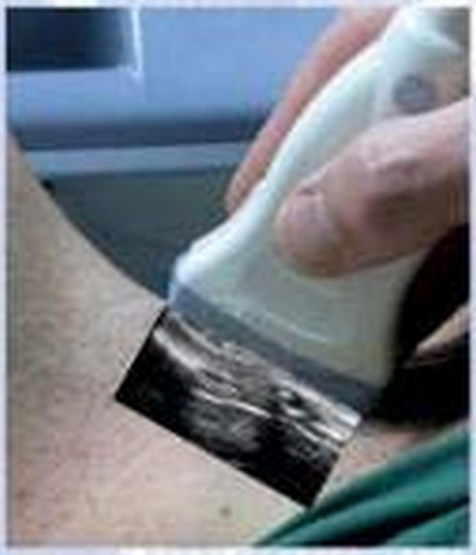
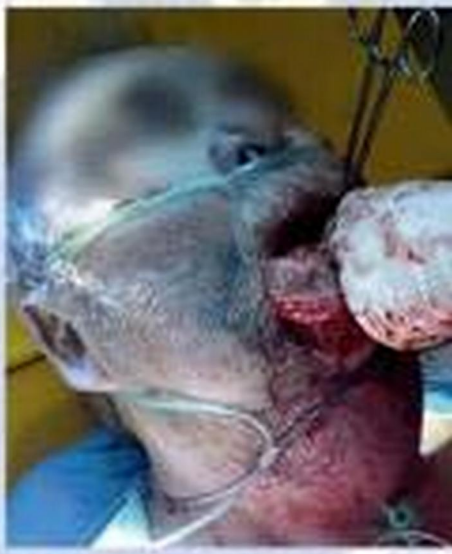
Core Topics in Airway Management

Edited by **Tim Cook** and **Michael Seltz Kristensen**

THIRD EDITION

CAMBRIDGE Medicine

Core Topics in Airway Management

Third Edition

Management of the airway is an important and challenging aspect of many clinicians' work and is a source of complications and litigation.

The new edition of this popular book remains a clear, practical and highly-illustrated guide to all necessary aspects of airway management. The book has been updated throughout, to cover all changes to best practice and clinical management, and provides extensive coverage of the key skills and knowledge required to manage airways in a wide variety of patients and clinical settings. The best of the previous editions has been preserved, whilst new chapters on videolaryngoscopy, awake tracheal intubation, lung separation, airway ultrasonography, airway management in an epidemic and many more have been added.

This is an essential text for anyone who manages the airway including trainees and specialists in anaesthesia, emergency medicine, intensive care medicine, prehospital medicine as well as nurses and other healthcare professionals.

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Foreword

Sixteen years have passed since we began recruiting the contributors to the first edition of *Core Topics in Airway Management* (2005). We are delighted that a third edition has been deemed useful and even more delighted with the editors appointed.

It is not surprising that opinions and techniques have changed, but the extent of the change is remarkable (videolaryngoscopy was covered in two lines in the first edition). It is also true that patients have changed, being often fatter and older and having more co-morbidities.

Problems with the airway are almost uniquely distressing and dangerous, and everybody involved wants to know the best way to ensure safety. We wrote in the first edition that there was 'an uneasy combination of art and science' in management of the airway. The contributors to this edition have done a great deal to solidify the evidence for best practice.

We dedicated the first edition to Dr Archie Brain, so we got something right all those years ago.

Adrian Pearce

Ian Calder

Preface to the Third Edition

The Essence of Airway Management – and Why You Should Read This Book

It is a great honour and pleasure for us to have been asked to take over editing the third edition of this book, so successfully previously edited by Ian Calder and Adrian Pearce. It is now almost 10 years since the last edition of the book was published and it is clearly time for an update.

In this new third edition we have updated only a small number of chapters – most have been completely rewritten to ensure they are up to date and relevant. While a few chapters have been combined we have added several new ones to ensure the book fully covers the range of challenges encountered during modern airway management. There are new full chapters on the epidemiology of airway complications, ultrasonography, videolaryngoscopy, combined techniques, expiratory ventilatory assist, airway management for robotic surgery, during CPR, for the bloody and bleeding airway and during pre-hospital emergency medicine.

We have strived to make this book clinically useful and globally applicable, not too dependent on national strategies or regional cultural or even legal considerations. In order to achieve that almost all chapters are co-authored by two or three individual expert authors with different backgrounds from different countries and often different continents. We are proud that the authors represent institutions from close to 20 different countries including from Europe, North America and Asia. Writing a chapter for a textbook is a labour of love and not one done for reward. We thank every single author for their expertise, knowledge, communication skills and for their patience in enabling us to produce this book.

From the cutting of the umbilical cord and for the rest of a human's life, the airway – from the tip of the nose and mouth to the lungs – must be kept open or within minutes the human will die or suffer irreversible damage. Airway management therefore is the essence of anaesthesia and without getting it right all that follows is arguably futile. The book covers all important topics in airway management and strikes a balance between focussing on the elective patient, routine and specialist settings and emergency airway management. It does not include excessive focus on airway equipment itself but rather emphasises the practicalities of use, suitable techniques and their limitations.

Throughout medicine it is increasingly recognised that understanding equipment and learning techniques is but one part of delivering safe medical care. For that reason, the book starts with several new chapters examining the epidemiology of airway complications, airway assessment both clinical and virtual, airway planning and strategies. The importance of training, human factors/ergonomics and crisis management is mentioned in almost all chapters but each of these topics has a chapter bringing the topic together towards the end of the book.

We hope the book will appeal to and inform everyone who directly manages the airway irrespective of their parent specialty. It is also written for all those who work with airway managers and care for patients who have undergone airway procedures. The book is written with patient safety and comfort as central goals in care.

We hope that readers will understand the broader goals of airway management by a team and become confident in mastering the strategies and techniques described. We hope you will be able to introduce those relevant to your own practice. In the future it will be you who we must trust to manage our airways, and the airways of our loved ones, should it become necessary.

Tim Cook, Bath, UK

Michael Seltz Kristensen, Copenhagen, Denmark

Chapter 1

Anatomy

John Picard

Individual flowers may be pretty. But in a bouquet, it's their relation to each other which makes the arrangement beautiful: context is key. The same is true of topological anatomy: context makes for clinical relevance. This chapter offers a selective account of the functional adult head and neck anatomy as it applies to anaesthetic clinical practice.

Mouth Opening and the Temporomandibular Joint

Cooking and cutlery both evolved after us; while our ancestors lived without tools or open fires, biting hard and opening the mouth wide were both advantageous.

A strong bite and a wide gape may seem to be conflicting ambitions. A firm bite, for instance, depends on a single, fused mandible, and on muscles inserting some way from the joint to gain greater leverage, as in humans. (In snakes, in contrast, each of the two halves of the mandible and the maxilla move independently from the skull and from each other, and their muscles insert close to the relevant joints, to give an enormous gape, but a weak bite.) An adequate gape is nevertheless achieved in most humans by subluxation. When the jaw is closed, the head of the mandible rests in the mandibular fossa in the temporal bone. But as the jaw opens, the head of the mandible is pulled out of the fossa by the lateral pterygoids (Figure 1.1). Rather than turning on its head, the mandible swivels on an axis which runs through the mandibular foramina (i.e. close to the insertion sites of temporalis and masseter).

This shift in the axis of rotation allows both strong bite and wide gape: at the limit of closure, as the molars meet, the jaw is turning on the temporomandibular joint, and masseter and temporalis are working with leverage. But at the jaw's widest opening, it turns about the muscles' insertion sites; they are not so passively stretched, and the bones of the joint do not so impinge on one another.

Overenthusiastic openers of the mouth may sometimes find their jaw becomes stuck in subluxation (during assessment for anaesthesia, for example). The patient is left phonating like a distant gargle, with the mouth wide open; to return the jaw to its joint, it suffices to push firmly on the mandible's molars posteriorly and inferiorly.

Gape may be reduced by abnormal skin around the mouth (e.g. scleroderma), by excessive tone in masseter (e.g. induced by a neighbouring abscess) or by disease in the temporomandibular joint itself (e.g. rheumatoid arthritis).

Mouth opening ability also depends on craniocervical flexion and extension. Head extension facilitates opening. Normal humans extend about 26° from the neutral position at the craniocervical junction to achieve maximal mouth opening. If cervical extension, from the neutral position, is prevented a subject can be expected to lose about one third of their normal interdental distance. Patients with poor craniocervical extension therefore suffer a 'double whammy' in terms of airway management.

The Oral Cavity and Oropharynx

The oral cavity is dominated by the tongue, and for anaesthetists, little else counts but its size. It may be swollen acutely (as in angioneurotic oedema) but is also susceptible to disproportionate enlargement by trisomy 21, myxoedema, acromegaly, tumours and glycogen storage diseases, among others.

Angioneurotic oedema can cause such swelling as to fill the entire pharynx, preventing both nasal and mouth breathing and making a front of neck airway necessary for survival. Less dramatically, a large tongue (relative to the submandibular space) can hinder direct laryngoscopy. That is, manoeuvred with reasonable force, the laryngoscope blade should squeeze the posterior tongue so as to achieve a direct view of the glottis. If the tongue is too large, or the jaw

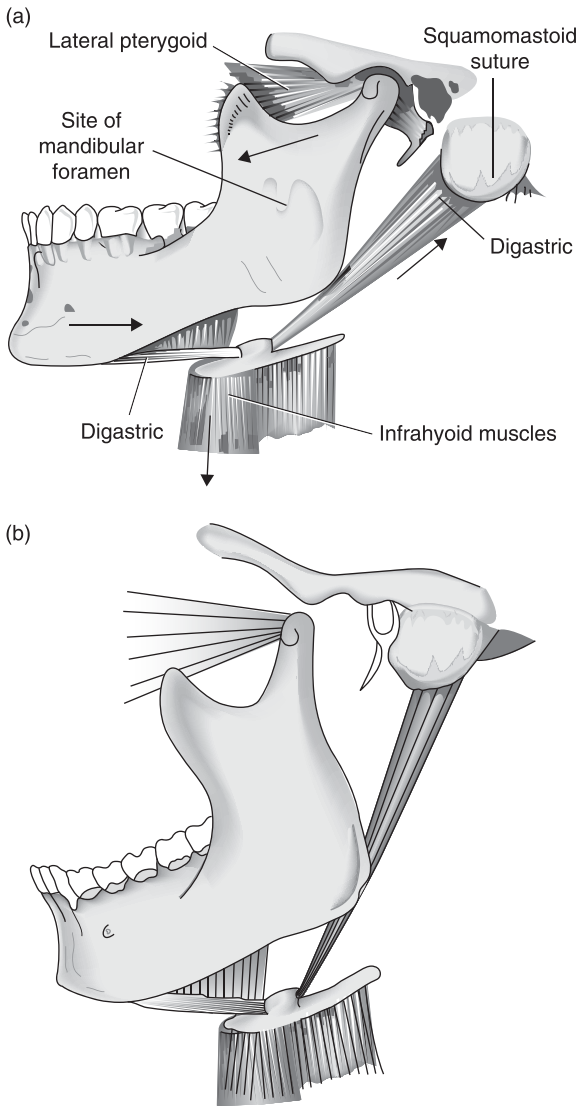


Figure 1.1 (a) Mandible and muscle actions. (b) Mandibular movement for opening the mouth wide.

hypotrophied, it may not be possible directly to see the glottis over the compressed tongue.

Within the oral cavity, the tongue is like a thrust stage in a theatre. It is surrounded by two tiers of teeth (stalls and royal circle), and a series of wings and flies (Figure 1.2).

Each tooth consists of calcified dentine, cementum and enamel surrounding a cavity filled (if the tooth is alive) with vessels and nerves. Each tooth is held in its socket in the jaw by a periodontal ligament. If a tooth is inadvertently knocked out, the sooner it is returned to its socket the better. If the root is clean, the tooth can simply be put back in; if dirty, the root should first be rinsed with saline or whole milk. A dentist will then be able to splint the tooth in place. If a displaced tooth cannot be immediately replaced, whole milk is the best storage medium; a dental cavity exposed too long to saline, or worse water, dies. Calcification of the periodontal ligament is then inevitable, and the tooth will become brittle and discoloured, and may fracture, loosen or fall out again.

The stage's side wings are formed by mucosal folds running over palatoglossal and palatopharyngeal muscles (from anterior posteriorly). Between the two folds on each side lie the tonsils (which may be invisible in adults, but in children may be so large as to meet, 'kiss', in the midline, hampering laryngoscopy). The glossopharyngeal nerve runs under the mucosa of the base of the palatoglossal arch (towards the posterior tongue) and can be blocked there. Just as in the theatre, so in the oral cavity: confusion surrounds the wings. Properly called the palatoglossal and palatopharyngeal arches, they are also commonly called fauces and pillars. They are all the same thing.

Access to the stage's flies is controlled by the soft palate, a flap of soft tissue which can move up to separate the nasopharynx from the mouth and oropharynx (during swallowing), or move down to separate/shield the pharynx from the mouth (during chewing).

The soft tissues which surround the pharyngeal airway are themselves contained by bony structures (the maxilla, the mandible, the vertebrae and the base of the skull). When awake, tone in the pharyngeal musculature maintains airway patency. But once a patient is asleep, sedated or anaesthetised, muscular tone falls, and airway patency may depend on the relative sizes of these bones and of the soft tissues within them. Patients with more soft tissue, a shorter mandible or squatter cervical vertebrae may be at particular risk of obstructive sleep apnoea.

The Nose and Nasal Cavities

The nasal cavities have evolved to humidify and warm air before directing it to the pharynx and thence towards the lungs; all roles likely to be subverted by anaesthetists. Nevertheless, the anatomies of both inside and outside of the nose have anaesthetic relevance.

The nose encases the two nasal cavities which each lead from nostril to nasopharynx. Each cavity is lined by a mucous membrane of peculiar vascularity; luxuriant perfusion limits local cooling and desiccation despite

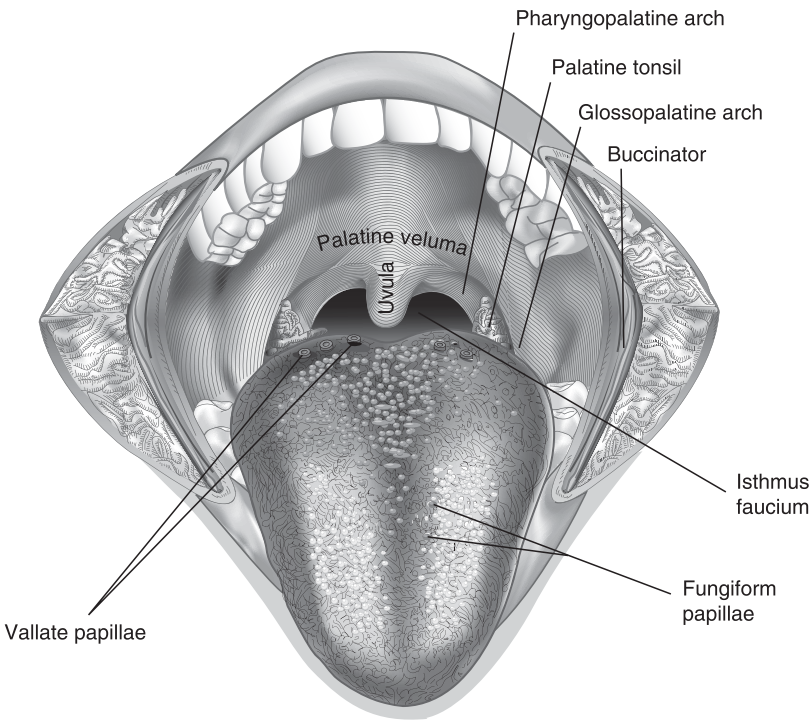


Figure 1.2 The mouth.

evaporation. It also means minimal trauma can cause profuse bleeding.

