3.1 INTRODUCTION

3.1.1 What are the fundamentals of an awake, bronchoscopically facilitated intubation?

Awake bronchoscopic intubation, if it is to be performed rapidly and with minimal patient discomfort, requires an in-depth knowledge of the anatomy of the airway, adequate regional anesthesia, and dexterity with bronchoscopic manipulation. In order to achieve optimal regional anesthesia of the airway and avoid complications, a thorough knowledge of the local anesthetics employed and techniques of administration is necessary. The primary requirement for successful awake intubation is effective regional anesthesia of the airway.1

3.2 AIRWAY ANATOMY

3.2.1 Why is knowledge of upper airway anatomy beneficial in airway management?

Knowledge of the structure, function, and pathophysiology of the upper airway permits the practitioner to anticipate potential life-threatening problems and better utilize the full spectrum of airway management techniques.2 Functionally, the upper airway can be considered to consist of the nasal cavities, pharynx, larynx, and trachea (see Figure 3-1).3 The oral cavity provides an alternate access route to the pharynx.

3.2.2 The nose

Anatomically, the nose can be divided into an external component and the nasal cavity.4 The external nose consists of a bony vault posterior superiorly, a cartilaginous vault anteriorly, and the lobule at the inferior-anterior aspect (see Figure 3-2).5 The cavity of the nose is divided into bilateral compartments by the nasal septum and continues posteriorly from the nostrils (nares), to communicate with the nasopharynx at the posterior aspect of the septum (the choanae) (see Figures 3-3 to 3-5).3 The nasal vestibule is a small dilatation located immediately inside the nostrils.34 Each nasal cavity is bounded by a floor, a roof, and medial and lateral walls.3-5 The roof of the nasal cavity extends posteriorly from the bridge of the nose, and consists of the lateral nasal cartilages, the nasal bones and spine of the frontal bone, the cribiform plate of the ethmoid, and the inferior aspect of the sphenoid (see Figure 3-2).3,4,6 The nasal septum forms the medial wall, and is formed by the quadrilateral cartilage, the perpendicular plate of the ethmoid, and the vomer (see Figure 3-5).3 The lateral wall is formed anterior-inferiorly by the frontal process of the maxilla, the nasal bones anterior-superiorly, the nasal aspect of the ethmoid superiorly, and the perpendicular plate of the palate and medial pterygoid plate posteriorly.3,5 A series of three horizontal scroll-like ridges (conchae or turbinates) project medially from the lateral walls of the nasal cavities, each of which overhangs a corresponding groove or meatus (see Figures 3-2 to 3-4).3,8 Septal deviation is common, may be associated with compensatory hypertrophy of the turbinates, and can produce nasal obstruction.3,4 The paranasal sinuses and the nasolacrimal duct empty into the nasal cavity through ostia in the lateral wall.3 Obstruction of the ostia of the paranasal sinuses can occur with prolonged nasal intubation and can cause sinusitis.3,4 The floor of each nasal cavity is concave and is formed by the palatine process of the maxilla and the horizontal
FIGURE 3-1. Sagittal view of the upper airway.

FIGURE 3-2. Bony components of lateral nasal wall.
**FIGURE 3-3.** Coronal section of the maxillary sinus. The position of a nasotracheal tube (ETT) is shown in the right nasal cavity.

**FIGURE 3-4.** Lateral nasal wall.

**FIGURE 3-5.** Medial nasal wall.
plate of the palatine bone.\textsuperscript{3,4} The floor extends posteriorly in a transverse plane from the vestibule.

The major nasal airway is located below the inferior turbinate, declines slightly front to back (approximately 20 degrees), and a nasotracheal tube or flexible endoscope should be directed backward and slightly inferiorly along the floor of the nose.\textsuperscript{2-4} Occasionally, the posterior aspect of the inferior turbinate may be hypertrophied and resistance to the passage of a nasotracheal tube may be encountered at this location.\textsuperscript{4} Alternating counterclockwise/clockwise rotation of the tube changes the orientation of the bevel and may facilitate negotiation of the nasal cavity (see Chapter 11).

