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...
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
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.
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 incompressible (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
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 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
facing anteriorly; their corrugations distinguish the trachea from the smooth oesophagus. Not only do the rings help disoriented 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.

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.
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 zygoapophysial 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.

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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 (\(D_O\_2\)), 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 (\(P,O_2\)) 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 (\(P,O_2\)) is likely to be normal if breathing air, or elevated if breathing supplemental oxygen. \(P,O_2\) 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 \(D_O\_2\) 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 \(D_O\_2\) is simply proportional to the product of the three 'Barcroft
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 $Q$ are not uniquely independent variables; it is merely the product, $Q[Hb]$, 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: