The upper airway during anaesthesia

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Upper airway obstruction is common during both anaesthesia and sleep. Obstruction is caused by loss of muscle tone present in the awake state. The velopharynx, a particularly narrow segment, is especially predisposed to obstruction in both states. Patients with a tendency to upper airway obstruction during sleep are vulnerable during anaesthesia and sedation. Loss of wakefulness is compounded by depression of airway muscle activity by the agents, and depression of the ability to arouse, so they cannot respond adequately to asphyxia. Identifying the patient at risk is vital. Previous anaesthetic history and investigations of the upper airway are helpful, and a history of upper airway compromise during sleep (snoring, obstructive apnoeas) should be sought. Beyond these, risk identification is essentially a search for factors that narrow the airway. These include obesity, maxillary hypoplasia, mandibular retrusion, bulbar muscle weakness and specific obstructive lesions such as nasal obstruction or adenotonsillar hypertrophy. Such abnormalities not only increase vulnerability to upper airway obstruction during sleep or anaesthesia, but also make intubation difficult. While problems with airway maintenance may be obviated during anaesthesia by the use of aids such as the laryngeal mask airway (LMA†), identification of risk and caution are keys to management, and the airway should be secured before anaesthesia where doubt exists. If tracheal intubation is needed, spontaneous breathing until intubation is an important principle. Every anaesthetist should have in mind a plan for failed intubation or, worse, failed ventilation.

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Collapse of the upper airway is common during anaesthesia, and airway maintenance is a fundamental anaesthetic skill. Anaesthetists recognise an airway as ‘difficult’ if it is difficult to maintain airway patency without tracheal intubation or intubation itself is problematic. Such problems often coexist because the tendency of the upper airway to collapse during anaesthesia is related to anatomical factors, which also make instrumentation difficult. Retrognathia for example both confines the soft tissues and changes their configuration.

The skeletal and neuromuscular characteristics that predispose to these difficulties also predispose to upper airway obstruction during sleep. A tendency to obstruction during anaesthesia and during sleep is related¹² and the anatomical features that predispose to difficult intubation also predispose to upper airway obstruction during sleep.¹⁶

During wakefulness, airway patency is protected by pharyngeal muscle tone. With both sleep and anaesthesia there is a loss of muscle tone as a result of decreased cortical influences and chemoreceptor drive, together with modulation of mechanoreceptor input. These changes predispose to partial or complete upper airway obstruction, particularly in those with already narrow, compliant upper airways.

In sleep, the changes are particularly evident in rapid eye movement (REM) sleep, when the loss of muscle tone can be profound. Where partial or complete obstruction occurs, the event is terminated by arousal, which is usually brief (<15 s), as a result of the accompanying return of muscle tone. Once patency is restored, sleep resumes, and along with it the tendency to obstruct again. These cyclical obstructions terminated by arousals provide the pathophysiological basis of obstructive sleep apnoea (OSA), a common complaint with substantial associated morbidity.⁴⁸

In the case of anaesthesia, the decrease in muscle tone associated with loss of wakefulness is compounded by specific drug-induced inhibition of upper airway neural and

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anaesthesia from examination of the airway in the awake

The upper airway has a framework of bone and cartilage with attached soft tissue structures, beginning at the nose and lips and ending at the larynx. Where not supported by bone or cartilage, the upper airway may collapse, because muscle tone provides rigidity and this tone can change, as with sleep or anaesthesia. It is these state-related changes and the influence of dynamic factors related to airflow through narrow, potentially collapsible, segments that limit the ability to predict possible collapse during sleep or anaesthesia from examination of the airway in the awake state.

Sites of upper airway obstruction

Collapse will occur at points of narrowing and/or flaccidity. Tonsillar hypertrophy is a common predisposing factor to OSA in children, but adults rarely have specific pathology to explain the disease. In general, adults with OSA have narrower airways than normal subjects, with the velopharynx often the smallest and most compliant segment in both. This is the most common site of collapse during sleep, although collapse may occur at several sites depending on specific predisposing factors.