The anterior and posterior ethmoidal branches of the internal carotid artery supply the anterior-superior aspect of the nasal cavity and the sphenopalatine branch of the external carotid supplies the posterior-inferior aspect.\textsuperscript{3,9} The vestibule receives blood supply from both the anterior ethmoidal and sphenopalatine arteries as well as from nasal branches of the superior labial branch of the facial artery (see Figures 3-6 A and B).\textsuperscript{3,9} Anastomoses between vessels from these three different sources occur particularly at the anterior-inferior aspect of the septum (Little’s area or Kiesselbach’s plexus), and this is a common site of epistaxis.\textsuperscript{3,4,9} Tintinalli reported moderate to severe epistaxis in 7\% of 71 attempted emergency nasotracheal intubations.\textsuperscript{10} The single case of severe epistaxis in this series occurred in a patient with cirrhosis. Minimal epistaxis has been reported in 11\% to 40\% of nasal intubations.\textsuperscript{10,11} In a series of 99 patients undergoing nasotracheal intubation for oromaxillofacial surgery, epistaxis occurred in 6 patients but was sufficient to result in a visible accumulation of blood in the pharynx in only 1 patient.\textsuperscript{12} Of 175 anesthetists who underwent

![Diagram of blood supply to nasal cavity and septum](image)

**FIGURE 3-6.** (A and B.) Blood supply to mucosa of lateral nasal wall and septum.
nasotracheal intubation at a training course, minor nasal bleeding was seen in 20 during endoscopy or after extubation. None of the 20 subjects required suction to control bleeding or clear the airway, and the bleeding did not interfere with endoscopy. During nasal intubation passing the tube with the bevel at the tip facing the septum directs the leading edge away from the vascular septum; however, the optimum orientation of the tube is controversial (see Blind Nasal Intubation section in Chapter 11). Perforation into the submucosal space can occur and lead to hematoma and abscess formation in the retropharyngeal space. Excessive force must be avoided.

Common sensation to the nasal cavities is supplied by the ophthalmic and maxillary divisions of the trigeminal nerve. The posterior aspect of the septum is innervated by the short and long sphenopalatine branches of the maxillary nerve. Anteriorly the septum and the lateral wall is supplied by the anterior ethmoidal branch of the ophthalmic nerve (see Figures 3-7A and B). The posterior-superior aspect of the lateral wall is innervated by the short sphenopalatine nerve and the inferior aspect by the posterolateral nasal branches of the sphenopalatine nerve. Anteriorly, the floor of the nose is supplied by the anterior-superior dental branch of the infraorbital nerve and posteriorly by the greater palatine. Rooterls of the olfactory nerve located in the roof of the nose adjacent to the cribriform plate transmit the sense of smell.

In addition to being a respiratory pathway, the nose humidifies and warms inspired air, houses the olfactory receptors, removes bacteria, dust, and other particles from inspired air, and acts as a voice resonator.

3.2.3 The mouth
Anatomically, the mouth consists of (1) the vestibule which is bounded externally by the lips and cheeks and internally by the gums and teeth and (2) the mouth cavity. The mouth cavity is bounded by the alveolar arches and the teeth anteriorly and laterally, the hard palate and the anterior aspect of the soft palate above, and the anterior two-thirds of the tongue and the reflection of its mucosa.
onto the floor of the mouth and mandible below.\textsuperscript{3,4} Posteriorly, the oral cavity opens into the oropharynx at the oropharyngeal isthmus.\textsuperscript{3,5} The anterior two-thirds of the palate (hard palate) is composed of the palatine plates of the maxillae and the horizontal plates of the palatine bones (see Figure 3-2).\textsuperscript{3,4,15} Posteriorly the hard palate is continuous with the soft palate, which is composed of a tough, fibrous sheath and extends to a free posterior border. In the midline, the soft palate ends in the uvula\textsuperscript{3,4,15} then curves laterally to blend into the lateral pharyngeal wall at the palatoglossal and palatopharyngeal folds (anterior and posterior tonsillar pillars), respectively.\textsuperscript{3,4,15} The anterior and inferior aspect of the soft palate faces the mouth cavity and oropharynx, whereas the posterior and superior aspect is part of the nasopharynx.\textsuperscript{3,4,15} The uvula is a valuable midline landmark during bronchoscopic intubation through the mouth. Movement of the soft palate is controlled by five paired muscles including palatoglossus and palatopharyngeus, which descend in their respective folds to blend with the side of the tongue (palatoglossus) and the side wall of the pharynx (palatopharyngeus) and serve to approximate the folds.\textsuperscript{3,4} These folds can be used as landmarks for transmucosal glossopharyngeal nerve blocks. The palatine muscles help to isolate the nasopharynx from the mouth during swallowing and phonation.\textsuperscript{3,4} Paralysis permits regurgitation of food into the nasopharynx and results in nasal speech.\textsuperscript{3,4} Sensation to the palate is primarily supplied by the trigeminal nerve; however the glossopharyngeal nerve supplies the most posterior aspect (see Figure 3-8).\textsuperscript{3,4}

