EFFECT OF GENERAL ANAESTHESIA ON THE PHARYNX


SUMMARY
Conventional lateral radiography was used in 18 elderly male patients to investigate the changes induced by general anaesthesia in the upper airway. The effect of tongue traction under anaesthesia was studied similarly in another 11 patients. Following induction of anaesthesia, there were highly significant approximations to the posterior pharyngeal wall of the soft palate (median change 1.3 mm, 95% confidence interval (CI) 0.3-2.6 mm; P = 0.006), tongue base (mean change 6.5 mm, 95% CI 5.3-7.7 mm; P < 0.001) and epiglottis (mean change 3.8 mm, 95% CI 3.1-4.5 mm; P < 0.001). Apparent radiographic occlusion of the airway occurred most consistently at the level of the soft palate (17 of 18 patients), sometimes at the level of the epiglottis (four patients), but the tongue base did not touch the posterior pharyngeal wall in any patient. Traction on the tongue failed to clear the nasopharyngeal obstruction. Attempted inspiration under anaesthesia caused major secondary collapse of the pharynx, with multiple sites of obstruction, similar to that found in obstructive sleep apnoea.

KEY WORDS

Obstruction of the upper airway under general anaesthesia has been recognized for more than a century, as has the effectiveness of head extension and anterior displacement of the mandible (the Esmarch–Heiberg manoeuvre) in clearing the airway [1]. Until recently this obstruction was attributed to posterior shift of the base of the tongue [2, 3], and it has been shown that both the normal phasic genioglossus electromyogram [4] and hypoglossal nerve activity [5] are depressed markedly during anaesthesia in the cat. The efficacy of the Esmarch–Heiberg manoeuvre also fits in well with this explanation of airway obstruction, as it displaces the insertion of genioglossus (and hence the tongue) anteriorly. The effect of anaesthesia seems to be paralleled by obstructive apnoea during sleep, when the genioglossus electromyogram may be depressed also during rapid eye movement (REM) sleep [6, 7].

However, more recent work in the anaesthetized human subject has challenged this view and implicated the epiglottis as a major cause of airway obstruction [8]. Our objective was to investigate anatomical changes in the pharynx during anaesthesia using a non-invasive technique in order to avoid distortion of pharyngeal structures by either mechanical displacement or reflex activity. We chose conventional lateral soft tissue radiography as providing the best compromise between practicability and fine discriminatory imaging of the air–tissue interface.

PATIENTS AND METHODS
The study was approved by the hospital Ethics Committee, but restricted to male patients older than 60 yr (range 63–84 yr), presenting for routine urological procedures under general anaesthesia. All were in good general health with no symptoms suggestive of upper airway pathology or ob-

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structive sleep apnoea. Verbal consent was obtained on the day before the study. The majority of patients were unpremedicated, but four received i.m. papaveretum and one morphine 1 h before induction of anaesthesia, and three received oral temazepam 2 h before induction.

Eighteen patients were studied initially. Radiographs were taken in the anaesthetic room of the operating theatre, using a Dean 38 mobile x-ray unit (Dean GEC X-ray Operators Ltd, Wembley, U.K.). Exposure factors were in the range 62–65 kV (peak) and 12–16 mAs, depending on patient build. Exposure times were 60 ms. The focal film distance was 150 cm and the patient midline was 26 cm from the film. This produced a magnification factor of 17%, correction for which was made in the airway measurements. All radiographs were taken by the same radiographer (S.J.T.).

After insertion of an i.v. cannula, the subjects were instructed to breathe normally through the nose, with the mouth closed, while lying supine with the head resting on a ring support in a comfortably neutral position. The occiput was supported 3–5 cm above the plane of the table. A total of five radiographs were taken.

The first 10 patients were instructed to stop breathing for a few seconds at the end of a natural expiration while the first radiograph was taken, and at the end of a natural inspiration while the second radiograph was taken. The remaining eight patients were instructed to continue normal tidal breathing throughout, and radiographs were taken in mid-expiration and mid-inspiration as judged by motion of the epigastrium.

