Anesthesiology
1996;85:787–93

Effects of General Anesthesia and Paralysis on Upper Airway Changes Due to Head Position in Humans

Murali Sivarajan, M.B., B.S.,* James V. Joy, M.D.†

Background: **In supine patients with their heads in flexion, general anesthesia causes posterior displacement of upper airway structures that is associated with airway obstruction, and extension of the head helps restore patency. However, the independent effects of head position, general anesthesia, and muscle paralysis on upper airway structures are not known.**

Methods: **Lateral radiographs of the neck were taken in supine patients with the head in flexion and extension, during consciousness, and after induction of general anesthesia and muscle paralysis. The following measurements were made: distances from the horizontal plane to the epiglottis, the hyoid, and the thyroid cartilage to detect anteroposterior displacements; distances from the transverse plane to the hyoid and the thyroid cartilage to detect cephalocaudad displacements; and widths of the oropharynx, the laryngeal vestibule, and the laryngeal sinus.**

Results: **With the head in flexion, anesthesia and paralysis compared with the conscious state caused posterior displacement of the epiglottis, narrowing of the oropharynx, and widening of the laryngeal vestibule. With the head in extension, anesthesia and paralysis compared with the conscious state caused anterior displacements of the epiglottis, the hyoid, and the thyroid cartilage, narrowing of the oropharynx, and widening of the laryngeal vestibule and the laryngeal sinus.**

Conclusion: **Loss of tonic muscular activity due to anesthesia and paralysis results in anteroposterior displacements of the upper airway structures with flexion and extension of the head that are in the same direction as that of the mandible. Anesthesia and paralysis also widen the dimensions of the larynx. These changes might have implications for instrumentation and protection of the airway during general anesthesia or unconsciousness. (Key words: Anesthesia, general Muscle paralysis, larynx: dimensions. Upper airway.)**

INDUCTION of general anesthesia or loss of consciousness in supine patients with their heads in flexion or in the neutral position causes posterior displacements of upper airway structures that are associated with airway obstruction.[1–4] Posterior displacement is passive and perhaps due to gravity when the tonic activity of muscles that maintain airway structures in an anterior position is abolished on loss of consciousness.[3,5] However, a recent study reported that if general anesthesia is induced in supine patients with their heads in extension, the epiglottis and the hyoid are displaced anteriorly.[6] Extension of the head after anesthesia is induced reestablishes patency of the airway by restoring airway structures from the posteriorly displaced position,[1–3] but any displacement anterior to the neutral position affects visualization and instrumentation of the airway. Displacement of the airway structures in the cephalocaudad axis may also be expected with head movement. The effects of general anesthesia and muscle paralysis and head position on upper airway structures have not been examined in a single study. To determine these effects, lateral radiographs of the neck were taken in supine patients with their heads in flexion and extension when they were awake and after anesthesia and paralysis were induced.

Methods

Study participants were 45- to 75-y-old men scheduled for elective operations under general anesthesia. The study was approved by the Human Subjects Committees of the University of Washington and the Seattle VA Medical Center, and informed consent was obtained from each patient. Patients with a history of gastroesophageal reflux or obstructive sleep apnea or difficult orotracheal intubation were excluded. After establishing intravenous access, patients were positioned on the operating table with the head resting on a pillow to provide approximately 20-degree flexion of

*Associate Professor.

†Clinical Assistant Professor.

Received from the Seattle Veterans Affairs Medical Center and the Department of Anesthesiology, University of Washington, Seattle, Washington. Submitted for publication December 7, 1995. Accepted for publication June 20, 1996. Supported by the Office of Research and Development, Medical Research Service, Department of Veterans' Affairs.