The anterior two-thirds of the body of the tongue occupies most of the floor of the mouth.\textsuperscript{2,4,6} The posterior third of the tongue lies in the oropharynx and is separated from the anterior two-thirds by a V-shaped groove on the dorsal aspect of the tongue, the sulcus terminalis.\textsuperscript{3} The posterior third of the tongue has abundant lymphoid nodules, the lingual tonsil,\textsuperscript{6} and hypertrophy of this lymphoid tissue can make intubation by direct laryngoscopy difficult or impossible.\textsuperscript{16} The tongue is also subdivided by a median vertical fibrous septum represented on the dorsum of the tongue by a shallow midline groove,\textsuperscript{15} another useful landmark during bronchoscopic intubation (see Figure 3-9). The tongue musculature is divided into intrinsic

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3-8}
\caption{The sensory distribution of the glossopharyngeal nerve. (Reproduced with permission from Basmajian JV: Grant’s Method of Anatomy, 8th edn. Baltimore: Williams and Wilkins [after Edwards], 1981.)}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3-9}
\caption{Horizontal section below lingua of mandible: superior view.}
\end{figure}
muscles that alter the shape of the tongue\textsuperscript{14} and extrinsic muscles that move the tongue as a whole (see Figure 3-10).\textsuperscript{2,3,15} The extrinsic muscles connect the tongue to the symphysis of the mandible (genioglossus), hyoid (hyoglossus), styloid process (styloglossus), and the soft palate (palatoglossus).\textsuperscript{14,15} In the supine unconscious individual, a decrease in genioglossal tone allows the tongue to move posteriorly and airway obstruction can occur. Sensation to the anterior two-thirds of the tongue is supplied by the lingual branch of the mandibular nerve,\textsuperscript{3,15} whereas sensation to the posterior third is supplied by the glossopharyngeal and superior laryngeal branches of the vagus.\textsuperscript{6,15} Stimulation of the posterior third of the tongue during awake intubation typically provokes the gag reflex and reflex secretions, and can be particularly problematic during bronchoscopic intubation. The tongue receives its blood supply from the lingual branch of the external carotid\textsuperscript{15} and is a very vascular structure.\textsuperscript{6} At the lateral aspect of the tongue, the mucous membrane is reflected onto the floor of the mouth and extends laterally to reach the gingiva, the "lingual sulcus."\textsuperscript{3} The "buccal sulcus" lies between the teeth and the cheek. Deep to the mucous membrane in the floor of the mouth on either side of the tongue anteriorly lie the sublingual glands, and deep to these structures lies the mylohyoid muscle which forms a sling to support the floor of the mouth (see Figure 3-11).\textsuperscript{3,6,7,15} The submandibular gland straddles the mylohyoid muscle posteriorly.\textsuperscript{3} Both the lingual and the hypoglossal nerves travel in the floor of the mouth lateral to the tongue in the lingual sulcus.\textsuperscript{3} Lingual branches of the glossopharyngeal nerve lie deep to the mucosa of the palatoglossal arch (anterior tonsillar pillar) at the lateral aspect of the tongue and can be blocked in this location (see Figure 3-12). The mylohyoid muscle divides the floor of the mouth into two potential spaces: the submandibular space below the muscle and the sublingual space above.\textsuperscript{2} Hematoma formation or infection in either of these fascial spaces can displace the tongue superiorly and posteriorly to produce airway compromise and can make intubation difficult (eg, Ludwig’s angina).

The mandible consists of a horseshoe-shaped body anteriorly and two rami posteriorly, which extend superiorly to end in a condylar head and a coronoid process with an intervening mandibular notch (see Figure 3-13).\textsuperscript{3} The condylar head articulates with the mandibular fossa of the temporal bone at

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3-10.png}
\caption{Extrinsic muscles of the tongue.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure3-11.png}
\end{figure}
Two types of movement occur at the TMJ—rotation and a forward gliding or forward translation—thereby opening the mouth (see Figure 3-14). Normal mandibular opening in the adult is about 4 cm, or at least two finger breadths, between the upper and lower incisors. Decreased mandibular mobility and anatomic variants, in particular micrognathia, can make intubation by direct laryngoscopy difficult or impossible.