The mucosa's innervation is so complex as to make topical anaesthesia the most practical option for even the most ardent regional anaesthetist (no less than nine nerves innervate each cavity). That said, simply pouring a local anaesthetic solution down the nostrils of a supine anaesthetised patient is profoundly unanatomical: the medicine can be directed to its target by gravity. Before functional endoscopic sinus surgery, for example, if the solution is to reach the cephalad reaches of the nasal cavity, the head must be tilted back (with Trendelenburg tilt and a pillow below the shoulders). To direct solution along the projected path of an optical bronchoscope, less Trendelenburg is necessary. Moreover, some sensory fibres pass through the contralateral sphenopalatine ganglion. It is therefore sensible to apply local anaesthetic to both nostrils, even if only one is to be subjected to a foreign body.

Each nasal cavity is divided by three turbinates (more properly conchae) which extend medially from the cavity's lateral wall (Figure 1.3). The space between the floor of the nasal cavity and the inferior concha is larger than that between inferior and middle conchae. Furthermore, the ostia (holes) through which the sinuses drain into the nose are all cephalad to the inferior concha. For both reasons, a tracheal tube which runs through the nasal cavity may be best placed along its floor, being less likely to cause damage, or to obstruct drainage and cause sinusitis. On the other hand, an optical bronchoscope advanced between middle and inferior conchae may execute a gentler turn inferiorly toward the glottis.

The damage that can be done by tubes passed blindly through the nose is remarkable; entire conchae have been amputated, and tubes passed into the brain through fractures in the skull base. Clearly tracheal tubes should be of as small a diameter as possible, while bleeding diatheses and basal skull fractures are important relative contraindications to nasal intubation. If a tracheal tube is nevertheless to be directed through the nose, using a flexible optical bronchoscope may reduce the risk of damage.

The nose's external profile also determines how tightly a face mask can fit. Given too large a nasal bone, gas escapes around the mask's sides, and too small, gas escapes at the midline.

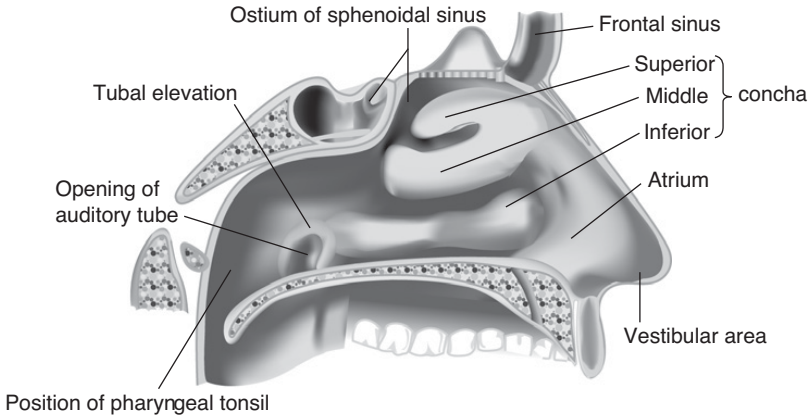


Figure 1.3 The lateral nose.

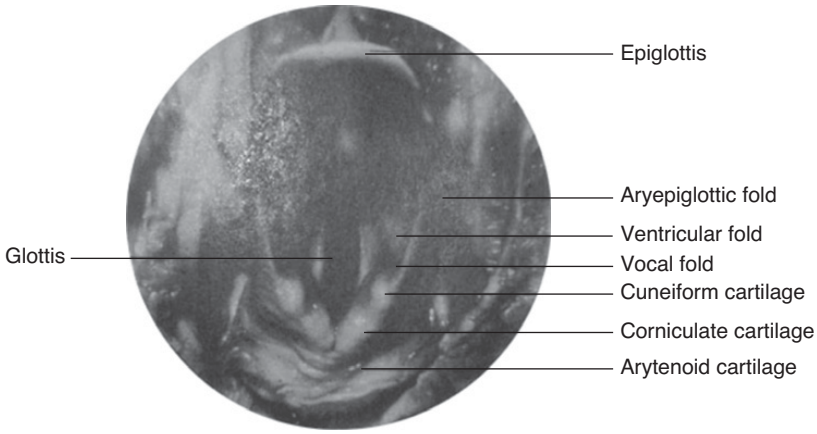


Figure 1.4 Anatomical specimen of adult human larynx.

Glottis and Epiglottis

The human larynx is often declared the organ of speech (Figure 1.4). More extraordinary still, it allows singing. Its intrinsic musculature is accordingly complex, but not always relevant to the anaesthetist simply aiming for the cavity the muscles surround. That said, a naming of the parts seen on laryngoscopy allows accurate description of abnormality. Just as for a glutton before fancy chocolates, only a few details of the box are relevant; the key is to get in, past the epiglottis and past the cords themselves, without doing undue damage on the way.

The epiglottis has evolved to shield the glottis not from anaesthetists, but from nutrients headed towards the oesophagus. It works like the flexible lid of a pedal bin. Generally, it is half open, to allow breathing. But on swallowing the epiglottis and larynx come together. Like the lid closing on the bin, the larger and more flexible the epiglottis, the better it can fit the glottis, but the more it can frustrate direct laryngoscopy. Given adequate anaesthesia, the tip of a laryngoscope placed in the vallecula and drawn anteriorly will generally also pull the epiglottis sufficiently far anteriorly to reveal the glottis. But if an anaesthetised patient is in the supine position, and the epiglottis is long and flaccid, it may fall to hide the cords unless it too is scooped above the laryngoscope's blade (Figure 1.5). Alternatively, the tip of a McCoy laryngoscope blade can be deployed to apply anterior pressure at the root of the epiglottis. Conversely, if the tissue around the epiglottis is incompliant (after radiotherapy, for instance), deploying the McCoy blade's tip may simply push the laryngoscope's blade posteriorly, hindering direct laryngoscopy rather than making it easier (see Chapter 14). A Miller straight blade can be placed posteriorly to a flaccid epiglottis to lift it out of the way.

A hypertrophied lingual tonsil or a tumour at the root of the tongue may also push the epiglottis

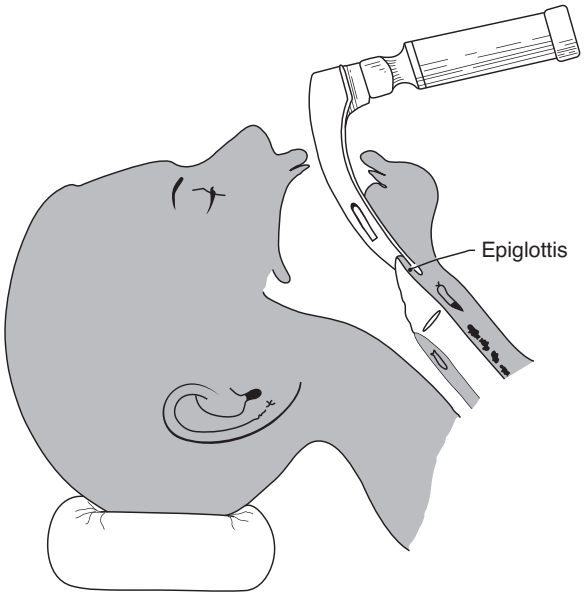


Figure 1.5 The laryngoscope.

posteriorly to obstruct the glottis, just as a bin's lid may be pushed down. While asymptomatic and imperceptible during a standard examination, such an enlarged tonsil may severely hamper airway control (see Chapter 14).

The mucosa of the larynx above the cords is supplied by the internal laryngeal nerve, which branches off the superior laryngeal nerve just lateral to the greater cornu of the hyoid bone. It then plunges deep to the thyrohyoid membrane. It can be blocked by local anaesthetic injected through a needle gingerly walked off the hyoid and then passed through the perceptible resistance of the membrane. As it is purely sensory, it can be blocked without fear of attendant paresis.

But below the cords, the mucosa is innervated by the recurrent laryngeal nerve, which also supplies almost all the intrinsic muscles of the larynx. Transection of the recurrent laryngeal nerve partially adducts the cord, and – worse – less extreme surgical damage of the nerve can cause the cord to adduct more extremely, across the midline. So, anatomy dictates that the mucosa below the cords is anaesthetised topically, if at all.

The ends of the vocal cords themselves are fixed anteriorly to the thyroid cartilage. But their posterior ends each attach to an arytenoid complex which moves like a cam on the cricoid cartilage. A few degrees' turn tightens the cord to raise the voice's pitch; more extreme movements adduct the cords (in laryngospasm) to protect the trachea from aspiration or to thwart the anaesthetist. With force, an arytenoid may be knocked off the cricoid cartilage – a remediable hoarse voice and sore throat are the results.

Subglottic Airway: Cricothyroid Puncture and Tracheostomy

'If you cannot go through it, go round it': if teeth, tongue, epiglottis or glottis obstruct the path to the cords, then it may be easier to reach the trachea directly through skin, either by cricothyroid puncture or by tracheostomy.

As the trachea must run posteriorly from the glottis to reach the carina in the mediastinum, it is most superficial at its start. Indeed, the defect between the thyroid and the cricoid cartilages is easily palpable in a slim normal neck, and is covered only by skin, loose areolar tissue and the fibrous cricothyroid membrane (Figure 1.6). So, in theory, a needle or cannula can be passed into the trachea here without risk of haemorrhage from anterior structures. The cricoid cartilage is the only ring-shaped cartilage in the upper airway and the posterior part is broader than the anterior part, thus to some extent preventing a needle or scalpel from penetrating into the oesophagus at the level of the cricothyroid membrane.

More caudally a larger tube can be passed into the trachea without undue force (either surgically or with a percutaneous technique). But again, the oesophagus runs directly behind the trachea, where the cartilages are C-shaped instead of complete rings, and can be damaged through the posterior wall in a percutaneous approach. Moreover, the trachea is far from subcutaneous as it approaches the sternum: the thyroid isthmus lies over the second, third and fourth tracheal rings; from there the inferior thyroid veins drain the gland, running close to the midline towards the chest – and in a short neck, the left brachiocephalic vein and artery may poke above the sternum as they cross the trachea. The position of these vessels, and indeed the trachea and the cricothyroid membrane, can usefully be identified by ultrasound before cricothyroidotomy or tracheostomy.

Trachea and Bronchial Tree

Like a jetliner's wing, the trachea's apparent simplicity belies its complexity. It is held open by the tracheal cartilages. These are shaped like a C, with the curve

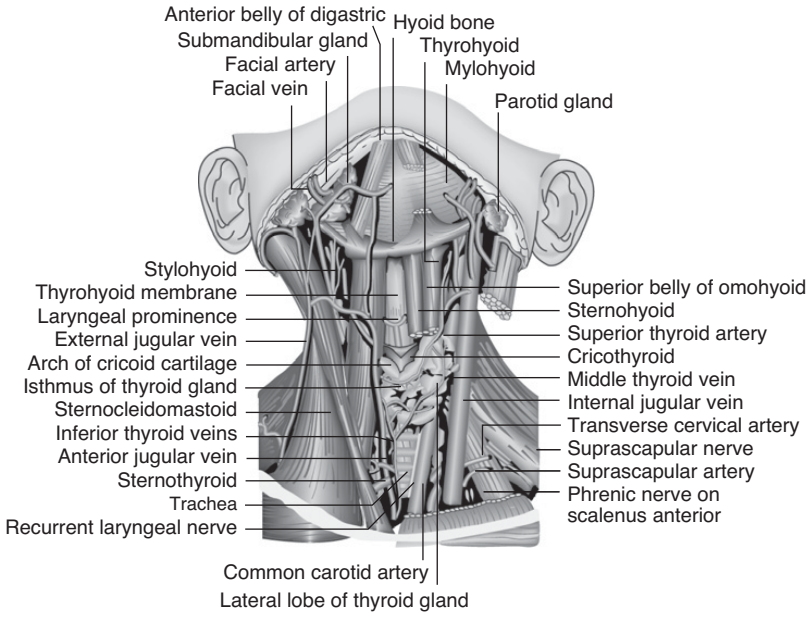


Figure 1.6 Thyroid gland and the front of the neck.

facing anteriorly; their corrugations distinguish the trachea from the smooth oesophagus. Not only do the rings help disorientated bronchoscopists, it also enables the tracheal bore to vary. The two ends of each C are joined by the trachealis muscle, which forms the posterior wall of the trachea. If the muscle tightens the trachea's radius is reduced (as the points of the C are drawn together), airway resistance rises and the volume of the dead space falls; conversely, airway resistance falls and the dead space swells as the muscle relaxes. So, just as in a wing, the trachea's shape can be optimised for different flow rates.

As the bronchial tree ramifies beyond the trachea (Figure 1.7), its initial divisions are crucially asymmetric. The carina itself is on the left of the midline; the left main bronchus is narrower and runs off closer to the horizontal than the right; all conspire to send aspirated material towards the right main bronchus. Moreover, in an adult the left main bronchus is some 4.5 cm long while the right main bronchus runs just 2.5 cm, or less, before giving off the bronchus to the right upper lobe. Clearly a larger target is easier to hit. It is therefore easier to isolate the lungs without occluding a lobar bronchus, if the left rather than the right main bronchus is the target (see Chapter 27).

The trachea is shortened by cervical flexion and lengthened by cervical extension. If a tracheal tube is anchored at the mouth, and rests above the carina when the neck is in the neutral position, it may stimulate the carina or even pass into a bronchus if the neck is flexed.

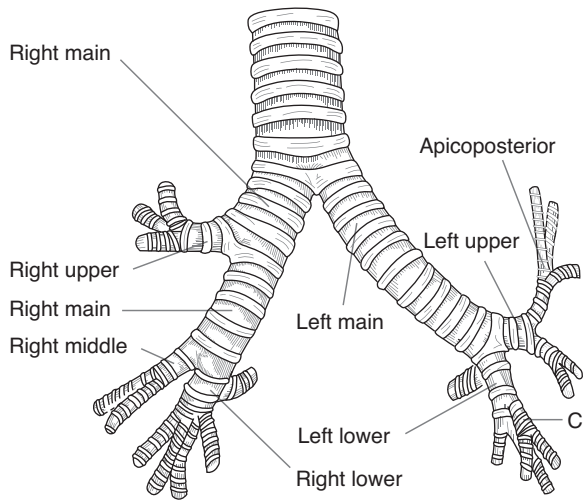


Figure 1.7 Main, lobar and segmental bronchi.

Cervical Spine

As in owls, so in humans: our two eyes face in the same direction, so our cervical spines have evolved particular mobility and strength to bear the heavy head, and allow it to turn relative to the body, while protecting the spinal cord within.

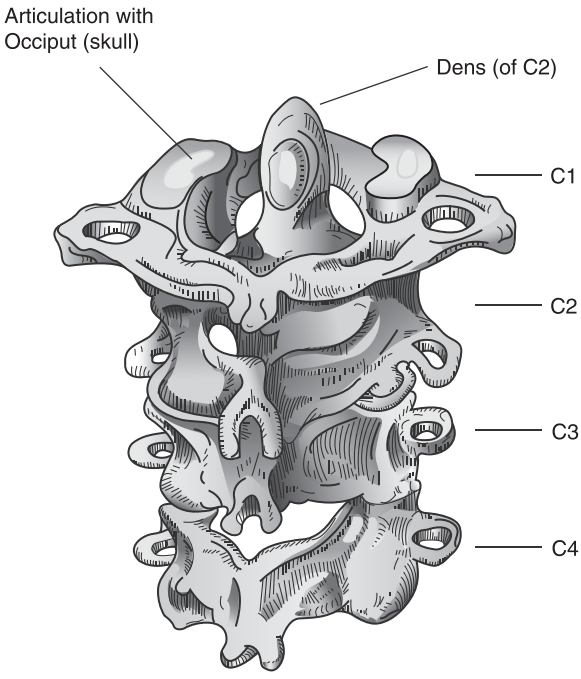


Figure 1.8 Atlas and axis.