Address correspondence to Dr. Sivarajan: Department of Anesthesiology, University of Washington, Seattle, Washington 98195-6540. Address electronic mail to: murali@u.washington.edu.

the neck. Using a skin-marking pen, a line joining the lateral canthus of the eye to the tragus of the ear (canthus-tragus line) was drawn on the side of the patient's face. The angle subtended by this line to the horizontal plane of the operating table represented the degree of flexion or extension of the head.[3] A 25-mm-long radioopaque marker was affixed to the side of the neck as a reference scale for making subsequent measurements on the radiographs. A Phillips BV 25 fluoroscopy machine (Eindhoven, the Netherlands) was used to obtain lateral radiographs of the neck, and the images were recorded on a videotape. First, lateral radiographs of the neck were taken at end expiration with the head in the neutral (flexed) position (angle of canthus-tragus line to horizontal plane, 90°) and after maximal voluntary extension. While radiographs were being taken, patients were asked to close their mouths and clench their teeth gently to obtain apposition of the mandible to the maxilla. The degree of flexion and extension of the head were measured using an orthopedic goniometer. Two milligrams metocurine was administered intravenously to prevent muscle fasciculations due to succinylcholine. General anesthesia and muscle paralysis were induced by intravenous administration of 3 to 5 mg/kg sodium thiopental and 1.5 mg/kg succinylcholine. After loss of consciousness and cessation of breathing, lateral radiographs of the neck were obtained with the head returned to the same degree of flexion and extension as when the patients were awake, with constant manual support of the chin to obtain apposition of the mandible to the maxilla. The study was terminated and anesthetic and operative procedures were performed. Radiation exposure did not exceed 30 s in all but one patient, delivering a total dose less than 500 millirems of ionizing radiation at the skin.

A horizontal plane passing through the anterior-inferior end of the fourth cervical vertebra (C4) and a transverse plane passing through the anterior-inferior end of the second cervical vertebra were drawn on each radiograph (fig. 1). The plane of the anterior surface of C4 was also drawn and the angle subtended by this plane to the horizontal was measured and designated as the C4 tilt (fig. 1). Using the radioopaque marker as the reference scale, the following measurements were made: vertical distances from the horizontal plane to the epiglottis, the hyoid, and the thyroid cartilage to detect anteroposterior displacements; vertical distances from the transverse plane to the hyoid and the thyroid cartilage to detect cephalocaudad displacements; and widths of the oropharynx, the laryngeal vestibule, and the laryngeal sinus (fig. 1). In each patient, the displacements of the same point on the anatomic structures were measured in all four radiographs. Usually the tip of the epiglottis and the anterior-inferior points on the body of the hyoid and the thyroid cartilages were used to measure the displacements. For the oropharyngeal width, anteroposterior dimensions of the air shadows at a fixed distance superior to the epiglottis were measured in all four radiographs. For the width of the laryngeal vestibule, anteroposterior dimensions of the air shadows at the base of the epiglottis were measured in all four radiographs because the base of the epiglottis forms the anterior boundary of the laryngeal vestibule.[7] The laryngeal sinus is bounded superiorly by the vestibular folds and inferiorly by the vocal folds and is seen as a biconvex air shadow inferior to the base of the epiglottis.[8] The maximum width of the laryngeal sinuses were measured in all the radiographs. The distance from C4 to a fixed point on the mandible was also measured in flexion radiographs only because the mandible was out of the field in most of the radiographs when the head was extended.