After the first two radiographs had been taken, anaesthesia was induced with thiopentone 3–5 mg/kg body weight and a third radiograph taken during the apnoeic pause. On re-establishment of spontaneous breathing, an oxygen–nitrous oxide–enflurane mixture was administered via a Magill attachment and conventional facemask. The inspired oxygen concentration was approximately 30%, and that of enflurane 3%. Head extension, with or without mandibular displacement, was required in all subjects to maintain airway patency after establishment of spontaneous breathing under anaesthesia; these manoeuvres were discontinued transiently and the head returned to the neutral position, in order to take the radiographs after re-establishment of spontaneous breathing at a light plane of surgical anaesthesia. For the first 10 patients these were at end-expiration and end-inspiration, and for the last eight patients in mid-expiration and mid-inspiration.

In another 11 patients (using the same criteria for inclusion in the study), both expiratory films were omitted, but an additional radiograph was taken under anaesthesia while firm traction was applied to the tip of the tongue using wide bore suction tubing to which a negative pressure of 200 mm Hg was applied.

Figure 1 depicts a pair of representative radiographs which illustrate the radiographic

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**Fig. 1.** Typical radiological appearances, before (left) and after (right) induction of anaesthesia.
anatomy of the peripharyngeal structures and demonstrate the typical changes which occurred after induction of anaesthesia.

Preliminary analysis of the radiographic findings consisted of making measurements of the width of the airspace in the sagittal plane at four standard sites (fig. 2):

$A$: the minimal distance from posterior pharyngeal wall to posterior aspect of soft palate;
$B$: the minimal distance from posterior pharyngeal wall to base of tongue;
$C$: the minimal distance from posterior pharyngeal wall to epiglottis;
$D$: the minimal distance from anterior aspect of soft palate to dorsum of tongue.

These measurements were made for all films; in the case of the pre-anaesthetic and apnoea films, two additional measurements were made:
$E$: the vertical height of the superior surface of the greater cornu of the hyoid bone above the lowest point of the body of the fourth cervical vertebra;
$F$: the vertical height of the tip of the epiglottis above the lowest point of the body of the fourth cervical vertebra.

In addition to these basic measurements, we constructed diagrams of a "mean airway" for each phase of the study (pre-induction expiratory, pre-induction inspiratory, apnoea, post-induction expiratory and post-induction inspiratory). Tracings were made of the outline of the pharynx and hyoid bone from transilluminated films, using as reference bony landmarks the anterior-most point of the body of the first cervical vertebra, and a line passing from it through the mid point of the anterior border of the fifth cervical vertebra. From these tracings, mean airway diagrams were drawn by superimposing tracings of individual patients' films on a single sheet, on which the visually estimated mean outline was then drawn manually. Validation of the accuracy of these diagrams was tested by comparing the means of the direct measurements $A$, $B$, $C$ and $D$ with the corresponding measurements on the mean diagrams; there was agreement in all cases to within 1 mm.

Statistical analysis of the six standard measurements was by paired Student's $t$ test and two-way analysis of variance as appropriate, except in the case of measurement $A$, for which the data distribution was markedly non-Gaussian even after log transformation. In this case analysis was by the Wilcoxon matched pairs signed rank test.

RESULTS

Before induction of anaesthesia there were no significant phasic changes in sagittal airway configuration between either end or mid phase of breathing; this is the subject of a separate publication.
Table 1. Anaesthesia (apnoea) vs awake control and attempted inspiration during anaesthesia. Measurements are as shown in figure 2. Geometric means and their 95% confidence intervals (in parentheses) based on the pooled estimate of the within-subject variability obtained from the analysis of variance.

