Fine lingerie itself is rather tedious: it is the context that makes it exciting. The same is true for anatomy: topology alone is for idiots-savants. The following lines instead offer a selective account of the functional anatomy of the adult head, neck and airway as it applies to anaesthetic clinical practice.

The mouth

The mouth is dominated by the tongue, a muscular instrument of pleasure – gastronomic and linguistic. For anaesthetists, little else counts but its size. It may be swollen acutely (as in angioneurotic oedema), but is also susceptible to disproportionate enlargement in trisomy 21, myxoedema, acromegaly 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 percutaneous subglottic 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 allow a direct view of the glottis. If the tongue is too large, or the jaw hypotrophied, it may not be possible to see the glottis over the compressed tongue.

Within the mouth, 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 trapdoors, wings and flies (Figure 1.1).

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 floor of the mouth can be opened like a trap by a surgeon. During maxillo-facial surgery, for example, oral and nasal tubes may both obstruct surgical access. (Fractures may further relatively contraindicate nasal intubation.) If long-term ventilatory support is unlikely, then a tracheostomy can be avoided by a submandibular intubation: a plane is developed from the submandibular triangle (between anterior and posterior bellies of digastric) to the floor of the mouth, avoiding the salivary apparatus and the lingual nerve, and a tracheal tube passed from the oral cavity despite the closed mouth.

The stage’s side wings are formed by mucosal folds running over palatoglossal and palatopharyngeal muscles (from anterior backwards). 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 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 mouth: 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 mouth (during chewing).

The nose has evolved to humidify and warm air before directing it to the nasopharynx and thence towards the lungs; all roles likely to be subverted by anaesthetists. Nevertheless the anatomy of both inside and outside of the nose has 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; this luxurient perfusion limits local cooling and dessication despite 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 anaesthetized patient is profoundly unanatomical: the solution 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 a fibrescope, less Trendelenburg is necessary. Moreover, some

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**Figure 1.1** The mouth.
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 laterally from the midline (Figure 1.2). The space between the floor of the nose and the inferior concha is larger than that between inferior and middle conchae. Moreover the more caudad a fibroscope’s path, the less acutely it must turn past the soft palate towards the glottis. For both reasons, keeping a ‘scope to the floor’ of the nose should facilitate its passage. Furthermore, the ostia (holes) through which the sinuses drain into the nose are all cephalad to the inferior concha. A foreign body running caudad to it may therefore be less likely to obstruct drainage or cause sinusitis.

The damage that can be done by tubes passed blindly through the nose is remarkable; entire conchae have been amputated, and the brain directly oxygenated by tubes passed into it through fractures in the skull base. Clearly endotracheal tubes should be of as small a diameter as possible while bleeding diatheses and basal skull fractures are contraindications to nasal intubation. The nose’s profile also determines how tightly a face mask can fit. Too large a nasal bone, gas escapes around the mask’s sides, and too small, gas escapes at the midline.

**Glottis and epiglottis**

The human larynx is often declared the organ of speech (Figure 1.3). 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.

The epiglottis has evolved to shield the glottis not from anaesthetists, but from nutrients headed towards the stomach. It works like the flexible lid of a pedal bin. Generally it is half open, to allow respiration. 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 anaesthetized 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 1.4).

The mucosa of the larynx above the cords is supplied by the internal laryngeal nerve; below the cords, the mucosa is innervated by the recurrent laryngeal nerve, which also supplies all the intrinsic muscles of the larynx (bar cricothyroid, innervated by the external laryngeal nerve). As it is purely sensory, the internal laryngeal nerve can be blocked without fear of attendant paresis. But 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 anaesthetized topically, if at all.

**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 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 cartilage and the first tracheal ring (the cricoid) is easily palpable in a normal neck, and is covered only by skin, loose areolar
tissue and the fibrous cricothyroid membrane (Figure 1.5). So, in theory, a needle or cannula can be passed into the trachea here without risk of haemorrhage from anterior structures. But posteriorly the oesophagus runs directly behind the trachea, and the needle can perforate the posterior wall of the trachea. Moreover, the gap between cricoid and thyroid cartilages will not admit a tube wide enough to allow conventional ventilation: some form of jetting device must be used.