It is not surprising that the velopharynx is also the most common site of collapse during anaesthesia. This has been shown radiologically during enflurane anaesthesia, by magnetic resonance imaging (MRI) during propofol anaesthesia and by pharyngeal manometry during isoflurane anaesthesia. In the latter study, 14 of 16 subjects had this pattern, with only two obstructing retrolingually. The principal site of collapse was not affected by depth of anaesthesia. These findings contrast with those of an earlier radiological study (and the traditional anaesthetic view) which attributed upper airway obstruction during anaesthesia to retrolingual collapse. This difference may be because the earlier study was of paralysed patients. During spontaneous ventilation, the potentially collapsible pharynx is affected by dynamic effects of negative intraluminal pressures during inspiration, which favour collapse. Such conditions more closely approximate those of sleep.

Prediction of the site of collapse during sleep from measurements during wakefulness has been largely unsuccessful, presumably because of marked differences in the level of resting central drive from cortical influences, chemosensitivity and the gain of upper airway reflexes. It is likely that these differences are less between sleep and anaesthesia, suggesting that behaviour under anaesthesia, including site of collapse, may predict behaviour during sleep. Hence patients vulnerable to OSA could be identified during anaesthesia, and locating the site of collapse might predict those most likely to benefit from surgical treatment such as palatal surgery.

The genesis of upper airway collapse under anaesthesia

Structurally, the upper airway consists of a collapsible segment (the pharynx) situated between two rigid tubes (nasal and tracheal). These correspond to the basic elements of a Starling resistor (Fig. 1) which is a useful model of upper airway pressure–flow relationships. Flow through the collapsible segment depends on how the pressures upstream and downstream of it relate to the pressure surrounding it (tissue pressure). Three different states can apply. (i) When both upstream and downstream pressures exceed the tissue pressure, the segment will remain distended and flow will vary with pressure gradient from upstream to downstream. (ii) When the tissue pressure is greater than the pressure downstream of the collapsible segment but is exceeded by upstream pressure, flow limitation will occur. Flow rate will be independent of downstream pressure, and is determined by the gradient...
between upstream and tissue pressures. There is a near-constant pressure at the site of flow limitation that approximates tissue pressure, hence the resistance of the pathway between the point of application of the upstream pressure and the site of flow limitation is important. (iii) Where tissue pressure exceeds upstream pressure then the segment is occluded and no flow occurs. The upstream pressure at which flow ceases is the critical closing pressure \((P_{\text{crit}})\) of the collapsible segment and provides a measure of its collapsibility.

Studies of the human upper airway during sleep and anaesthesia demonstrate the validity of this model. In 16 subjects breathing spontaneously at end-tidal isoflurane levels of 0.4%, 0.8% and 1.2%, airway patency was maintained with continuous positive airway pressure applied via a nasal mask with the mouth occluded. As this upstream pressure was systematically reduced, a value was reached where inspiratory flow limitation first appeared. This was recognized by independence from downstream pressure (Figs 2 and 3) and therefore inspiratory effort. Further decreases in upstream pressure caused proportionate reductions in maximum inspiratory flow rates, until a critical level was reached \((P_{\text{crit}})\), at which complete collapse (no flow) was apparent. \(P_{\text{crit}}\) varied between individuals, reflecting the variable collapsibility of the upper airway: in some it was greater than atmospheric pressure, indicating that such an airway would obstruct without support; in others a negative pressure had to be applied to produce obstruction, indicating relative resistance to collapse. Indeed, \(P_{\text{crit}}\) was greater than atmospheric pressure in 8 of 16 subjects. These patients did not have any known predisposition to upper airway obstruction, indicating that vulnerability to collapse of the passive upper airway is remarkably common. Such vulnerability is likely to relate to anatomical factors that reduce pharyngeal calibre by increased flaccidity of the pharyngeal walls or increased pharyngeal tissue pressures (see ‘Patient factors contributing to upper airway obstruction’ below). Even when \(P_{\text{crit}}\) was less than atmospheric pressure, indicating a relatively stable airway, flow limitation was evident at atmospheric pressure (see Fig. 3, left panel).

