusion-delivery systems.

Example 1

A 70 kg patient requires a dobutamine infusion. The dobutamine entry in Table 8.2, shows that diluting 210 mg ($3 \times 70$) in 50 ml 5% dextrose gives a concentration for which 1 ml/hr is equivalent to 1 µg/kg/min. Start the infusion at 5 mls/hr.

Example 2

A 30 kg patient requires a GTN infusion. The GTN entry in Table 8.2, shows that diluting 9 mg ($3 \times 30/10$) in 50 ml 5% dextrose gives a concentration for which 1 ml/hr is equivalent to $0.1 \mu g/kg/min$. Start the infusion at 5 mls/hr.

8.1.2 Bolus dosage

In emergency situations it is sometimes necessary to give IV bolus doses of these drugs (typically for GTN, adrenaline, noradrenaline). Consequently, it is very important that the dilution of the maintenance infusion used should simplify this, i.e., allow an IV bolus to be given manually and quickly using a syringe, so as not to be dependent on the electronic delivery system which can sometimes fail. An important ‘feature’ of the ‘single strength’ dilutions shown in Table 8.2 is that they facilitate the manual administration of appropriate bolus doses, quickly, safely and easily; typically using a 1 ml syringe (see examples below).

I have found empirically that an appropriate initial bolus dose is a volume roughly equivalent to 1–2 minutes worth of the current maintenance infusion (depending on the severity of the problem). In practice the 1-minute bolus volume is easily calculated since an infusion rate of 6 ml/hr is equivalent to $0.1 \text{ ml/min}$; a maintenance infusion rate of $R \text{ ml/hr}$ is therefore equivalent to $0.1 \times R/6 \text{ ml in one minute}$. For example, a 1-minute bolus volume for an infusion running at 12 mls/hr is $0.1 \times 12/6 = 0.2 \text{ ml}$. In general the bolus volume can be easily and conveniently delivered using a 1 ml syringe—always readily available on any ward. If the effect of the initial bolus is inadequate, sequentially double the bolus volume until a therapeutic bolus volume is found.
8.1.3 Noradrenaline

Example 1

Consider a 70 kg routine surgical patient requiring a noradrenaline infusion to counteract the effects of an epidural (typical requirement: single strength noradrenaline running at 2–8 mls/hr).

**Step 1:** Dilute $\frac{3\times70}{100}$ mg noradrenaline into 50 mls saline, (i.e., approximately 2 mg in 50 mls), and start the infusion at 5 mls/hr (equivalent to 0.05 µg/kg/min). Remember to include a three-way tap and a filled labelled 1 ml syringe, and connect the infusion to the central-line port as a separate dedicated infusion.

**Step 2:** Now fill the central-line deadspace cautiously, using 0.1–0.2 ml increments, until a bolus results in a transient rise in blood pressure. Then titrate the infusion using increments of about 2–4 mls/hr to obtain a suitable blood pressure, using small boluses via a 1 ml syringe as necessary to gain control. If the patient is significantly ‘septic’ then the infusion rate may well need to be increased, up to 50 mls/hr as necessary. A useful rule-of-thumb is to increase the infusion rate in steps of 20%, but to decrease it in steps of only 10%.

**Notes:** If there is occasion to rescue a low BP of, say, 60–70 mm Hg, then a bolus of approximately 1–2 mins worth of the infusion will be an appropriate first choice test dose. For example, if the current infusion rate is 6 mls/hr then consider a test bolus of about 0.1–0.2 mls (since an infusion of 6 mls/hr is equivalent to 0.1 ml/min).

Example 2

Consider a 70 kg septic ITU patient on double-strength noradrenaline at 12 mls/hr, and suppose you are now called because the infusion has suddenly stopped, and the blood pressure is falling.

This amount of noradrenaline is not unusual in the ITU—even the use of ‘quad’ strength is not uncommon. However, what is not widely appreciated is that if a ‘high-dose’ noradrenaline infusion stops suddenly (e.g., battery failure; line occlusion; bag/syringe empty etc.) the blood pressure fall will be rapid and profound—for example, falling to about 40–50 mmHg within 1 minute or so—and so this problem constitutes a significant emergency and must be addressed immediately. The essence of the problem, therefore, is how to administer 2 minutes worth of noradrenaline to the patient within less than 1 minute from a standing start.

**Step 1:** Note the infusion rate:— 12 mls/hr in this case.

**Step 2:** Divide the current infusion rate by 6 to determine the number of 0.1 ml units the patient would normally be receiving per minute $^3$ (this is the minimum bolus volume). If large infusion flow rates become necessary, then consider using twice the concentration (or more) with an appropriately reduced rate (one can happily use a ‘single’ strength dilution up to 50 mls/hr or so in a theatre setting).

$^2$An infusion of 6 mls/hr is equivalent to 0.1 ml/min.
Thus, in this example we have $12/6 = 2$, and so the ‘1-minute’ bolus volume is $2 \times 0.1 \text{mls} = 0.2 \text{mls}$.

**Step 3:** Fill a 1 ml syringe directly from the infusion bag/reservoir/pump and inject 2 mins worth of noradrenaline infusion as a bolus—0.4 ml in this case $(2 \times 0.2)$—down a separate CVP/IV line and flush it in with 10 ml saline. If there is already significant bradycardia, then atropine and temporary cardiac massage may also be necessary to help the drug get around. Titrate against the blood pressure, approximately 0.2 mls/min, as necessary, until the original infusion problem is fixed and ‘up and running’ again.

**Notes:** In this particular setting, since the bolus volume is so small and a rapid response is required, the bolus must be flushed in with, say, 10–20 ml saline, in order to guarantee that the bolus is delivered into the circulation (i.e., so the drug cannot inadvertently remain within the CVP line). Consequently, a drug-free CVP-line must be used, and if any uncertainty exists, then give the bolus (initially at least) into a peripheral vein.

This scenario occurs not infrequently, particularly in patients returning from theatre with their noradrenaline syringe driver on ‘empty’. You then have to sort out the severe hypotension and maintain an adequate blood pressure manually for about 5 mins or so while the nurses set up a new noradrenaline infusion. Fortunately, when a syringe driver alarms and stops there are usually several mls left in the syringe/system which can be used to fill your 1 ml syringe.

**Example 3**

Postoperative use of noradrenaline in patients with a history of hypertension. A patient with a long history of hypertension requiring quadruple drug antihypertensive therapy, was admitted postoperatively to ITU (following a nephrectomy; preop creatinine 150), with a blood pressure of 110–120 mmHg and poor urine output. From the notes it was clear that her usual blood pressure was approximately 160–170 mmHg, and so a CVP line was inserted and noradrenaline used to push the blood pressure up to 160 mmHg, with the effect that her urine output increased significantly and her creatinine stabilised out at 160–170. The noradrenaline was weaned down over about 4 days (during which her pre-op antihypertensive drugs wore off) by which time her blood pressure stabilised at about 160 mmHg and she was returned to the ward. Her normal antihypertensive therapy was then gradually re-introduced.

8.1.4 References


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4Avoid using the existing noradrenaline CVP line, since you need to guarantee that (a) only the bolus dose is given, and (b) it is delivered into the circulation—you can not flush the bolus with saline down the existing noradrenaline infusion line. Much safer to give it initially via a separate line, until you have control of the blood pressure.

• Cooper DW (2005). Ephedrine, phenylephrine and fetal acidosis. *Anaesthesia*; 60, 1237–1238. [10 refs]


• Roth JV (2006). The use of vasopressin bolus to treat refractory hypotension secondary to reperfusion during orthotopic liver transplantation. *Anesthesia & Analgesia*; 103 (July), 261. [6 refs]

[Used an infusion of vasopressin 4 U/h, plus two boluses of 0.4 U, in a 83 Kg man relatively refractory to noradrenaline (20 mls/hr of ‘single-strength’ noradrenaline).]


8.2 Somatostatin analogues & carcinoid

An good overview with useful references is presented by Mason (2001).

8.2.1 Octreotide

Octreotide is a synthetic analogue of the hypothalamic release-inhibiting hormone somatostatin (BNF), and is a key drug in the management of patients with carcinoid syndrome (Farling and Durairaju 2004; Vaughan and Brunner 1997; Battershill PE and Clissold SP 1989). It is used to counteract serotonin (5HT) and kinin activity and to gain full control of symptoms (subcutaneously (adults): initially 50 µg 1–2 times/day increasing to 200 µg 3 times/day). Octreotide is increasingly being used in the treatment of congenital and postoperative chylothorax (Roehr CC, Jung A, Proquitté H et al. 2006).

8.2.2 Ketanserin

Ketanserin is a selective antagonist of anti-5HT₂, the α₁-adrenoreceptor, and the H₁-histamine receptor (Koopmans et al. 2005; Hughes and Hodkinson 1989). Ketanserin reduces portal pressure in animals (Chernow 1988). A 10 mg bolus IV (adults) has been used successfully in carcinoid crisis to block mediators (Koopmans et al. 2005, Hughes and Hodkinson 1989). Alternatively (adults), ketanserin 10 mg given over 3 mins followed by an infusion of 3 mg/hr is suggested by Mason (2001), Fischler et al. (1983), Hughes and Hodkinson (1989).

8.2.3 Carcinoid tumours

Carcinoid (neuroendocrine) tumours have their origin in the endocrine argentaffine cells of the small bowel mucosa, which are part of the so-called APUD family (Amine content, Precursor Uptake, and Decarboxylation). Carcinoid is characterised biochemically by an increased urinary excretion of 5-hydroxyindole-acetic acid (5-HIAA; Mol wt 191), the normal range being 10–47 µMol/24 hrs (1·9–8·9 mg/24 hrs).

A range of peptides, kinins and prostaglandins can be secreted, including kallikrein, bradykinin, serotonin (5HT), histamine, and substance-P. Less commonly secreted are insulin, ACTH, MSH, gastrin and glucagon. The carcinoid syndrome symptoms of diarrhoea, sedation, hypertension are thought to be due to serotonin; flushing, hypotension and bronchospasm are thought to be due to bradykinin (Mason and Steane 1976).

8.2.4 Anaesthesia for bronchial carcinoid resection

The anaesthetic management of such patients consists primarily of (a) using the drug octreotide for preoperative symptom control (2 days–2 weeks) and also as part of the premedication regimen, (b) avoiding drugs which liberate histamine (e.g., atracurium,
morphine), (c) using small boluses of octreotide (adults: 10–20 µg) as necessary to counteract symptomatic episodes (flushing, hypotension, bronchospasm). Ketanserin (adults: 10 mg bolus IV) has been used successfully in carcinoid crisis to block mediators (Koopmans et al. 2005, Hughes and Hodkinson 1989). Steroids are often used perioperatively. Bradykinin may be the key mediator of hypotension in carcinoid syndrome (Veal et al. 1994).

**Preoperative control of symptoms:** Octreotide is typically given subcutaneously in doses (adults) of 50–200 µg 8-hrly.

**Premedication:** Octreotide 50–100 µg subcutaneously 1 hr preoperatively (adults).

**Induction:** Insert the arterial line under local prior to induction. Try to avoid the use of rigid bronchoscopy by the surgeons on induction in order to minimise airway stimulation—suggest they consider flexible bronchoscopy via a single-lumen tube if necessary.

**Intra-operatively** (adults): Use octreotide intravenously as required to control exacerbations of bronchospasm, hypotension with flushing, and bradycardia (100 µg diluted in 10–20 mls saline). Titrate using small boluses of approximately 10–20 µg. Bradycardia associated with heart-block has been reported following a 100 µg bolus (Dilger et al. 2004).

Acute hypertension may be due to serotonin (5HT) (blocked by ketanserin), while acute hypotension may be due to bradykinin (blocked by octreotide). Have a syringe of adrenaline (1/10,000) immediately available to treat episodes of profound bradycardia or asystolic arrest

Consider ondansetron (give slowly) for its anti-5HT activity (Wilde and Markham 1996). Consider using chlorpheniramine (anti-H₁) and ranitidine (anti-H₂) (Vaughan, Howard and Brunner 2000, Mason and Steane 1976). Monitor blood glucose (carcinoid tumours may secrete insulin or glucagon). Epidural bupivicaine + fentanyl is fine.

The vasopressors of choice in carcinoid surgery are most probably phenylephrine, metaraminol, and also methoxamine (not currently available), since they are not catecholamines (Koopmans et al. 2005). However, I have used a noradrenaline infusion in carcinoid cases on several occasions uneventfully.

**Example of intraoperative hypotension**

Figure 8.1 shows an episode of extreme hypotension during a thoracic carcinoid resection. During this operation there were two such episodes (one cardiac standstill) which responded rapidly to open cardiac massage, adrenaline (2 ml of 1/10,000) and 20 µg octreotide via the CVP line. Although a noradrenaline infusion was being used at the time (to counteract the epidural), each of these episodes appeared to be related to surgical manipulation; no noradrenaline boluses were given at any stage.