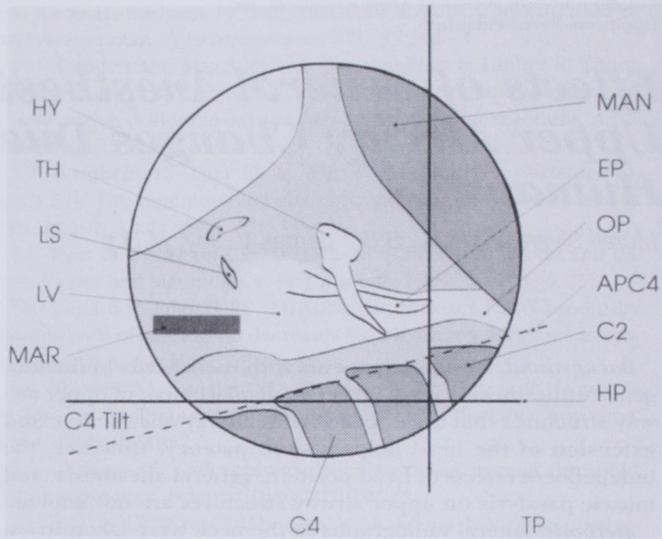

Fig. 1. Drawing from a radiograph showing structures and measurement planes. Structures: MAN = mandible; EP = epiglottis; OP = oropharynx; C2 = second cervical vertebra; C4 = fourth cervical vertebra; MAR = radioopaque marker (25 mm long); LV = laryngeal vestibule; LS = laryngeal sinus; TH = thyroid cartilage; HY = hyoid. Measurement planes: HP = horizontal plane; TP = transverse plane; APC4 = anterior plane of C4; C4 tilt = angle subtended by APC4 to the horizontal plane.

Because extension of the head at the atlantoccipital joint is terminated when the spinous process of the atlas contacts the occiput, Nichol and Zuck[9] have suggested that attempts to extend the head further would bow the cervical spine. Because measurements in this study were made during the conscious state with the head in maximal voluntary extension, it was assumed that extension of the head beyond this range during anesthesia and paralysis would bow the cervical spine. Thus the C4 tilt was taken as a measure of the constancy of head position, especially in extension. The C4 to mandibular distance was used as a measure of constancy of head flexion. Excluded from analyses were radiographs of patients in which these values during anesthesia and paralysis differed by more than 5% from values during the conscious state. With each head position, measurements made after induction of anesthesia and paralysis were compared with those made when the patient was awake using a Student's t test for paired values. Measurements with flexion of the head were also compared with those with extension of the head during anesthesia and paralysis using a Student's t test for paired values. Changes were considered significant when P was less than 0.05. To validate these measurements further, all the radiographs were coded, and the anterioposterior displacements of the epiglottis, the hyoid, and the thyroid cartilages were measured by the other investigator (J.V.J.), who was unaware of the code. Intraclass correlation coefficients between the measurements made by the two observers were then determined.[10] Correlations were considered significant when P was less than 0.01.

Table 1. Positions of Airway Structures and Dimensions of the Upper Airway during the Conscious State and after Induction of Anesthesia and Paralysis with the Head in *Flexion*

	Conscious State	Anesthesia and Paralysis
HP to epiglottis (mm)	21.4 ± 1.1	19.5 ± 1.4*
HP to hyoid (mm)	44.4 ± 2.1	45.6 ± 2.2
HP to thyroid cartilage (mm)	36.6 ± 2.4	34.8 ± 2.6
TP to hyoid (mm)	40.7 ± 3.4	43.3 ± 3.7
TP to thyroid cartilage (mm)	77.1 ± 5.0	81.2 ± 5.5
Oropharynx (mm)	10.9 ± 1.8	8.3 ± 1.7†
Laryngeal vestibule (mm)	16.0 ± 0.9	18.6 ± 0.6†
Laryngeal sinus (mm)	1.6 ± 0.9	3.3 ± 0.9
C4 to mandible (mm)	45.6 ± 5.6	46.4 ± 5.9
C4 tilt (°)	8.0 ± 1.6	7.7 ± 1.6

HP = horizontal plane; TP = transverse plane; C4 = fourth cervical vertebra; C4 tilt-angle subtended by the plane of the anterior surface of C4 vertebra to the horizontal.

Values are mean ± SEM; N = 10 for all measurements except those of the thyroid cartilage and laryngeal sinus, which were made in only eight and seven subjects, respectively.

* $P < 0.01$.

† $P < 0.05$.