The variability in upper airway collapsibility in anaesthetized patients is also found in sleep. In general, \(P_{\text{crit}}\) during sleep is greater in patients with sleep apnoea than in those who simply snore, who, in turn, have greater values than normal subjects. The tendency to upper airway...
obstruction during sleep and during anaesthesia is related, and sleep apnoea is more prevalent in those with greater closing pressure under anaesthesia.12

Since vulnerability to collapse of the flaccid upper airway is common, in many people airway patency during wakefulness must depend on activity of the upper airway dilator muscles. These muscles (of which the genioglossus is the most influential) demonstrate tonic and phasic activity in the awake state. The phasic activity is present during inspiration and, together with background tonic activity, acts to resist airway narrowing when pharyngeal intraluminal pressure decreases during inspiration. This activity is greater during wakefulness in patients with snoring and, particularly, OSA, reflecting the underlying vulnerability of their airways to collapse.30

Upper airway muscle activity depends partly on centrally mediated drive from the brainstem and partly on reflexes originating within the upper airway. For these reflexes, the most important local stimulus is negative intrapharyngeal pressure.28 Topical anaesthesia of the pharynx increases upper airway obstruction during sleep.6 With sleep and with anaesthesia, upper airway muscle activity is reduced by reduced central drive, because of decreased cortical influences and decreased chemosensitivity. A ‘dose–response’ effect is evident during sleep, with some preservation of upper airway muscle tone present in non-REM sleep. In REM sleep, the loss of muscle tone can be profound, and OSA is usually at its most severe during this sleep stage.18

In addition to its central effects, anaesthesia also directly inhibits upper airway muscle and neural activity, including inhibition of laryngeal respiratory-modulated mechanoreceptors, and therefore upper airway reflexes,33 35 and depresses protective arousal responses. In both sleep and anaesthesia there appears to be greater depression of upper airway muscles than of the diaphragm, so that breathing efforts continue when upper airway muscle activity is markedly reduced. This increases the vulnerability to upper airway collapse during inspiration, as air is accelerated across a disproportionately floppy upper airway. Because of these sleep-associated effects, hypoglossal nerve stimulation by an implanted nerve stimulator has been investigated as a therapy for OSA.43

**Effect of individual anaesthetic agents**

Changes during anaesthesia appear to vary with the agent used. Clearly, with neuromuscular blockade profound relaxation of skeletal muscle activity is present. This effect is dose dependent and a hierarchy of effect is observed, with the diaphragm more resistant to neuromuscular blockade than non-diaphragmatic respiratory muscles,8 including the upper airway muscles.6 This indicates relative safety margins for neuromuscular transmission between these muscle groups, with a particularly high margin for the diaphragm, consistent with its importance for survival.

During anaesthesia with spontaneous ventilation, this same hierarchy is seen, with more inhibition of upper airway relative to diaphragm activity, again reflecting the functional reserve of the diaphragm. This has been found in experimental animals with subanaesthetic concentrations of halothane, pentobarbital,20 thiopental, diazepam36 and ethanol.4 Studies with anaesthetic concentrations of halothane found differential suppression of phrenic and hypoglossal activity, with a dose-related decrease in both, but to a greater extent for the hypoglossal nerve.36 When the activity of respiratory muscles is considered, halothane has a greater depressant effect on genioglossus activity than on the intercostals, with least effect on the diaphragm.38 With opioids these differential effects are less apparent, and hypoglossal and phrenic nerve activity are reduced by similar amounts.5 With ketamine the situation is less clear, one study demonstrating differential effects,20 whereas another did not.36

In humans, sedation with midazolam powerfully inhibits upper airway activity.9 37 This is less apparent with ketamine.9 Propofol sedation is also associated with pharyngeal dysfunction46 and upper airway obstruction.29 However, dose–response relationships have not yet been clearly defined for any of these agents in man, so that it is not known to what degree changes in anaesthetic depth affect upper airway collapsibility. Such a relationship has then been seen with thiopental, with a progressive decrease in upper airway electromyographic activity with increasing dose.10

With isoflurane there is no clear dose–response relationship at anaesthetic concentrations, since genioglossus activity is absent even during light planes of anaesthesia, returning abruptly with emergence.12 However, disturbance of swallowing occurs at subanaesthetic concentrations,46