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5 Alternatively ketanserin 10 mg over 3 mins followed by an infusion of 3 mg/hr (Mason 2001).
6 1 mg in 10 mls saline.
7 See letter by Cherian and Maguire 2005.
8 Since o–dihydroxybenzine is called catechol, sympathomimetic amines having the same OH-substitutions in the benzine ring (i.e., in both the 3- and 4- positions) are termed catecholamines (e.g., dopamine, dobutamine, noradrenaline, adrenaline, isoprenaline).
Figure 8.1: Anaesthetic record during a thoracic carcinoid resection in an adult, showing an episode of bradycardia and extreme hypotension which responded to cardiac massage, bolus of adrenaline (2 ml 1/10,000) and 20 µg octreotide—see text. Notice the transient reduction in ET\(_{CO_2}\) due to low cardiac output associated with the period of hypotension. The patient made a full and uneventful recovery. Datex AS/3 monitor; data points at 5 sec intervals; BP, NIBP, CVP mmHg; \(P_{plateau}\) cm H\(_2\)O; TV mls; SAT, \(F_{I\text{O}_2}\), ET\(_{CO_2}\), \(F_{IN\text{O}_2}\), MAC\(_{age}\), VAP (sevoflurane) \%. 
8.2.5 References


- Carcinoid Cancer Foundation (http://www.carcinoid.org/): This web site is mainly for patients and has lots of general information. However, it also has a good medical section (http://www.carcinoid.org/content/medical-reviews/) which has several useful articles.

- Cherian A and Maguire M (2005). Transient blindness following intravenous ondansetron. Anaesthesia; 60, 938–939. [advise slow IV injection; 5HT₃ and 5HT₃A receptors are involved in modulation of retinal signals]


• Vaughan DJ, Howard JM and Brunner MD (2000). Refractory hypotension during carcinoid resection. *Anaesthesia*; 55, 927 [letter] [see also: Cortinez 2000].


8.3 Haemostatic drugs

Note that the hospital has a ‘major haemorrhage’ guideline. See also review by Mahdy and Webster (2004) discussing the role of agents for reducing perioperative blood loss and transfusion requirements. Consider the cell-saver.


Recombinant factor VIIa

The hospital has a *Guideline for use of Factor VIIa* for use in massive haemorrhage.


8.4 Remifentanil

Remifentanil (*Ultiva*) is an ultra short-acting narcotic, used either as a bolus or as an infusion. Remifentanil pharmacokinetics are not modified by hepatic or renal failure (Absalom and Struys 2006). Duration of action when given as a bolus is approximately 10 mins. See the excellent review by Scott and Perry (2005).

8.4.1 Bolus

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dilution (in saline)</th>
<th>Bolus dose (adults)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remifentanil (Ultiva)</td>
<td>1 mg/20 mls</td>
<td>0.5–1.5 µg/kg</td>
</tr>
<tr>
<td>1 mg ampoule</td>
<td>(50 µg/ml)</td>
<td></td>
</tr>
</tbody>
</table>
Induction of anaesthesia

Remifentanil is an extremely useful co-induction agent for intubation and for bronchoscopy. It is very cardio-stable and reduces the propofol induction dose by about 50%. Typical co-induction dose for 70 kg adult: 70–100 µg (in 25–50 µg increments over 2 mins). Use approximately half the dose in the elderly. Watch out for (a) respiratory depression, especially in the elderly (only give during pre-oxygenation), (b) bradycardia (have atropine handy), (c) hypotension.

During anaesthesia

Bradycardia seems to be most pronounced during anaesthesia with volatiles, so once anaesthesia has been established then titrate initially with 10–20 µg boluses (70 kg adult) in order to determine the effective dose.

8.4.2 Infusion

For sedation and analgesia in ventilated patients in an ITU setting, the recommendation is to start at about 0.1 µg/kg/min and titrate by ±20% depending on the response, as shown in the following Table.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dilution (5% Dex or saline)</th>
<th>Infusion (adults)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Remifentanil (Ultiva)</td>
<td>3 × kg ( \frac{mg}{100 mL} ) in 50 mLs</td>
<td>start at 10 mLs/hr ( (0.1 , \mu g/kg/min) )</td>
</tr>
</tbody>
</table>

For anaesthesia, any recommendation of specific ranges of MAC to supplement a remifentanil infusion should really be based on a review of the growing literature of observed MAC reductions (see: Breslin et al. 2001, Lang et al. 1996, van Delden et al. 2002).

For TCI techniques see Absalom and Struys (2007). The Minto 3-compartment model (Minto, Schnider, Egan et al. 1997; Minto, Schnider and Shafer 1997) most commonly used for remifentanil, uses age and lean body mass (LBM) as co-variables, and so requires entry of weight, height and gender as well (Absalom and Struys 2007, p. 16).

8.4.3 References


This article attempts to determine the appropriate remifentanil infusion rate associated with sevoflurane at 1·5, 1·0 and 0·5 MAC. They found that a remifentanil infusion of 0·34 (range 0·20–0·49 µg/kg/min) requires a MAC of 0·5 in a population of mean age 30 years and mean BMI 25·1 for operations of mean duration 57 mins.


Their conclusion (majority of patients in the age-range 31–55) was that isoflurane (in oxygen only) concentrations of 0·4–0·5 % (i.e., 0·4 MAC40 approx from my charts) required a remifentanil infusion rate of 0·15–0·3 µg/kg/min for adequate anaesthesia (plasma level of 4–8 ng/ml).


This article determined the appropriate remifentanil infusion rate associated with sevoflurane (with no nitrous oxide) at 1·2, 0·8 and 0·4 MAC. They found that a remifentanil infusion of 0·23 (range 0·12–0·73 µg/kg/min) requires a MAC of 0·8 in a population of mean age 47 years. Their conclusion was that the optimum combination (for anaesthesia quality and recovery rate) was sevoflurane ET 1·24 % (i.e., MAC47 = 0·7 approx from my charts) and remifentanil 0·23 µg/kg/min (I have converted their µg/kg/hr to µg/kg/min).

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9See Figures 9.1, 9.2, 9.3.
Supporting technologies

Here we address some of the supporting technologies associated with thoracic anaesthesia. The motivation is historical—to highlight some of the key references and see how discoveries were made. Since our subject is thoracic anaesthesia the recent historical overview by Brodsky (2005) is an excellent starting point.

Unfortunately the historical background of medical discoveries tends to be insufficiently emphasised, but time spent reading original papers and early documentation is usually well rewarded, often revealing not only scientists with extreme focus and mental tenacity but also the occasional hand of serendipity.

Two insights are commonly cited as influencing discovery and subsequent innovative development. The first is awareness of the problem, immortalised by Louis Pasteur (1822–1895) in the succinct but misquoted phrase Chance favours the prepared mind. The second is appreciating the significance of a discovery. For example, although Crawford Long (1815–1878) was apparently the first person to use ether for a surgical operation, we really owe the initial development of clinical anaesthesia to William Morton (1819–1868) who appreciated its value to humanity, and to John Snow (1813–1858) who laid the scientific foundations of inhalational anaesthesia.

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1See the memorable September 2002 issue of the ASA Newsletter (http://www.asahq.org/) on the history of monitoring.
3See accounts by Comroe (1977), Swazey and Reeds (1978) and Watson (1968) in Section 1.2 (History).
4The full sentence actually used by Pasteur in his inaugural address (1854) as the professor of chemistry at the Faculté des Sciences at Lille, was In the field of observation, chance only favours those minds which have been prepared. (Mackay A (1977). The harvest of a quiet eye; a selection of scientific quotations. Institute of Physics, London; ISBN 0-85498-031-8, page 116.)
6It now appears that there were in fact several other people experimenting with ether at that time (see Section 9.4.5) Stone, Meyer and Alston 2010.
7See more on John Snow in Section 9.4.1.
9.1 Serendipity

The word serendipity is often used in the context of discovery and innovation; its original usage was very specific—discovering, quite by accident, something that you were not looking for (Remer 1965). The word is ascribed to Horace Walpole (1717–1797), who used it in 1754 in a letter to Horace Mann, then living in Florence (Toynbee 1903; Remer 1965, Boyle 2000).

This discovery indeed is almost of that kind I call serendipity, a very expressive word, which as I have nothing better to tell you, I shall endeavour to explain to you: you will understand it better by the derivation than by the definition. I once read a silly fairy tale, called The three princes of serendip:9 as their highnesses travelled, they were always making discoveries, by accidents and sagacity, of things which they were not in quest of: for instance, one of them discovered that a mule 10 blind of the right eye had travelled the same road lately, because the grass was eaten only on the left side, where it was worse than on the right—now do you understand serendipity? One of the most remarkable instances of this accidental sagacity (for you must observe that no discovery of a thing you are looking for, comes under this description) was of my Lord Shaftsbury, who happening to dine at Lord Chancellor Clarendon’s, found out the marriage of the Duke of York and Mrs Hyde, by the respect with which her mother treated her at table.

Horace Walpole — January 28, 1754

From: Toynbee (1903)

The animal in the original story was actually a camel. The relevant extract from the 1964 English translation of the 1557 Italian version (Boyle 2000), runs as follows.

Misfortune befalls the princes when a camel driver stops them on the road and asks them if they have seen one of his camels. Although they have not, they have noticed signs that suggest a camel has passed along the road. Ever ready to dazzle with their wit and sagacity, the princes mystify the camel driver by asking him if the lost camel is blind in one eye, missing a tooth and lame. The camel driver, impressed by the accuracy of the description, immediately hurries off in pursuit of the animal.

After a fruitless search, and feeling deceived, he returns to the princes, who reassure him by supplying further information. The camel, they say, carried a load of butter on one side and honey on the other, and was ridden by a pregnant woman. Concluding that the princes have stolen the camel, the driver has them imprisoned. It is only after the driver’s neighbour finds the camel that they are released.

The princes are brought before the Emperor Beramo, who asks them how they could give such an accurate description of a camel they have never seen. It is clear from the princes’ reply that they had brilliantly interpreted the scant evidence observed along the road.

8Letter dated 28 January, 1754—see Toynbee (1903).
9Sri Lanka.
10A camel in the English version.
As the grass had been eaten on one side of the road where it was less verdant, the princes deduced that the camel was blind on the other side. Because there were lumps of chewed grass on the road the size of a camel’s tooth, presumably they had fallen through the gap left by a missing tooth. The tracks showed the prints of only three feet, the fourth being dragged, indicating that the animal was lame. That butter was carried on one side of the camel and honey on the other was clear because ants had been attracted to melted butter on one side of the road and flies to spilled honey on the other.

Boyle (2000)


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### 9.2 Tuohy needle with Huber point and Lee markings

Not only is the modern epidural needle a fusion of three primary ideas, but it actually started life as spinal needle. Of the many designs which have been developed few have endured (see the excellent review by Frölich *et al.* 2001).

Edward Boyce Tuohy (1908–1959) was an anaesthetist at the Mayo Clinic (Rochester, Minnesota, USA), interested in continuous spinal anaesthesia (Maltby 2002). Although this technique was first described by Henry Dean in 1906, it was mainly developed during the 1940s by William Lemmon (Maltby, 2002). However Lemmon’s technique was far from ideal since it required the needle to remain in position (tip in the CSF) during anaesthesia in order to allow intermittent top-ups as required.

Tuohy’s idea was to develop a method of introducing a catheter into the lumbar CSF space via a spinal needle to facilitate continuous spinal anaesthesia—the needle itself could then be removed once the catheter was in place. Continuous caudal anaesthesia via a caudal catheter was already fairly widely used, and lumbar subarachnoid catheters were also sometimes used for drainage in meningitis.

In 1944 Tuohy described this technique using an ordinary 15-gauge spinal needle (Maltby 2002), and only later, in 1945, did he decide to incorporate the Huber point (designed by Ralph L Huber (1890–1953) and made by Becton Dickinson) taking advantage of its lateral opening which allowed the catheter to be directed sideways into the
space (Tuohy 1945). In 1949, both MM Curbelo and Charles Flowers described using the Tuohy needle for epidural anaesthesia. Tuohy was later appointed professor of anaesthesia at the Georgetown Medical Centre in Washington, DC.

Finally, we owe the 1 cm markings on the standard Tuohy needle to the English anaesthetist John Alfred Lee (1906–1989). He added them so that anaesthetists would know fairly accurately the depth of the needle tip, and hoped that this small refinement might reduce the number of dural taps (Lee 1960; Maltby 2002).


### 9.3 Pulse oximetry

For a detailed review of the long and fascinating history of blood gases, oximetry and pulse oximetry see Severinghaus (2002; 1986) and Severinghaus and Astrup (1987).