Both the mobility and strength are crucial to anaesthetic practice: if pathology limits mobility, management of the airway is typically hampered; if the cervical spine is weakened, inappropriate management of the airway may catastrophically damage the cord.

The three most cephalad bones together form the occipito-atlanto-axial complex (Figure 1.8). Most of the neck's movement occurs between these three bones, both in normal life and during direct laryngoscopy.

Working caudad, the occipital condyles rest on the lateral masses of atlas like the rails of a rocking chair stuck in tram tracks: the head can flex forward at the joint (until the odontoid hits the skull) and extend backwards; some abduction is possible, but rotation is not. Atlas, however, turns around the axial odontoid peg which occupies the anterior third of the space within the axis. Posterior movement of atlas over axis is limited by the axial anterior arch impinging on the peg.

Otherwise ligaments are responsible for the stability of the joints:

- The alar ligaments run from the sides of the peg to the foramen magnum – depending on which way the head is turned, one or other tightens and so limits rotation.
- The transverse band of the cruciform ligament – said to be the strongest ligament in the body – runs behind the peg from one side of atlas to the other – it stops atlas moving anteriorly over axis.
- The tectorial membrane runs as a fibrous sheet from the back of the body of the peg to insert around the anterior half of the foramen magnum – running anterior to the axis around which the head nods, it tightens as the head is extended.

Below the axis, in the 'subaxial' spine, the vertebrae assume a more conventional form. They articulate at the zygoapophyseal joints ('facet joints') between each bone's facets. Flexion is limited by the ligaments between the posterior parts of the vertebrae; extension by the anterior longitudinal ligament and the intervertebral disc capsules.

Direct laryngoscopy is classically facilitated by bringing oral, pharyngeal and laryngeal axes into line. In practice, that means extension at the occipito-atlanto-axial complex and very moderate flexion in the subaxial cervical spine. A normal spine and cord will typically tolerate the forces applied by a gentle anaesthetist.

But after trauma, or with disease or malformation, the cervical spine may be either fixed or abnormally mobile. Ankylosing spondylitis, surgical fusion, or fixation may (for example) all frustrate the anaesthetist hoping to align the oral, pharyngeal and laryngeal axes, and so indicate the need for more artful management of the airway.

At the other extreme, trauma or ligamentous laxity may make the cervical spine so especially mobile as to jeopardise the spinal cord or medulla. Here anatomy is paramount, determining which manoeuvres are safe, and which dangerous. For example, in rheumatoid arthritis, the cruciform ligament may become lax; flexion of the occipito-atlanto-axial complex is then especially dangerous (atlas may move anteriorly on axis, impaling the cord between the peg and the posterior arch of atlas). But if the peg is fractured at its base, atlas is freer to move relative to axis, and both extension and flexion of the occipito-atlanto-axial complex will be dangerous.

Similarly, turning a patient from the supine position to prone will expose the patient to different dangers according to anatomy. Generally, the volume of the vertebral canal is increased in flexion, easing pressure on a cord compressed by, for example, ligamentous hypertrophy. But in bilateral facet fracture dislocation, flexion can precipitate anterior subluxation of the cephalad vertebra, disastrously guillotining the cord.

Summary

Gentle subluxation of the temporomandibular joint facilitates passive mouth opening. Direct laryngoscopy entails extension of the intricate occipito-atlanto-axial joint. Passage through the cricothyroid membrane offers the easiest percutaneous access to the airway in an emergency. The oesophagus lies behind the trachea at this level, and it may be punctured by a needle or scalpel passed posteriorly through the trachea, though the posterior arch of the cricoid cartilage may protect at the level of the cricothyroid membrane. Anatomy determines what manoeuvres will be especially dangerous in cervical instability.

Acknowledgements

Christian Ulbricht and Peter and Ellie Clarke nobly and generously improved an earlier draft; all remaining mistakes are mine alone.

Chapter 2

Physiology of Apnoea, Hypoxia and Airway Reflexes

Andrew D. Farmery and Jeremy A. Langton

Hypoxia

Humans are adapted to tolerate oxygen pressures which range from what might be called 'sea-level normoxia', down to the modest hypoxia of high-altitude living. Humans are not adapted to hyperoxic conditions and these are increasingly recognised as harmful. Why then should the airway specialist be concerned about hypoxia, and seek to counter it with hyperoxic exposure?

Classification of Hypoxia

'Cellular respiration' occurs at the level of the mitochondria, when electrons are passed from an electron donor (reduced nicotinamide adenine dinucleotide (NADH)) via the mitochondrial respiratory cytochromes to 'reduce' molecular oxygen (O_2). The energy from this redox reaction is used to phosphorylate adenosine diphosphate (ADP), thereby generating the universal energy source, adenosine triphosphate (ATP), which powers all active biological processes. If molecular oxygen cannot be reduced in this way, this bit of biochemistry fails and cellular hypoxia occurs. Based on Barcroft's original classification, four separate causes of cellular hypoxia can be considered. Three of these four factors affect oxygen delivery to the tissues ($\dot{D}O_2$), which is described mathematically by the equation in Box 2.1. Derangements of each of the terms on the right-hand side of this equation will reduce oxygen delivery to tissues.

The fourth cause of cellular hypoxia in our classification is *histotoxic hypoxia*. An example of this is cyanide or carbon monoxide poisoning. In histotoxic hypoxia, there is not (or there need not be) any deficit in oxygen delivery. Cellular and mitochondrial partial pressure of oxygen (PO_2) may be more than adequate, but the deficit lies in the reduction of molecular oxygen due to a failure of electron transfer. In order to fully understand the classification of hypoxia, it is useful to consider the example of carbon monoxide poisoning.

What Is the Mechanism of Death in Severe Carbon Monoxide Poisoning?

Let us consider each of the factors of Barcroft's classification.

Hypoxaemic hypoxia is not likely to be the cause. Assuming no lung damage has occurred, this patient's arterial oxygen (P_aO_2) is likely to be normal if breathing air, or elevated if breathing supplemental oxygen. P_aO_2 is determined by the gas-exchanging properties of the lung and is unaffected by haemoglobin concentration or by the nature of the haemoglobin species present.

Anaemic hypoxia. The presence of carboxyhaemoglobin, which has no oxygen-carrying capacity, will certainly reduce the amount of normal oxygen-carrying haemoglobin. But normal oxyhaemoglobin will still be in the majority, and the $\dot{D}O_2$ will be more than adequate. Counter to popular understanding perhaps, the presence of carboxyhaemoglobin is not the problem here.

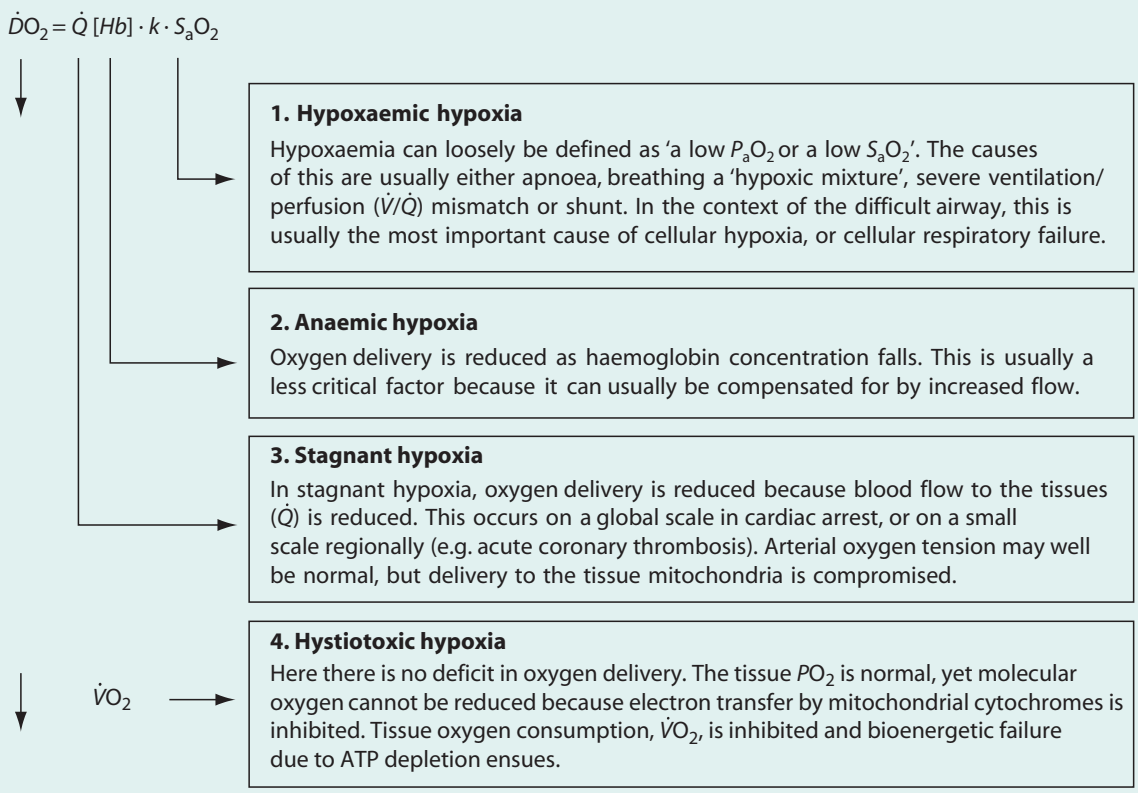
Stagnant hypoxia is unlikely to be a cause, since the cardiac output is likely to be elevated as a compensatory mechanism.

So, what is the cause of death? The underlying mechanism of cellular death in this case is *histotoxic hypoxia*. Just as carbon monoxide has a high affinity for the haem group in haemoglobin, it also has a high affinity for the iron-containing haem flavoproteins in mitochondrial respiratory cytochromes. Once bound, electron transfer is interrupted and tissue oxygen, albeit in abundant supply, cannot be reduced and bioenergetic failure supervenes. In carbon monoxide poisoning, the presence of carboxyhaemoglobin merely serves as a marker of carbon monoxide exposure. It is not usually part of the mechanism of death.

Differential Effects of Deficits in Oxygen Delivery

The equation in Box 2.1 shows that $\dot{D}O_2$ is simply proportional to the product of the three 'Barcroft

BOX 2.1 Barcroft's classification of hypoxia



$\dot{Q}$ is the cardiac output, [*Hb*] is the haemoglobin concentration and S_aO_2 is the arterial oxyhaemoglobin saturation. The constant, *k*, can be ignored in this analysis. Deficiencies in $\dot{Q}$, [*Hb*] and S_aO_2 produce *stagnant, anaemic and hypoxaemic* hypoxia, respectively.

variables'. It would, therefore, appear that any given deficit in $\dot{D}O_2$ should cause identical degrees of cellular hypoxia regardless of whether the deficit is due to hypoxaemia, anaemia or low blood flow. We shall see below that whereas $\dot{D}O_2$ deficits due to anaemic and stagnant hypoxia have virtually identical consequences, $\dot{D}O_2$ deficits due to hypoxaemic hypoxia are very distinct and uniquely important.

Anaemic and Stagnant $\dot{D}O_2$ Deficits

Experimental and theoretical models show that the variables [*Hb*] and $\dot{Q}$ are not uniquely independent variables; it is merely the product, $\dot{Q}$ [*Hb*], which determines oxygen delivery and cellular oxygenation. For example, if haemoglobin concentration is halved and blood flow doubled, oxygen delivery and cellular oxygenation remain unchanged. This is because these variables simply determine the flux of oxygen to the tissues, and they have no other significance beyond this point.

Hypoxaemic $\dot{D}O_2$ Deficits

Reductions in $\dot{D}O_2$ due to hypoxaemia are much more impactful than if an equal $\dot{D}O_2$ reduction were due to anaemic or stagnant causes. This seems counterintuitive if considered in terms of Barcroft's classification, because this focusses on oxygen delivery (bulk oxygen flux) to the tissue capillaries.

From the lung to the capillary, oxygen transport is by *convection*, whereas from capillary to cell/mitochondrion, oxygen transport is by *diffusion*. It is the PO_2 in the capillary which drives the diffusion of oxygen from capillary to cell. So, the effects of hypoxaemia are twofold:

not only does it reduce oxygen flux along the arterial tree (via a reduced S_aO_2), but it also impairs oxygen delivery beyond the tissue capillary (via a reduced PO_2).

The PO_2 at the cellular level is around 3–10 mmHg (0.4–1.3 kPa), and at the mitochondrion it is around 1 mmHg (0.13 kPa). The PO_2 in tissue capillaries may be around 40 mmHg (5.3 kPa) and this PO_2 gradient drives oxygen from capillary to mitochondrion according to Fick's law of diffusion. Figure 2.1 shows the effect of reducing $\dot{D}O_2$ on the cell's ability to take up and consume oxygen ($\dot{V}O_2$), and how this differs depending on whether the fall in $\dot{D}O_2$ is achieved via anaemic/stagnant or hypoxaemic mechanisms. It can be seen that as $\dot{D}O_2$ falls, $\dot{V}O_2$ remains constant until a critical $\dot{D}O_2$, $\dot{D}O_{2crit}$, is reached, below which cellular oxygen uptake and utilisation are diminished. $\dot{D}O_{2crit}$ represents the oxygen delivery at which cellular hypoxia begins. In normal tissue (bold lines), cellular hypoxia is seen to begin when $\dot{D}O_2$ falls to 0.4 L min^{-1} for hypoxaemic hypoxia, whereas the cell can tolerate a lower $\dot{D}O_2$ if the mechanism is anaemic or stagnant. In other words, cells are more vulnerable to hypoxaemic hypoxia.

According to Fick's law, diffusive oxygen flux depends not only on the partial pressure gradient, but also on the distance between capillary and cell, and this may be increased in oedematous states (where the interstitium occupies a greater volume, separating capillary from cell), and in capillary de-recruitment due to shock (where, if a cell's nearest capillary is de-recruited, its new nearest patent capillary will now be a greater distance away). This may explain why the difference between stagnant/anaemic and hypoxaemic hypoxia on cellular oxygen uptake is exaggerated in states of reduced diffusive conductance. This effect is also shown in Figure 2.1 (feint curves).

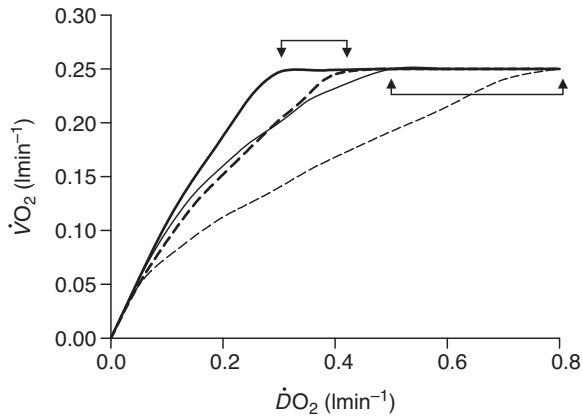


Figure 2.1 Plot of cellular O_2 consumption ($\dot{V}O_2$) vs. bulk O_2 delivery ($\dot{D}O_2$). Solid lines represent stagnant/anaemic hypoxia. Broken lines represent hypoxaemic hypoxia. Bold lines show normal relationship for tissue without significant barrier to O_2 diffusion from capillary to cell. Feint lines represent tissue with significant diffusional resistance, as in oedema or shock. As $\dot{D}O_2$ falls, $\dot{V}O_2$ initially remains constant and satisfies the normal metabolic requirement (0.25 l min^{-1}). When $\dot{D}O_2$ falls to a critical value, $\dot{D}_{crit}$ (shown by arrows), cellular O_2 consumption falls and cellular hypoxia begins. The difference in $\dot{D}_{crit}$ between hypoxaemic and stagnant/anaemic hypoxia is shown to increase when a diffusional barrier exists. (Redrawn from Farmery and Whiteley (2001).)