### 3.2.4 The pharynx

The pharynx is a U-shaped musculofascial tube which extends from the base of the skull to the lower border of the cricoid cartilage where at the level of the sixth cervical vertebrae it is continuous with the esophagus (see Figure 3-1). Posteriorly, it rests against the prevertebral fascia. Anteriorly, it communicates with the nasal cavity, mouth, and the larynx at the naso-, oro-, and laryngopharynx, respectively (see Figure 3-15). From the inner aspect outward, the pharynx consists of mucosa, submucosa, muscle, and a loose areolar sheath, the buccopharyngeal fascia. This buccopharyngeal fascia is the thin fibrous capsule of the pharynx, contains the plexi of pharyngeal veins and nerves, and is continuous with the areolar sheath of the buccinator muscles and the
adventitia of esophagus. Superiorly, it is attached to the base of the skull. Edema associated with infection in the floor of the mouth, such as Ludwig’s angina, is limited by the buccopharyngeal fascia, can spread into the pharynx and larynx, and may lead to airway obstruction. The muscular layer of the pharynx is made up primarily of three paired constrictor muscles that curve around the pharyngeal lumen and telescope into one another (see Figure 3-16). The inferior constrictor consists of an upper oblique part and a lower transverse part (the cricopharyngeus) that is continuous with the esophagus and functions as an upper esophageal sphincter. The junction of the pharynx with the esophagus is the narrowest part of the gastrointestinal tract and is a common place for foreign bodies to impact. A prominent arch of the atlas vertebra (C1) may protrude anteriorly into the nasopharynx and during nasal intubation, the endotracheal tube can impact the mucosa and resist advancement at this level. Rotation of the tube will facilitate passage around this prominence. However, on occasion, digital manipulation through the mouth may be required, or it may be necessary to first pass a soft nasal trumpet into the pharynx beyond the anterior tubercle of the atlas.

FIGURE 3-15. Opened posterior view of the pharynx.
A nasogastric tube cut off at its proximal end can then be passed into the oropharynx through the trumpet and the trumpet removed. A nasal endotracheal tube can then be passed over the nasogastric tube beyond the tubercle of the atlas. The NG tube can then be removed and the endotracheal tube passed into the trachea.

The oropharynx extends from the soft palate to the epiglottis and lies behind the mouth cavity and posterior third of the tongue. The palatoglossal folds arch downward from the soft palate to the junction of the anterior two-thirds and posterior third of the tongue and provide the dividing line between the mouth and oropharynx (see Figure 3-9). This oropharyngeal isthmus is completed by the soft palate and the sulcus terminalis of the tongue. The palatoglossal folds are innervated by the superior laryngeal branch of the vagus. As seen during direct laryngoscopy, the larynx can be conceptualized to be a smaller cylinder eccentrically placed within and at the anterior aspect of the larger cylindrical pharynx. Laterally, the piriform fossae are bounded by the thyroid cartilage and the thyrohyoid membrane. Superior to the piriform fossae, the median glossoepiglottic fold connects the epiglottis to the tongue in the midline and the lateral glossoepiglottic folds connect it to the pharyngeal wall. The depressions formed between these folds are termed the valleculae and are considered to be within the oropharynx. During direct laryngoscopy, the Macintosh blade is inserted into the base of the vallecula to engage the glossoepiglottic ligament (beneath the glossoepiglottic fold) and thereby move the epiglottis anteriorly to expose the glottis.

Sensation to the nasopharynx and oropharynx is supplied primarily by the glossoepiglottic nerve. This glossoepiglottic nerve enters the neck in company with the internal carotid artery and the internal jugular vein. At the level of the styloid process, it leaves this position and winds anteriorly and inferiorly lateral to stylopharyngeus which it supplies (see Figure 3-18). The nerve then passes forward between the superior and middle constrictors and gives off pharyngeal branches as well as lingual branches to the posterior third of the tongue (see Figure 3-12). Glossoepiglottic nerve blocks can be performed posterior to the midline of the palatoglossal fold or at the base of the palatoglossal fold in the mouth. The maxillary branch of the trigeminal nerve supplies sensation to the roof of the nasopharynx and contributes to the sensory supply of the soft palate and the adjacent part of the tonsil.

The laryngopharynx receives sensory innervation from the internal branch of the superior laryngeal or the nerve can be approached percutaneously. It has been said that the superior aspect (pharyngeal surface) of the epiglottis is innervated by the glossoepiglottic nerve, whereas the inferior aspect (laryngeal surface) receives sensory innervation from the superior laryngeal nerve. Others have stated that both the surfaces of the epiglottis are innervated by the superior laryngeal branch of the vagus.