The first practical oximeter was the eight-wavelength ear-oximeter developed in 1964 by Robert Shaw and subsequently marketed by Hewlett-Packard in 1970 (Moyle 1994). In 1971 Takuo Aoyagi (1936–), a Japanese biomedical engineer at the Nihon Kohden Corporation (Tokyo), used the pulsatility of the absorption signal to separate arterial and tissue absorption and determine arterial saturation. Severinghaus describes it thus:

> Takuo Aoyagi . . . attempted to eliminate arterial pulsatile ‘noise’ in his earpiece dye dilution curves by subtracting infra-red signals. He observed that the compensated noise varied with oxygen saturation and realised that it might be used to compute the arterial oxygen saturation.

Severinghaus (1989)
The first commercial pulse oximeter appeared in 1970 (Moyle 1994), and in 1982 the Stanford anaesthetist William New (1942–), with Jack Lloyd and engineer Jim Corenham, founded Nellcor Incorporated to mass-produce clinically useful pulse-oximeters (Rendall-Baker and Bause 2002).

If a finger pulse-oximeter fails owing to significant peripheral vasoconstriction, then an ear-probe will usually be satisfactory. Alternatively, a digital nerve block may help (Erasmus 2003).


• Rendell-Baker L and Bause GS (2002). Cardiorespiratory monitoring: a pictorial sampler. ASA Newsletter; 66, No. 9 (September) (http://www.asahq.org/)


The name ‘Nellcor’ was derived from a synthesis of the surnames NEw, LLoyd, CORenham (Rendall-Baker and Bause 2002).


9.4 MAC

*Ether contributes other benefits besides preventing the pain. It keeps patients still, who otherwise would not be.*  

John Snow  


### 9.4.1 History

**John Snow**

John Snow (1813–1858) appears to have been the first person to appreciate the importance of controlling the inspired concentration of volatile anaesthetics, and within five years of William Morton’s ether demonstration he had single-handedly established the scientific foundations underpinning the pharmacokinetics of volatile anaesthetics. Snow was a London-based GP with hospital connections, and had been interested for a long time in the use of inhalation agents on respiration. He initially investigated the use of carbon dioxide, and had been experimenting with inhaled ether since 1843 believing it to be a useful medicine for improving circulation. In 1846 he published an article entitled “Pathological effects of atmospheres” (Maltby 2002; Vinten-Johansen et al. 2003).

Consequently, following Morton’s ether demonstration (October 16, 1846) at the Massachusetts General Hospital (Boston, USA), and the subsequent demonstration in London in December 1846, Snow found himself in the right place at the right time. Furthermore, with his interest in chemistry and recent researches into inhaled ether he also found himself to be just the right person to get involved in this new anaesthesia phenomenon. Since his GP work was not very profitable, Snow decided to take up anaesthesia.

Snow was extraordinarily industrious and productive. By mid 1847 he had (a) defined and published the temperature characteristics of ether vapour (January 1847), (b) designed

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12 This quotation, which heads Eger’s own chapter on MAC, is from: Snow 1847 (part 4).

13 William Morton (1819–1868) gave his demonstration on October 16, 1846. For an excellent account of the history and background see MacQuitty (1969).
an ether inhaler, (c) defined a range of clinical stages of anaesthesia (his five ‘degrees of narcotism’), and (d) performed many animal experiments with a view to determining the effects of different inspired concentrations of both ether and chloroform. Snow appreciated the significance of knowing the saturated vapour pressure, and went on to show that the amount of volatile agent required to produce anaesthesia was inversely related to its solubility in the blood.

Snow published his findings in an eighteen-part series of articles in the journal London Medical Gazette during the period 1848 to 1851, entitled “On narcotism by the inhalation of vapours”. In the following extracts (all from Part–I of the series) Snow describes the effects on a mouse of a sequence of step increases in the inspired concentration of ether (from approximately 1.2% to 4.7%). Notice the detail of his observations, and how he pays particular attention to how well the mouse breathes (I have added a calculated concentration in MAC$_{ether/mouse}$ at each stage to make it easier to follow his experiments).

I consider, however, that I have found a plan of determining more exactly the [required] proportion of ether and of other volatile substances present in the blood in the different degrees of narcotism. It consists of ascertaining the most diluted mixture of vapour and of air that will suffice to produce any particular amount of narcotism; and is founded on the following considerations, and corroborated by its agreeing with the comparative physiological strength of the various substances.

… The plan which I adopted to ascertain the smallest quantity of vapour, in proportion to the air, that would produce a given effect, was to weigh a small quantity of the volatile liquid in a little bottle, and introduce it into a large glass jar covered with a plate of glass; and having taken care that the resulting vapour was equally diffused through the air, to introduce an animal so small, that the jar would represent a capacious apartment for it, and wait for that period when the effects of the vapour no longer increased. …

Exp. 17. — Two grains of ether were put into a jar holding 200 cubic inches [1.16%, 0.36 MAC], and the vapour diffused equally, when a tame mouse was introduced, and allowed to remain a quarter of an hour, but it was not appreciably affected.

Exp. 18. — Another mouse was placed in the same jar, with three grains of ether, being a grain and a half to each 100 cubic inches [1.75%, 0.55 MAC]. In a minute and a half it was unable to stand, but continued to move its limbs occasionally. It remained eight minutes without becoming further affected. When taken out it was sensible to pinching, but fell over on its side in attempting to walk. In a minute and a half the effect of the ether appeared to have gone off entirely.

Exp. 19. — A white mouse in the same jar, with four grains of ether [2.33%, 0.72 MAC], was unable to stand at the end of a minute, and at the end of another minute ceased to move, but continued to move its limbs occasionally. It remained five minutes without becoming further affected. When taken out it was sensible to pinching, but fell over on its side in attempting to walk. In a minute and a half the effect of the ether appeared to have gone off entirely.

\[ 	ext{MAC}_{ether/mouse} = 3.2\% \] (Eger 1974, p. 5).
Exp. 20. — Five grains of ether, being two and a half grains to each 100 cubic inches [2.92 %, 0.91 MAC], were diffused throughout the same jar, and a mouse put in. It became rather more quickly insensible than the one in the last experiment. It was allowed to remain eight minutes. It moved its foot a very little when pinched, and recovered in the course of four minutes.

Exp. 21. — A white mouse was placed in the same jar with six grains of ether [3.5 %, 1.1 MAC]. In a minute and a half it was lying insensible. At the end of three minutes the breathing became laborious, and accompanied by a kind of stertor. It continued in this state till taken out, at the end of seven minutes, when it was found to be totally insensible to pinching. The breathing improved at the end of a minute; it began to move at the end of three minutes; and five minutes after its removal it had recovered.

Exp. 22. — The same mouse was put into this jar on the following day, with seven grains of ether, being 3.5 grs to the 100 cubic inches [4.08 %, 1.28 MAC]. Stertorous breathing came on sooner than before; it seemed at the point of death when four minutes had elapsed; and being then taken out, was longer in recovering than after the last experiment.

Exp. 23. — Two or three days afterwards the same mouse was placed in the jar, with eight grains of ether, being 4 grains to the 100 cubic inches [4.66 %, 1.46 MAC]. It became insensible in half a minute. In two minutes and a half the breathing became difficult, and at a little more than three minutes it appeared that the breathing was about to cease, and the mouse was taken out. In a minute or two the breathing improved, and in the course of five minutes from its removal it had recovered.

... We find from the eighteenth experiment, that a grain and a half of ether for each 100 cubic inches of air, is sufficient to induce the second degree of narcotism in the mouse; and a grain and a half of ether make 1.9 cubic inches of vapour, of sp. gr. 2.586. Now the ether I employed boiled at 96°[F].\footnote{96°F = 35.5°C, \( \left( \frac{F - 32}{9} \right) = \frac{C}{5} \). Pure diethyl-ether boils at 34.51°C (CRC Handbook of chemistry and physics; 1972).} At this temperature, consequently, its vapour would exclude the air entirely; and the ether vapour in contact with the liquid giving it off, could only be raised to 100° by such a pressure as would cause the boiling point of the ether to rise to that temperature. That pressure would be equal to 32.4 inches of mercury [1.082 Atm.], or 2.4 inches above the usual barometrical pressure; and the vapour would be condensed somewhat, so that the space of 100 cubic inches [at 1.082 Atm.] would contain 108 cubic inches at the usual pressure [1 Atm.]. This is the quantity, then, with which we have to compare 1.9 cubic inches, in order to ascertain the degree of saturation of the space in the air-cells of the lungs, and also of the blood; and by calculation, as when treating of chloroform,

\[ 1.9 \text{ to } 108 \text{ as } 0.0175 \text{ is to } 1 \]

So that we find 0.0175 [1.75 %], or 1/57th, to be the amount of saturation of the blood by ether necessary to produce the second degree of narcotism;\footnote{Snow (1848a)}
Notice the interesting way in which Snow calculates the vapour concentration resulting from 1·5 grains\(^{16}\) of liquid diethyl-ether\(^{17}\) in 100 cubic inches\(^{18}\) of air at 100\(^{\circ}\)F\(^{19}\) as 1·75 %. My own calculation runs as follows. Since the molecular weight of diethyl-ether is 74·12, the volume of pure ether vapour at STP occupied by 96·75 mg liquid ether (1·5 grains) is given by\(^{20}\)

\[
22·4 \times \frac{96·75}{74·12} = 29·24 \text{ cc}
\]

If we now correct this volume for a temperature of 100\(^{\circ}\)F (37·7\(^{\circ}\)C) we obtain \(29·24 \times 310·7/273 = 33·3 \text{ cc.}\) Adding this volume of pure vapour to 100 cubic inches of air\(^{22}\) (also at 37·7\(^{\circ}\)C) gives a concentration of

\[
\frac{33·3}{33·3 + (100 \times 2·54^3 \times 310·7/273)} = 0·01754 \equiv 1·75 \%
\]

However, we have made some simplifying assumptions (e.g., constant pressure and complete mixing), and since Snow only used a glass jar with a simple lid, it is likely that some of the mixture escaped from the jar before mixing was complete.\(^{23}\)

It is clear from these extracts from Snow’s publications, that Snow was seeking the inspired concentration associated with each of his five ‘degrees of narcotism’, and that he was guided by two key principles, namely (a) to determine ‘the most diluted mixture’ which gave these effects (i.e., the minimum concentration), and (b) waiting until ‘the effect no longer increased’ (i.e., at equilibrium).

Snow’s pharmacological approach of linking particular inspired concentrations of vapour to particular states or depths of anaesthesia, and then using this information to try and deliver a safer form of anaesthesia by controlling the inspired vapour concentration was, therefore, strikingly similar to our modern use of MAC.\(^{24}\) His experiments were carefully performed, observed, and well documented—in fact so much so that they even allow us to make a reasonably accurate estimate of MAC for the mouse. For example, Snow’s experiments 20 and 21 suggest that the inspired concentration of ether associated with 50 % movement was between 2·9 % and 3·5 %, giving an estimate close to the modern value of MAC\(_{\text{ether/mouse}}\) of 3·2 %.

\(^{16}\) 1 grain = 64·5 mg.
\(^{17}\) \(\text{CH}_3\text{CH}_2\text{OCH}_2\text{CH}_3\); molecular weight = 74·12; BP = 34·51 \(^{\circ}\)C.
\(^{18}\) 1 cubic inch = 2·54^3 = 16·38 cc.
\(^{19}\) 100 \(^{\circ}\)F = 37·7 \(^{\circ}\)C.
\(^{20}\) 1 gm mol of vapour occupies 22·4 L at STP.
\(^{21}\) Note that this is roughly mouse body temperature.
\(^{22}\) We keep the pressure constant and assume complete mixing.
\(^{23}\) Please email me if you improve on this analysis.
\(^{24}\) The first paper on MAC was by Merkel and Eger (1963). The definition of MAC is as follows:– the minimum alveolar concentration, at equilibrium, and at 1 atmosphere pressure, which prevents movement in 50 % of patients to a standard surgical incision. For an excellent early overview of MAC see Chapter 1 in the timeless classic book by Eger (1974).
100 years or so later in the early 1960s Eger and Severinghaus embodied Snow’s concepts in the form of MAC (Merkel and Eger 1963; Saidman and Eger 1964; Eger, Saidman, and Brandstater 1965a).

**Edmond Eger**

In 1960 Edmond Eger joined the San Francisco Department of Anaesthesia, and became a ‘Research Fellow’ to John Severinghaus (Eger 2002, Maltby 2002). Eger and Dr Giles Merkel (Research Fellow) were given the task of defining the properties of a new volatile anaesthetic agent called halopropane. Eger describes the early steps as follows.

From studies John [Severinghaus] and others had performed with carbon dioxide, we knew that measuring the end-tidal concentration of a gas gave us a handle on the arterial partial pressure for that gas. Also, the work of Kety and Schmidt indicated that the cerebral partial pressure of an inert gas should rapidly equilibrate with the partial pressure in arterial blood. So, if we measured the end-tidal concentration of halopropane and held it stable for a sufficient period of time, the end-tidal concentration would give us a measure of the anesthetic partial pressure at its site of action. With that, we had the first part of MAC.