The Rate of Arterial Desaturation in Apnoea

We have seen that hypoxaemic hypoxia is of particular importance in the development of cellular hypoxia and clearly in the context of the difficult airway, the principal cause of hypoxaemia is airway obstruction. It is important to understand the mechanisms by which hypoxaemia develops, and the factors which determine the rate of this process.

As soon as apnoea (with an obstructed airway) occurs, alveolar and hence pulmonary capillary PO_2 begins to fall. In apnoea, the process of gas exchange between alveolus and pulmonary capillary becomes non-linear. The rising partial pressure of carbon dioxide (PCO_2) and falling pH associated with carbon dioxide accumulation continually shifts the oxygen–haemoglobin dissociation curve adding yet more non-linearity to the process of arterial desaturation. The time lag between changes in PO_2 feeding through into changes in mixed venous PO_2 enhances the complexity of the mathematical model further. Figure 2.2 shows the effects of six different physiological derangements on the rate of arterial desaturation in obstructed apnoea. Figure 2.2(a) shows that desaturation is exaggerated in small lung volumes (as might occur in supine anaesthetised patients). Figure 2.2(b) shows that the value of the initial alveolar oxygen concentration at the onset of apnoea is also important. Due to the various mathematical non-linearities in the system, the lower the initial alveolar oxygen tension, the greater the rate of desaturation. This has important implications for patients who have periods of partial airway obstruction (and hence diminished alveolar PO_2 (P_AO_2)) before obstructing completely. It also underpins the value of thorough pre-oxygenation before procedures where there is a significant risk of obstructed apnoea. Figure 2.2(c) shows that while shunt diminishes the value of S_aO_2 at any given time during apnoea, the rate of desaturation is unaltered. Figure 2.2(d) shows that increased

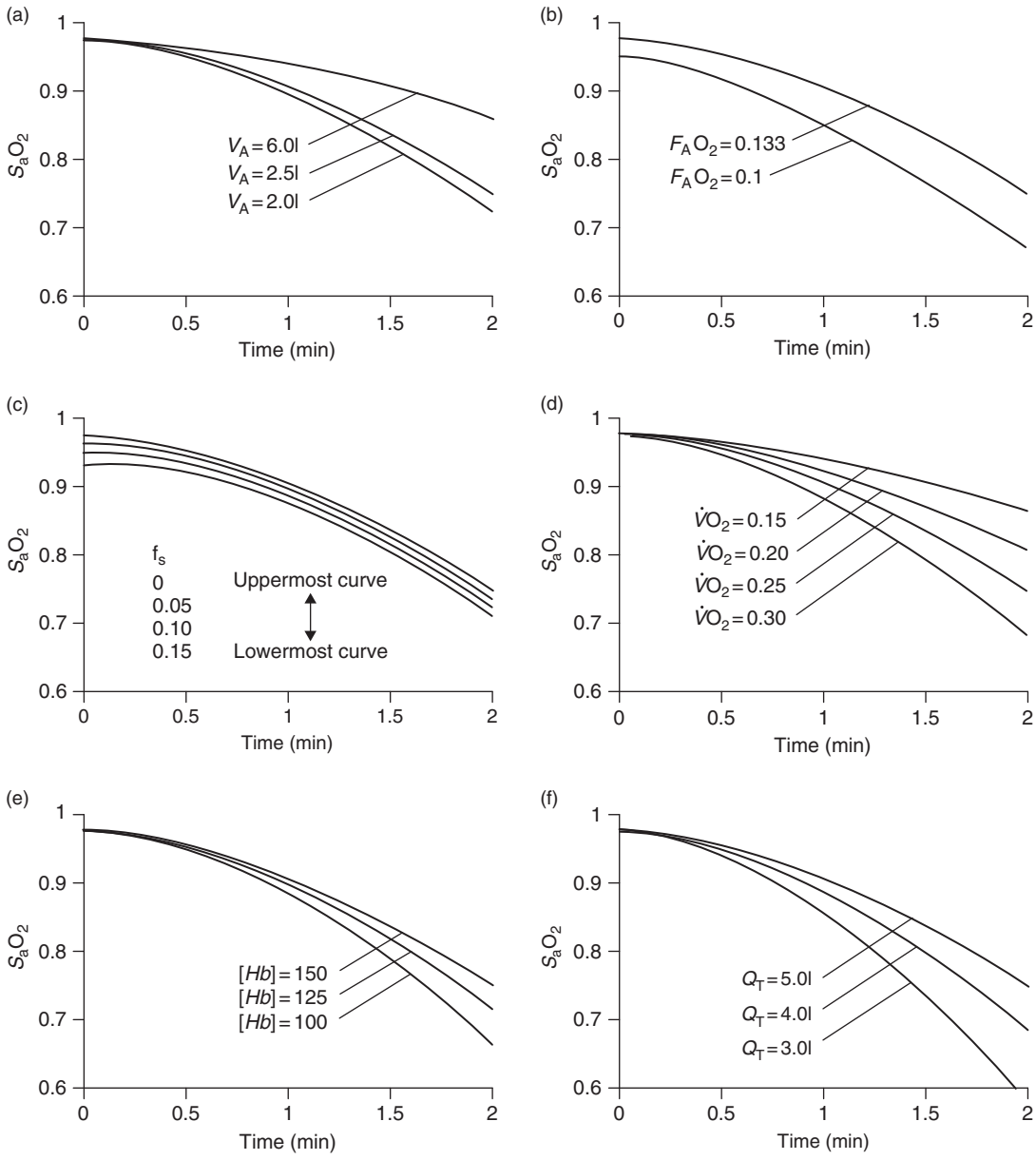


Figure 2.2 (a) Effect of lung volume (V_A in litres) on the time course of S_aO_2 in apnoea. (b) Effect of initial F_AO_2 on the time course of S_aO_2 in apnoea. (c) Effect of shunt fraction (f_s) ranging from 0% to 15% on the time course of S_aO_2 in apnoea. (d) Effect of O_2 consumption rate ($\dot{V}O_2$) ranging from 0.15 to 0.3 litre min^{-1} on the time course of S_aO_2 in apnoea. (e) Effect of haemoglobin concentration ([*Hb*] in g $litre^{-1}$) on the time course of S_aO_2 in apnoea. (f) Effect of total blood volume (Q_T) on the time course of S_aO_2 in apnoea. (Reproduced with permission from Farmery and Roe (1996).)

metabolic rates (as may occur in sepsis, or when struggling to breathe in severe airway obstruction) increases the rate of arterial desaturation, and this effect is exaggerated as desaturation proceeds. Figure 2.2(e) and (f) show how both diminished cardiac output and reduced haemoglobin concentrations increase the rate of arterial desaturation in apnoea. This is partly because haemoglobin acts as an oxygen reservoir. The effect of cardiac output is complex. So, not only does arterial hypoxaemia have a unique importance in terms of cellular hypoxia (as discussed above and in Figure 2.1), but in apnoea, anaemia and

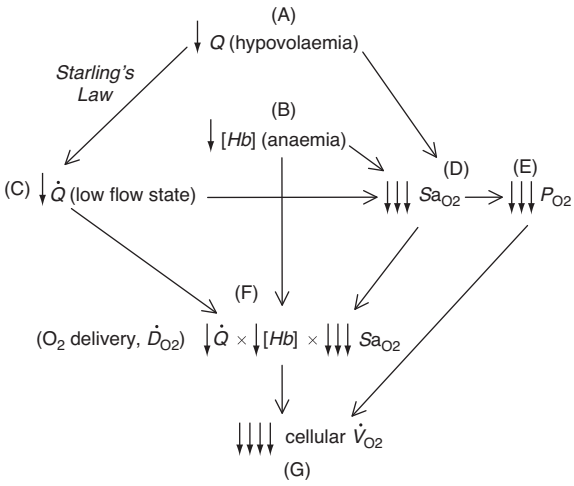


Figure 2.3 Reductions in both [*Hb*] and $\dot{Q}$ as shown in (A) not only reduce oxygen delivery (B) directly (path A–B), but also indirectly via path A–C–B during apnoea because the rate of arterial desaturation during apnoea is increased by anaemia and low output. The combination of this direct and indirect effect is to produce an exaggerated reduction in oxygen delivery (B). The reduction in oxygen delivery may reduce cellular oxygen uptake (path B–D), as predicted by the solid lines in Figure 2.1. In addition, the hypoxaemic conditions (point C) contribute independently to exaggerating the reduction of cellular oxygen uptake (via path C–D) as predicted by the broken lines in Figure 2.1.

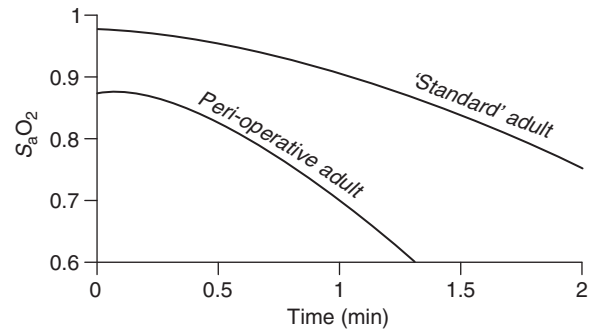


Figure 2.4 Rate of arterial oxyhaemoglobin desaturation with combination of small derangements in pathophysiological variables as might be seen in a peri-operative adult. Haemoglobin = 10 g dl^{-1}, cardiac output = 4 l min^{-1}, initial P_AO_2 = 10 kPa, initial P_ACO_2 = 8 kPa, alveolar volume = 2.0 litres, shunt fraction (f_s) = 0.1. (Reproduced with permission from Farmery and Roe (1996).)

low flow states *compound* the reduction in S_aO_2 and also markedly exaggerate the reduction in oxygen delivery, which is the product of all three of these terms. The interplay of these factors is depicted in Figure 2.3.

Also of note is the fact that small derangements in each of the physiological factors in Figure 2.2 combine to produce a larger overall effect on the rate of arterial desaturation. An example of this might be a 'typical' sick patient about to undergo induction of anaesthesia. This is shown in Figure 2.4.

Pre-oxygenation and Hyperoxia

The use of supplemental oxygen, and particularly high flow, high fraction oxygen is commonplace in anaesthesia. In addition to its use in the anaesthetic room and operating theatre it is routinely used in awake patients in the recovery room and sometimes on the wards post-operatively. Such practice is often regarded as a hallmark of high quality medical care. Recent provocative evidence, however, suggests that hyperoxia is associated with adverse outcomes in stroke, myocardial infarction and cardiac arrest. Although there is no good evidence to support discontinuation of this practice in the perioperative setting, there are good reasons to consider reserving pre-oxygenation for only those cases who are at increased risk of difficult airway management and desaturation.

Classic pre-oxygenation aims to increase body oxygen stores to their maximum, so that periods of apnoea are tolerated for longer before critical hypoxia occurs. Hyperoxia is an unavoidable consequence of it, rather than its aim. The body store which can be most increased is the lung and airways, whose nitrogen can be exchanged for oxygen. This store holds oxygen at high partial pressures and can release most of it at usefully high partial pressures. The second most significant oxygen store, which is often overlooked, is the arterial and venous blood, which comprise a store which is almost half that of the (denitrogenated) lung. This store (mainly the venous blood) can be increased by almost 20% by pre-oxygenation. Unlike the lung store, the blood store is filled at normoxic partial pressures. It requires moderately hypoxic conditions for the store to be unloaded. As such it buffers the speed of hypoxic decline at these modestly hypoxic levels.

The necessary evil of pre-oxygenation is analogous to the practice of feasting before a period of fasting. An assessment of the risks and benefits dictate that the risks of gluttonous feasting are probably justified by the increased likelihood of surviving prolonged starvation immediately following. This risk–benefit analysis is only valid, however, if the risk of starvation is real and appreciable. Likewise, pre-oxygenation should be undertaken after careful consideration of the risks imposed by not doing so.

There are two elements within the practice of pre-oxygenation:

- *Supplying 100% inspired oxygen*: efficient pre-oxygenation involves a close-fitting face mask to avoid air entrainment. Correct face mask application can be confirmed by seeing a full reservoir bag which moves with respiration. The bag is an essential component of the circuit because it provides the reservoir necessary when the patient's peak inspiratory flow (> 30 L min^{-1}) exceeds the fresh gas flow. The fresh gas flow should be high enough to minimise any rebreathing of nitrogen. A small amount of rebreathing of carbon dioxide is not relevant here, since it makes little difference to the oxygenation, so circle systems are no better than a Bain system in this respect. In fact, the larger circuit volume of the circle system means that nitrogen elimination is slower than a Bain system for the same fresh gas flow.
- *Time required for effective denitrogenation with 100% oxygen:* at the end of a quiet expiration the lung volume at functional residual capacity (FRC) may be 2000–2500 mL. This will be affected by patient position or disease processes and may be much reduced by obesity, pregnancy or a distended bowel. On breathing 100% oxygen, the wash-in of oxygen is exponential. The time constant (τ) of this wash-in process is the ratio of FRC or alveolar volume to alveolar ventilation ($V_A/\dot{V}_A$). Given an alveolar minute ventilation of 4 L min^{-1} and FRC volume of 2.0 L we can estimate the time constant to be 2.0/4 = 0.5 minutes.

Exponential wash-in of oxygen during pre-oxygenation (typical values)

- Time constant (τ) of exponential process = $V_A/\dot{V}_A$

  = 2.0/4

  = 0.5 minutes

- After one time constant (0.5 minutes) pre-oxygenation is 37% complete
- After two time constants (1.0 minutes) pre-oxygenation is 68% complete
- After three time constants (1.5 minutes) pre-oxygenation is 95% complete

It is, therefore, reasonable to continue pre-oxygenation for at least three time constants to ensure maximal pre-oxygenation. It should be noted that patients with a small FRC will pre-oxygenate more quickly than normal but the oxygen store contained in the FRC will be reduced. Increasing alveolar minute ventilation (four to eight deep or vital capacity breaths) increases the rate of increase in P_AO_2 and is extremely useful when time for pre-oxygenation is limited. Administering opioids such as fentanyl before pre-oxygenation may lengthen the time required to achieve a high P_AO_2. Pre-oxygenation is also discussed in Chapter 8.

In any particular patient, the magnitude of the alveolar minute ventilation and FRC is unknown. It is, therefore, useful to monitor the process of denitrogenation by measuring end-tidal FO_2. An end-tidal FO_2 of 90–91% indicates maximal pre-oxygenation and a store of oxygen in the FRC of approximately 2000 mL. The overall increase in oxygen stores in the blood and lungs with pre-oxygenation is from 1200 mL (air) to 3500 mL.

Desaturation following the Use of Suxamethonium

The American Society of Anesthesiologists (ASA) difficult airway algorithm recommends that if initial attempts at tracheal intubation after the induction of general anaesthesia are unsuccessful, the anaesthetist should 'consider the advisability of awakening the patient'. 'Awakening' more realistically means allowing return to an unparalysed state that permits spontaneous ventilation. This is considered to be safe practice. However, to what level might arterial saturation fall before spontaneous ventilation resumes? Using a combination of clinical data and a theoretical model, Benumof demonstrated that during complete obstructive apnoea, and in the 'cannot intubate, cannot ventilate/oxygenate' situation, critical haemoglobin desaturation occurs before the time to functional recovery for various patients receiving 1 mg kg^{-1} of intravenous suxamethonium.

Figure 2.5 shows that in all but the 'normal' adult, critical desaturation occurs long before recovery of even 10% of neuromuscular function.

