One of the first skills that a trainee in anaesthesia is expected to acquire is the ability to maintain the anaesthetized patient’s airway patent. The current concept of the mechanism of airway obstruction in patients who are comatose, anaesthetized or have received neuromuscular blocking agents, is that the tongue “falls back” and occludes the lower pharynx. Several well known textbooks contain diagrams that are artist’s impressions illustrating this belief. Although this concept is supported commonly by reference to the classic radiological studies of paralysed patients carried out by Morikawa, Safar and DeCarlo [1], careful study of their report suggests otherwise. These authors showed that head tilt, with stretch of the structures between larynx and chin, was the most effective means of increasing hypopharyngeal patency. It is difficult to understand how this manoeuvre can “pull the tongue forward” to clear the airway. Everyday clinical experience emphasizes the relative unimportance of the action of gravity on the tongue in the development of airway obstruction, by the frequent need to maintain head extension, for example in patients lying on their side while recovering from anaesthesia, or in supine anaesthetized patients in whom an oral airway has been placed, which one might expect should overcome the problem of the tongue position. Using ultrasound to visualize the tongue, there seems to be little change in tongue position immediately after induction of anaesthesia with i.v. agents, although airway obstruction may supervene [2].

A recent upsurge in interest in the upper airway and the factors that regulate its patency has come from the recognition of sleep apnoea syndromes and the sudden infant death syndrome, both of which may be related to abnormalities in airway patency. How can we apply this information to improve our management of the anaesthetized patient’s airway? Unfortunately, a great many of the data available are difficult to interpret and apply directly to the circumstances of anaesthesia. Techniques used commonly to study the airway are electromyography, measurement of pressure-flow relationships, and the method of acoustic reflection to assess airway area. In some circumstances, imaging by fiberoptic endoscopes, radiology, ultrasound, axial tomography or magnetic resonance has been used. Electromyography indicates muscle activity, but electrical activity does not necessarily indicate that the muscle is shortening, particularly in this region where so many muscles have almost opposing actions. The pressure-flow relationships in this region are complex, as the flow is often in transition from laminar to turbulent patterns, but it is possible to detect airway obstruction at different regions of the airway in normal human subjects during sleep [3]. Acoustic reflection is a promising and relatively non-invasive method that analyses reflections of a sound pulse projected down the airway [4]. The results agree reasonably well with those obtained by axial tomography, but the apparatus is unwieldy and requires the use of 80% helium to improve the resolution [5].

Although it appears that muscle activity may be a major factor in maintaining airway patency, it remains to be determined which of the many muscles are most important, and the main site or sites at which collapse occurs. The studies of Nandi and co-workers reported in this issue, [6], and those of others [3, 7], suggest that there may be more than one site. Investigation of this topic is complicated by the degree of reflex control of the activity of these muscles, and the possibility that some measuring devices may themselves influence the observations. For example, the use of a mouthpiece or similar apparatus alters breathing patterns and possibly muscle activation [8], and topical anaesthesia of the nose can cause abnormal breathing during sleep [9]. Although investigations with endoscopy may be possible in well anaesthetized patients, reflex responses may
be exaggerated in circumstances such as barbiturate anaesthesia. Finally, the use of different forms of anaesthesia in many of the animal studies may be responsible for some of the contrasting results obtained [10].

Most human investigations have been of electrical activity of the genioglossus muscle, first studied by placing electrodes into the tongue either via the floor of the mouth or percutaneously [11], and later by surface electrodes placed either below the chin [12] or in a moulded mouthpiece [13]. Early reports were qualitative, but they suggested that there could be continuous activity of the tongue, particularly in its posterior part. Activity of the genioglossus was augmented during inspiration [11]. During rapid eye movement sleep, the activity became profoundly less, as does the activity in other skeletal muscles, and it was in this phase of sleep that obstructive apnoea was likely to occur. Airway patency was therefore attributed to activity of the tongue. It was argued that because activation of this muscle caused protrusion of the tongue, lack of activity would lead to a posterior movement. However, the mere association of loss of genioglossal activity with reduction of airway patency does not imply that the first event causes the second. Indeed, many muscles in the upper airway show an increase in activity during inspiration, such as the alae nasi [14], the tensor palatini [15], intrinsic laryngeal muscles such as posterior cricoarytenoid [16], and the strap muscles of the neck such as geniohyoid [17], sternohyoid, and sternothyroid [18]. As activity in many of the upper airway muscles changes in a similar way during ventilation it is possible that the activity of the tongue merely reflects changes in activity of the muscles that are more directly responsible for airway patency, rather than having an important effect itself.