The second part was not hard to come by. … Movement. A categorical response, seemed just the thing … So we married the end-tidal concentration with movement–no movement as an index of anesthesia, and MAC was born.

Everything except the name. John’s group met every Monday morning to discuss the previous week’s work and what might be done in the coming week … At one of these Giles and I told of our technique for determining the minimal alveolar anesthetic concentration, and John connected this to the ratio of the speed of an airplane relative to the speed of sound (a MAAC ratio). John now says it never was clear why we chose MAC rather than MAAC. I don’t remember either, except that we wanted to emphasise the word “alveolar”. Besides, voicing “MAAC” might make us sound like bleating sheep rather than anesthesiologists.

The next step was to determine MAC in humans. … The result was the series of articles that were published in 1965 (Eger, Saidman and Brandstater 1965a, 1965b; Eger *et al.* 1965).

**John Severinghaus**

Severinghaus (Severinghaus 2009, Maltby 2002) recalled this episode in a recent journal interview as follows (Kofke 2003).

Dr. Eger was interested in the relative potency of anesthetics. He wanted a way to compare them numerically in terms of their alveolar concentrations at the time of establishment of a minimal level of anesthesia to permit surgery. It was clear to all that for each patient or animal, there was a critical alveolar (and thus arterial and ultimately brain) pressure of an agent that just prevented a motor response to pain. He
believed this would be a relatively invariant number between patients. This would be the minimal alveolar anesthetic concentration. I recalled that in aviation, a similar index, Mach, was the ratio of an aircraft’s speed to the speed of sound.\textsuperscript{26} A hypersonic flight was defined, for example, as Mach 2, twice the speed of sound. I suggested the same symbol be used for the ratio of concentration of the anesthetic in the alveoli (as determined in the airway at end expiration) to that critical no-movement level, which would be defined as 1 MAC, originally MAAC. It still should be MAAC since we can’t agree on whether the single ‘A’ refers to alveolar or anesthetic or both.

Kofke (2003)

William Mapleson

In 1979, a far-sighted William Mapleson anticipated the increasingly central role of MAC with respect to how anaesthetists delivered a given depth of anaesthesia, as follows (see also Maltby 2002).

\ldots To this end, the anaesthetist will be invited to set his flows of oxygen and nitrous oxide in the normal way and then to set the brain tension of anaesthetic he requires, not in kPa or mmHg, but in total MAC units.

Mapleson (1979).

More recently the clinical utility of MAC has been extended by establishing its variation with age (Mapleson, 1996), temperature (Eger 2001) and hair colour (Liem \textit{et al.} 2004).

9.4.2 Age-corrected MAC

Although several factors are known to be associated with altered anaesthetic requirements,\textsuperscript{27} age is the most important owing to the increasingly large age-range met with in clinical practice.

While age has long been known to influence anaesthetic requirement (Gregory, Eger and Munson 1969), the exact variation of MAC with age was formalised only recently by Mapleson (1996), following a meta-analysis of the available data (see Table 9.1). In particular, Mapleson showed that semi-log plots of MAC against age (age \(\geq 1\) year) for all inhalational agents are linear and parallel, and hence it is probable that all the inhalational agents achieve their effects by a similar mechanism. On this basis, therefore, Mapleson derived the following relationship between age and MAC from the pooled data,

\[
MAC_{age} = MAC_{40} \times 10^{-0.00269(age-40)}
\]

which expresses MAC for a given age as a function of that at 40 years (MAC\(_{40}\)).

The computed real-time MAC as displayed by the Datex AS/3 and S/5 anaesthesia monitors relates to normothermic patients aged approximately 35 years-old. However,

\textsuperscript{26}Severinghaus worked on radar technology during World War II (Kofke 2003).

\textsuperscript{27}The key factors are narcotics (see section on remifentanil in the appendix), age, temperature, pregnancy, and hair colour (Liem \textit{et al.} 2004, showed that patients with red hair had a 19 \% increased MAC requirement).
Table 9.1: MAC data based on age ≥ 1 year. The 95% confidence limits (CL) for ages 1 and 80 years are up to 1% greater than at MAC$_{40}$ (from Mapleson 1996). * For the CO$_2$ value see Eisele and Eger (1967).

<table>
<thead>
<tr>
<th>Agent</th>
<th>1 year</th>
<th>40 years</th>
<th>80 years</th>
<th>95% CL (± % MAC$_{40}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Halothane</td>
<td>0.95</td>
<td>0.75</td>
<td>0.58</td>
<td>6</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.49</td>
<td>1.17</td>
<td>0.91</td>
<td>6</td>
</tr>
<tr>
<td>Enflurane</td>
<td>2.08</td>
<td>1.63</td>
<td>1.27</td>
<td>17</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>2.29</td>
<td>1.80</td>
<td>1.40</td>
<td>6</td>
</tr>
<tr>
<td>Desflurane</td>
<td>8.3</td>
<td>6.6</td>
<td>5.1</td>
<td>10</td>
</tr>
<tr>
<td>Carbon dioxide*</td>
<td>—</td>
<td>≈ 30</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Xenon</td>
<td>92</td>
<td>72</td>
<td>57</td>
<td>16</td>
</tr>
<tr>
<td>Nitrous oxide</td>
<td>133</td>
<td>104</td>
<td>81</td>
<td>8</td>
</tr>
</tbody>
</table>

since many of the thoracic patients are quite elderly it is more appropriate clinically to use an age-corrected MAC. A real-time software version which incorporates nitrous oxide is shown in Figure 9.5 (page 159). Print versions in the form of graphs which allow for nitrous oxide use have been designed (Nickalls and Mapleson 2003), a separate chart being used for each volatile agent as shown in Figures 9.1–9.3. The use of nitrous oxide is accommodated in the charts by offsetting the right-hand N$_2$O scales vertically by the amount given by

\[ F_{E_{N_2O}} = \frac{\text{MAC}_{\text{age, volatile}}}{\text{MAC}_{\text{age, N}_2\text{O}}} \]

The print versions are available for download and also in Allman and Wilson (2006). A nomogram by Lerou (2004) also gives age-corrected MAC.

Software

The iso-MAC information is also available for some hand-held devices, for example, as the software MACpalm and ACTc.

**MACpalm**: The MACpalm program is available from [http://www.medicaldownload.com/medicalsoftware/macpalm.html](http://www.medicaldownload.com/medicalsoftware/macpalm.html). The installation is described in the MACpalm manual.

**ACTc**: The Anesthesia Clinical Tutor and Calculator (ACTc) program is available from [http://www.gasshead.com/](http://www.gasshead.com/). A manual is available at [http://www.gasshead.com/content/TutorACTc.pdf](http://www.gasshead.com/content/TutorACTc.pdf)

28These allow anyone to confidently use the common volatile agents with patients of any age without any guesswork or the need for superhuman memory. The motivation for developing a convenient graphic version arose from my wanting a paper-equivalent for use when working at another hospital, since I then had no access to my own real-time computer version based in the thoracic theatre at the City Hospital.

29[http://www.nickalls.org/dick/xenon/rwdXenon.html#workstation-mac](http://www.nickalls.org/dick/xenon/rwdXenon.html#workstation-mac)
Figure 9.1:
Age-related iso-MAC curves drawn using the data of Mapleson (1996). The dots on the iso-MAC curves are to help alignment. The left-hand ordinate scale indicates the end-expired isoflurane concentration when using an oxygen/air mixture. The two right-hand ordinate scales indicate the end-expired isoflurane concentration when using nitrous oxide 50 % and 67 % in oxygen. The vertical shifts for the nitrous oxide 50 % and 67 % scales are 0·56 and 0·75 respectively. For a given age and MAC the associated end-expired isoflurane concentration is read from the appropriate ordinate scale. For example, a MAC of 1·2 for a 60-year old patient using isoflurane and nitrous oxide 67 % in oxygen requires an end-expired isoflurane concentration of approximately 0·5 %.
Figure 9.2:
Figure 9.3:
Figure 9.4:
An example of one of the new age and temperature-corrected MAC charts (see Section 9.4.3 for details). A Perl program prompts the user for agent name and patient age and then prints the chart out in the operating theatre.
9.4.3 Temperature corrected MAC

It is well established that MAC decreases as body temperature decreases. In fact even John Snow was aware of the influence of temperature, as the following extract shows.

As the narcotism of frogs, by vapour too much diluted to affect animals of warm blood, depends merely on their temperature, it follows that by warming them, they ought to be put into the same condition, in this respect, as the higher classes of animals; and although I have not raised their temperature to the same degree, I have found that as it is increased, they cease to be affected by dilute vapour that would narcotise them at a lower temperature.

Snow (1848a)

However, it is less well known that (a) MAC decreases linearly with core temperature fall (approximately 2–5 % reduction in MAC per degree centigrade from 37°C), and (b) the rate of MAC decrease with temperature fall is considerably more for vapours than for gases; for example the change in MAC /°C for halothane and cyclopropane in dogs is 5·3 % and 2 % respectively (Eger, Saidman, and Brandstater 1965b; Eger 1974). In humans the linear rate of fall of MAC with temperature in the clinical range is approximately 5 % per degree centigrade for isoflurane, sevoflurane and desflurane, while that for nitrous oxide shows essentially no change (Eger 2001).

These findings can be used to combine both age and temperature correction for MAC in a single chart—most easily done by creating a separate chart for each year of age—as shown in Figure 9.4. The additional functionality of such a chart is particularly useful, since the end-tidal agent requirement is most likely to be underestimated in young patients with a pyrexia. In practice, it is a simple matter to create and print this combined age and temperature-corrected iso-MAC chart for a specific patient on demand in the operating theatre.

For example, suppose we wish to deliver 1·2 MAC to a 15 year-old patient with a temperature of 39°C. The Datex AS/3 and S/5 monitors show a value of 1·2 MAC for an end-tidal sevoflurane concentration (in air) of approximately 2·2 %, whereas the age/temperature correction chart (see Figure 9.4) indicates that to achieve the same MAC value in this patient (age 15 yrs; temp 39°C) actually requires an end-tidal value of 2·8 % (i.e., a 27 % increase compared with the Datex displayed value).

9.4.4 Dosage and MAC correction

Awareness

The problem of awareness and the need for research in this area is often highlighted (Bergman et al. 2002, Guidry 2005, Leslie and Davidson 2010) and, as one might expect, data gathered by automatic anaesthesia management systems (AIMS) has been particularly

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30 This temperature work relates to chloroform—see his Experiment 16 (Snow 1848a).
31 I have written a Perl program for this which is freely available. This program and separate charts for the ages 0–120 yrs are available on the thoracic CD-ROM in PDF format.
useful in this regard. For example, using archived AIMS data, Driscoll et al. (2007) were not only able to establish volatile agent underdosing as the cause of awareness in three cases, but were also able to show that the clinicians’ manual component of the relevant parts of the anaesthesia records were unreliable and failed to reflect events accurately.

More recently, the role of underdosing as a cause of awareness during anaesthesia has been highlighted in two large studies (Ghoneim et al. 2009, Xu et al. 2009), both of which found that awareness was associated with reduced drug dosage, being younger, and with non-volatile anaesthetic techniques. Similarly, Kent (2010) found that the two main causes of awareness in the Closed Claims database were light anaesthesia and anaesthetic delivery problems.

Unfortunately EEG depth of consciousness monitoring techniques (e.g., BIS) are still problematic and unreliable (Mychaskiw et al. 2001, Rampersad and Mulroy 2005). Furthermore, in spite of several well publicised large neuromonitoring trials and surveys (Ekman et al. 2004, Myles et al. 2004; Sebel et al. 1997; Sebel et al. 2004; Avidan et al. 2008) there are still no data to suggest, in those cases where volatile agents are used, that neuromonitoring offers any advantage with regard to preventing awareness, over the rigorous implementation of ‘corrected’ MAC monitoring. Indeed, it is significant that the study of 20,000 patients by Sebel et al. (2004) actually showed that the BIS-monitored cohort had a higher incidence of awareness (0·18%) than the control cohort (0·1%) (McCulloch 2005). In the BIS/MAC study by Avidan et al. (2008) the authors concluded that “...Our findings do not support routine BIS monitoring as part of standard practice.” Indeed, the associated editorial by Orser (2008) expressed concern regarding BIS-like devices, as follows.

... the delegation of critical elements of patient care to a “black box” approach, in which decisive factors are under proprietary control, must be avoided.”