From this analysis, it is clear that in a complete cannot intubate, cannot ventilate/oxygenate situation, it is not appropriate to wait for the return of spontaneous ventilation, but rather a rescue option should be pursued immediately. Benumof points out that this

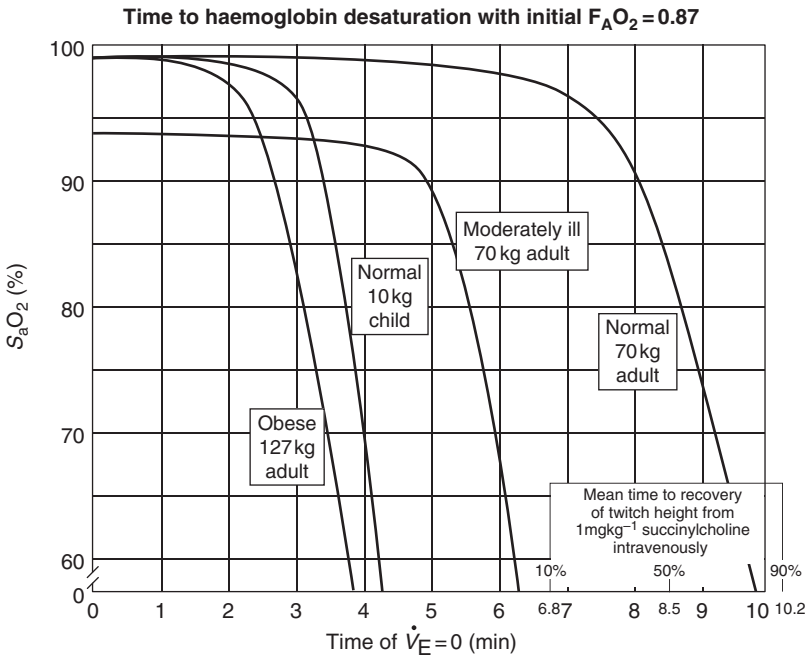


Figure 2.5 S_aO_2 vs. time of apnoea for various types of patients. (Reproduced with permission from Benumof et al. (1997).)

analysis ignores the central respiratory depressant effects of the concomitantly administered general anaesthetics, and so this should be regarded as an underestimation of the time to functional recovery.

The Final Common Pathway of Cellular Hypoxia: Membrane Potential and Cell Death

Venous PO_2 is a reasonable indicator of *capillary* and hence *tissue* PO_2. In many respects, measuring venous PO_2 (either mixed venous, or organ-specific venous such as jugular venous) is more useful in evaluating tissue oxygenation than measuring P_aO_2. Experimental and clinical evidence suggests that consciousness is lost when jugular venous PO_2 (and hence 'tissue PO_2' in the watershed of this drainage) falls below 20 mmHg (2.7 kPa). It is this PO_2 which drives diffusion of oxygen to its final destination in the mitochondria, where the PO_2 may be a fraction of a mmHg. With this degree of mitochondrial hypoxia, electron transfer cannot proceed (there is insufficient available molecular oxygen to accept electrons). This redox reaction falters and there is insufficient energy production to power the generation of ATP. We discuss the events which follow the onset of cellular bioenergetic failure.

Tissues vary in their sensitivity to hypoxia, but cortical neurones are particularly sensitive. They (along with the myocardium) are perhaps the most clinically important and are therefore the most studied. It is said that 'hypoxia stops the machine and wrecks the machinery'. As far as neurones and the myocardium are concerned this aphorism means that hypoxia initially arrests cellular function. For a period the integrity of the cell and its viability remain intact. If hypoxia is reversed, function will resume. However, sustained hypoxia wrecks the machinery. Via numerous and complex mechanisms, and in neurones particularly, an accelerating series of destructive events ensues, which results in cell death. The length of this process is highly variable depending on the tissue, the metabolic rate, blood flow and many other factors. However, it may be as short as 4 minutes for some neurones.

Anoxia and Membrane Potential

In general, living cells can be characterised by possession of a resting membrane potential whereas dead cells have no resting membrane potential. The effect of anoxia on resting membrane potential depends on the nature of the anoxic insult. In ischaemia (as in stroke), the tissue is deprived of oxygen and blood flow, whereas in airway obstruction, hypoxaemia occurs while blood flow (and glucose supply) continues, and this may have more deleterious effects.

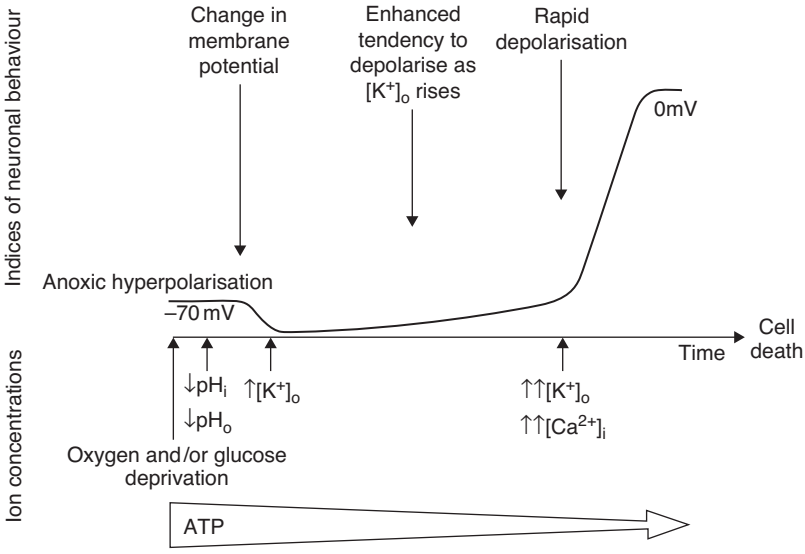


Figure 2.6 Membrane potential changes induced by cellular hypoxia. Intra- and extracellular pH changes are the first to be observed. Changes in membrane potential occur between 15 and 90 seconds. This is usually hyperpolarisation due to increased K^+ channel conductance. K^+ then leaks from within the cell. This causes an increase in extracellular $[K^+]$, especially if perfusion is limited (as in ischaemia), since the extracellular space is not washed out of ions and metabolites. The increasing extracellular $[K^+]$ causes gradual membrane depolarisation which in turn activates voltage-sensitive Ca^{2+} channels, contributing further to the depolarisation. The increasing acidosis and increasing depolarisation triggers Ca^{2+} release from intracellular stores, which in turn triggers synaptic release of glutamate. The release of this massive amount of glutamate stimulates ligand-gated cation channels whose opening coincides with a very rapid phase of membrane depolarisation. At this point, the Na^+–K^+-ATPase pump has ceased to operate and membrane potential is lost irretrievably.

One of the first metabolic features of mitochondrial bioenergetic failure is the depletion in ATP and accumulation of NADH. Small amounts of ATP can be generated from the glycolytic pathway, but this requires oxidised nicotinamide adenine dinucleotide (NAD^+), which is in short supply. However, the necessary NAD^+ can be generated by converting pyruvate to lactate, thus facilitating limited ATP production anaerobically. The intracellular acidosis which results from anaerobic metabolism is one of the first changes to be detected following cellular anoxia. If the nature of the hypoxia is *hypoxaemic hypoxia*, then blood flow will be preserved and there will be an abundant supply of glucose which will exacerbate the acidosis. Hyperglycaemic patients are particularly at risk.

Shortly after the onset of intracellular acidosis, the membrane potential of neurones begins to change. This is shown in Figure 2.6. The effect is variable, but the majority hyperpolarise. It is thought that this is due to an increase in K^+ channel conductance. The mechanisms are not clear, but possibilities include activation of ATP-sensitive K^+ channels (increased conductance in low ATP states), activation of direct oxygen-sensitive K^+ channels or activation of pH-sensitive K^+ channels. Hyperpolarisation of neurones renders them less susceptible to synaptic activation, and this may manifest as loss of consciousness (i.e. 'the machine stops').

From this point, membrane potential changes from hyperpolarisation to slow depolarisation. The mechanism of this is thought to be that the increased K^+ conductance (which initially hyperpolarised the membrane) enables K^+ efflux out of the cell down its concentration gradient. This escaped K^+ is normally removed from the extracellular space by the Na^+–K^+-ATPase, but as this pump begins to fail, extracellular $[K^+]$ increases and, as can be predicted by the Nernst equation, resting membrane potential begins to depolarise. As the membrane potential depolarises further, Ca^{2+} channels are activated and Ca^{2+} influx contributes to an acceleration of the depolarisation.

At this point, these electrophysiological effects are reversible if oxygenation is restored. If not, a cascade of irreversible events ensues.

Within a short time, membrane depolarisation becomes very rapid. This coincides with a number of cellular events: the failure of the Na^+–K^+-ATPase pump, massive release of Ca^{2+} from intracellular stores triggering massive release of excitatory neurotransmitters (principally glutamate) from synaptic vesicles, which in turn stimulate glutamate receptor-linked ion channels triggering further cation influx into the cell. Beyond this point, cell survival is unlikely. The machine is wrecked.

The time course for these events is variable. It is quickest for neurones exposed to ischaemia (arrested

flow) under hyperglycaemic and hyperthermic conditions, where the process may be a matter of only 1 to 4 minutes. Under hypoxaemic conditions with preserved flow and normoglycaemia, the process may take between 4 and 15 minutes depending on the degree and abruptness of the insult.

Summary: Hypoxia

Tissue hypoxia can be fatal despite normoxaemia (cyanide and carbon monoxide poisoning). Hypoxaemic hypoxia (airway obstruction) is more damaging to cells than anaemic or stagnant hypoxia. Oxygen saturation will fall more quickly in an apnoeic sick patient. Waiting for spontaneous ventilation to return may not be a sensible option. An end-tidal oxygen fraction of > 90% indicates maximum pre-oxygenation. Pre-oxygenation achieves its end by increasing the store of oxygen in the lung and the blood. Initial hyperoxia is an unwanted consequence.

Airway Reflexes

Upper airway reflexes are important to anaesthetists, as a clear airway enables safe ventilation of the lungs and oxygenation of the patient. It also provides a means by which the depth of inhalation anaesthesia can be rapidly altered. An increase in the sensitivity of airway reflexes during induction of anaesthesia increases the likelihood of laryngeal spasm and coughing. This may impair the smooth administration of inhalation anaesthesia and when severe may be life-threatening. During recovery from anaesthesia the larynx plays a primary role in the protection of the lungs from aspiration of foreign material.

Reflexes from the Nose

The nasal mucosa receives sensory innervation from the trigeminal nerve (cranial nerve V) via branches of the anterior ethmoidal and maxillary nerves. There are not clearly structurally identified sensory end organs in the nose; however, it is thought that non-myelinated nerve endings in the sub-epithelium mediate the nasal reflexes. Airborne chemical irritants cause discharges in the trigeminal nerves and these responses may be responsible for nasal reflexes such as sneezing and apnoea. The apnoeic reflex is part of the complex diving response, caused by the physiological stimulus of water being applied to the face or into the nose. Apnoea can also be induced by odours or irritants and this response has been identified in all mammalian species. Apnoea is associated with cardiovascular changes and complete laryngeal closure, which occurs as part of the diving response.

Chemical, mechanical stimuli and mediators such as histamine can cause sneezing when applied to the nasal mucosa. Local application of capsaicin, which depletes substance P-containing nerves of their neuropeptide, can prevent the sneeze due to the inhaled irritants, suggesting that non-myelinated nerves may be the receptors. Positive pressure applied to the nose and nasopharynx can stimulate breathing in humans and experimental animals. In addition, nasal irritation can cause bronchoconstriction or bronchodilation by two afferent pathways.

Anaesthetic vapours stimulate the nasal mucosa and elicit nasally mediated reflexes. Enflurane may produce the most marked influence on the breathing pattern. Following the start of insufflation of enflurane or isoflurane into the nose, there is a decrease in tidal volume with a prolongation in the expiratory time. Halothane has the least effect. Inhalation induction of anaesthesia using the volatile agents may be associated with breath-holding, coughing and laryngospasm. It is likely that these reflexes arise from stimulation of upper airway receptors. The nose is an important reflexogenic area, and stimulation of the nasal mucosa may cause some of the most frequently seen airway problems during anaesthesia.

Reflexes from the Pharynx and Nasopharynx

The nasopharynx is supplied by the maxillary nerve (V), and the glossopharyngeal (IX) nerve via the pharyngeal branch provides sensory innervation to the mucous membrane below the nasopharynx. Stimulation of the pharynx and nasopharynx may cause powerful reflexes including hypertension and diaphragmatic contraction.

Reflexes from the Larynx

The innervation of the larynx is by the superior laryngeal nerve (X) and to a lesser extent by the recurrent laryngeal nerves (X). The internal branch of the superior laryngeal nerve contains afferent fibres from the cranial portion of the larynx. The recurrent laryngeal nerve provides afferent innervation to the subglottic area of the larynx. There are many nerve fibres which are thought to be sensory in

almost all areas of the laryngeal mucosa and also in some deeper structures. There have been various types of nerve ending identified in the laryngeal mucosa, including myelinated and non-myelinated fibres in the mucosa and submucosa. The posterior supraglottic region has the highest density of free nerve endings, with the afferent fibres being transmitted via the superior laryngeal nerve. Laryngeal afferent neurones with receptive fields in the epiglottis can be activated by a range of stimuli, including water, but mechanical stimuli are the most effective. The sensory units are thought to consist of free nerve endings that lie between the mucosal cells of the airway epithelium.

Laryngospasm

Laryngospasm is a common and potentially dangerous complication of general anaesthesia. It is defined as 'occlusion of the glottis by the action of the intrinsic laryngeal muscles' and is considered to be present when inflation of the lungs is hindered or made impossible by unwanted muscular action of the larynx.

Laryngeal spasm is essentially a protective reflex to prevent foreign material reaching lower down into the lungs. The laryngeal muscles are striated, the most important muscles involved in the production of laryngeal spasm being the lateral cricoarytenoid, thyroarytenoid (adductors of the glottis) and the cricothyroid (tensor of the vocal cords).

During laryngeal spasm in a human, either the true vocal cords alone or the true and false cords become apposed in the midline and close the glottis. There are thought to be two initiators of laryngeal spasm during general anaesthesia. First, direct irritation of the vocal cords may be caused by a sudden increase in concentration of irritant anaesthetic vapour or direct contact with blood or saliva, and second, by traction on abdominal and pelvic viscera. There are many reports in the literature of the inhalation of irritant vapours producing laryngeal spasm, coughing and bronchospasm. Anaesthetic agents may sensitise the receptors.

This complication is not uncommon and may be life-threatening. In a large study of 156,064 general anaesthetics the overall incidence in all patient groups was 8.7/1000 patients. The incidence was high in children aged between 0 and 9 years, with a peak incidence of 27.6/1000 in infants aged 1–3 months. Animal work has demonstrated laryngeal adductor hyperexcitability in early life and a similar developmental neuronal imbalance may occur in humans.