3.2.5 The larynx

The larynx is a complex structure made up of a framework of cartilages and fibroelastic membranes covered by a layer of muscles and lined by mucous membrane. It functions as an open valve during respiration, a partially closed valve during phonation, a closed valve during swallowing, and to produce increased intrathoracic pressure when effort is required (Valsalva maneuver). It extends from its oblique entrance or aditus to the lower border of the cricoid cartilage and bulges posteriorly into the laryngopharynx (see Figure 3-15). It is suspended from the hyoid bone which is itself attached to the mandible, tongue, and the base of the skull. The laryngeal cartilages include the thyroid, cricoid, epiglottic and the paired arytenoid, corniculate, and cuneiform cartilages (see Figure 3-19). The quadrilateral laminae of the thyroid cartilage...
meet in the midline anteriorly to form the thyroid prominence (Adam’s apple). Superiorly, the thyroid cartilage is attached to the hyoid by the thyrohyoid membrane. Posteriorly the lower horns of the thyroid cartilage articulate with the posteriorly oriented signet ring-shaped cricoid cartilage. Anteriorly, the thyroid cartilage is attached to the cricoid by the cricothyroid membrane, a suitable site for emergency surgical airway access in the adult. The cricoid cartilage is the only complete skeletal ring of the airway and can be used to provide cricoid pressure (Sellick maneuver) during rapid sequence induction/intubation. The paired arytenoid cartilages articulate with the superior aspect of the cricoid cartilage posteriorly. The corniculate cartilages in turn articulate with the apices of the pyramidal shaped arytenoids. The shallow depression between the two corniculate cartilages (the posterior commissure) is a useful landmark during laryngoscopy. The cuneiform cartilages are located lateral to the corniculate cartilages and lie within the aryepiglottic folds. The leaf-shaped epiglottis is attached directly to the thyroid cartilage inferiorly and the hyoid bone superiorly by the hyoepiglottic ligament. The remaining framework of the larynx consists of two paired fibroelastic folds, the quadrangular and triangular membranes (see Figure 3-20). The quadrangular membrane spans the space between the lateral border of the epiglottis and the arytenoid cartilage and extends posteriorly to attach to the arytenoid cartilage. The inferior border of the triangular ligament is attached obliquely to the cricoid cartilage whereas the upper border is free and thickened to form the vocal ligament (true vocal cord). In coronal section, the relationship of the true vocal cords to the false vocal cords and the laryngeal ventricle or sinus (between the true and false cords) can be readily appreciated (see Figure 3-21). The aryepiglottic, the vestibular (false cords), and the vocal folds (true cords) form a trilevel sphincter mechanism that regulates and protects the airway. The folds also divide the larynx into three spaces: the supraglottic compartment or vestibule above the false cords, the glottic compartment between the false and true cords, and the infraglottic compartment between the true cords and the lower border of the cricoid. The absence of a submucosal layer at the vocal ligament causes the cords to appear white and limits the extent to which they can swell in edematous conditions.

The average distance from the incisors to the vocal cords is 12 to 16 cm. A complex arrangement of intrinsic muscles alters the configuration of the laryngeal folds. The cricothyroid muscle is classified by itself as the only extrinsic muscle of the larynx; muscles that
Elevate or depress the larynx as a whole (e.g., during swallowing) are considered to be accessory laryngeal muscles.3,9

The larynx receives its nerve supply from the superior and recurrent laryngeal nerves.3,4 The superior laryngeal nerve arises from the vagus just below the pharyngeal plexus, passes medial to both the internal and external carotids, and then divides into a large sensory internal and a small motor external branch which supplies the cricothyroid muscle (see Figure 3-22).3,4,7 The internal branch pierces the thyrohyoid membrane to provide sensation to the laryngeal mucosa above the level of the false cords3,9,21,22 or true cords.6,7,20

On the right side, the recurrent laryngeal nerve leaves the vagus as it crosses the subclavian artery, loops posteriorly under the artery, and ascends to the larynx in the groove between the esophagus and
trachea. On the left, the nerve leaves the vagus as it crosses the aortic arch and similarly loops posteriorly under the arch and then runs superiorly between the esophagus and the trachea to reach the larynx.3,4 The recurrent laryngeal nerves supply all the intrinsic muscles of the larynx and provide sensation below the level of the false3,9,21,22 or true cords.6,7,20 Hoarseness produced by damage to the superior laryngeal nerve is usually temporary as the contralateral cord exerts a compensatory action.3,4 Damage to the recurrent laryngeal nerve also produces hoarseness, and if both nerves are affected, severe airway obstruction can occur.3,15

The cricothyroid membrane or ligament can be identified in the anterior neck as a concavity between the convex inferior border of the thyroid cartilage and the superior portion of the cricoid cartilage (see Figures 3-19 and 3-23). The space is trapezoidal in shape with a cross-sectional area of 2.9 cm² and a mean height of 9 mm (range 5-12 mm).26,27 The average vertical distance between the true cords and the midpoint of the cricothyroid membrane is 13 mm in the adult.26 The vertical distance from the lower border of the thyroid cartilage to the vocal cords is 5 to 11 mm.27,28 The cricothyroid branches of the superior thyroid arteries run transversely across the membrane, usually the upper third,27,28 and tributaries of the anterior jugular veins occasionally run anterior to the membrane, although considerable variation exists in the vascular pattern.27 During cricothyrotomy or membrane puncture, the cricothyroid membrane should be traversed at its inferior third to minimize vascular injury.27,28