Since the MAC paradigm has been so successful (White, 2003), and because there are no known convincingly documented cases of awareness which are not associated with possible underdosing, it was recently suggested (Nickalls and Mahajan 2010)

... that the time has come to reformulate the concept and adopt a new and pragmatic working premis, namely, that all cases of awareness are due to underdosing unless there is convincing verifiable information to the contrary. ... We ... must confront the problem of underdosing by putting in place systems which we can have confidence in to deliver an adequate dose, implementing the latest alarms (Umesh 2009), algorithms (Mashour, Esaki Vandervest et al. 2009), and corrections for age (Mapleson 1996, Nickalls and Mapleson 2003, Eger 2001), temperature (Eger 2001), and so forth as they come available.

In practice, however, there are studies showing a very low incidence of awareness even without using brain function monitoring (Pollard et al. 2007), and when volatile agents are used the end-tidal MAC approach is still the most reliable method for avoiding awareness,

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and is recommended by the Royal College of Anaesthetists (see RCOA 2006). Indeed, anaesthetists can easily be made even more aware of the current MAC status simply by using a real-time colour-coded dial-display of corrected MAC (see Figure 9.5).

![Figure 9.5:](image)

**Left:** Example of the real-time age-corrected MAC-widget displayed by the author’s open-source anaesthesia workstation software \(^a\) interfaced to the Datex S/5 monitor. If the corrected MAC is too low (as shown in this case—total \(\text{MAC} \approx 0.7\)) then, in addition to sounding an audible alarm, the dial of the MAC-widget turns red.

**Right:** Screenshot showing the MAC widget displaying a white dial (corrected MAC in the normal range). The MAC-widget software can easily be run on a laptop interfaced to an anaesthesia monitor.

\(^a\)© Nickalls RWD, Dales S and Nice AK (1996–2011).

What is the minimum MAC multiple which avoids awareness? 1 MAC has long been regarded as a significant boundary since isoflurane at 1 MAC was shown to prevent implicit memory during surgery (Dwyer *et al.* 1992). More recently this problem was addressed by Hardman and Aitkenhead (2005), who stated that a stable end-tidal value greater than 1 MAC makes awareness extremely unlikely, as follows.

Risk of awareness correlates with depth of anaesthesia. . . . Fortunately, clinical investigations have shown a reasonably reliable association between recall and MAC; patients exhaling more than 0.8 MAC are unlikely to recall intraoperative events, and spontaneous recall is virtually eliminated if > 1 MAC is exhaled, except after a sudden increase in inspired concentration.

Hardman and Aitkenhead (2005)

That the end-tidal agent concentration should be maintained \(\geq 1 \text{MAC}_{\text{age}}\) in order to reliably avoid awareness is consistent with a recent fMRI finding by Kerssens *et al.* (2005), namely that while auditory activation in a group of 6 subjects (mean age 23 years) was detected
when breathing 1% end-tidal sevoflurane in oxygen/air (0.5 MAC$_{age}$), such activation was absent when breathing 2% end-tidal sevoflurane (i.e., when breathing 1 MAC, since MAC$_{23}$ = 2%).

It is significant, therefore, that in the BIS/MAC study by Avidan et al. (2008) the end-tidal agent concentration was less than 0.7 MAC in three of the four cases of definite awareness, and in seven of the nine cases of possible awareness. Thus the lower acceptable limit of 0.5 MAC suggested by Eger and Sonner (2005), Myles (2007) would seem to be far too low to reliably prevent awareness. Consequently the values suggested by Hardman and Aitkenhead (2005)—see above—particularly when age and temperature corrected, would seem to be the best current advice.

Note that the MAC value displayed by monitors having no age and temperature correction is most likely to underestimate the true MAC requirement in young patients with a high temperature (Section 9.4.3) and (possibly) with red hair (Liem et al. 2004). Indeed, it may be significant, therefore, that the two patients with documented awareness during BIS monitoring with sevoflurane reported by Ekman et al. (2004) were quite young (aged 16 and 22 years). Unfortunately the details of the aware patients who received volatile agents in the non-BIS group were not given.

Research

In all work relating to depth of anaesthesia or awareness, it is essential to collect accurate real-time end-tidal anaesthetic gas (ETAG) concentrations and core temperatures using automated anaesthesia record keeping (AARK) equipment, drugs and doses used, as well as patient age, height, gender and hair-colour. The presented awareness-related data must be sufficient to enable readers to calculate the corrected MAC for each patient; consequently the observed ETAG concentrations for each patient should always be presented. Where MAC corrections have been applied, the literature source of the corrections used must be indicated. Furthermore, mean age-corrected values need to be correctly determined; for example, the age correction for MAC is non-linear and hence the mean MAC value for a group must be derived from the corrected MAC of each individual subject/patient.

In view of the importance of determining minimum MAC values for reliably preventing awareness, journal editors should ensure that awareness-related MAC data presented in journal articles are sufficient to allow readers to determine the corrected MAC values for each patient (Nickalls and Mahajan 2010).

Data sharing

Ideally all cases of inadvertent awareness should be documented, and archived, together with all the anaesthesia data associated with such cases (including all machine-automated end-tidal data) and placed in the public domain. Such a database could then form the basis of research, and may therefore, help “...recognise those few cases which may suggest either that the accepted dosage threshold should be raised or, perhaps, a significant pharmacogenetic difference” (Nickalls and Mahajan 2010).
However, although original data documented in journals is still often difficult to access from the authors or organisation (Wicherts et al. 2006, Anon 2006, Kaiser 2008), fortunately the climate of opinion is now strongly in favour of data-sharing, with funding organisations increasingly stipulating that authors place their data in open-access repositories within a set period after publication in peer-reviewed journals (Short 2007, Wadman 2009). Nevertheless, we should continue to press for even more safeguards; for example, the adoption by anesthesia journals of an authorship policy specifying the preservation and sharing of original data (Anon 2009a). Authors and researchers need to embrace the new culture of ‘integrity, access and stewardship’ (Anon 2009b)—not only making the data available, but safe-guarding it as well.

9.4.5 References

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• Eger EI and Sonner JM (2005). How likely is awareness during anesthesia? *Anesthesia and Analgesia;* 100, 1544. [see reply by Bowdle et al. (2005)]

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• Liem EB, Lin C-M, Suleman M, Doufas AG, Veauthier JM, Loyd G and Sessler DI (2004). Anesthetic requirement is increased in redheads. *Anesthesiology*; 101, 279–83. [MAC requirement is increased by 19 %]


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• Short R (2007). Open access will mean peer review becomes “the job of the many, not the select few”. *British Medical Journal*; 334 (17 February), 330.


• Snow J (1848b). On narcotism by the inhalation of vapours (Part I). *London Medical Gazette*; 6NS 33, 850–854 [included in Snow 1848b]


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336NS indicates New Series No 6.


9.5 Arterial line

9.5.1 History

The first direct measurement of arterial blood pressure is generally said to have been in 1733 by Stephen Hales (1677–1761) using a glass tube 9 feet long connected flexibly (using the trachea of a goose) to the femoral and carotid arteries of horses (Comroe 1977, pp. 15–17). Some eight years earlier the physician and mathematician Daniel Bernoulli (1700–1782) was measuring fluid pressure in pipes using a narrow tube (Quinney 1997). In 1828 Jean-Louis Poiseuille (1799–1869) developed this technique further (see also: Zuck 1997) using a U-tube filled with mercury to determine the pressure at various points along the aorta (his later researches into flow in small tubes led to his famous Poiseuille’s Law).

The first clinically useful placement of an arterial catheter for this purpose was developed by Peterson et al. (1949). They described their method as follows.

A small plastic catheter, inserted into an artery through a needle, is left in the artery when the needle is withdrawn. Attached to a capacitance manometer, this technique permits recording for long periods of time without discomfort and allows relatively free mobility of the subject.

Comroe (1977), p. 36

Use of the strain-gauge (Tomlinson, 1876) for transducing arterial pressure was first described a few years earlier by Lambert and Wood (1947). See letter by Kannan (2005) for recent use of ultrasound to facilitate arterial line placement.


CHAPTER 9. SUPPORTING TECHNOLOGIES

9.5.2 Anatomy

An extensive collateral network of superficial and deep palmar arches normally connects the radial and ulna arteries in the hand. However, in 58% of patients the palmar arches are incomplete, and of these ‘incomplete’ cases approximately 4% will suffer significant vascular insufficiency if the radial artery is removed or occluded (Cable, Mullany & Schaff 1999; Lippert H and Pabst R 1985) — see Allen test below.

Riekkinen HV, Karkola KO and Kankainen A (2003). The radial artery is larger than the ulna. *Annals of Thoracic Surgery*, 75, 882–884. [mean internal diameter is 3·1 mm (range: 2–4 mm)]

9.5.3 Allen test

Edgar V Allen (1900–1961) was a physician at the Mayo Clinic, and co-author of a textbook on vascular medicine (Allen, Barker and Hines 1946). He described a simple test (the so-called Allen test) to reveal ulnar or radial artery occlusion at the wrist (Allen 1929). His description with respect to the ulnar artery is as follows.

If obstruction of the ulnar artery is suspected, the radial arteries are located by their pulsations; the examiner places one thumb lightly over each radial, with the four fingers of each hand behind the patient’s wrist, thus holding the wrist lightly between the thumb and fingers. The patient closes his hands as tightly as possible for a period of 1 minute in order to squeeze the blood out of the hand; the examiner compresses each wrist between the thumb and fingers, thus occluding the radial arteries; the patient quickly extends his fingers partially while compression of the radial arteries is maintained by the examiner. The return of color to the hand and fingers is noted. In individuals with an intact arterial tree the pallor is quickly replaced by rubor of a degree higher than normal.

Cable, Mullany & Schaff (1999)

Before ‘harvesting’ or even cannulating the radial or ulnar artery, the adequacy of collateral vessels should be assessed by an Allen test (after EV Allen 1929), to determine if they alone can adequately supply the hand. If a vessel fails the Allen test, then the other vessel certainly should not be harvested, and probably ought not to be cannulated either.

Modified Allen test

Erroneous results can arise if the test is not performed correctly (Greenhow 1972). Note that the wrist must not be hyperextended as this can cause vessel occlusion and invalidate the test (Fuhrman et al. 1992). The slightly more controlled so-called ‘modified’ Allen test (Vaghadia et al. 1988) is now generally used (compressing both arteries simultaneously), in conjunction with the following ‘pass’ or ‘fail’ times.

[both] the ulnar and radial arteries are compressed at the wrist for ≥ 30 secs to induce hand ischaemia, while the hand is drained of blood by tight clenching. The test vessel is then released and the time to adequate perfusion of the tips of the fingers and thumb noted. The vessel is said to pass or fail the test as follows: pass (< 5 secs); equivocal (6–10 secs); fail (> 10 secs)

Royse et al. (1999)

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34The radial artery is sometimes removed (known as ‘harvesting’) for use in coronary-artery or temporal-artery bypass procedures—see Royse et al. (1999).
Note that the vessels should actually be compressed just proximal to the point where the tip of the cannula is expected to lie, in order to detect essential collateral branches distal to the likely cannula tip position (Gandhi and Reynolds 1983). Plethysmography is more sensitive than either the Allen test or pulse-oximetry (Fuhrman et al. 1992).


35 Buerger’s disease.
36 Emergency Department
9.5.4 Systolic pressure variation

Real-time measurement of systolic-diastolic pressure variation with respect to spontaneous ventilation or with IPPV may be useful as an index of hypovolaemia, either via pulse oximetry waveform variation or via direct arterial measurement.


9.5.5 Complications

Complications from arterial cannulation are rare, but therapeutic vasodilation in the form of stellate ganglion block (Gyanendra et al. 1998), or use of intra-arterial phentolamine (Burrell 1977) or papaverine is sometimes required.

- Burrell AR (1977). Treatment of ischaemia following radial artery cannulation. *Anaesthesia and Intensive Care*; 5, 388. [used intra-arterial phentolamine (diluted to 0.5 mg/ml) and gave a 3 ml bolus, followed a while later by a 5 ml bolus.].
9.6 Central venous catheter

9.6.1 History

The development of intravascular catheters and the techniques for inserting them has resulted in important advances both diagnostically (angiography) and therapeutically (CVP & Hickman lines; angioplasty).

Werner Forssmann

In the 1920s Werner Forssmann (1904–1979), then a surgical resident, was looking for safer ways of delivering cardio-active drugs to the heart (i.e., other than via direct needle injection), and eventually he hit upon the idea of using a long IV catheter. In 1929, when he was only 26, Forssmann tested this idea in a Berlin hospital, by inserting a long urinary catheter via the antecubital fossa into his own right atrium and confirmed its intra-cardiac position radiologically. He went on to use this method for injecting intra-cardiac contrast in animals, paving the way for diagnostic cardiac angiography, for which he was awarded the Nobel Prize in 1956, together with Cournand (1895–1988) & Richards (1895–1973). The following translation of part of Fossmann’s original article (Forssmann, 1929) is by Luft (1994).