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**Fig 3** Relationship between maximal inspiratory flow and nasal mask (upstream) pressure for flow-limited breaths from two subjects breathing spontaneously at end-tidal isoflurane levels of 1.2%. The left panel demonstrates data from a subject with a relatively stable upper airway; note that the critical closing pressure (\(P_{\text{crit}}\)) is subatmospheric. The right panel demonstrates data from a subject with an unstable airway; note that \(P_{\text{crit}}\) exceeds atmospheric pressure.
suggesting subtle pharyngeal impairment, so that there could be a definable dose–response effect at these low drug levels. A differential effect on upper airway muscles relative to the diaphragm is clearly found at anaesthetic concentrations, with inspiratory efforts continuing when upper airway activity is profoundly depressed.11 The presence of measurable effects at relatively low concentrations of many anaesthetic drugs highlights the susceptibility of the patient with a vulnerable upper airway to obstruction in the recovery room after anaesthesia is complete, or elsewhere where narcotic analgesics or sedation are administered.

**Table 1** Known and suspected predisposing conditions for obstructive sleep apnoea (adapted from Loadsman and Hillman,26 with permission)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>Acquired obesity, Prader–Willi syndrome</td>
</tr>
<tr>
<td>Genetic predisposition</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>Male gender</td>
<td></td>
</tr>
<tr>
<td>Alcohol, sedatives, analgesics, anaesthetics</td>
<td></td>
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<tr>
<td>Smoking</td>
<td></td>
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<tr>
<td>Nasal obstruction</td>
<td></td>
</tr>
<tr>
<td>Pharyngeal obstruction</td>
<td>Septal deviation, chronic nasal congestion</td>
</tr>
<tr>
<td>Craniofacial abnormality</td>
<td>Tonsillar and adenoidal hypertrophy</td>
</tr>
<tr>
<td>Laryngeal obstruction</td>
<td>Laryngomalacia</td>
</tr>
<tr>
<td>Endocrine/metabolic</td>
<td>Hypothyroidism, androgen therapy, Cushing’s syndrome</td>
</tr>
<tr>
<td>Neuromuscular disorders</td>
<td>Stroke, cerebral palsy, head injury, Shy–Drager syndrome, poliomyelitis, muscular dystrophies, myotonic dystrophy, tetraplegia</td>
</tr>
<tr>
<td>Connective tissue disorders</td>
<td>Marfan’s syndrome</td>
</tr>
<tr>
<td>Storage diseases</td>
<td>Mucopolysaccharidoses</td>
</tr>
<tr>
<td>Chronic renal failure</td>
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Patient factors predisposing to upper airway collapse during anaesthesia or sleep

Factors that narrow the pharynx, increase pressure around it, reduce pressure within it or make its walls more compliant will increase upper airway obstruction during sleep and anaesthesia. These factors may exist in the lumen (e.g. a foreign body), in the pharyngeal wall (e.g. bulbar muscle weakness), or outside the pharynx. Factors acting outside the pharynx include direct actions (e.g. lateral pharyngeal fat pads in obesity, narrow skeletal confines) or indirect actions (e.g. loss of longitudinal traction on the pharynx from neck flexion or obesity-related reduction in lung volumes). A narrow airway is particularly vulnerable to collapse for three reasons. Firstly, Laplace’s law requires that with a small radius of curvature, a greater expanding force (transmural pressure) is needed to maintain sufficient wall tension to oppose collapse. Secondly, the absolute change in calibre before airway closure occurs is less if the calibre is already small. Thirdly, the greater resistance of a narrow airway requires generation of more negative intraluminal pressures during inspiration, increasing the tendency to collapse. These considerations are relevant to conditions that predispose to upper airway obstruction when upper airway dilator muscle activity is reduced with sleep or when under the influence of sedative or anaesthetic drugs (Table 1).

Characteristics that reflect propensity to upper airway collapse include obesity, macroglossia, micrognathia and maxillary hypoplasia.15 These may be present in otherwise normal individuals or as part of a disease syndrome, such as acromegaly, Down’s syndrome, Pierre–Robin syndrome or other craniofacial abnormalities. Familial predispositions are common,41 reflecting common morphological characteristics.