3.2.6 The trachea

The trachea extends inferiorly from its junction with the larynx at the lower border of the cricoid cartilage to the carina (see Figure 3-24).3,4,29 It is approximately 10 to 15 cm in length in the adult and about 13 to 20 mm in diameter.3,4,6,22,24 The inferior half of the trachea lies within the superior mediastinum.6 The cervical trachea is in the midline; however the intrathoracic portion is deviated to the right by the aortic arch.4 The patency of the trachea is maintained by 16 to 20 U-shaped rings of hyaline cartilage6 joined by fibroelastic tissue and closed posteriorly by the trachealis muscle.3,4 Longitudinal mucosal markings can be seen posteriorly when the trachea is viewed through the bronchoscope, and these can be used for spatial orientation. The average distance from the central incisors to the carina is 27 cm in the adult male and 23 cm in the adult female.17 The distance from the nostrils to the carina is an additional 4 cm.17 Tracheotomy is usually performed between the second and third or third and fourth tracheal rings.4,27,30

3.2.7 How is the anatomy of the pediatric airway different from that of the adult?

Awake intubation is most often performed in the adult population, and this chapter on preparation for awake intubation is directed to this age group. Management of the pediatric airway does require consideration of anatomic differences in children. The reader is directed to the pediatric sections of the text for further information (see Chapter 42 and Figure 42-5).
3.3 LOCAL ANESTHESIA OF THE AIRWAY

3.3.1 What drugs are useful for airway anesthesia? What are their toxicities and associated complications?

Lidocaine, tetracaine, cocaine, benzocaine, and dyclonine have all been used to produce topical anesthesia of the airway, and lidocaine is commonly used to perform glossopharyngeal and superior laryngeal nerve blocks.

3.3.2 Lidocaine

Introduced in 1948, lidocaine is the prototypical member of the amide class of local anesthetics and is metabolized in the liver by mixed function oxidases. The principle metabolite is monoethylglycinexylidide (MEGX) which has a local anesthetic effect and side effect profile similar to lidocaine. Hepatic metabolism of lidocaine appears to be limited by liver perfusion as well as parenchymal disease such as cirrhosis and the clearance of lidocaine is reduced in the presence of cardiac and hepatic insufficiency. Orally ingested lidocaine undergoes first pass metabolism in the liver and about 35% of an oral dose reaches the systemic circulation. Local anesthetics are probably the local anesthetics most commonly used for regional anesthesia of the airway and have also been used extensively in the past for the treatment of ventricular arrhythmias.

Following application to the mucous membranes of the airway, lidocaine produces an anesthetic effect which is limited to the mucous membrane in 1 to 2 minutes. The peak anesthetic effect occurs within 2 to 5 minutes, and the duration of the airway anesthesia is said to be 30 to 40 minutes, 20 to 40 minutes, or 15 to 30 minutes. Watanabe et al found the duration of anesthesia following the application of lidocaine to the oral mucosa to be 40 minutes with glycopyrrolate pretreatment, and 20 minutes without it. Schonemann et al sprayed 10% (100 mg·mL⁻¹) lidocaine onto the oral mucosa of the lower lip and demonstrated a hypoalgesic effect that lasted 14 minutes. Maximum hypalgesia was observed after 4 to 5 minutes. Adriani et al found that the maximum effective concentration of topical lidocaine applied to the tongue was 4% (40 mg·mL⁻¹), and that this concentration had a latent period of 2 minutes, and a duration of effect of 15.2 minutes. The latent period has a profound significance when bronchoscopic intubation is performed using a spray-as-you-go technique and suggests that a significant risk of insufficient anesthesia may exist. Concentrations of lidocaine of 1% (10 mg·mL⁻¹) to 10% (100 mg·mL⁻¹) have been used for topical anesthesia of the airway. Excellent topical anesthesia of the airway in the adult can be produced by 4% lidocaine, but at 2% (20 mg·mL⁻¹) concentration, topical anesthesia may be inadequate, and 1% lidocaine has been found to be insufficient for airway instrumentation. Increasing the concentration beyond the optimum level does not affect the latent period or duration of action, and increasing the...
dose to a given area of mucosa beyond 15 mg of lidocaine per square centimeter does not increase the anesthetic effect. Importantly, topical epinephrine penetrates mucous membranes poorly, has no significant local effect, does not prolong the duration of topical local anesthesia, and will not slow the rate of anesthetic absorption.