In cases of shock, such as those engendered by sudden cardiac standstill, or during anesthetic emergencies and poisonings, it may be desirable to deliver medications directly to the heart itself. . . . Nevertheless intracardiac puncture is a dangerous procedure for several reasons, including injury to the coronary arteries and its branches, pericardial tamponade, injury to the diaphragm, and pneumothorax. . . . For these reasons I considered a new method to approach the heart in a less dangerous fashion, namely the catheterisation of the right heart from the venous system.

Experiments on a cadaver were productive. I was able to catheterize any vein in the antecubital fossa and was able to regularly reach the right ventricle. . . . I next undertook experiments on a living subject, namely on myself. I first convinced a colleague to puncture a vein in my right antecubital fossa with a large needle. I next advanced a well-oiled ureteral catheter size 4 Charriere in diameter through the needle into the vein. The catheter allowed itself to be advanced with trivial ease to 35 cm. Because my friend objected to our proceeding with these experiments further, we broke them off even though I felt perfectly well. One week later I tried again alone. I anesthetized my own left antecubital fossa and because I was not able to manipulate the needle by myself I constructed a “cut-down” and advanced the catheter along its full 65 cm length. From surface estimates, I reasoned that the catheter tip would be at the level of the heart.

I documented the position of the catheter with roentgenograms that I obtained by standing in front of the fluoroscope while observing the catheter in a mirror held by a nurse. In conclusion, I would like to point out the utility of this technique in providing new opportunities to research the metabolic activities and actions of the heart.

Forssmann (1929) [From: Luft (1994)]
André Cournand and Dickinson Richards

Cournand and Richards extended Forssmann’s intravascular catheter concept and developed long single and double-lumen catheters which allowed them to sample blood and pressures from the right heart and pulmonary artery (c. 1940s). They also determined approximate left atrial pressures by wedging the catheters by pushing them as far as they would go (i.e., not balloon-occlusion wedge pressures as determined by Swan and Ganz in 1967). They studied cardio-pulmonary physiology and patho-physiology, and showed that hypoxia, sufficient to make the arterial oxygen saturation less than 80 %, resulted in significant pulmonary vasoconstriction and a rise in pulmonary artery pressure (Cournand 1956; Richards 1956). Interestingly, they also showed that an infusion of acetylcholine (0.5 mg/min) into the pulmonary artery reversed the pulmonary vasoconstriction while not affecting the systemic blood pressure (Harris et al. 1956; Cournand 1956).

Sven-Ivar Seldinger

Radiologists often need to insert long large-diameter catheters into arteries in order to inject contrast into distant vessels. In the early 1950s, however, the two existing techniques had significant shortcomings. For example, the catheter-through-needle technique was associated with a significant leak at the vessel entry point (catheter smaller than needle), and the long narrow catheters made it difficult to inject contrast fast enough to be effective. The catheter-over-needle technique was only feasible with quite short catheters (since the needle did not bend, and long needles were difficult to manipulate safely).

In 1952 Sven-Ivar Seldinger (1921–1998) a Swedish radiologist at the Karolinska Hospital, Stockholm overcame these difficulties by developing his catheter-over-guidewire technique (Seldinger 1953; Seldinger 1987; Higgs et al. 2005; Greitz 1999). He actually used a guidewire with a straight flexible tip. Seldinger described the process of development as follows.

However, rightly or not, some people considered the procedure [translumbar aortography] hazardous and searched for a technique where a catheter could be inserted via a peripheral artery. Surgical cut down methods had been reported . . . and Bierman et al. (1951) . . . suggested a percutaneous technique in which a catheter was inserted through a puncture instrument into the femoral artery and advanced to the aorta. The catheter had to be wide enough to permit a very rapid injection. If not, the contrast medium would be so diluted by the voluminous aortic bloodflow that diagnostic angiographs would not be obtained. In turn a very wide-bore puncture instrument, with consequent risk of trauma, was required.

Thus there was obviously a need for an improved percutaneous method for aortography, and one of the requirements to the solution was an increased bore of the catheter. . . . There existed a “puncture equipment” named after Cournand, consisting of an inner sharp needle in an outer blunt cannula, the edge exceeding the cannula by one or two mm. One alternative was to use a flexible catheter instead of the cannula, but it would certainly be tricky to handle an inner needle, half a meter or more long. I avoided this
trouble by cutting a side hole on a polythene catheter at such a level that a cutting needle of convenient length, when inserted through it, exceeded the tip of the catheter by one or two mm. After some moulding of the catheter and a minute incision in the skin, this instrument could be inserted into the artery by percutaneous puncture. Some obvious disadvantages were inherent in this technique. For instance, the thin-walled catheters were so flexible that, sometimes it was impossible to advance them further into the vessel. This difficulty could often be overcome. When intravascular position was obtained, the needle could be withdrawn from the side hole and replaced by a semi-flexible metal wire which was introduced through the entire length of the catheter to support it.

Now! After an unsuccessful attempt to use this technique I found myself, disappointed and sad, with three objects in my hand—a needle, a wire and a catheter—and, in a split second, I realised in what sequence I should use them: needle in—wire in—needle off—catheter on wire—catheter in—catheter advance—wire off.

I have been asked how this idea turned up and I can quote Phokion, the Greek: “I had a severe attack of common sense.”

The tools could not be less complicated; they could be found among the instruments of any hospital and, if necessary, could be completed at the nearest ironmonger’s. Any handy person could use them.

With the ‘beginner’s luck’ the first angiography performed with this technique was a success: a subclavian arteriography, with one single exposure, the catheter introduced through the brachial artery after puncture at the cubital level, which revealed a parathyroid adenoma, unsuccessfully searched for by the surgeon in the mediastinum,

With my permission, the Head of the Department, Knut Lindblom, reported on the technique at the Radiological Congress of Northern Europe which took place in Helsinki one week later, in June 1952.

Seldinger (1987)

The January 1984 issue of the American Journal of Roentgenology (volume 142) celebrated the 30th anniversary of the Seldinger Technique with a series of articles on Seldinger. The article by Doby (1984) gives an excellent historical overview, and includes some detailed sketches by Seldinger himself relating to his development of the technique.

Stanley Baum and Herbert Abrams

A not uncommon problem associated with the straight guidewire, particularly when cannulating the femoral artery, was failure to advance easily. This problem was largely overcome in 1964 by Baum and Abrams’ development of the J-tipped catheter which is threaded over the guidewire (Baum and Abrams 1964). Once the catheter has been

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38See their web site at http://www.ajronline.org/
39The main articles are listed in the references.
positioned above the obstruction then the catheter is changed (by reinserting the guidewire) for a special angiography catheter.

Charles Dotter

At approximately the same time the American radiologist Charles Dotter (1920–1985), widely regarded as the father of interventional radiology, was beginning to lay the foundations of this new speciality at the Oregon Health State University, in conjunction with his student Melvin Judkins.

In 1963 Dotter inadvertently unblocked an occluded right iliac artery while passing a catheter through it in order to reach the aorta for an abdominal aortogram, and realised that intravascular catheterisation can be used therapeutically as well as diagnostically. On 16 January 1964, Dotter, together with Judkins, performed the first deliberate dilation of an arterial obstruction, and thereafter developed the tools and techniques for what is now known as transluminal angioplasty (Payne 2001). Dotter also developed the first safety J-tipped guidewire (Judkins et al. 1967), flow-guided catheter, an intravascular biopsy catheter, and intravascular coils which were the forerunner of expandable stents (http://www.ohsu.edu/dotter/ctdotter.htm).

PICC catheters—Broviac JW and Hickman

With the advent of intensive care, intravenous nutrition and chemotherapy central catheters were increasingly used for long periods of time, leading to significant catheter-related infections. This prompted engineers to address design and materials issues, leading to new long-term so-called PICC catheters, first by Broviac et al. (1973) and later by Hickman et al. (1979). Special valved catheters (Croshong catheter) were developed by Bard Access Systems.

9.6.2 References

- Cournand AF (1956). Control of the pulmonary circulation in man with some remarks on methodology. (Nobel Lecture)

40 see http://www.ptca.org/nv/history.html, and also http://www.ohsu.edu/dotter/
41 PICC — Peripherally Inserted Central Catheter.
• Forsmann W (1929). Catheterization of the right heart. Klinische Wochenschrift; 8 (No. 45), 2085–2087. [from Luft 1994]


• Richards DW (1956). The contribution of right heart catheterization to physiology and medicine, with some observations on the pathophysiology of pulmonary heart disease. (Nobel Lecture)


• (1984) — special ‘Seldinger’ issue of the American Journal of Roentgenology; 142 (Jan):
  – The Seldinger technique. American Journal of Roentgenology; 142 (Jan), 5–7. [a reproduction of Seldinger’s original article]
  – Testimonials to Seldinger. American Journal of Roentgenology; 142 (Jan), 8–11. [reflections by Dotter CT, Grainger RG, Nordenström B, Abrams HL, and Athanasousis CA]
9.6.3 Optimum position

The current view regarding the optimum location of the tip of a CVP-catheter is driven by the need to avoid the possibility of the catheter migrating into the pericardium. Consequently the tip should be above the pericardial reflection on to the SVC (Chalkiadis and Goucke 1998), which is generally held to be at the level of the carina (T4–T5; sternal angle)—i.e., above the left and right atria. Ryu et al. (2007) give a simple landmark-based method for safely positioning the tip of the CVP line in relation to the carina. Several articles have appeared recently describing the use of ultrasound to facilitate CVP-line placement, including a good editorial by Scott (2004).

Techniques for correcting/relocating subclavian and internal-jugular catheters which have taken an aberrant course are addressed by Pattnaik and Bodra (1999); they highlight an article by Kayal et al. (1989) who used ultrasound while flushing with saline to detect when the tip of the catheter is in the correct vessel. Pattnaik and Bodra (1999) suggest listening with a stethoscope is useful. An alternative approach to the ‘aberrant catheter’ problem, might be to consider placing a new J-wire into the same vein via the proximal lumen (hopefully in the internal jugular vein), removing the misplaced CVP line and then railroading a new one—with luck the new wire will be in a better location (one could check the new wire position with an x-ray first perhaps).

Occasionally a CVP line inserted via the left IJ vein will go down the left internal mammary vein; quite how this happens is not clear since the curved tip of the J-wire should prevent the wire from going down a small vessel. The distal lumen in such cases is typically associated with difficult aspiration and poor CVP waveform. Since redirecting a misplaced CVP line can be difficult, consider monitoring the CVP via one of the more proximal lumens—withdrawling the line slightly if necessary—until you see a good CVP waveform. Consequently, always X-ray a left IJ line before considering railroading a Swan-sheath over it. For information and video clips relating to CVP insertion technique see the Clinical Cases web-site (http://clinicalcases.org/).

9.6.4 Anatomy

- Davidson A, Blumgard C, Paes ML and Enever G (1993). Posture and internal jugular vein size studied with the ‘Siterite’ ultrasound device. Br. J. Anaesth.; 71, 771P. [November issue] [gives a useful table of depth and diameter of the vein for various amounts of head tilt. My own working of their data gives the mean depth of the middle of the vein as 1-64 cm, which is equivalent to a distance of 2.3 cm at 45 degrees to the skin]

I have not tried this as yet, but it seems as though it ought to work.
9.6.5  Position of CVP tip


- Chalkiadis GA and Goucke CR (1998). Depth of central venous catheter insertion in adults: an audit and assessment of a technique to improve tip position. *Anaesthesia and Intensive Care* 26, 61–66. [they used the subclavian method—their tailored technique (8 cm distal from the tip of needle) gave a mean distance from the skin of 13·2 cm (range: 11·5–15 cms; n=73)]


9.6.6 General

- Chalkiadis GA and Goucke CR (1998). Depth of central venous catheter insertion in adults: an audit and assessment of a technique to improve tip position. *Anaesthesia and Intensive Care; 26*, 61–66. [they used the subclavian method—their tailored technique (8 cm distal from the tip of needle) gave a mean distance from the skin of 13.2 cm (range: 11.5–15 cms; n=73)]


- Kitagawa N et al. (2004). Proper shoulder position for subclavian venepuncture. *Anesthesiology; 101*, 1306–1312. [evidence from CT studies suggests that best position is with the shoulder pushed inferiorly]


- Nandwani N, Fairfield MC, Krarup K and Thompson J (1997). The effect of laryngeal mask airway insertion on the position of the internal jugular vein. *Anaesthesia; 52*, 77–83. [no lateral movement, but perhaps some slight anterior movement 0.6–1.1 cm, mean 0.8 cm]


- Pattnaik SK and Bodra R (1999). Another ‘whoosh’ test. *Anaesthesia; 54*, 1224–1225. [they describe gradually withdrawing the the malpositioned central line while listening for the disappearance of the distal ‘whoosh’ sound (caused by flushing it with saline) with a stethoscope over the vein. Also list other useful references on this theme (6 refs)].