Other risk factors are a history of asthma or of upper respiratory tract infection (URTI) and smoking. In children with a history of recent URTI the incidence of laryngeal spasm was increased to 95.8/1000

Factors Affecting the Sensitivity of Upper Airway Reflexes

Using low inspired concentrations of ammonia vapour as an irritant chemical stimulus allows study of the upper airway in a repeatable and reliable manner by measuring inspiratory flow patterns. The lowest concentration of ammonia required to elicit a response is termed the threshold concentration (NH3TR). A low value of NH3TR indicates sensitive or reactive airways, whereas a higher NH3TR value represents a reduced upper airway reflex sensitivity and a depression of airway reflexes. Studying the sensitivity of upper airway reflexes in subjects suffering with, and recovering from, a URTI showed increased sensitivity until day 15 (Figure 2.7). This coincided with the presence of symptoms. Upper respiratory tract infections cause acute mucosal oedema followed by shedding of epithelial cells. Loss of airway epithelium can extend down to the basement

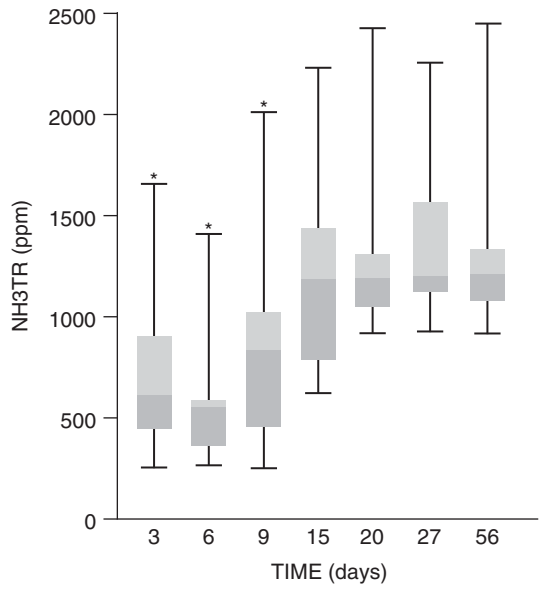


Figure 2.7 The effect of a URTI on upper airway reactivity. Ammonia threshold concentration (NH3TR) in volunteers with an upper respiratory tract infection (URTI) showing the median, interquartile range and the 10th and 90th percentiles $^{**}P < 0.01$ (Wilcoxon). (From Nandwani et al. (1997).)

membrane and may persist for up to 3 weeks. The mechanism for upper airway hyper-reactivity following viral infection may be due to increased exposure of intraepithelial sensory receptors to inhaled irritants. Bronchial reactivity to experimentally inhaled histamine is also increased during a URTI and persists for up to 7 weeks. The high incidence of laryngospasm during inhalation anaesthesia is likely to be due to a direct effect of irritant gases and vapours on the airways. In a cross-sectional study of inhalation anaesthesia the incidence of laryngeal spasm was 12/1000 cases but during isoflurane anaesthesia was 29/1000.

Cigarette smoking also has an effect on the sensitivity of upper airway reflexes (Figure 2.8). Following abstinence, sensitivity is unaltered after 24 hours but then increases over the next 48 hours achieving a consistent change by day 10. It is known that chronic cigarette smokers develop dysplasia of the respiratory epithelium, which may disrupt the integrity of the respiratory epithelium. In addition, smokers have depressed production of salivary epidermal growth factor, which is known to stimulate epithelial proliferation. The evidence for epithelial injury or inflammation causing increased airway sensitivity comes from work on the lower airway reflexes after damaging epithelium mechanically or chemically. Both ozone and acute smoke exposure have been shown to increase tracheal mucosal permeability with increased airway responsiveness. Nebulised lidocaine administered preoperatively, prior to induction of anaesthesia, significantly improved the quality of induction of anaesthesia in smokers.

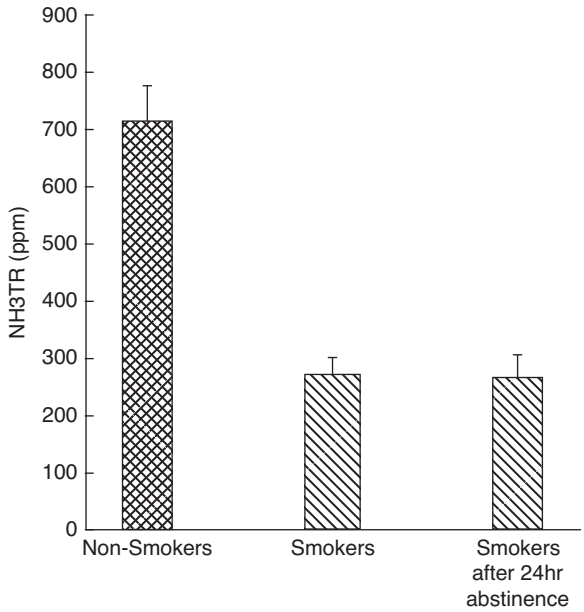


Figure 2.8 Mean (SEM) ammonia thresholds in 20 non-smokers and in 20 smokers before and after 24hr abstinence. ***P =< 0.001. (From Erskine et al. (1994).)

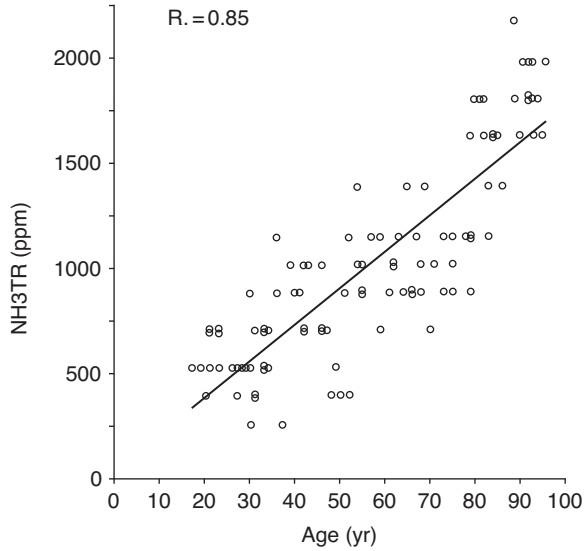


Figure 2.9 Correlation between age and ammonia threshold. Correlation coefficient +0.85. (From Erskine et al. (1993).)

Age affects laryngeal reflexes, with reactivity reducing with advancing age (Figure 2.9). Laryngeal reflexes in the elderly are less active, both during induction of anaesthesia and in the recovery room, compared to a younger patient, suggesting that airway protection may be impaired in the elderly: sensitivity of airway reflexes decreases by a factor of three between the third and ninth decade of life.

Anaesthetic Agents and Laryngeal Reflexes

Inhalation Anaesthetic Agents

The respiratory tract is hypersensitive to stimuli arising during light general anaesthesia. The historical volatile agents ether and halothane produced laryngeal spasm and isoflurane was also irritant. In modern practice desflurane is the most significantly irritant volatile agent, with sevoflurane being the least irritant.

Sevoflurane does not elicit the cough reflex and is the preferential agent for inhalation induction of anaesthesia.

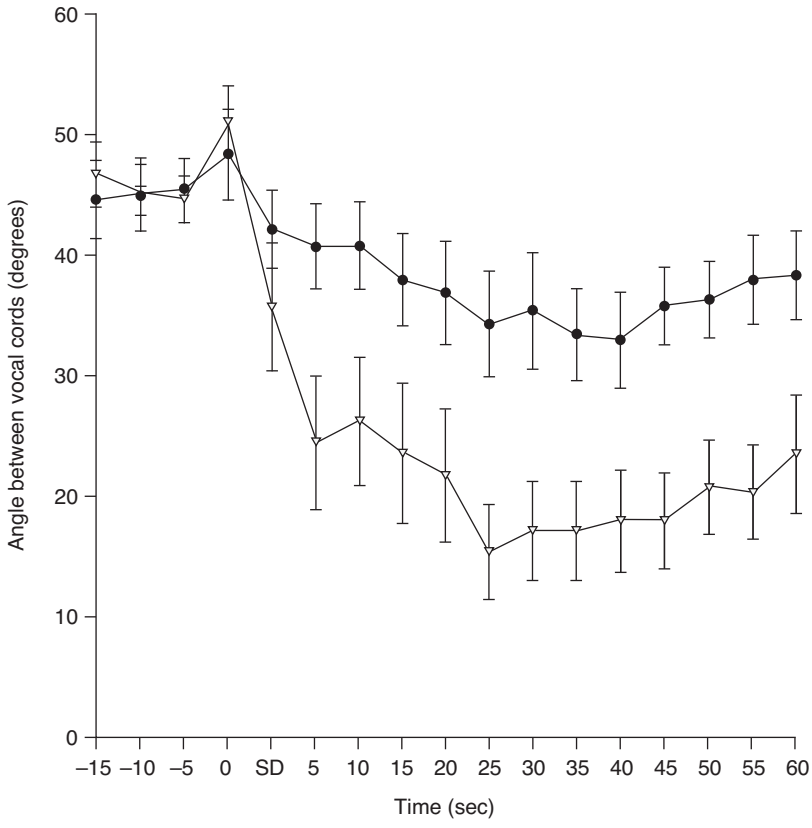


Figure 2.10 Angle between vocal cords after induction of anaesthesia with propofol or thiopentone (mean, SEM). Time 0 = start of the injection of thiopentone or propofol. SD = syringe drop. Circles: propofol; open triangle: thiopentone. (From Barker et al. (1992).)

Intravenous Anaesthetic Agents

Thiopentone

Early work with thiopentone conducted in an animal model found that most of the animals would cough, sneeze or hiccup during thiopentone anaesthesia. Inspection of the glottis in these animals revealed hyperactive adducted vocal cords and lifting the epiglottis elicited complete closure of the glottis. The administration of large doses of atropine (3–5 mg kg^{-1}) would lead to relaxation of the vocal cords and it was concluded that the closure of the glottis following intravenous barbiturates was probably mediated via the parasympathetic nervous system. Following induction of anaesthesia with thiopentone vagal reflexes or increased nerve sensitivity can cause closure of the glottis and a hyperactive state of the laryngeal reflex.

Propofol

Propofol is associated with minimal airway reflex activation. Indeed, the vocal cords may remain abducted enabling tracheal intubation using propofol alone, in contrast to thiopentone after which the vocal cords of > 50% of subjects are closed). Airway manipulation, insertion of airways and laryngeal mask insertion are more easily tolerated following induction of anaesthesia with propofol than other induction agents (Figure 2.10).

Opioids

These depress airway reactivity. Fentanyl has been shown to depress airway reflex responses in a dose-related manner and to reduce desflurane-induced airway irritability. Remifentanil improves the intubating conditions in children during sevoflurane anaesthesia. There have also been many studies showing that remifentanil and alfentanil improve conditions for laryngeal mask insertion and during awake intubation.

Benzodiazepines

Benzodiazepines are widely used to produce short-term sedation and anxiolysis to facilitate endoscopy and minor surgical procedures. However, it is known that these drugs reduce the sensitivity of upper airway reflexes. This may impair the ability of the patient to protect their lower airway from aspiration. Using ammonia challenges, it has been shown that diazepam

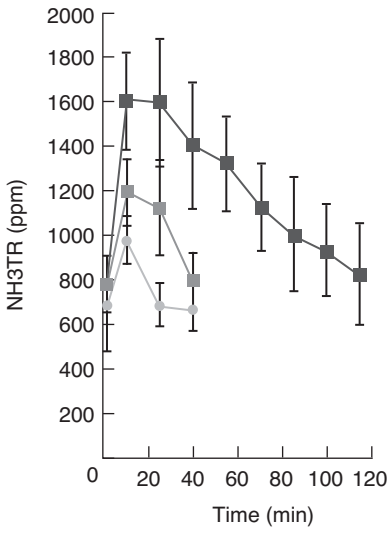


Figure 2.11 Effects of topical vocal cord lignocaine, nebulised lignocaine and oral benzocaine lozenges, on upper airway threshold response to ammonia stimulation (NH3TR) (mean, 95% CI). Black squares represent directly applied lidocaine, grey squares nebulised lidocaine and light grey circles benzocaine lozenges. (From Raphael et al. (1996).).

(0.2 mg kg^{-1}) and midazolam (0.07 mg kg^{-1}) produce significant depression of upper airway reflex sensitivity within 10 minutes of administration, with baseline values regained within 60 minutes. This is significantly reversed by flumazenil (300 μg) administered 10 minutes after midazolam. Oral diazepam has a similar impact on upper airway reflexes from 30 to 150 minutes after administration. Benzodiazepines also impair airway maintenance by reducing the tonic contraction of genioglossus, whose activity is essential to keep the tongue away from the posterior pharyngeal wall. Benzodiazepines should not be considered safe agents in airway obstruction.

Local Anaesthetic Agents

Local anaesthetic agents may be applied to the airway to facilitate awake intubation or to reduce the reflex physiological effects during tracheal intubation and extubation. Benzocaine lozenges produce a significant effect within 10 minutes returning to normal within 25 minutes. Directly applied lidocaine produces a significant effect lasting 100 minutes and nebulised lidocaine lasts 30 minutes.

Intravenous lidocaine 1.5 mg kg^{-1} (when plasma concentrations were > 4.7 μg mL^{-1}) reduced responses to tracheal irritation to only brief apnoea, and other reflex responses were completely suppressed.

Summary: Reflexes

Upper airway reflexes are important to anaesthetists. Anaesthetic agents produce changes in the sensitivity of upper airway reflexes. Propofol is associated with depression of upper airway reflexes. Ageing leads to a gradual reduction in sensitivity of upper airway reflexes. Cigarette smoking increases the sensitivity of upper airway reflexes, a change which persists for up to 2 weeks following cessation of smoking.

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Chapter 3

The Epidemiology of Airway Management Complications

Johannes M. Huitink and Tim Cook

Overview

Anaesthetists and others who manage the airway are trained airway specialists who strive to prevent harm to our patients, but airway management complications may still occur. Sometimes these happen because of patient factors, sometimes because management is suboptimal and, most often, because of a combination of these two. Recent developments and technology should have made anaesthesia safer but complications during airway management still lead to consequences such as cancelled operations, unplanned intensive care admission, airway trauma, brain damage or even death. Modern anaesthesia has become considerably safer with mortality attributable to anaesthesia falling at least 10-fold in the last few decades. Improvements in airway safety almost certainly contribute to this.

How much airway complications contribute to anaesthesia-attributable mortality and morbidity is unknown and will vary considerably according to the location. In well-resourced environments mortality is low: a Japanese study reported anaesthesia-related deaths in a low-risk population to be 10 per million, 40% of which were airway related. In contrast, in a low-resource environment, in Togo, post-operative mortality rate was 1 in 38 at 24 hours; 93% of deaths were judged avoidable with half attributed to anaesthesia including 30% due to respiratory management. It is likely that much airway-related morbidity and mortality is avoidable, but only with availability of trained personnel and sufficient equipment. Airway complications related to airway management performed outside the operating room (OR) environment are several-fold more common than in the OR. Consequently, although only a modest proportion of airway management takes place in the intensive care unit (ICU) up to a quarter of major events occur in that location.

Details of many specific complications, avoidance strategies and management plans are included in other chapters, so this chapter provides an overview of complications occurring during airway management, focussing particularly on epidemiology and patterns.

Despite almost universal concern amongst anaesthetists about avoidance of airway complications the reality is that serious complications are infrequent enough that individual practitioners will only encounter complications infrequently. For that reason, databases likely provide the most reliable sources of information on complications. In this chapter we will focus on information from clinical practice databases including

- the UK 4th National Audit Project: major complications of airway management (NAP4)
- a Dutch prospective database of airway complications (mini-NAP)
- data from litigation databases including that of the UK National Health Service NHS Litigation Authority (NHSLA) and the USA American Society of Anesthesiology Closed Claims Project (ASACCP).

Common Airway Complications – Difficulty and Failure

Incidences of airway management failure will vary depending on definitions, operator experience and the patient population examined: difficult laryngoscopy occurs in 1 in 16 unselected elective patients but this rises to 1 in 5 in patients having cervical spine surgery. Failure and complications occur disproportionately commonly in ICU and in the emergency department (ED) where failure may be 10-fold higher than during anaesthesia.

Common complications of airway management are listed in Table 3.1. Difficulty and failure of a primary airway procedure should be considered complications, especially as they inevitably precede the vast majority of airway complications leading to

Table 3.1 Estimates of rates of airway procedural failure derived from the literature. None of these rates are 'hard and fast': rates may be much higher in selected groups, in emergency settings, in the hands of inexperienced airway managers or in low-resource settings

Complication	Location or setting	Approximate incidence
Difficulty		
Face mask ventilation	Anaesthesia	1 in 50–100
Ventilation via SGA	Anaesthesia	1 in 10
Intubation (low-risk group)	Anaesthesia	1 in 18
Intubation (high-risk group)	Anaesthesia	1 in 5
Face mask ventilation and laryngoscopy	Anaesthesia	1 in 250
Intubation	ED	1 in 12
Intubation	ICU	1 in 3
Failure		
Face mask ventilation	Anaesthesia	1 in 600
Ventilation via SGA	Anaesthesia	1 in 50
Intubation	Anaesthesia	1 in 200–1500
	ICU	>1 in 100
	ED	>1 in 100
CICO	Anaesthesia	1 in 5000
Front of neck airway	Anaesthesia	1 in 50,000
	ED	1 in 400

CICO, cannot intubate, cannot oxygenate; SGA, supraglottic airway.

patient harm. Failed tracheal intubation likely occurs at some point in almost every airway-related fatality.