Table 2. Positions of Airway Structures and Dimensions of the Upper Airway during the Conscious State and after Induction of Anesthesia and Paralysis with the Head in *Extension*

	Conscious State	Anesthesia and Paralysis
HP to epiglottis (mm)	28.6 ± 1.7	32.2 ± 0.7*
HP to hyoid (mm)	54.2 ± 2.3	60.6 ± 2.9†
HP to thyroid cartilage (mm)	39.0 ± 2.3	42.8 ± 3.4*
TP to hyoid (mm)	19.7 ± 3.1	22.6 ± 4.0
TP to thyroid cartilage (mm)	61.1 ± 5.5	61.7 ± 6.8
Oropharynx (mm)	19.9 ± 2.3	16.9 ± 1.5*
Laryngeal vestibule (mm)	25.6 ± 1.2	29.5 ± 1.1†
Laryngeal sinus (mm)	3.1 ± 0.8	5.4 ± 0.4*
C4 tilt (°)	9.0 ± 1.8	8.8 ± 1.8

HP = horizontal plane; TP = transverse plane; C4 tilt = angle subtended by the plane of the anterior surface of C4 vertebra to the horizontal.

Values are mean ± SEM; N = 10 for all measurements except those of the thyroid cartilage and laryngeal sinus, which were made in only eight and seven subjects, respectively.

* $P < 0.05$.

† $P < 0.01$.

Results

Radiographs from three patients were excluded from analyses because of inconstant head flexion between the conscious state and during anesthesia and paralysis as measured by the C4 to mandibular distance. Head extension was constant in all the radiographs as measured by the C4 tilt. Tables 1 and 2 present data from the remaining ten patients. The thyroid cartilages and the laryngeal sinuses were out of the field of the radiographs in two and three patients, respectively.

With the head in the flexed or neutral position, induction of anesthesia and paralysis resulted in a significant decrease (−9%) in the horizontal plane to epiglottis distance, indicating posterior displacement of the epiglottis. There were also a significant decrease (−24%) in the width of the oropharynx and a significant in-

Table 3. Intraclass Correlation Coefficients between Measurements Made by the Two Observers

	Conscious State		Anesthesia and Paralysis	
	Flexion	Extension	Flexion	Extension
HP to epiglottis	0.82*	0.93†	0.92†	0.80*
HP to hyoid	0.98†	0.99†	0.97†	0.99†
HP to thyroid cartilage	0.98†	0.99†	0.97†	0.99†

HP = horizontal plane.
* $P < 0.001$.
† $P < 0.0001$.

crease (+15%) in the width of the laryngeal vestibule (table 1). Only one patient had complete occlusion of the oropharynx when anesthesia and paralysis were induced with his head in flexion.

With the head in extension, induction of anesthesia and paralysis significantly increased the distances from the horizontal plane to the epiglottis (+13%), to the hyoid (+12%), and to the thyroid cartilage (+10%), indicating anterior displacements of these structures. The width of the oropharynx was significantly decreased (−15%), and the widths of the laryngeal vestibule and the laryngeal sinus were significantly increased (+15% and +74%, respectively) during anesthesia and paralysis compared with the conscious state (table 2).

Extension of the head during anesthesia and paralysis compared with flexion significantly increased the distances from the horizontal plane to the epiglottis (+165%), to the hyoid (+132%), and to the thyroid cartilage (+123%), indicating anterior displacements of these structures and significant decreases in the distances from the transverse plane to the hyoid (−48%) and to the thyroid cartilage (−24%), also indicating cephalad movement of these structures. Extension of the head during anesthesia and paralysis also significantly increased the widths of the oropharynx (+203%), the laryngeal vestibule (+158%), and the laryngeal sinus (+165%).

Intraclass correlations between measurements made by the two observers blinded to the other's findings were highly significant (table 3). Figure 2 shows radiographs from one patient, and figures 3 and 4 are composite drawings showing typical changes in the upper airway when anesthesia and paralysis were induced with the head in flexion and extension.