Even in studies in which airway patency has been assessed before and after muscle activity has been abolished by nerve section, differentiation of the action of different muscles has not been established [17]. However, muscles that influence the position of the hyoid have been shown to influence airway patency. Stimulation of these muscles improves stability of the airway [18], and direct measurement of the length of these muscles shows that pharyngeal volume is increased as they shorten [19]. These studies in animals support observations in man during halothane anaesthesia, in which airway obstruction was attributed also to posterior movement of the epiglottis [20]. Relaxation of the neck strap muscles would cause such a movement. Obstruction may be overcome by extension of the head, to increase the passive tension in the muscles of the anterior neck, and so pull the hyoid forwards and with it the structures of the anterior pharyngeal wall, restoring airway patency—a mechanism recognized at least since the 18th century [21].

Is the activation of these muscles of the upper airway associated with actual dilatation of the pharynx? In animals anaesthetized with barbiturate there is evidence of upper airway dilatation during inspiration [22]. In addition to the effects of the neck muscles, however, the thoracic muscles may also influence the upper airway. During inspiration with the diaphragm, traction by the thorax on the strap muscles of the neck may increase airway size [23]. In conscious normal subjects, a reduction in lung volume by active exhalation from functional residual capacity to residual volume is associated with a reduction in area of the pharynx, by about 20% [24].

What factors influence activation of the airway muscles? Stimulation of ventilation by hypercapnia is associated with increased activity of the airway muscles [25], and lung volume-related reflexes that prolong inspiration also affect upper airway muscle action [26]. In addition, the upper airway is richly supplied with receptors that respond to pressure and flow [27] and a reduction in pressure in the upper airway such as might result from obstruction causes reflex activation of the tongue and neck strap muscles [28]. In normal human subjects, topical anaesthesia of the upper airway increases the incidence of episodes of airway obstruction during sleep, particularly if the pharynx is anaesthetized [29]. Hence, at least in some circumstances, upper airway reflex responses may contribute to airway patency. These different influences make it difficult, in the presence of partial or complete airway obstruction, to attribute increases in muscle activity to increased chemical drive, a reflex response to decreased pressure or flow in the airway, or a combination of these factors. In particular, studies of sleep apnoea patients may be difficult to interpret. Such patients have reduced pharyngeal size [24] and thus may have greater changes in upper airway pressure with ventilation, and hence reflex activation of the muscles of the airway, whereas normal subjects may not. Similar considerations could apply to older, male, and
overweight patients who frequently have reduced airway dimensions [30] and are recognized by anaesthetists as having "difficult airways". In fact, during quiet breathing in normal subjects, genioglossus is relatively inactive and not always phasic [31], even in the supine position where airway size is reduced [32].

Anaesthesia or sedation often reduces respiratory drive, chemical responses and airway reflex activity, and it is not surprising that airway muscle activity is reduced. Although several studies have shown that increasing depth of anaesthesia causes a progressive reduction in upper airway motor activity [33, 34], there are few data comparing either the pattern or magnitude of activity in the conscious with the anaesthetized state. In awake cats with fine wire electrodes implanted in the tongue, tonic activity was more common than phasic activity [25], and in men with implanted electrodes, tonic activity was more common, although some phasic activity could be caused by maximal inspiration [Douglas, Warren and Drummond, unpublished observations]. Thiopentone causes a reduction in activity and a change in the pattern of activity from tonic to phasic [31]. As the reduction in activity of upper airway muscles seems to be greater than in the diaphragm, it is possible that the imbalance may predispose the upper airway to collapse [35], particularly if the onset of activity in airway muscles does not precede that in the diaphragm. Such a sequence of events has been demonstrated by the application of a hypoxic stimulus during sleep [36, 37].

Can we now add science to craft in airway maintenance? The changes in soft palate position observed by Nandi and co-workers [6] may be relevant: certainly, intranasal obstruction or reduced pressure at the nose can worsen airway obstruction during sleep [35, 38]. Exactly the same manoeuvres that help improve the airway in the anaesthetized patient also establish naso-phyanggeal patency [39], and hence might reduce this tendency to airway occlusion. If the exact effects of airway maintenance manoeuvres can be estimated, then simpler and less invasive apparatus for airway management may be practical. The acoustic reflection technique may be useful for this because it permits frequent rapid measurements. Techniques such as nasal CPAP [40] or pharyngeal insufflation [41] should be investigated cautiously. Most important, simple formal clinical studies may be of value in defining the frequency, extent and factors associated with airway problems.

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REFERENCES