Neuromuscular disorders that decrease upper airway muscle tone predispose to upper airway obstruction, as do endocrine, connective tissue and storage diseases affecting upper airway calibre.

Specific lesions in the upper airway provide other predispositions to upper airway obstruction that are site specific. Examples include nasal obstruction, tonsillar and adenoidal hypertrophy, pharyngeal and laryngeal tumours, foreign bodies, haematoma, oedema and laryngomalacia.

Patient habits such as smoking, and alcohol and sedative consumption also predispose to collapse through their effects on muscle tone or airway calibre. The supine posture is a further predisposing factor, as the effects of gravity increase the extraluminal compressive forces exerted by the tongue, soft palate and related structures.

**Assessment of the upper airway**

**Clinical findings**

Identification of patients at risk of upper airway obstruction during sleep or anaesthesia or of difficult intubation is a search for the factors referred to in the previous section. Many pathological processes are predictably associated with difficult airway maintenance or intubation, such as epiglottitis and tumours of the larynx or pharynx. Not all difficult airways are associated with major pathology, but result from more subtle anatomical predisposition, modulated by neuromuscular forces. Such difficulties may only become manifest with the state-related decreases in neuromuscular activation that accompany sleep and anaesthesia. Examination of the awake patient may not reveal these influences or the dynamic effect of airflow across potentially vulnerable segments, reducing the capacity to identify airways at risk.

Features that anaesthetists associate with difficult intubation are also associated with upper airway obstruction during sleep.16 However, although the upper airway may readily obstruct during sleep or anaesthesia, and these propensities are related,12 difficult intubation is a much rarer phenomenon. Hence measurements that reasonably reliably
predict vulnerability to collapse, such as obesity and increased neck circumference, or others referred to below are much less accurate predictors of difficult intubation.

In the absence of obvious pathology, the most reliable predictor of difficult intubation is previously documented difficulty, and previous anaesthetic records should always be sought for guidance. Problems with airway maintenance or intubation should always be recorded in the notes and in emergency medical record systems such as MedicAlert, and the patient should be advised.

Where such information is not available then the anaesthetist depends on clinical indicators to suggest the possibility of difficulty, but should recognise their limitations. Particular note should be made of obesity, increased neck circumference, decreased neck length, limited head/neck extension, impaired nasal patency, crowded pharyngeal appearance (high Mallampati score, macroglossia, tonsillar hypertrophy), dental abnormalities, limited mouth opening, maxillary hypoplasia, mandibular hypoplasia/retrusion, decreased thyromental distance and increased mandibular angle.

As many of the airway abnormalities that predispose to difficulty during anaesthesia also cause problems during sleep, a sleep history is useful, paying particular attention to the cardinal symptoms of OSA: snoring, witnessed apnoeas and disrupted sleep with excessive daytime somnolence.

**Investigations**

Where concern exists regarding upper airway calibre and function, further information may come from radiographic, endoscopic and physiological evaluation. Usually these investigations have been performed awake, and so the potential effects of state-related changes in neuromuscular function accompanying anaesthesia need to be considered when interpreting the findings.

**CT and MRI.** Studies using computed tomography (CT) and MRI scanning have improved our knowledge of upper airway function under anaesthesia and the pathogenesis of sleep apnoea. While the chest radiograph can indicate tracheal deviation or compression, both CT and MRI scanning give accurate images of the upper airway, which allow its calibre to be examined from end to end. Although useful generally, such scans are particularly helpful to anaesthetists in examining the influence of clinically identified abnormalities on upper airway configuration. For example, the effect of tumours that are displacing or constricting the airway can be quantified. This information is usually available in such cases as they present for surgery and provides invaluable insights for the anaesthetist preparing for the challenges in airway management that they may present.