Discussion

Because airway structures are displaced with head movement, it was necessary to ensure constancy of flexion and extension of the head between the conscious state and during anesthesia and paralysis. This was accomplished in two ways. First, as described in Methods, in each patient during the conscious state and during anesthesia and paralysis, radiographs were made at the same degrees of flexion and extension as measured by the angles subtended by a line joining the lateral canthus of the eye and the tragus of the ear to the horizontal plane of the operating table. Second, objective measurements of C4 to mandibular distance and C4 tilt in the radiographs were used to test constancy of head flexion and extension, respectively, and radiographs from three patients were excluded from analyses because of inconstant head flexion between the conscious state and during anesthesia and paralysis. Thus this study, in which the effects of anesthesia and paralysis on upper airway structures with two different head positions were measured in the same patients, shows that upper airway structures are displaced in the direction in which the mandible is displaced in the anteroposterior plane. That is, with flexion of the head and a relative posterior station of the mandible, induction of anesthesia and paralysis resulted in posterior

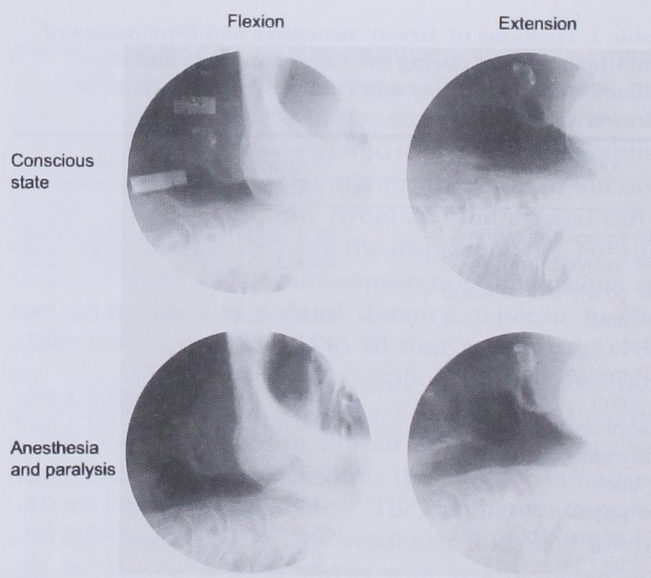

Fig. 2. Lateral radiographs from one patient showing slight posterior displacement of the epiglottis after anesthesia and paralysis were induced with the head flexed, and moderate anterior displacements of the epiglottis, the hyoid, and the thyroid cartilages after anesthesia and paralysis were induced with the head extended. There are also increases in the dimensions of the air shadows outlining the laryngeal vestibules and the laryngeal sinuses after anesthesia and paralysis were induced.

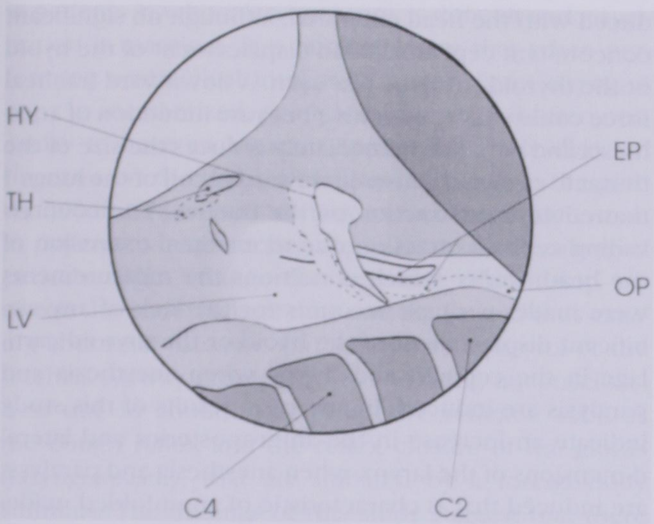

Fig. 3. Composite drawing from the radiographs taken with the head *flexed*, showing the positions of structures during consciousness (in continuous outline) and after anesthesia and paralysis were induced (in dashed outline).

displacement of upper airway structures relative to their position during the conscious state and, with the head in extension and an anterior station of the mandible, induction of anesthesia and paralysis resulted in anterior displacements of upper airway structures relative to their position during the conscious state. However, extension of the head, which moves the mandible anteriorly as well as in the cephalad direction, did not result in cephalad displacement of upper airway structures, as evidenced by nonsignificant changes in the distances from the transverse plane to the hyoid and the thyroid cartilage. There were also increases in the widths of the laryngeal vestibule and the laryngeal sinus indicating increases in the dimensions of the larynx. These findings suggest that skeletal muscle tone is important for the position of upper airway structures and for the dimensions of the larynx during the conscious state.