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, 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 may poke above the sternum as it crosses the trachea.

**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. The most cephalad of these (the cricoid) forms a complete ring. (Indeed, cricoid means ‘like a ring’.) The remainder are each shaped like a C, with the curve facing anteriorly. Not only does this help disoriented bronchoscopists, it also allows 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 optimized for different flow rates (Figure 1.6).

As the bronchial tree ramifies beyond the trachea, 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 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.

Mouth opening and the temporo-mandibular joint

Hominids evolved before cutlery: so, until the Stone Age, 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 (Figure 1.7). (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 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. 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 temporo-mandibular joint, and masseter and temporalis are working with leverage. But at the jaw’s widest opening, it turns about their insertion sites; they are not so passively stretched and the bones of the joint do not so impinge on one another. The lower limit of normal inter-incisor distance has been found to be 37 mm in young adults. Mouth opening declines with age and in general females have slightly smaller inter-incisor distances.

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

Cervical spine

Mobility and strength also characterize the cervical spine. The mobility stems from the arrangement of so many bones over a comparatively short distance (as at the wrist); the strength from the geometry of the joints’ articular surfaces and from the ligaments which bind them.

The joints between occiput, atlas (C1) and axis (C2) are unlike others in the vertebral column. Working caudad, the occipital condyles rest on the lateral masses of atlas like the rails of a rocking horse stuck in tram tracks: the head can flex forward at the joint (until the odontoid hits the skull) and extend backwards; some abduction is allowed, but rotation is not possible. Atlas, however, turns around the axial odontoid peg. Posterior movement of atlas over axis is obviously limited by the axial anterior arch impinging on the peg (Figure 1.8).

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 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.

The functional unit formed by the base of the skull, the atlas and the axis, is often known as the ‘occipito-atlanto-axial complex’. Normal movement at this complex permits easy airway management, both for mask anaesthesia (‘chin lift’) and direct laryngoscopy. Quantification of the movements of the cervical spine is not simple. There is considerable variation in the population and the range of all movements declines with age. Wilson suggested the use of a pencil placed on, and at right angles to, the forehead. The angle swept by the pencil during flexion/extension should be more than 80°. Another method is to compare the movement of a line between the canthus of the eye and tragus of the ear and also a horizontal or vertical line. The normal
range of extension from the neutral position is about 60°. An attractive but sadly, generally impractical, test is to ask the subject to drink from a narrow champagne flute. Patients with poor cranio-cervical movement find this difficult.

Below the axis, in the ‘subaxial’ spine, the vertebrae assume a more conventional form. They articulate at the zygapophyseal joints between each bone’s facets. Extension and flexion are both limited by the bones impinging on one another, either at the facet joints, or in the anterior midline.

Like the other subjects in this chapter, the normal cervical spine is largely relevant only to the extent that it obstructs anaesthetists’ access to the airway. 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 minimal movement in the subaxial cervical spine.

**Key points**

- The landmarks associated with the cricothyroid membrane offer the easiest emergency site for percutaneous airway access.
- The oesophagus lies behind the trachea and is easily perforated by needles introduced into the trachea.
- Normal mouth opening is a complex phenomenon.
- The occipito-atlanto-axial complex has a profound influence on airway management.

**Further reading**


Ethical constraints make the study of this all important topics very difficult. Simple questions, such as how long will an apnoeic patient survive? – cannot be answered with precision.

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 (O₂). 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 biological processes. If molecular O₂ 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 O₂ delivery to the tissues (ΔO₂), 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 O₂ 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 O₂ delivery. Cellular and mitochondrial partial pressure of O₂ (PO₂) may be more than adequate, but the deficit lies in the reduction of molecular O₂ 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?