Local anesthetics applied to the mucous membranes of the airway are rapidly absorbed into the circulation. The extent of systemic absorption depends on the site and technique of application, tissue vascularity, the total dose administered, the state of the mucosa, the concomitant use of drying agents, the amount of mucus present, the rate and depth of respiration, the state of the circulation, the patient’s disease state, and individual variation. Slower uptake occurs from the more proximal parts of the respiratory tract. Absorption is particularly rapid when local anesthetics are applied to the tracheobronchial tree, whereas decreased rates of absorption occur in the upper airway secondary to decreased vascularity and surface area. Absorption from alveoli may approximate IV administration due to osmotic relationships in the pulmonary vascular bed designed to prevent the collection of fluid in the alveolar spaces. The therapeutic serum concentration of lidocaine when the drug is used as an antiarrhythmic is usually considered to be 1.5 to 4.0 μg·mL⁻¹, or 1.5 to 5 μg·mL⁻¹. As serum lidocaine concentrations increase however, systemic toxicity is produced. The commonly accepted toxic plasma concentration of lidocaine has been said to be 5 μg·mL⁻¹. At the upper limit of the antiarrhythmic therapeutic range, lightheadedness, tinnitus, and circumoral and tongue numbness can occur. As serum concentrations continue to rise, visual disturbances and muscle twitching occur and can be followed by generalized seizure activity. Seizures are most frequently seen at plasma concentrations more than 8 to 10 μg·mL⁻¹, although they
have occurred at plasma concentrations as low as 6 µg·mL⁻¹.⁴⁷ Cardiorespiratory arrest can occur at plasma lidocaine concentrations of 20 to 25 µg·mL⁻¹.⁴⁷,⁵⁵ Levels in arterial blood have been shown to be 20% to 30% higher than those in venous samples⁴⁸,⁵⁶,⁵⁷ and more closely correlate with CNS effects.⁵⁸ However, most clinicians consider venous blood levels to be reliable indicators of clinical toxicity.⁴⁷ Hypersensitivity reactions to lidocaine, although exceedingly rare, can also occur and can be catastrophic.⁴⁹ Clinically significant methemoglobinemia has been reported in association with lidocaine administration, although these occurrences have been extremely rare.⁵⁹,⁶⁰ A 2007 review of the literature found 12 episodes of methemoglobinemia related to lidocaine without associated prilocaine or benzocaine use.⁶¹ In seven of these cases an oxidative drug had been administered concomitantly. In one case the episode occurred within 24 hours of exposure to benzocaine, and one case occurred after the chronic abuse of lidocaine gel. Of the remaining three cases, one developed cyanosis about 21 hours after lidocaine injection.⁶² Methemoglobin levels were not reported but the patient responded to methylene blue.⁶³

The maximum safe dose of topical lidocaine has been stated to be 4 mg·kg⁻¹,³,⁴,⁶,⁷⁻⁰ mg·kg⁻¹,¹³ and 6 mg·kg⁻¹.¹⁷ However, when topically applied to the mucous membranes of the airway, these maximal doses can only be interpreted when the method of topical administration is known. The exact dose of lidocaine delivered to the airway when an inhalational technique is used is difficult to measure,⁶⁴ and the dose administered may bear little relation to the dose actually absorbed.⁷¹ Lidocaine administered to the airway by nebulization has been reported to produce low peak plasma concentrations as compared to other techniques.⁴⁷,⁴⁸,⁵⁸,⁷² This has been attributed to the loss of up to 50% of the nebulized solution to the environment with continuous nebulization.⁵⁸,⁷³ Absorption of local anesthetics administered by aerosols is also dependent on droplet size. Typical nebulizers produce droplets that range from 1 to 20 µm in diameter.⁷⁴ The peak deposition of aerosol droplets occurs in the peripheral airway for droplets of about 2 µm, in bronchioles for droplets of about 8 µm, in bronchi for droplets of about 15 µm, and in the upper airway for droplets larger than 40 µm.⁷⁵ Higher oxygen flow rates through nebulizers create smaller droplets (<30 µm) that travel further distally into the bronchial tree and increase the rate of absorption.⁸¹