The decrease in the width of the oropharynx without a loss of patency in all but one patient correlates with previous reports concluding that obstruction of the oropharynx due to posterior displacement of the tongue was not a major mechanism for the loss of airway patency when anesthesia is induced.[4,11,12] Posterior displacement of the epiglottis on induction of anesthesia with the head in flexion or in the neutral position was also reported before[3,4] and could perhaps be attributed to passive movement due to gravity. Anterior movement of the epiglottis and the hyoid when anesthesia and paralysis were induced with the head in extension could be attributed to the loss of muscle tone resulting in anterior movements of these structures as they are drawn forward by the stretched platysma and the deep cervical fascia attached to the mandible. This implies that the balance of muscular forces during consciousness with the head extended maintains the upper airway structures, especially the larynx, in a relative posterior position. The stylohyoid, the stylopharyngeus, and the posterior belly of the digastric muscles exerting posterior traction on the thyrohyoid apparatus are opposed by the geniohyoid, the mylohyoid, and the anterior belly of the digastric muscles exerting anterior traction.[13] The middle and the inferior constrictor muscles, which also pull the thyrohyoid apparatus toward the posterior pharyngeal wall, have no functional antagonists. Perhaps the tensions of these constrictor muscles maintain the hyoid and the thyroid cartilage in a posterior position during consciousness.

The chin was manually supported to appose the mandible to the maxilla during anesthesia and paralysis to maintain the same position of the mandible in relation to the maxilla as during consciousness. Without the manual support of the chin, the mandible might be expected to lie in a posterior station, especially when the head is extended. Without manual support of the chin during anesthesia and paralysis, there may be greater posterior displacement of the epiglottis with

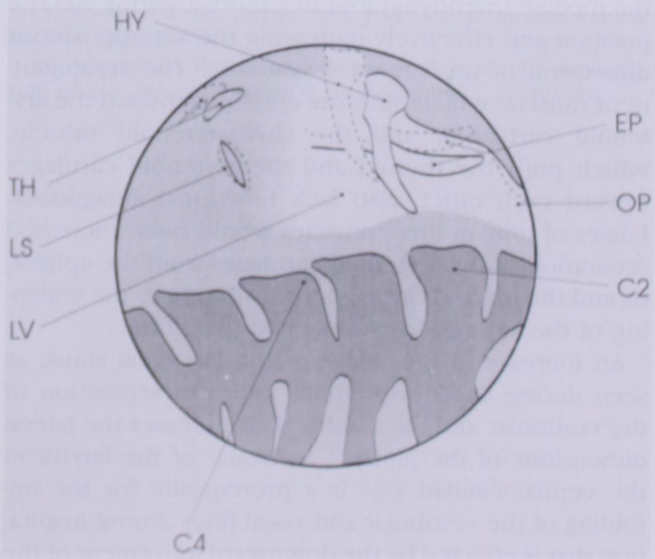

Fig. 4. Composite drawing from radiographs taken with the head *extended*, showing the positions of structures during consciousness (in continuous outline) and after anesthesia and paralysis were induced (in dashed outline).

the head in flexion and lack of any anterior displacements of the airway structures when patients' heads are extended compared with when patients are conscious.