<table>
<thead>
<tr>
<th>Measurement (mm)</th>
<th>Awake</th>
<th>Apnoea</th>
<th>Inspiration</th>
<th>P vs apnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>A*</td>
<td>1.3</td>
<td>0.0</td>
<td>0.0</td>
<td>0.006†</td>
</tr>
<tr>
<td>(0.3-2.6)</td>
<td>(0.0-0.0)</td>
<td>(0.0-0.0)</td>
<td></td>
<td>0.9†</td>
</tr>
<tr>
<td>B</td>
<td>9.6</td>
<td>3.0</td>
<td>1.2</td>
<td>0.001</td>
</tr>
<tr>
<td>(6.8-13.2)</td>
<td>(2.0-4.4)</td>
<td>(0.7-2.0)</td>
<td></td>
<td>0.006</td>
</tr>
<tr>
<td>C</td>
<td>5.5</td>
<td>1.8</td>
<td>0.5</td>
<td>0.001</td>
</tr>
<tr>
<td>(3.8-7.5)</td>
<td>(1.1-2.6)</td>
<td>(0.2-1.0)</td>
<td></td>
<td>0.002</td>
</tr>
<tr>
<td>D</td>
<td>1.0</td>
<td>0.9</td>
<td>0.4</td>
<td>0.8</td>
</tr>
<tr>
<td>(0.5-1.6)</td>
<td>(0.4-1.5)</td>
<td>(0.1-0.9)</td>
<td></td>
<td>0.13</td>
</tr>
</tbody>
</table>

Apnoeic phase following induction of anaesthesia (fig. 3)

Following induction of anaesthesia, there were highly significant posterior displacements of the soft palate, base of tongue and epiglottis (table 1). The displacement of the palate (median change 1.3 mm, 95% confidence interval (CI) 0.3-2.6 mm) resulted in apparent airway occlusion in all but one subject; that of the epiglottis (mean change 3.8 mm, 95% CI 3.1-4.5 mm) in only four subjects; and that of the tongue (mean change 6.5 mm, 95% CI 5.3-7.7 mm) in no subject. There was no significant change in the gap between posterior surface of tongue and soft palate. In addition, there were significant (P < 0.05) caudad shifts of both the epiglottis (mean change 3.8 mm, 95% CI 0.5-7.0 mm) and the hyoid bone (mean change 3.4 mm, 95% CI 0.02-6.7 mm).

Expiratory phase during anaesthesia

After spontaneous breathing under anaesthesia was established, there was a barely significant anterior movement of the epiglottis during expiration, but the other measurements were not significantly different from those during the apnoeic phase.

Inspiratory phase during anaesthesia (fig. 4)

After the re-establishment of spontaneous breathing during surgical anaesthesia, an attempted inspiration with the head in neutral position and the jaw unsupported resulted in extensive collapse of the pharynx, with significant reductions in most of its dimensions (table 1). The tongue touched the posterior pharyngeal wall in seven, and the epiglottis in 10 of 18 patients.

Effect of tongue traction (fig. 5)

In the second series of 11 subjects, tongue traction was used to establish if the posterior shift in the soft palate was passively related to posterior displacement of the tongue. However, while tongue traction was highly effective in drawing the tongue base anteriorly, it had no demonstrable effect on the soft palate.

Discussion

We are aware of only three studies [2, 9, 10] that have used conventional radiography to assess changes in pharyngeal configuration and airway patency in the anaesthetized, non-paralysed patient. Two of these involved very small numbers of subjects; none attempted any quantification of changes in anatomical dimensions.

In our study the primary and almost universal change was a posterior movement of the soft palate, which gave the radiological appearance of occlusion of the nasopharynx in 17 of 18 patients. Morikawa, Safar and DeCarlo [3] reported a mean posterior movement of the soft palate of 2.6 mm with occlusion in five of 10 patients following induction of anaesthesia and paralysis, and a mean posterior movement of the tongue of 5.5 mm compared with 6.5 mm (95% CI 5.3-7.7 mm) in our study, and occlusion in three of 10 patients. In contrast, we have failed to demonstrate obstruction between tongue and posterior pharyngeal wall in a single patient, except during attempted inspiration.

The similarity of our results to those of Morikawa in other respects suggests that anaesthesia, even without paralysis, markedly re-
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- apnoea
- inspiration

**FIG. 4.** Changes in pharyngeal outline in attempted inspiration during anaesthesia, but with the head and jaw in neutral position.

- apnoea
- tongue retraction

**FIG. 5.** Changes in pharyngeal outline during anaesthesia when firm traction was applied to the tongue. Note that the outline of the soft palate is unchanged.