• Stickle BR and McFarlane H (1997). Prediction of a small internal jugular vein by external jugular vein diameter. *Anaesthesia*, 52, 220–222. [if the external jugular vein is greater than 7 mm diam, then the internal jugular vein is likely to have a diameter less than 7 mm, and so may be difficult to find]

• Tripathi M, Dubey PK and Ambesh SP (2005). Direction of the J-tip of the guidewire in Seldinger technique is a significant factor in misplacement of subclavian vein catheter: a randomised controlled study. *Anesthesia and Analgesia*; 100, 21–24.


### 9.6.7 Ultrasound guided


• Habib FA and McKenney MG (2004). Surgeon-performed ultrasound in the ICU setting. *Surgical Clinics of North America*; 84, 1151–1179. [see section on CVP line placement 1165–1166, with screen images]

• Hall AP and Russell WC (2005). Towards safer central venous access: ultrasound guidance and sound advice. *Anaesthesia*; 60, 1–4. [see also correspondence from Reavley P (2005)]


### 9.6.8 External jugular vein

If the external jugular vein distends on head-down position, then a Venflon in this site adequately reflects CVP providing the chest is not open. Placing a central catheter via this route has a high failure rate.


### 9.6.9 Axillary vein


### 9.6.10 Femoral vein

There are many papers in the literature showing that CVP is accurately reflected by inferior vena cava and common iliac venous pressure measurements in supine patients (both adult and paediatric), providing the transducer is zeroed at the usual right-atrial level on the mid-axillary line. Measurements of inferior vena cava pressures seem to be approximately 0.5 mm Hg lower than superior vena cava pressures on average, and rarely more than 3 mm Hg different, even in patients with high PEEP or raised mean airway pressures (Desmond 2003). Femoral CVP results may be less accurate in patients with significantly raised intra-abdominal pressure [the references below are from Desmond (2003)].


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I thank Dr Mofolashade Enebeli-Cliffe for drawing my attention to many of these references.
9.6.11 Complications

These are mostly related to air embolism, vessel damage from needle or dilator or kinked guide-wire, introducing the guide-wire outside the vessel, catheter knotting, dysrhythmias, pneumothorax and cardiac tamponade. Unusual anatomy and failure to use ultrasound visualisation appear to be prominent factors in many complications (see Section 9.6.4).

Guide-wire problems

The guide-wire is easily kinked, and once kinked it can not be straightened and can easily damage/tear the vein if introduced into it. A simple test to check for such kinking while trying to advance the dilator (over the wire) is to intermittently check that you can slide the wire back and forth (say, ± 1 cm or so) inside the introducer. Any difficulty in sliding the wire back and forth is a good sign that the wire may have become kinked—in which case withdraw the guide-wire carefully to bring the kink to the skin for inspection.

In my experience, the guide-wire is most easily damaged/kinked when using the femoral approach in fat patients, since in these cases one often has to press the Sonosite probe down quite firmly (and hence distort the subcutaneous tissue) in order to see the vein clearly. The kinking of the guide-wire usually occurs while trying to introduce the dilator through the subcutaneous tissue, since in fat patients the path of the guide-wire here becomes quite curved into a sigmoid shape once the Sonosite probe is removed. In my experience in this setting, it is best to have an assistant replace the Sonosite probe and press down as before (i.e., to straighten the subcutaneous path of the guide-wire) while introducing the dilator.

If a dialysis catheter guide-wire does become kinked in a difficult case, it is often possible to rescue the situation and exchange it safely, since the guidewire will generally still be in the vein even when pulled back slightly to bring the kink to the skin. The idea is to first railroad an ordinary CVP line over the wire and into the vein (i.e., with the kink still showing just above the skin), replace the damaged guide-wire with a new dialysis catheter guide-wire, then remove the CVP line, and then continue with the dialysis line as before.

- Dhanani J, Senthuran S, Olivotto R, Boots RJ and Lipman J (2007). The entrapped central venous catheter. Br. J. Anaesth.; 98, 89–92. [a new catheter pierced an existing catheter in the same vein; interventional radiology used for diagnosis and determination of a removal strategy; the literature is reviewed; 11 refs]

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44 Persistence of the left superior vena cava (LSVC)—which is asymptomatic—is thought to be the most common anomaly of the venous circulation and can be a significant hazard with regard to CVP line placement (see paper by Schummer, Schummer and Gerald (2002) listed in Section 9.6.4).

45 Since the guide-wire is initially introduced via a straight needle, its path to the vein will only remain straight (after removing the needle) while the Sonosite probe is pressing down over the vein.

46 It is therefore, a good idea to introduce plenty of guide-wire into the vein before starting to thread the dilator.


### 9.7 Pulmonary artery catheter

#### 9.7.1 History

**Michael Lategola and Hermann Rahn**

Balloon-flotation pulmonary artery catheters were first used by the American physiologists Lategola and Rahn for pressure recording, blood sampling and vessel occlusion in experiments with dogs (Lategola and Rahn 1953). Since their balloon covered the distal tip they were only able to measure pressures proximal to the occluded vessel. Thermodilution cardiac output was first described in animals the following year (Fegler 1954).

**Ronald Bradley**

The first person to describe the use of a pulmonary-artery catheter in man was Ronald Bradley, a physician at St. Thomas’ Hospital, London (Bradley 1964). He used an extremely narrow catheter (0.63 mm diam) having no balloon, to determine pulmonary artery pressures and waveforms, and later went on to determine thermodilution cardiac output in man using a thermistor-tipped catheter (Branthwaite and Bradley 1968), and suggested the use of PA-diastolic pressure as an index of mean left-atrial pressure (Jenkins, Bradley and Branthwaite 1970). Bradley also wrote an excellent book on the physiology of heart failure (Bradley 1977).

**Harold Swan and William Ganz**

Bradley’s catheters had no balloon and were extremely difficult to position, and so the technique remained clinically impractical until Swan *et al.* (1970) developed the modern
balloon-flotation catheter. Since Swan wished to determine left atrial pressure he arranged that pressures could be measured distal to the balloon. Oddly enough Swan fails to credit Bradley and Branthwaite (1968) with the thermodilution technique in man—instead he credits this to Ganz (Swan 1991).

Swan (1922–2005) graduated from St. Thomas’ Hospital Medical School, London, in 1945, and went on to become the Director of the Division of Cardiology at Cedars-Sinai Medical Center in Los Angeles, California. He has set on record the key early ideas and development of the flow-directed balloon-tipped catheter (Swan 1991; 2005) as follows.

In 1950 as a lecturer in physiology at St. Thomas’ Hospital of the University of London, I had come to know a young medical student, Ronald Bradley, who was completing a bachelor’s degree in physiology. I had noted a paper published in the *Lancet* (Bradley 1964), in which Bradley had claimed that it was possible to catheterize the pulmonary artery for measurement of pressures using an extremely fine Portex tubing . . . and I therefore attempted to place it. . . . In our hands this approach had little success . . .

In the fall of 1967, I had the occasion to take my (then young) children to the beach in Santa Monica. On the previous evening, I had spent a frustrating hour with an extraordinary pleasant but elderly lady in an unsuccessful attempt to place one of Bradley’s catheters. It was a hot Saturday and the sailboats on the water were becalmed. However, approximately half a mile offshore, I noted a boat with a large spinnaker well set and moving through the water at a reasonable velocity. The idea then came to me to put a sail or a parachute on the end of a highly flexible catheter and thereby increase the frequency of passage of the device into the pulmonary artery. I felt convinced that this approach would allow for the rapid and safe placement of a flotation catheter without the use of fluoroscopy and would solve the problem of arrhythmias.

. . . I had been appointed a consultant to the Edwards Laboratories, then a small manufacturing company whose products included the Starr-Edwards heart valve and the Fogarty embolectomy catheter. . . . I brought my concept to the attention of Mr David Chonette and Mr Will Perrie. They had the facilities for extrusion of catheters of different sizes . . . To test the concept, however, they had the ability to manufacture balloons (as for the Fogarty catheter) and suggested that, as a first effort, a double-lumen extruded catheter should be manufactured with one lumen available to inflate a flotation balloon. This proved to be acceptable and they agreed to fabricate five such catheters.

. . . As luck would have it, when the Edwards Laboratories delivered their first catheters, Willie [Ganz] was finishing an experiment with his animal in good condition. I brought the prize catheters to the laboratory and connected the pressure lumen to an appropriate strain gauge manometer. The catheter was then introduced via the exposed jugular vein into the right atrium and, observing with fluoroscopy, the balloon was inflated. It immediately disappeared, and the technician reported no change in the recorded pressure. I immediately assumed an inadequacy of balloon tensile strength and mentally blamed faulty construction by the Edwards Laboratory. However, repeat visualization revealed that the catheter had migrated in one heartbeat through the right heart and was recording the wedge pressure in a distal pulmonary artery. Deflation of the balloon allowed its prompt return to the superior vena cava. . . . Willie Ganz accepted the
responsibility of clearing up the many technical details, but the concept was proved and the new device was born.

... A triple-lumen catheter allowed measurement of simultaneous pressures in the wedge position (the pulmonary occluded pressure) and in the right atrium. With a slight modification, a thermistor was inserted close to the guiding balloon and the thermodilution technique of Willie Ganz (Ganz et al. 1971) for determination of cardiac output was applied.

Swan (1991)

Swan (1922–2005) died on 7th February, 2005, and his last paper (Swan 2005) appeared in the October 2005 issue of *Anesthesiology*. A photograph of Swan can be found on the *Anesthesiology* web site.47


47http://journals.lww.com/anesthesiology/; follow the ‘enhancements index’ link → 2005.

• Jenkins BS, Bradley RD and Branthwaite MA (1970). Evaluation of pulmonary arterial end diastolic pressure as an indirect estimate of left atrial mean pressure. *Circulation*; 42, 75.


• Shaw TJI (1979). The Swan-Ganz pulmonary artery catheter: incidence of complications, with particular references to ventricular dysrhythmias, and their prevention. *Anaesthesia*; 34, 651–656. [Swan’s final paper—includes some interesting historical detail regarding the development of the PA catheter]

• Swan HJC (2005). The pulmonary artery catheter in anesthesia practice. *Anesthesiology*; 103, 890–893. [Swan’s final paper—includes some interesting historical detail regarding the development of the PA catheter]


### 9.7.2 Decline in use

In recent years the use of pulmonary artery catheters has declined somewhat, partially owing to lack of good evidence that it improves outcome (ESCAPE committee 2005; Shah et al. 2005; Hall 2005), and partly owing to new non-invasive cardiac output monitoring devices (e.g., PiCCO, LiDCO, oesophageal doppler).


## 9.8 Computers & information technology

The first anaesthetic machine to incorporate a microprocessor was in 1976 (Katz 2006), and since then computers have progressively influenced anaesthesia delivery and patient safety. One of the next major influences on anaesthesia practice is likely to be related to data processing, particularly in the areas of smart alarms and decision support. While development and take-up in the operating theatre is almost imperceptible just now, the future surely lies in computers offering anaesthetists seriously useful facilities. The initial motivation with regard to data handling lay in automating the anaesthesia record. However, while this technology has been effectively solved for over 15 years (see Kenny 1990), the take-up by anaesthetists in the UK remains almost zero.

### 9.8.1 History of the anaesthesia record

The documentation of events, procedures undertaken, physiological parameters (*vital signs*) which are associated with the process of anaesthesia (for example, in conjunction with surgery or an intensive care setting) is known as the Anaesthesia Record. This record serves two main functions, namely (a) medical (the moment-to-moment drug history and vital-signs serves as a useful practical aid), and (b) medico-legal (the anaesthesia record is a legal document in its own right, setting out the facts as they unfold during an anaesthetic).

**Background**

Effective surgical anaesthesia using inhaled diethyl-ether (“ether”) was first established in 1842 by Crawford Long (1815–1878) in a handful of unpublicised cases. Some four years later in 1846 ether anaesthesia was rediscovered and popularised by William Morton (1819–1868), who gave a public demonstration on 16th October 1846 at the Massachusetts General Hospital (Boston, USA).

Subsequently, John Snow (1813–1858), Joseph Clover (1825–1882), and Mounier (1855) demonstrated the importance of monitoring the pulse and respiration during anaesthesia (Ellis 1995; Rushman, Davies and Atkinson 1996), but it was not until 1894, at the Massachusetts General Hospital, Boston, that surgeons Ernst A Codman (1869–1940) and Harvey Cushing (1869–1939) established the practice of keeping a careful *written* record (on graph paper) of the patient’s pulse and respiration rate during operations—known as the ‘ether chart’ (Beecher 1940; Hirsch and Smith 1986). Apparently this was prompted by a death under anaesthesia in 1893 (Rushman, Davies and Atkinson 1996, p. 128). In 1901 they started including measurements of the arterial blood pressure using the newly described apparatus of Scipione Riva-Rocci (1863–1937) of Turin (Cushing 1902; Cushing 1903; Rushman, Davies and Atkinson 1996, p. 157).
Ralph Waters (1936; 1942) championed and emphasised the importance of written anaesthetic records, and later Noseworthy (1945) produced special cards on which to record anaesthetic details (see Rushman, Davies and Atkinson 1996, p. 111, for an illustration).