Risk factors for failure of airway management and complications are described in Table 3.2.

Procedural difficulty is also associated with secondary complications – including airway swelling, trauma, pulmonary aspiration and development of airway obstruction and the cannot intubate, cannot ventilate/oxygenate (CICV/CICO) situation. Avoidance of primary difficulty is therefore at the heart of complication avoidance.

'Composite Airway Failure'

It is essential to understand that in a patient in whom one airway technique fails, the risk of failure of other techniques is increased – so called 'composite failures'.

- After failed face mask ventilation intubation failure increases more than 10-fold.
- After failed intubation, face mask ventilation fails in approximately 1 in 10.
- After SGA insertion the risk of difficult mask ventilation rises threefold.

To minimise the risk of complications, when one technique is predicted to be difficult it is important to focus carefully on assessing the likely ease of other techniques that may be used for rescue.

National Level Complications: Lessons from NAP4

NAP4 examined major complications of airway management in the UK. This lengthy document cannot be adequately summarised here and is signposted in the Further Reading.

The aims of NAP4 were:

- To examine the extent of major complications of airway management
- To characterise these problems
- To capture recurrent themes and causes
- To make recommendations to improve airway management at national, organisational and individual levels. The former two are the basis of *institutional preparedness* and the latter of *personal preparedness.*

Key Points from NAP4

NAP4 was a 1-year national registry of major airway complications during anaesthesia and in ICU or ED. It only included cases leading to

- death
- brain damage
- emergency front of neck airway (eFONA)
- ICU admission or prolongation of ICU stay

Table 3.2 Risk factors for airway complications

Factor	Note
Difficult airway	While seemingly obvious that a difficult airway is a cause of complications it is more complex. Approximately half of difficult intubations are not predicted. Tests have low sensitivity. Previous difficult intubation is the best predictor of future difficult intubation and should never be ignored. Most airway complications, however, occur in patients who are not predicted and may not have anatomically difficult airways
Obesity	Obesity is repeatedly identified as a risk factor for difficulty for all types of airway management in all settings. Increased risk likely starts as low as BMI 35 kg m^{-2}. Reduced safe apnoea time and progression to severe hypoxaemia is the greatest factor
Emergency	Urgency of airway management and factors such as use of cricoid force increase risk of failure and complications up to 10-fold
Outside OR	All locations outside the OR are associated with a marked increase in risk of failure and complications. Multiple reasons are described in the text
Head and neck surgery	The combination of airway abnormality due to disease and treatment increases risk many-fold. The need for a shared airway and blood in the airway at extubation add further difficulty
Reduced mouth opening	Decreases access for laryngoscope, SGA and airway adjuncts
Reduced neck movement	Reduces mouth opening. Increases difficulty in optimal positioning for FMV and intubation. Hyperangulated videolaryngoscopy is useful to overcome the problem
Previous radiotherapy	Increases difficulty in optimal positioning for FMV and intubation. Often associated with reduced neck movement. Hinders anatomical recognition and performance of eFONA
Repetition of failing technique	Repetition of the same airway technique that has already failed is illogical – after a failed attempt at intubation subsequent attempts have an approximately 80% failure rate – but is consistently seen in airway disasters. Also termed 'failure to transition' to describe the failure to move to the next step of the airway algorithm
Lack of strategy	Safe airway management requires a series of plans each consequent on the failure of the preceding plan. A lack of a strategy, communicated to all, leads to repetition of failing techniques and chaotic airway management
Communication issues	Common in airway disasters. Failure to ensure a strategy is understood by all involved, including the transition points
Poor decision making	This often involves choosing a poor primary plan and lacking a strategy for failure. During difficulty, a combination of repetition, missing algorithm steps and using unfamiliar techniques is often seen
Untrained personnel	Training and knowledge are fundamental prerequisites for avoidance of airway complications. Training is not the same as seniority – seniors are more commonly involved in airway mismanagement than junior staff

BMI, body mass index; eFONA, emergency front of neck airway; FMV, face mask ventilation.

As such it only captured the airway events with the worst outcomes, the very 'tip of the iceberg' – lesser complications or 'rescued events' were not captured.

A concurrent denominator survey enabled a national incidence of events to be calculated (Table 3.3). NAP4 studied the complications of approximately 3 million anaesthetics. There were 133 anaesthesia events, 36 in ICU and 15 in ED.

Amongst ≈3 million general anaesthetics, there were 16 airway-related deaths and 3 cases of persistent brain damage. Incidences are reported in Tables 3.3 and 3.4 according to the degree of injury. The rarity of such events is noteworthy and is a key reason why assessing safety in airway management is so difficult – no individual's or department's practice is likely to shed light on high level safety and this is also true for almost any randomised clinical trial (RCT).

Important themes identified in NAP4 included:

- Omitting an assessment of potential airway difficulty and risk of aspiration, and the failure to tailor the anaesthetic technique appropriately, contributed to poor outcomes.
- Poor planning and 'failure to plan for failure' were common in events. Responses to unexpected

Table 3.3 Point estimates for anaesthesia airway complications in NAP4

	Risk of event	
Included event	46 per million	1:22,000
Death	5.6 per million	1: 180,000
Death and brain damage	6.6 per million	1: 150,000

Table 3.4 Point estimates of risk of complications by airway device in NAP4

Primary airway device	Events	Death and brain damage
Any	1:22,000	1:150,000
Tracheal tube	1:12,000	1:110,000
Supraglottic airway	1:46,000	1:200,000
Face mask	1:22,000	1:150,000

difficulty and failure were unstructured. Airway managers started with only a single plan. Airway *strategies* were advocated; a logical sequence of plans, designed to manage failure at each step and to achieve oxygenation, ventilation and prevent aspiration.

- Difficult or failing techniques were regularly managed with repeated attempts, especially tracheal intubation. This was associated with deterioration from a 'cannot intubate, *can* oxygenate' to a 'cannot intubate, *cannot* oxygenate' (CICO) situation. NAP4 strongly advocates adopting a limited number of attempts as part of any strategy.
- Decisions made and techniques chosen were sometimes illogical, including using routine care in cases of known difficulty and avoiding awake intubation when strongly indicated. Lack of judgement, skills, experience, confidence and equipment all contributed.
- Anaesthetists often used 'their usual technique' when this was not in the patient's interest. Best care may require involvement of colleagues with other skill sets.
- Quality of care was judged to be 'poor' or 'good and poor' in three quarters of cases. In a secondary study, human factors were identified in all cases (mean of four factors per case). Poor judgement, education and training were the most common contributory factors.
- Delayed, difficult or failed intubation was the primary event in almost half of reports and intubation difficulty and failure likely occurred at some point in all cases.
- SGAs (most often first generation) were used in the face of high aspiration risk or marked obesity and aspiration followed. Use by junior doctors and accepting a poorly functioning airway were themes in non-aspiration SGA events. Use of an SGA to avoid anticipated difficult tracheal intubation, without a rescue plan or strategy, was followed by problems: unstructured management of difficulty followed and some of these patients died. Fatalities would likely have been avoided by an awake intubation technique or tracheal intubation through the SGA (see Chapter 13).
- Obese and morbidly obese patients were over-represented throughout NAP4. This finding has been replicated and reinforced in other important studies (see Chapter 24).
- Head and neck cases accounted for 40% of all anaesthesia cases and the need for multidisciplinary communication and senior anaesthetic and surgical involvement was emphasised.
- Many reported cases involved the obstructed airway. CICO was common in these. Human factors were plentiful including poor planning, communication, equipment, teamwork and situation awareness. Awake tracheostomy was too infrequently considered. When problems occurred transition to eFONA was often slow, even when part of the strategy.
- Transition to eFONA when required was often delayed and eFONA often failed.
- In anaesthesia events aspiration was the commonest cause of death (51% of reports of death or brain damage). Half of these cases involved tracheal intubation. Poor judgement and ignoring risk assessment were causative in many cases.
- Unrecognised oesophageal intubation occurred in all locations and accounted for 1 in 16 cases. It was emphasised that harm from oesophageal intubation is preventable by capnography, even in cardiac arrest. (This is discussed further below.)

- One quarter of events took place during emergence and recovery: all were associated with airway obstruction and many with post-obstructive pulmonary oedema. Blood in the airway and a suboptimal airway during maintenance were common precipitants.

ED and ICU

- A quarter of airway events occurred in the ICU or ED. Estimates are that the rate of events leading to death or brain damage were, compared with anaesthesia, 35-fold higher in ED and 55-fold higher in ICU.
- Permanent harm or death followed 61% of ICU reports, 33% of ED reports and 14% of anaesthesia reports.
- In the ICU, much morbidity and mortality followed airway displacement, especially of tracheostomy and in obese patients. Delayed recognition and lack of a structured plan for such an event was prominent (see Chapter 28).
- In the ED, most complications followed rapid sequence induction.
- Suboptimal care, including preventable deaths, was especially common in ICU and ED reports. Issues included not recognising at-risk patients; poor planning; inadequate or inaccessible skilled staff and equipment; slow recognition of problems; unstructured responses; and poorly prepared institutional and individual strategies for managing predictable airway complications.
- Failure to use capnography in ventilated patients or to interpret it correctly (and consequent failure to identify airway displacement or misplacement) contributed to more than 70% of ICU-related deaths.

Lessons from Databases and Registries

Airway-related databases and registries (whether permanent or short term as part of a trial) are sources of useful information but differ from routine databases that generally gather data about a large number of routine cases. Registries more often collect smaller datasets relating to a focussed area, e.g. patient populations, diseases, procedures or complications. There is a degree of overlap between databases and registries. Both can provide useful information about patterns of airway complications and in some cases detailed information.

Databases that include information from routine cases are useful as they generally create a complete dataset and this puts rarer complications into context and provides a denominator enabling calculation of incidences. However, they need to be very large to gather sufficient numbers of cases of interest to be useful. Limitations include: the effort required to collect such a volume of data; that the data is often collected for other purposes (e.g. for financial or administrative purposes) so that clinical information may be of secondary purpose, leading to omissions or perverse associations; databases from a single or atypical institution may not be generalisable. Selective databases and registries are more focussed, and a greater proportion of cases are likely relevant to those interrogating the dataset. This provides economy of effort in collection and analysis. Their limitations include that: they lack denominators so cannot (by themselves) provide incidences; and the methods by which cases are captured may lead to uncertainty over whether all cases are included.

A range of airway-related databases have been established in the last decade and they are now starting to provide important data about rates of complications and identifying risk factors for harm and insight into the efficacy of various rescue techniques. Some examples are listed in Table 3.5.

For both databases and registries, the expansion of digital capacity over the last decade has been a huge benefit but with this comes responsibility for protection of personal data and effective information governance.

Notable findings from these databases (in some cases contradicting findings from RCTs and meta-analyses) are the identification of higher rates of airway difficulty than previously reported and the poor predictability of difficult tracheal intubation (NAP4), the clinical importance of avoiding multiple intubations (Sakle's group and APRICOT) and the value of videolaryngoscopy for airway rescue in children (PeDI and APRICOT).

Lessons from Fatalities and Sentinel Cases

Airway complications leading to fatality are often reviewed at multiple levels from local, regional, coronial, legal or even at a national registry level. They may appear in media or academic publications. Other than registries and national audits, such reviews are

Table 3.5 A selection of useful airway databases providing information on the epidemiology and patterns of airway complications and their management

Database or registry	Area of practice and year started	Data source	Notes
DAD The Danish Anaesthesia Database	Anaesthesia (Denmark) 2012	Routine data from > 70% of cases nationally	Detailed data on > 600,000 cases. Useful incidences and risk factors of e.g. difficult mask ventilation, difficult intubation and eFONA
NAP4 4th National Audit Project	Anaesthesia, ED, ICU (UK) 2009	One-year registry of all UK hospitals	Captured complications of 2.8 million anaesthetics. Contemporaneous denominator survey enabled incidence and risk factor determination https://www.nationalauditprojects.org.uk/NAP4_home
APRICOT (Anaesthesia PRactice In Children Observational Trial: European prospective multicentre observational study: Epidemiology of severe critical events)	Paediatric anaesthesia (Europe) 2014	One-off collection of routine data from > 30,000 paediatric anaesthetics in 250 hospitals	A large database trial exploring the incidence of severe critical events during and immediately after anaesthesia http://www.esahq.org/apricot
ASACCP American Society of Anesthesiologists Closed Claims Project	Anaesthesia (USA) 1984	Rolling database of closed litigation cases from USA	Database covers perhaps 50% of litigation claims, with considerable temporal delay. Not restricted to airway topics https://www.aqihq.org/ACCMain.aspx
University of Arizona College of Medicine registry	ED (USA) 2007	Single centre database of all ED intubations	Run by Drs J Sakles and J Moiser. Well reported and comprising > 6000 cases.
NZEMN-ANZEDAR New Zealand Emergency Medicine Network-Australian New Zealand Emergency Department Airway Registry	ED (Australia/New Zealand) 2015	Database of all intubations in ED in 40 + contributing units	http://www.thesharpend.org/airway-registry
NEAR National Emergency Airway Registry	ED (USA, Canada, Singapore) 2003	Database of all intubations in ED in > 20 contributing units	Including data on 30,000 intubations http://www.nearstudy.net
NEAR4KIDS National Emergency Airway Registry for Children	Paediatric ICU (USA, Canada) 2010	22 specialised paediatric hospitals	Collecting data on all tracheal intubations and analysing risk and incidence of difficulty and including > 2000 difficult intubations http://www.near.edu/near4kids/welcome.cfm

Table 3.5 (cont.)

Database or registry	Area of practice and year started	Data source	Notes
PeDI Pediatric Difficult Intubation registry	Paediatric anaesthesia (USA) 2012	13 children's hospitals in the USA	North American registry of difficult intubation in specialised centres with data on > 1000 difficult intubations
PeAR (PaEdiatric Airway Registry)	Paediatric anaesthesia (Europe) 2019	UK initially, expanding to Europe	A recently established registry of difficult airway management (focussing on laryngoscopy) in paediatrics https://w3.abdn.ac.uk/clsm/pear/home.aspx
The Airway App	*Specific to FONA* Anaesthesia/ED/ICU/PHEM (Global) 2016	Self-reported cases	A novel open platform for reporting anonymised data on eFONA either on-line or using a smartphone app. Currently with approximately 200 cases http://www.airwaycollaboration.org/
RCoA-DAS FONA database	*Specific to FONA* Anaesthesia/ICU/ED (UK) 2020	Self-reported cases	Due 2020

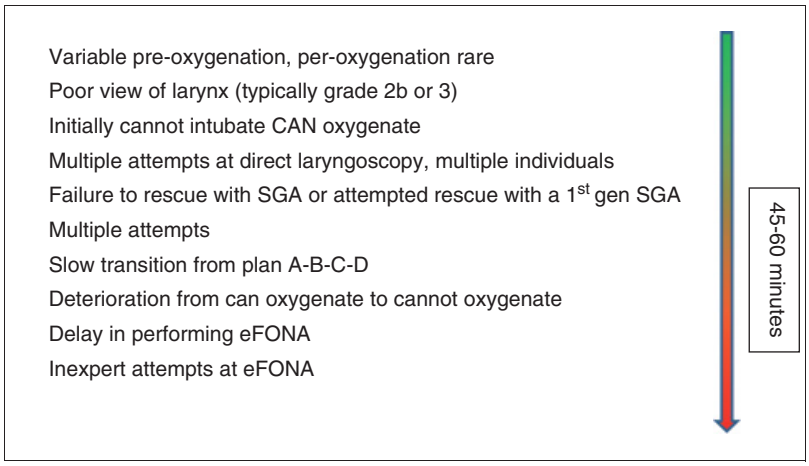

Figure 3.1 A typical timeline for the development of a major complication of airway management. Notable features include an airway that initially appears manageable, repeated attempts at the same procedure, failure to progress through an airway algorithm and development of the CICO situation. (Reproduced with permission from Wiley from Cook TM. Strategies for the prevention of airway complications – a narrative review. *Anaesthesia* 2018; 73: 93–111.)

usually single cases analysed by individual clinicians. Learning may or may not be of value. Through systematic collection of these cases and analysis common themes can be identified (as illustrated in Figure 3.1): NAP4 was an example of such a process.