Fink and Demarest[8] have advanced the concept of the larynx as an organ consisting of eight folds—the bilateral aryepiglottic, vestibular, and vocal folds and the unpaired median thyrohyoid and interarytenoid folds—that influence its dimensions. During effort closure (Valsalva maneuver) and swallowing, there is approximation of the hyoid and the thyroid cartilage and the folds are apposed to seal the laryngeal passage.[8] During deep inspiration, there is descent of thyroid cartilage, away from the hyoid, with wide separation of the folds to increase conductance.[8] This concept helps explain the increase in laryngeal dimensions when anesthesia and paralysis are induced with both head positions, even as the oropharyngeal dimension proximal to the larynx was decreased. The median thyrohyoid fold consisting of the base of the epiglottis and the connective tissue anterior to it forms the anterior boundary of the laryngeal vestibule.[7] Tension of the thyrohyoid muscle, approximating the hyoid and the thyroid cartilage, stresses the preepiglottic tissues, causing the median thyrohyoid fold to bulge posteriorly and decreasing the anteroposterior dimension of the laryngeal vestibule.[8] Loss of thyrohyoid muscle tone when anesthesia and paralysis are induced would relieve the stress on the preepiglottic tissue, restoring the median thyrohyoid fold to a more anterior, neutral position and effectively increasing the anteroposterior dimension of the laryngeal vestibule. The aryepiglotticus muscle, which pulls the epiglottis toward the arytenoid cartilages, and the thyroarytenoid muscle, which pulls the thyroid and the arytenoid cartilages toward each other, also lack functional antagonists. Losses of tone in these muscles would cause increased separation of the arytenoid cartilages from the epiglottis and the thyroid cartilage, contributing to the widening of the laryngeal vestibule and the glottis.

An increase in the width of the laryngeal sinus, as seen during deep inspiration, indicates separation of the vestibular and vocal folds that increases the lateral dimensions of the glottis.[8] A stretch of the larynx in the cephalocaudad axis is a prerequisite for the unfolding of the vestibular and vocal folds during inspiration that is effected by the downward movement of the trachea and the action of the sternothyroid muscle.[8] A significant increase in the width of the laryngeal sinus was observed when anesthesia and paralysis were induced with the head extended, although no significant concomitant cephalocaudad displacement of the hyoid or the thyroid cartilage was seen. A downward tracheal force could still be operative because induction of anesthesia and paralysis immediately reduces the size of the thoracic cage and causes an inward recoil of the lungs[14] that could exert traction on the trachea. The countervailing cephalad traction due to maximal extension of the head, under which conditions the measurements were made, perhaps accounts for the lack of any significant displacement of the hyoid or the thyroid cartilage in the cephalocaudad axis when anesthesia and paralysis are induced. In any case, results of this study indicate an increase in the anteroposterior and lateral dimensions of the larynx when anesthesia and paralysis are induced that is characteristic of an unfolded, wide-open larynx. These findings correlate remarkably with the widely abducted vestibular and vocal folds and a wide-open glottis that are seen clinically on direct laryngoscopy after anesthesia and paralysis are induced. Paralyses of the intrinsic muscles of the larynx, as in bilateral recurrent laryngeal nerve sections, result only in a partially open glottis with the vocal folds in the midposition,[15] suggesting that the paralyses of the muscles outside the larynx increase the glottic dimensions. To our knowledge, no other report exists on the effects of general anesthesia on laryngeal dimensions.

Extension of the head after induction of anesthesia and paralysis results in anterior movements of the epiglottis[2,3] and the hyoid cartilage,[3] and our study confirms these findings. Cephalad displacement of the hyoid and the thyroid cartilages that we observed when patients' heads were extended after induction of anesthesia and paralysis can be attributed to cephalad movement of the mandible, to which airway structures are attached by deep cervical fascia and muscles. Such anterior and cephalad displacements would be expected to result in unfolding of the larynx.[8] Our findings of increased dimensions of the laryngeal vestibule and laryngeal sinuses when the head is extended after anesthesia and paralysis are induced confirm such an unfolding of the larynx.