After an unsuccessful suicide attempt involving motor exhaust-gas inhalation, a patient is taken to hospital. He is alert and breathing O₂ enriched air via a Hudson mask. His haemoglobin concentration is 15 g dl⁻¹, and his carboxyhaemoglobin fraction is 33%. The patient later dies. What is the mechanism of his death?

Let us consider each of the factors of Barcroft’s classification in Box 2.1. Hypoxaemic hypoxia is not likely to be the cause. Assuming no lung damage has occurred, this patient’s arterial (PₐO₂) is likely to be normal if breathing air, or elevated if breathing O₂. PₐO₂ 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.

A common (and erroneous) answer to this question is that, since carbon monoxide has a very high affinity for haemoglobin, and that since carboxyhaemoglobin has no O₂ carrying capacity, O₂ delivery to tissues is compromised, resulting in cellular hypoxia and death. This is clearly erroneous since if total haemoglobin concentration is 15 g dl⁻¹ and the carboxyhaemoglobin fraction is 33% then there is 10 g dl⁻¹ of normal haemoglobin which, since the PₐO₂ is normal, is fully saturated. While this does constitute a form of functional anaemia, an anaemic hypoxia mechanism cannot realistically be implicated.
as a cause of death, since having a haemoglobin concentration of 10 g dl\(^{-1}\) is hardly fatal.

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

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 flavoprotein in mitochondrial respiratory cytochromes. Once bound, electron transfer is interrupted and tissue \(O_2\) cannot be reduced because electron transfer by mitochondrial cytochromes is inhibited. Tissue \(O_2\) consumption, \(\dot{V}O_2\), is inhibited and bioenergetic failure due to ATP depletion ensues.

**Box 2.1 Barcroft’s classification of hypoxia**

\[ DO_2 = \dot{Q} \cdot [Hb] \cdot S_aO_2 \]

1. Hypoxaemic hypoxia
   Hypoxaemia can loosely be defined as ‘a low \(P_{aO_2}\) or a low \(S_aO_2\)’. The causes of this are usually either apnoea, breathing a ‘hypoxic mixture’, severe ventilation/perfusion (\(V/Q\)) mismatch or shunt. In the context of the difficult airway, this is usually the most important cause of cellular hypoxia, or cellular respiratory failure.

2. Anaemic hypoxia
   \(O_2\) delivery is reduced as haemoglobin concentration falls. This is usually a less critical factor because it can usually be compensated for by increased flow.

3. Stagnant hypoxia
   In stagnant hypoxia, \(O_2\) delivery is reduced because blood flow to the tissues (\(\dot{Q}\)) is reduced. This occurs on a global scale in cardiac arrest, or on a small scale regionally (e.g. acute coronary thrombosis). Arterial \(O_2\) tension may well be normal, but delivery to the tissue mitochondria is compromised.

4. Histotoxic hypoxia
   Here there is no deficit in \(O_2\) delivery. The tissue \(P_{O_2}\) is normal, yet molecular \(O_2\) cannot be reduced because electron transfer by mitochondrial cytochromes is inhibited. Tissue \(O_2\) consumption, \(\dot{V}O_2\), is inhibited and bioenergetic failure due to ATP depletion ensues.

\(\dot{Q}\) 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.

**Differential effects of deficits in \(O_2\) delivery**

The equation in Box 2.1 shows that \(DO_2\) is simply proportional to the product of the three ‘Barcroft’ variables. It would, therefore, appear at first sight that any given deficit in \(DO_2\) should cause identical degrees of cellular hypoxia regardless of whether the deficit is in \(DO_2\) due to anaemia, low flow or hypoxaemia. We shall see below that whereas \(DO_2\) deficits due to anaemic and stagnant hypoxia have virtually identical consequences, \(DO_2\) deficits due to hypoxaemic hypoxia are very distinct and uniquely important.

**Anaemic and stagnant \(DO_2\) deficits**

Experimental and theoretical models show that the variables \([Hb]\) and \(\dot{Q}\) are not uniquely independent