Occasionally cases arise of such prominence that their analysis fosters national or international learning and those of Elaine Bromiley (https://emcrit.org/wp-content/uploads/ElaineBromileyAnonymousReport.pdf) and Gordon Ewing (https://www.scotcourts.gov.uk/search-judgments/judgment?id=328 e86a6-8980-69d2-b500-ff0000d74aa7) are two such cases where the analyses of their deaths have learning potential for all airway managers.

A limitation of 'fatal case review' is that as much or more may be learnt from reviewing cases of difficulty that were resolved with a favourable outcome. This rarely happens.

Lessons from Litigation

Another source of knowledge about major complications of airway management is from litigation databases and analyses of these are available from the USA, UK and Denmark. The strength of these analyses is that they focus on events that are important enough to patients to initiate litigation. There are several major weaknesses to such analyses: cases are often at least a decade old; the trigger for litigation may be unrelated to severity of injury or negligence; there is no denominator so incidences of events cannot be calculated. Providing these limitations are understood the data may be of significant value.

Anaesthesia is a low-litigation specialty (with claims some 40-fold less than against surgeons and obstetricians) and airway-related claims account for fewer than 10% of all anaesthesia claims. However, they are especially important because amongst anaesthesia claims they are among those strongly associated with the greatest patient harm and the greatest medicolegal costs and they often affect young patients, many of whom do not have predicted difficult airways. Understanding the claims illustrates avoidable harm within cases and teaches avoidance strategies (see Further Reading).

Table 3.6 provides a comparison of the distribution of litigation related to airway trauma in the American and UK legal systems.

Lessons from a Departmental Level: 'Mini-NAP4'

Even a moderately large hospital will only deliver approximately 15,000–20,000 anaesthetics per year and most anaesthetists will anaesthetise in the range of 250–500 patients per year. This means that local data collection systems are unreliable for collecting data on the most serious and uncommon events. However, it also means they are well placed to collect data on more frequent and so-called 'minor events': these are often minor in the opinion of anaesthetists but are of potential interest because they are the precursors of more serious events, are important to patients and may have health economic importance. There is surprisingly little published in this area.

Table 3.6 Comparison between claims of airway trauma reported in the American Society of Anaesthesiologist Closed Claims Project (ASACCP) in 1991, in 1999 and those notified to the UK National Health Service Litigation Authority (NHSLA)

	ASACCP 1991	ASACCP 1999	NHSLA 1995–2007
Percentage of all anaesthesia claims	5%	6%	3%*
Deaths	12%	8%	14%
Payments to claimant	60%	54%	61%
Laryngeal injury	33%	33%	36%
Pharyngeal injury	14%**	19%	32%
Oesophageal injury	14%**	18%***	14%****
Difficult airway	42%	39%	9%*****

* Denominator adjusted to exclude dental damage (as per ASACCP).

** Pharyngeal and oesophageal injuries were 28% combined, but were not subdivided: a 50:50 split is assumed.

*** 90% were perforations.

**** All were perforations.

***** Likely an underestimate of true incidence due to methodology.

From Cook TM, Scott S, Mihai R. Litigation following airway and respiratory-related anaesthetic morbidity and mortality: an analysis of claims against the NHS in England 1995–2007. *Anaesthesia* 2010; 65; 556–563. Reproduced with permission from Wiley

The 'mini-NAP4' study (see Further Reading) is an example of a study that collected such data. All cases were studied prospectively: researchers interviewed all anaesthetists at the end of every day to identify any problems and all cases where hypoxia occurred (identified in the central theatre monitoring system) led to investigation of the cause. Episodes were divided into airway problems (unavoidable events not leading to harm) and complications (avoidable events with the potential for or causing actual harm). Degree of harm was graded, with 'serious' harm equivalent to NAP4 entry criteria. The results are shown in Table 3.7, which includes potential changes that might be made to mitigate these complications. Events such as hypoxia in 1 in 64 cases, intubation difficulty in > 1 in 100 cases, accidental oesophageal intubation and airway obstruction in 1 in 561 cases and CICO in 1 in 2803 case are all notable. Twenty-four events (0.9%) would have triggered inclusion in NAP4 including 1 death, 1 CICO, 2 eFONAs and 12 unplanned ICU admissions. Intubation difficulty accounted for 23% of events, failed mask ventilation 3%, aspiration 1.8% and laryngospasm for 7%. Of note events were most common in healthy patients and in males, those aged > 40 or < 10 years and in patients with an elevated body mass index (BMI) (half of events in patients with BMI > 26 kg m^{-2}). More than two thirds (69%) of events occurred at induction, 12% during maintenance and 14% after surgery.

This is an important study and similar studies are underway in other countries. In an ideal world every department, and perhaps every airway manager, would know their own rate of such events.

Timing of Complications

More than half of airway problems and complications occur during induction of anaesthesia. However, up to one in five major airway complications occur during emergence and recovery and this period requires due care, especially in higher-risk patients including patients with blood in the airway. Post-operative complications are less frequent but for example late airway swelling may be difficult to manage and should not be underestimated.

Specific Complications

Oesophageal Intubation

If there is one complication above all that airway managers should seek to avoid it is perhaps unrecognised oesophageal intubation. When an intended tracheal tube is inadvertently placed in the oesophagus, this will result in severe hypoxaemia, followed by brain injury or death if it is not recognised and corrected within 3–5 minutes. In modern healthcare many would think such an event would be inconceivable,

Table 3.7 Incidence of airway management problems and complications derived from a continuous prospective 2-month study of 2803 patients at an academic teaching centre in the Netherlands

Event (N=2803)	Occurrence	Incidence (%)	Incidence (odds)	Potential solutions
Desaturation < 93%	44/2803	1.57%	1:64	HFNO
Unanticipated intubation problems	29/2803	1.03%	1:97	Triage
Bronchospasm	12/2803	0.43%	1:234	–
Unanticipated ICU admission because of airway complications	12/2803	0.43%	1:234	Triage
Laryngospasm	11/2803	0.39%	1:255	–
No seal with SGA	6/2803	0.21%	1:467	Second generation SGA
Accidental oesophageal intubation	5/2803	0.18%	1:561	–
Airway obstruction	5/2803	0.18%	1:561	–
Cannot mask ventilate	5/2803	0.18%	1:561	Triage
Aspiration	3/2803	0.11%	1:934	Triage, second generation SGAs, gastric ultrasound
Emergency surgical airway	2/2803	0.07%	1:1402	CICO kits
Blood clots in airway	2/2803	0.07%	1:1402	–
Epistaxis	1/2803	0.04%	1:2803	–
Death	1/2803	0.04%	1:2803	Triage
Cannot intubate, cannot oxygenate	1/2803	0.04%	1:2803	CICO kits
Dental injury	1/2803	0.04%	1:2803	–

HFNO, High flow nasal oxygen.

but in NAP4 unrecognised oesophageal intubation accounted for 6% of reports and eight deaths. Such deaths continue to be reported.

Most clinical tools to detect a misplaced tracheal tube are unreliable and this includes tube 'misting' and auscultation of the chest. However, capnography is close to 100% sensitive, such that whenever the capnograph trace is flat the tube should be assumed to be in the oesophagus until this possibility has been actively excluded. Importantly, even during cardiac arrest ventilation via a correctly placed tracheal tube will lead to a visible (attenuated) capnograph trace. In the UK a 'no trace wrong place' campaign has been launched to highlight the role of capnography in detecting oesophageal intubation (https://www.youtube.com/watch?v=t97G65bignQ&t=15s). Use of waveform capnography in all settings where intubation is performed, allied with appropriate training, has the potential to eliminate this complication. In skilled hands ultrasonography can also be used to rapidly detect oesophageal intubation (see Chapter 7).

Pulmonary Aspiration of Gastric Contents

In NAP4 (in line with previous historical reports) pulmonary aspiration was the commonest primary cause of anaesthesia reports, of deaths and of brain damage. Most cases included avoidable harm and were contributed to by poor judgement or educational deficit. Approximately half of the cases involved tracheal intubation (often involving inadequate preparation for management of the full stomach) and half involved SGAs (including use in the morbidly obese, in patients with full stomachs and a preponderance of cases were with first generation SGAs).

Avoidance of aspiration starts with assessment of the risk of aspiration and where such risk is identified it is essential that the anaesthetic technique chosen is appropriate. The advent of gastric ultrasound may prove to be an opportunity to make an important impact on this complication. The subject is discussed in Chapters 7 and 11.

High-Risk Patient Groups

Obesity

NAP4 shed a bright light on obesity as a risk factor for major airway complications. Both the obese and morbidly obese were significantly over-represented (two- to fourfold) in the incidence of complications and in all locations. Numerous other studies have confirmed these findings. Obese patients may have distortion of the oropharynx, limited neck extension and a higher incidence of co-morbid conditions: obesity is a risk factor for difficult mask ventilation, SGA insertion, intubation and eFONA procedures. While the difficulty of individual airway procedures likely increases with obesity, the overriding risk in obese patients is from increased likelihood of airway obstruction and the dramatically increased rate of hypoxia during apnoea. All airway managers should treat obese patients with a heightened caution. This is discussed further in Chapter 24.

Head and Neck Surgery

NAP4 also highlighted head and neck surgery as a key area of increased risk, accounting for more than 40% of all reported cases. Chapters 25 and 26 discuss this topic in detail.

Complications Related to Specific Airway Devices

Face Mask Ventilation

Failure is the greatest complication here, though poor technique may also precipitate gastric inflation, regurgitation and aspiration of gastric contents. When mask ventilation fails it may start a spiral of hypoxaemia and other airway failures. Where difficulty occurs, there is convincing evidence that muscle paralysis improves ease of mask ventilation.

Supraglottic Airways

The rate of complications from SGAs is low. Soft tissue injuries from the SGA include tongue ischaemia and neuropathies including unilateral and bilateral recurrent laryngeal, hypoglossal and lingual nerve injuries. These neuropraxias manifest as vocal cord paralysis, hemitongue paralysis and tongue anaesthesia, respectively. Most resolve over several weeks to months. The mechanism is likely to be direct mechanical compression of the nerve by the SGA. While it is logical that reduced SGA cuff pressures will reduce these events, they are so rare that this is not practical to test. Nitrous oxide diffuses into the SGA cuff, particularly if it is silicone.

Best practice is to

- select the appropriate size of SGA
- monitor SGA cuff pressure and avoid cuff pressures > 60–70 cmH_2O
- when using nitrous oxide, routinely measure cuff pressure after 20 minutes and again if the concentration of nitrous oxide is increased
- avoid extreme neck positions, especially extension
- use extreme caution if surgery is very prolonged, opinions differ as to whether 3 or even 8 hours are safe limits

Direct Laryngoscopy

A single attempt at direct laryngoscopy is rarely injurious. However, multiple attempts lead to airway swelling and may progress to development of a CICO situation (see Figure 3.1). Tracheal intubation should be undertaken with great care. Direct vocal cord injury and arytenoid dislocation are injuries that can permanently damage the patient's voice, which may have significant implications for them. Use of a small tube (e.g. 6.0–6.5 mm ID), with an atraumatic tip, and placement first time with an optimal view of the larynx will logically reduce these complications, the latter perhaps being an argument for routine use of videolaryngoscopy. Vocal cord granulomas have been reported to be as common as 1% after tracheal intubation, but it is hoped this is of historical relevance.

Bougies, Exchange Catheters and Stylets

When bougies and stylets are used for tracheal intubation, the risk of injury is increased. Perforation of the pharynx, oesophagus and trachea are all described (see Table 3.6). These may lead to mediastinitis and can be fatal. Use of soft and non-rigid bougies, avoidance of use of the hold-up sign and ensuring a rigid stylet never enters the glottis are all important precautions to prevent such wholly avoidable complications.

When bougies and exchange catheters (Aintree intubation catheters, airway exchange catheters) are used it is important that they are not inserted beyond the carina (23–25 cm from the lips). This is particularly important if oxygen is administered

via these devices (though that is generally not necessary) as there is a major risk of barotrauma if the exchange catheter wedges in the bronchial tree (see Chapter 15).

It is possible for plastic particles to be sheared off intubation catheters during railroading, especially if a large exchange catheter is used with a smaller tracheal tube. The clinical relevance of this is unknown but it is undesirable and avoidable.

Videolaryngoscopy

The most common complication specific to videolaryngoscopy is injury to the soft palate or posterior pharynx.

The mechanism of injury with the videolaryngoscope differs from that with traditional intubation; with videolaryngoscopy a hyperangulated blade or the styletted tube may be advanced without direct observation into a 'blind spot' in the posterior pharynx where it is not seen directly or by video and leads to tissue trauma. Such injury is reduced (or eliminated) by directly watching the blade or tube enter the airway as far as is possible and then immediately switching to the video screen to watch its further progress. Ensuring the styletted tracheal tube runs along the blade of the hyperangulated videolaryngoscope will not only reduce the risk of injury to posterior structures but also facilitate successful intubation (see Chapter 17).

If lacerations do occur, mild ones can be managed conservatively, whereas more substantial injuries may benefit from haemostasis and primary repair.

Double-Lumen Tube

Because of their size and relative difficulty in placement double-lumen tubes (DLTs) are associated with a greater risk of complications. Airway trauma, including vocal cord damage, dental damage, cuff damage or airway bleeding, is a particular issue.

Videolaryngoscopy and DLTs with an integrated camera within the DLT both have the potential to reduce complications but more studies are needed to confirm this.

Tracheostomy

Tracheostomies are perhaps the airway with the greatest risk of complications. Complications may occur at placement, but this is perhaps overstated. More important is the risk of complications during use with displacement (especially in critical care and in patients whose lungs are ventilated) and obstruction (especially on the wards) being prominent problems in critical airway events. These are discussed in Chapter 29.

Emergency Front of Neck Airway (eFONA)

eFONA is the final common pathway of all airway algorithms. When a needle-based technique is used the greatest procedural risks of harm are from failure and from barotrauma during high-pressure source ventilation and this increases dramatically in an emergency setting (see Further Reading). When a scalpel-based technique is used the greatest procedural risks are failure and bleeding. However, by far the commonest complication is hypoxaemic brain damage or death due to delay: whether outside or in hospitals, patients do not die from eFONA but from delay or failure to perform it.

Complications by Anatomical Location

Mouth and Oropharynx

Dental injury during laryngoscopy, often due to pressure on the maxillary incisors, is one of the most common complaints against anaesthetists: however, in a recent prospective study dental injury occurred in only 1:2803 cases. A careful history and documentation of the condition of dentition preoperatively, communication of risk and appropriately gentle care are all essential.

Minor bruising and lacerations to the lips, buccal mucosa, floor of mouth, palate, uvula and tongue are all relatively common, caused by direct tissue trauma from airway devices, especially the laryngoscope or tracheal tube. Careful technique during airway manipulations should reduce the incidence of injuries. Injuries should be documented and discussed with the patient.

Hyperangulated videolaryngoscopes enable an easier view of the larynx with lesser force exerted on soft tissues; whether this translates to lesser minor soft tissue injury is unknown.

Nasal Cavity

Epistaxis during nasotracheal intubation or instrumentation is relatively common (30–50%) and vasoconstrictors are recommended. It is usually self-limiting but severe blood loss has been reported. With forceful