These results have implications for visualization, instrumentation, and protection of the airway. Direct laryngoscopy and orotracheal intubation and fiberoptic bronchoscopy are often performed in patients receiving general anesthesia with the head extended and mandible held anteriorly with the laryngoscope or with manual support of the chin. An anteriorly shifted larynx might create difficulty in performing these maneuvers in patients in whom laryngoscopy is difficult and visualization is marginal. It is not surprising that when performing orotracheal intubation under direct laryngoscopy in anesthetized, paralyzed patients, it is often necessary to have an assistant apply posteriorly directed pressure on the thyroid cartilage to visualize the glottis.[16] Shifting levels of consciousness and muscle tone in patients whose trachea are intubated for prolonged periods might change the anatomic relationship between the endotracheal tube and the larynx, thus causing abnormal pressure and stress on the larynx or the trachea. Airway protection is lost during unconsciousness due to abolition of protective reflexes, such as the cough reflex and the reflex closure of the glottis (laryngospasm), that are initiated by a foreign-body stimulus. The findings of this study suggest that there may be an additional risk beyond that imposed by the loss of protective reflexes. An unfolded, wide-open larynx that results from anesthesia and paralysis would offer less resistance to a foreign body than would a folded, partially closed larynx.

Induction of anesthesia and paralysis with the head flexed causes posterior displacements of the epiglottis; with the head extended, it causes anterior displacement of the epiglottis, the hyoid, and the thyroid cartilage. Onset of anesthesia and paralysis also decreases oropharyngeal dimension and increases the dimensions of the larynx at both head positions. These changes might have implications for visualization and instrumentation of the larynx and for protection of the trachea from a foreign body during general anesthesia or unconsciousness associated with flaccid paralysis.

References

1. Safar PL, Escarraga A, Chang F: Upper airway obstruction in the unconscious patient. J Appl Physiol 1959; 14:760–4
2. Morikawa S, Safar P, De Carlo J: Influence of the head-jaw position upon upper airway patency. Anesthesiology 1961; 22:265–70
3. Boidin MP: Airway patency in the unconscious patient. Br J Anaesth 1985; 57:306–10
4. Nandi PR, Charlesworth CH, Taylor SJ, Nunn JF, Dore CJ: Effect of general anesthesia on the pharynx. Br J Anaesth 1991; 66:157–62
5. Drummond GB: Keep a clear airway. Br J Anaesth 1991; 66:153–6
6. Sivarajan M, Fink BR: The position and the state of the larynx during general anesthesia and muscle paralysis. Anesthesiology 1990; 72:439–42
7. Fink BR: The median thyrohyoid fold: A nomenclatural suggestion. J Anat 1976; 122:697–9
8. Fink BR, Demarest RJ: Laryngeal Biomechanics. Cambridge, Harvard University Press, 1978, pp 1–112
9. Nichol HC, Zuck D: Difficult laryngoscopy—The "anterior" larynx and the atlanto-occipital gap. Br J Anaesth 1983; 55:141–4
10. Ostle B: Statistics in Research. Basic Concepts and Techniques for Research Workers. Iowa City, Iowa State University Press, 1963, pp 222–43
11. Abernathy LJ, Allan PL, Drummond GB: Ultrasound assessment of the position of the tongue during induction of anesthesia. Br J Anaesth 1990; 65:744–8
12. Mathru M, Esch O, Lang J, Herbert ME, Chaljub G, Goodacre B, vanSonnenberg E: Magnetic resonance imaging of the upper airway. Effects of propofol anesthesia and nasal continuous positive airway pressure in humans. Anesthesiology 1996; 84:273–9
13. Tucker HM: The Larynx. New York, Thieme Medical Publishers, 1987, pp 21–32
14. Westbrook PR, Stubbs SE, Sessler AD, Rehder K, Hyatt RE: Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. J Appl Physiol 1973; 34:81–6
15. Tucker HM: The Larynx. New York, Thieme Medical Publishers, 1987, pp 235–58.
16. Atkinson RS, Rushman GB, Lee JA: A Synopsis of Anaesthesia, 10th edition. Bristol, Wright IOP Publishing, 1987, pp 203–26