It is known that electrical activity is present in genioglossus in the awake state [11, 12], and there is evidence in the cat for selective depression of the genioglossus electromyogram (relative to the diaphragm and intercostal muscles) under halothane anaesthesia [4], probably because of selective depression of hypoglossal nerve activity [5]. However, the relevance of genioglossus activity *per se* to airway obstruction under anaesthesia seems questionable in the light of our findings. Drummond [13] has failed also to demonstrate any correlation between genioglossus activity and airway patency in anaesthetized human subjects, and draws attention to the potential influence of complex interactions of various muscle groups on airway obstruction. So far as the soft palate is concerned (which on the basis of our findings may have greater relevance to airway obstruction under anaesthesia), there is no published work of which we are aware investigating the effects of anaesthetics on palatal neural or muscular activity.

The impression that the tongue is principally responsible for airway obstruction in the anaesthetized patient has probably been reinforced by the common observation that insertion of an artificial oropharyngeal airway usually relieves the obstruction. In fact this manoeuvre would be effective, even if the obstruction were solely at the nasopharynx, by providing an alternative route for airflow via the mouth rather than the nose. It is also possible that an occluded nasopharynx might contribute to airway obstruction even if the mouth was open, if the free edge of the tongue were to make a seal against the alveolar arch of the maxilla; this form of obstruction by the tongue would not be amenable to detection by lateral radiography.

There are several mechanisms by which the soft palate might occlude the nasopharynx after induction of anaesthesia. Tonic activity in palatoglossus and palatopharyngeus, perhaps in conjunction with tensor veli palatini, may be responsible for holding the palate away from the posterior pharyngeal wall in the awake state, and it is likely that anaesthesia abolishes or reduces activity in these muscles. Alternatively, anaesthesia (without paralysis) might activate levator palati as it does for the abdominal muscles [14]. However, the similarity of our results to those of Morikawa, Safar and DeCarlo [3] militates against this. A third possibility is derived from the presumed diminished activity of genioglossus.

Posterior movement of the tongue, which we have
demonstrated in the present study, could result in secondary posterior displacement of the soft palate, either by direct pressure or as a result of posterior movement of the glosso-laryngeal structures. Our observations on the effect of tongue traction (fig. 5) would seem to exclude this factor as a major influence. Subsidiary factors which we have not investigated include gravity, as suggested by Tomlin and Roberts [10], and surface adhesion between soft palate and posterior pharyngeal wall.

The study by Sivarajan and Fink [15], reporting anterior movement of laryngeal structures with anaesthesia, does not accord with our observations. However, their measurements from the third cervical vertebra would be at a variable angle to the cephalo-caudal axis and would therefore be influenced variably by caudad, in addition to anteroposterior, shifts of the epiglottis and hyoid.

In the light of our findings, it is not clear why artificial oral airways fail so often to provide a clear airway unless neck extension or jaw protrusion (or both) are used. It may be that, on the occasions when such devices fail, their tip is not within a clear airspace (as we have observed radiologically on one occasion); alternatively, these cases may represent obstruction by the epiglottis [8]. It is also possible that the configuration of the tongue may be altered by the airway in a manner that predisposes to airway obstruction. In any event, it seems likely that the nature of the obstruction varies between patients. Further studies will be required to determine why neck extension, jaw protrusion, or both, are often necessary when an artificial oral airway is in place.

The use of conventional radiography as a tool for exploring the upper airway clearly has the limitation of applying a two-dimensional imaging technique to a three-dimensional structure. Hence lateral channelling of airflow might theoretically occur in the presence of apparent midline occlusion. However, it is difficult to see how occlusion could exist in the presence of apparent patency on the radiograph. We therefore conclude that, in our series, the tongue was not responsible for airway obstruction, and that the soft palate would appear to have been the prime site, with possibly some contribution from the epiglottis. Attempted inspiration in the face of upstream obstruction causes major secondary pharyngeal collapse (fig. 4), as in obstructive sleep apnoea [16, 17].

REFERENCES

1. Heiberg J. A new expedient in administering chloroform. Medical Times and Gazette 1874 (January 10); 36.