**Lateral cephalometry.** Lateral cephalometry involves a true lateral radiograph of the head and neck, with penetration carefully determined to outline soft tissue and skeletal outlines. Allowance is made for magnification. Measurements are taken of craniofacial and soft tissue structures. Patients with OSA have a small retrroposed mandible, narrow posterior airway spaces, enlarged tongue and soft palate, inferior positioned hyoid bones and retroposition of their maxillae. Patients who are difficult to intubate share many of these characteristics. While readily available, lateral cephalometry requires expert evaluation and is used more to examine the site and cause of upper airway narrowing, for example when craniofacial surgery is proposed, than for clinical anaesthesia.

**Endoscopy.** Fibreoptic upper airway endoscopy allows the calibre and configuration of the airway to be seen directly and possible endoscope-assisted intubation to be assessed. Familiarity with airway endoscopy and fibreoptic-aided intubation is essential for good management of difficult airways. Laryngoscopic evaluation of the upper airway may already have been done during the surgical assessment, particularly of patients presenting for surgery to the upper airway, and this information should be obtained.

**Maximum inspiratory and expiratory flow volume curves.** In substantial upper airway obstruction, reduction in maximum respiratory flow rates occurs, particularly during inspiration as the negative intraluminal pressures favour collapse. A plateau in flow is often seen, indicating flow limitation. The reduction in maximum flow rates indicates the severity of the obstruction and the measurement is readily repeated, allowing changes with time and treatment to be assessed. This is not used for routine evaluation but is valuable for following the progress of patients with chronic conditions associated with substantial airway narrowing, usually clinically evident during wakefulness, that require intermittent treatment (e.g. subglottic stenoses).

**Recognition of upper airway obstruction**

The hallmark of upper airway obstruction is diminished or absent airflow in the presence of continued respiratory effort. Complete upper airway obstruction is silent, while partial obstruction will be accompanied by snoring (if supralaryngeal), or inspiratory stridor (if perilaryngeal). A consequence of continued inspiratory efforts against the partially or completely obstructed airway is the development of large negative intrathoracic pressure swings, which are often seen as a combination of diminished chest wall movement and distortion, with paradoxical (inward) motion of the anterior rib cage. In the laboratory (e.g. during polysomnography in a sleep clinic), obstruction is quantified by relating measures of breathing effort (chest wall motion or, more accurately, oesophageal pressure changes) with those of ventilation (oronasal airflow).
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Prevention and treatment of upper airway obstruction

Many pathological processes are predictably associated with difficult airway maintenance or intubation, such as epiglottitis and laryngeal or pharyngeal tumours. These can be extremely challenging for the anaesthetist. Their management is well described in standard anaesthetic textbooks and is beyond the scope of this article, which is primarily concerned with airway difficulty arising from more subtle variations in upper airway structure and function.

Anatomical positioning and posture

The supine posture can worsen upper airway obstruction because gravity affects the tongue and soft palate position, narrowing the retropalatal and retrolingual airways. Neck flexion, mouth opening and table tilt with the head down are also unfavourable because of loss of longitudinal tension on the upper airway.

Apart from avoiding these unfavourable circumstances where possible and practical, two commonly practised manoeuvres can be used to improve airway patency. Firstly, placing the head in the ‘sniffing the morning breeze’ position (lower cervical flexion, upper cervical extension with full extension of head on neck) increases longitudinal tension on the upper airway and decreases its collapsibility. Secondly, displacing the mandible forwards pulls the tongue forward and increases the calibre of the retrolingual airway and the retropalatal airway as the soft palate is also displaced forwards because it is coupled to the tongue via the faucets. The principle of mandibular advancement is now also applied to the treatment of OSA by dental splints designed for the purpose. Simply lifting the chin with mouth closure can also be effective in increasing pharyngeal dimensions by increasing longitudinal tension in the pharyngeal muscles and anterior neck structures. This action increases the anteroposterior distance between tongue and posterior pharyngeal wall and counteracts the tendency to collapse.

Continuous positive airway pressure (CPAP)

Applying positive pressure to the upper airway in order to splint it punitatively is a widely used principle in the treatment of OSA but is less systematically applied during anaesthesia, even though effective in this circumstance. While this is understandable intraoperatively where other more convenient techniques are available to the anaesthetist for airway management, applying PEEP when assisting ventilation with bag and mask is a strategy that can help relieve upper airway obstruction. It is probable that CPAP is underused in the recovery room, where the potential for upper airway obstruction is high, and further into the postoperative period, where many patients with the tendency to upper airway obstruction remain at increased risk because of the use of narcotic analgesic or sedative drugs. In this regard it is important to note that OSA remains notoriously under-diagnosed, and may first be identified as a problem under these circumstances.