**Automation**

An automated anaesthesia record is significantly superior to the usual hand-written record, since it samples data much more frequently and more accurately, and hence it has significant medico-legal advantages regarding the documentation of patient care, particularly during complicated and/or unstable cases.

The first mechanical device capable of printing an anaesthetic record was the *Nargraf* machine of 1930 developed by EI McKessons (Westhorpe 1989), which generated a semi-automated record of inspired oxygen, tidal volume and inspiratory gas pressure.

After this little of real technological significance was developed in the area of anaesthesia monitoring until the 1970s, when advances in chip technology gave rise to clinically useful portable electronic devices for measuring such things as arterial and central venous blood pressure, breath-by-breath concentrations of oxygen, carbon dioxide and inhalational anaesthetics, pulse oximetry, and of course, small computers.

From an interfacing point of view, a very significant and far reaching feature was incorporated into virtually all early medical monitoring devices, namely a specialised serial communications interface known as the RS-232 port.\(^{48}\) Equally significant, therefore, was the decision by IBM to incorporate the RS-232 port into the IBM Personal Computer which appeared in 1981. Fortunately all IBM-compatible PCs since then have also incorporated the RS-232 serial port.

Owing to the widespread use of the RS-232 interface in medical equipment it soon became a relatively easy matter to use a PC to access the numerous measured and derived parameters output by patient monitoring devices, and consequently anaesthetists increasingly explored methods for automating data collection and processing, with a view to developing useful trend displays of measured data, real-time calculation of derived parameters, and hard-copy data printouts.

The RS-232 interface is likely to be replaced at some stage by the Medical Interface Bus (MIB; IEEE-1073). This is a high-tech high-speed medical plug-and-play version of the familiar domestic USB interface, and will greatly facilitate medical device inter-connectivity, largely by allowing the relevant interface software to be more easily standardised.

**Guidelines**

The Royal College of Anaesthetists has published a summary of what data ought to be collected (in addition to the electronic data from the anaesthesia monitors) as part of the

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\(^{48}\)The Electronic Industries Association Recommended Standard 232. In 1986 the prefix RS was superceded by the prefix EIA. In 1988 the Telecommunications Industry Association (TIA) was formed by the merger of the US Telephone Suppliers Association and EIA/ITG, and subsequent documents are therefore prefixed by EIA/TIA. The 1991 revision was EIA/TIA-232-E. (Nickalls and Ramasubramanian 1995).
Anaesthesia Record (Adams 1996), building on the work of Lack et al. (1994). The extent to which these guidelines are actually being met has also been looked at (Smith 1997). The required record set which appears to be emerging, consists of a number of fields within the following general categories: pre-, per- and postoperative information, untoward events and hazard flags.

9.8.2 The anaesthesia workstation

It is clear that computerisation, both in the operating theatre and in ITU, has the potential to free anaesthetists and nurses from much of the work of documentation (e.g., drug doses, procedures, measured parameters etc.), releasing significant amounts of time which is better spent on direct patient care. Anaesthesia Information Management Systems (AIMS) which incorporate sophisticated record-keeping systems clearly offer the advantage of allowing the anaesthetists to concentrate fully on the patient, leading to enhanced vigilance and improved patient care and safety.

Much work has gone into studying the anaesthetists’s workload (Weinger et al. 1997; Byrne, Sellen and Jones 1998; Leedal and Smith 2005). For example, Kennedy et al. (1976) showed that anaesthetists commonly spend 10–15% of their time producing the handwritten record. Similarly, Smith (1997) pointed out that about 10% of the anaesthetists’ time was related to record keeping, and that if this were to increase then this would likely be to the patient’s detriment. A similar study by Wong et al. (2003) showed that an ICU information system reduced the time spent by nurses on documentation by 31%, with the significant benefit being that almost half of the time saved was transferred to patient assessment and direct patient care.

Secondary data processing by anaesthetists in the UK is well behind other countries, with electronic data collection being actively supported by foreign health organisations. For example, in 2001 the ‘summer’ newsletter of the Anesthesia Patient Safety Foundation (APSF) was devoted to Information systems in anaesthesia (Thys, 2001). In 2002 the APSF formally endorsed the use of automated anesthesia information management systems (AIMS) as the following quote indicates.

In this context it is heartening that the … APSF has recently endorsed the use of automated anesthesia information management systems (AIMS): “The Anesthesia Patient Safety Foundation endorses and advocates the use of automated record keeping in the perioperative period and the subsequent retrieval and analysis of that data to improve patient safety.”

Gage (2002)

Anaesthetists urgently need to harness the power of computing technology in a way which can help both in the operating theatre and in the clinic, most likely via some form of anaesthesia workstation. While such systems will probably be commercial, this is not necessarily the only route. Providing anaesthetists take some interest in the details, it is quite possible for useful systems to be developed along the Open Source model, as for
example, the immensely successful Linux operating system, and the excellent software tools \TeX, \LaTeX, Perl and others.

The emphasis for such a workstation needs to be on helping the anaesthetist give a safe anaesthetic during difficult circumstances. It would access data from a range of sources via the Medical Interface Bus (e.g., anaesthesia monitors, HIS) and then process the data in various ways; for example, generating the anaesthesia record, offering smart alarms, decision support and predictive physiological and pharmacokinetic modelling, as well as enabling data export, data storage and emergency communications.

For a long time now, even with a modest PC, it has been a simple matter to access high quality data from anaesthesia monitors (Nickalls and Ramasubramanian 1995; Nickalls 1998, Nickalls, Dales and Nice 2010) and create excellent anaesthesia records offering medico-legal security. These are relatively straightforward to write and get up and running, as, for example, the graphic record shown in Figure 8.1 (page 135) generated by the author’s open-source anaesthesia workstation software. With little additional work a theatre-based PC can also display warnings, equipment status information and value-added parameters; for example, real-time age-corrected MAC (Nickalls and Mapleson 2003), smart diabetes monitoring & management, as well as extensive general and drug information support (see Figure 9.5, page 159).

Although there has been widespread uptake of AIMS technology by anaesthetists, it is clear that the optimum interface design to facilitate easy and intuitive use is difficult to achieve. Interface design must minimise keyboard/mouse entries by the anaesthetists while maximising information display. All too often the user interface is awkward to use with the effect that time is wasted and data collection is incomplete (Driscoll et al., 2007). However, since anaesthesia practice is much the same the world over, it is to be expected that with sufficient computer-engineering research an optimum and intuitive interface will emerge given time. A typical example of current progress in making practical automated anaesthesia records and the involvement of XML is that described by Meyer-Bender et al. (2010). The wider adoption of AIMS technology also has the potential to bring about a significant reduction of intraoperative awareness (Nickalls and Mahajan 2010).

Of course commercial AIMS technology is available and can be extremely useful (for example, the NarKoData system —see Benson et al. 2000, and the Saturn Information System, Dräger—see Driscoll et al. 2007), but some can be far from ideal, and relatively unhelpful in facilitating anaesthesia-related activities, or even generating good quality records. These latter failings largely account for the poor take-up of commercial systems by anaesthetists in the UK. That said, improvements are of course being made all the time.

Computerisation also offers a significant research benefit. For example, in a study by Müller et al. (2002) anaesthetists were able to search the database of their automated anaesthesia record-keeper and establish useful risk factors predictive of subsequent inotropic support requirement following cardio-pulmonary bypass. Driscoll et al. (2007) used AIMS...
data to establish underdosage as the cause of awareness in three patients.

**Databases**

Extracting data from big databases requires a good data dictionary (Sanderson and Monk 2003) as, for example, the currently well advanced SNOMED Clinical Terms program (SNOMED-CT),\(^1\) which is a dynamic health care terminology infrastructure being developed as part of the NHS National Program for Information Technology (NPfIT). A demonstration program can be accessed from the SNOMED-CT home page.

Another NPfIT dictionary database of interest to anaesthetists is the Dictionary of Medicines and Devices (dm+d).\(^2\) This consists of a number of coordinated XML-encoded pharmaceutical databases, which also incorporate the associated SNOMED encoding. Of particular interest to anaesthetists is the Virtual Therapeutic Moiety (VTM) database of approximately 2000 official drug names which are to be used henceforth in all European computer interactions relating to drugs. This list is updated frequently and can be downloaded from the website (password required). This useful list was incorporated into the author’s experimental anaesthesia workstation used in the CHN thoracic theatres.

**The future**

The future holds the exciting prospect of developing sophisticated (and possibly Open Source) anaesthesia workstations giving anaesthetists access to good data displays and trends, sophisticated alarms (smart-alarms), real-time predictive modelling for drugs and physiological parameters, information management and decision-support systems (Sanderson, Watson and Russell 2005; Tarassenko, Hann and Young 2006, Berkenstadt et al. 2006). A possible view of the future was presented recently by John, Peter, Chacko et al. (2009). Finally, we note that since 2010 the NHS has been embracing the Open Source domain—this can only be a good sign for anaesthetists (see [http://www.ehealthopensource.org/](http://www.ehealthopensource.org/)).

### 9.8.3 References


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\(^1\) [http://www.ihtsdo.org/snomed-ct/](http://www.ihtsdo.org/snomed-ct/)

\(^2\) [http://www.dmd.nhs.uk/](http://www.dmd.nhs.uk/)


• Cushing HW (1902). On the avoidance of shock in major amputation by cocainization of large nerve trunks preliminary to their division. With observations on blood pressure changes in surgical cases. Annals of Surgery, 36, 321–345. [from Hirsch & Smith (1986)]


• Fulton JF (1946). Harvey Cushing: a biography. (C Thomas, Springfield, IL, USA).


  [chapters: Anesthesiology national CME program and ASA activities in simulation / Does simulation improve patient safety?: self-efficacy, competence, operational performance, and patient safety / Simulation applications for human factors and systems evaluation / Credentialing and certifying with simulation / Statewide simulation systems: the next step for anesthesiology? / Crew resource management and team training / Simulation: translation to improved team performance / Virtual worlds and team training / Virtual reality simulations / Credentialing and certifying with simulation / Debriefing with good judgment: combining rigorous feedback with genuine inquiry / Integration of standardized patients into simulation ]


• Mounier CCR (1855). *Acad. Sci. Paris; 40*, 530. [from Rushman, Davies and Atkinson (1996)]


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53 Wood Library-Museum of Anesthesiology.


• Noseworthy M (1937). *St. Thomas’s Hospital Reports (London);* **2**, 54. [from Rushman, Davies and Atkinson (1996)]


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CHAPTER 9. SUPPORTING TECHNOLOGIES


• Waters RM (1942). The evolution of anaesthesia. Proceedings of the Mayo Clinic; 17, 40. [from Hallén 1990]


Colophon

... \TeX is potentially the most significant invention in typesetting this century. It introduces a standard language in computer typography and in terms of importance could rank near the introduction of the Gutenberg press.

Gordon Bell (1979)\(^1\)

This booklet was typeset using the 2010 \TeX Live\(^2\) implementation of the open-source\(^3\) \LaTeX\ system\(^4\)\(^5\) on a PC (Mandriva2006-Linux operating system), and printed at 300 dpi from PDF files generated using PDF\LaTeX. The line drawing in Figure 5.1 and the iso-MAC charts were generated using mathsPic\(^6\). Image manipulation was achieved using standard Open Source utilities, including GIMP, DVIPS, fitps.pl, GhostScript, ps2pdf and epstopdf. The text-editor used was KILE 1.6. The index was compiled automatically using the TEX package makeindex.

The ‘hazard’ glyph shown in the margin (used on page 47) was created by the Stanford computer scientist Donald Knuth\(^7\) and featured prominently in Knuth’s \TeX books\(^8\). This delightful and unusual ‘dangerous-bend’ notation has a curious provenance; it was originally the brain-child of a remarkable and prolific group of mathematicians, known collectively as Bourbaki,\(^9\)\(^10\) who used a variant form of it to highlight the trickier mathematical sections in their series of books.

\(^1\)In the Forword to: Knuth, DE (1979). \TeX and \METAFONT, new directions in typesetting. (American Mathematical Society and Digital Press, Stanford).
\(^2\)http://www.tug.org/texlive/
\(^3\)The Open Source initiative defines nine requirements which software must comply with in order for it to be regarded as ‘Open Source’—see http://www.opensource.org/docs/definition.html.
\(^4\)\LaTeX is the de facto standard for the production of scientific documents (http://www.tug.org/).
\(^5\)For an overview of \TeX & \LaTeX see http://www.nickalls.org/dick/links/rwdnLinks.html#tex
\(^6\)http://www.ctan.org/tex-archive/graphics/mathspic/perl/
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