Airway aids

Oral and nasopharyngeal airways help by bypassing the obstruction (e.g. the oral airway in the case of retropalatal obstruction) or providing a conduit through it (e.g. the nasopharyngeal airway in the same circumstance). Often these prove to be an aid rather than a complete solution to provision of an adequate airway, and neck extension and forward traction on the mandible may also be needed to maintain patency.

The laryngeal mask airway (LMA), of which there are now several variants, can provide a conduit from the exterior to the rima glottidis. Its ease of introduction and reliable provision of a patent airway without the requirement of additional manoeuvres have made it an invaluable addition to anaesthetic equipment in the 19 years since its first description.

Tracheal tubes

Tracheal intubation remains the ‘gold standard’ for control of the airway. Once secured, the lower airway and gas-exchanging regions of the lung are directly accessed and control is complete. However, because of their common anatomical factors, airways that are difficult to maintain patent may also be difficult to intubate. Where the intubation is known (from previous experience) or predicted (from preoperative assessment – see above) to be difficult, then spontaneous ventilation should be preserved until intubation is accomplished. This is best done during wakefulness using a fibreoptic endoscope introduced under local anaesthesia, providing the patient is cooperative and trauma associated with bleeding does not obscure the view. Alternatively, it may be done in the anaesthetized spontaneously breathing patient either by direct laryngoscopy, often with the aid of a bougie, light wand or similar device, or via an LMA designed to allow passage of a fibroptic scope (e.g. the LMA-Fastrach). Tracheotomy under local anaesthesia is a more drastic solution that may be necessary if all else fails, or in the case of an obstructing lesion (such as carcinoma of the larynx) that does not allow the passage of even a relatively small tracheal tube.

When un-anticipated difficult intubation is encountered after neuromuscular blocking agents have been given, then subsequent events depend on how well ventilation and gas exchange can be maintained (ventilating with 100% oxygen) while attempting to solve the difficulty. Adequate oxygenation is the first consideration. If ventilation is well maintained by bag and mask, it may be possible to secure the airway using one of the methods specified above. The intubating LMA (LMA-Fastrach) is often useful, and even
more so if there is any difficulty maintaining ventilation. If problems with ventilation persist, this is an emergency and extra help should be obtained. An emergency airway needs to be established using transtracheal techniques, such as transtracheal jet ventilation or tracheotomy. Several algorithms that deal with such circumstances have been published, one of the most authoritative being from the American Society of Anesthesiologists.²

**Heliox**

Where the airway is extremely narrow, flow turbulence is a prominent feature. This is a density-dependent phenomenon and a gas of low density may relieve respiratory distress. Helium–oxygen mixtures fulfill this requirement.²⁷ A balance has to be struck between decreasing density of the carrier gas and reducing the inspired concentration of oxygen, hence the respective concentrations of helium and oxygen.¹⁷ This is determined from assessment of breathing comfort and arterial blood gas analysis. The treatment is expensive so this is a strategy for short-term use until other therapies can relieve the underlying problem.

**Summary**

The difficult upper airway causes problems with airway maintenance and/or instrumentation under anaesthesia. The skeletal and neuromuscular characteristics that predispose to these difficulties also predispose to upper airway obstruction during sleep.

The behaviour of the upper airway during sleep can indicate to the anaesthetist its likely behaviour during anaesthesia, where the effects of loss of wakefulness are compounded by specific anaesthesia-induced inhibition of upper airway neural and muscle activity and suppression of protective arousal responses. Conversely, difficult airway maintenance or intubation during anaesthesia suggest OSA, a common condition with substantial associated morbidity. Safe management of such cases involves identification of risk and cautious preparation, securing the airway before anaesthesia where doubt exists, and a systematic approach in the case of failed intubation or, worse, failed ventilation.

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