Droplets larger than 60 µm are preferred for airway anesthesia during awake intubation because they rain out in the proximal airway where the topical anesthesia is required.⁴¹ The droplet size produced by manually squeezing the bulb of the hand-held DeVilbiss #40 nebulizer tends to be much larger than that produced by conventional aerosol delivery systems and is dependent on the pressure generated in the atomizer.⁷⁶ The mean bulb pressure produced by a firm squeeze is 250 to 340 mm Hg (4.86–6.01 psi). The mass median diameter (MMD) of the droplets thus produced was found to be 6.2 to 12.0 µm with 28% to 50% of the particles being less than 6.2 µm in diameter, small enough to penetrate the tracheobronchial tree.⁷⁶ With increasing firmness of the manual squeeze and increased bulb pressure, these nebulizers increase output and decrease MMD. When an oxygen flow through the RD 15 DeVilbiss atomizer of 5.0 L·min⁻¹ was utilized, pressures at the inlet of the device of 3.8 to 4.0 psi were generated, and at 8 L·min⁻¹ oxygen flow pressure was 11.2 to 11.4 psi. This technique may produce smaller droplets leading to increased systemic absorption, although it has been shown that only about 7% to 12% of the nebulized dose of a drug actually reaches the lung.⁷²,⁷³ Furthermore, the anesthetic expectorated after gargle and atomizer administration must be subtracted from the total dose administered.

Many investigators have endeavored to link route of administration and dosage of lidocaine used for airway anesthesia to plasma levels and toxicity:

- In a study performed by Melby et al, 1.5 mg·kg⁻¹ of 4% lidocaine was injected into the endotracheal tube of six patients under general anesthesia. Peak serum lidocaine concentrations of 1.4 to 3.3 µg·mL⁻¹ occurred at 11.7 ± 5.2 minutes following administration. Three of the six patients experienced almost instantaneous absorption.⁷⁹
- Chu et al measured plasma lidocaine concentrations produced by tracheal spraying using 3.3 mg·kg⁻¹ of 4% lidocaine and IV injection of 1 mg·kg⁻¹ of 2% lidocaine. After tracheal spraying, maximum plasma lidocaine levels of 2.0 to 5.6 µg·mL⁻¹ were recorded at 15 to 20 minutes. Following IV administration, peak concentrations of 5.0 to 6.85 µg·mL⁻¹ were reached within 12 minutes.⁷⁵
- Curran et al measured the concentration of lidocaine in venous blood following tracheal as compared to laryngeal spraying in 10 patients under general anesthesia. Each group received 3 mL of 10% lidocaine. The peak lidocaine concentration in the tracheal group ranged from 1.9 to 8.2 µg·mL⁻¹ whereas in the laryngeal group the range was 0.4 to 2.5 µg·mL⁻¹. Lidocaine levels in the tracheal group tended to rise more rapidly and reach a peak earlier than in the laryngeal group.⁸⁰
- Eyres et al administered 4 mg·kg⁻¹ of 4% lidocaine into the larynx and immediate subglottic area of 96 children under general anesthesia and measured plasma lidocaine levels at 2, 4, 6, 10, 15, 20, and 30 minutes after administration. Mean peak lidocaine levels measured were 4.3 ± 1.9 µg·mL⁻¹ for those less than 1 year of age, 5.7 ± 2.0 for those 1 to 3 years of age, 5.3 ± 1.4 for those 3 to 5 years of age, and 5.3 ± 2.0 for those more than 5 years of age. Plasma lidocaine levels exceeded 8.0 µg·mL⁻¹ in 13 patients. The time to peak concentration varied from 8.5 ± 2.5 minutes in those less than 1 year of age to 11.7 ± 4.3 minutes in those more than 5 years of age.⁸¹
- Parks et al administered 6 mg·kg⁻¹ of 10% lidocaine to 10 ASA I volunteers via a nebulizer connected to a facemask powered by an oxygen flow of 6 L·min⁻¹. The mean peak serum concentration of lidocaine produced was 0.29 µg·mL⁻¹, and the highest measurement was 0.45 µg·mL⁻¹. The peak concentration occurred 30 minutes after nebulization was commenced.⁸⁸
- Chinn et al administered 10 mL of 4% lidocaine to five healthy subjects using a DeVilbiss 35B ultrasonic nebulizer connected to a Hudson oxygen mask.⁷³ Plasma lidocaine levels were measured at the start of nebulization, at 5-minute intervals for 30 minutes and then at 45, 60, 90, and 120 minutes after the start of nebulization. The mean peak plasma level occurred at 10 minutes and, as measured from the published graph, was 0.95 µg·mL⁻¹.⁷³
- Mostafa et al similarly administered 6 mg·kg⁻¹ of 10% lidocaine to 14 ASA I and II patients scheduled for head and neck surgery...
using a nebulizer system with a mouthpiece and an oxygen flow of 7 L-min\(^{-1}\). The mean plasma lidocaine level 10 minutes after nebulization was 0.95 ± 0.62 μg·mL\(^{-1}\), and at 20 minutes it was 0.68 ± 0.32 μg·mL\(^{-1}\).

- Sutherland and Williams administered 5 mL of 4% lidocaine to 20 adult patients using a standard nebulizer connected to a mouthpiece and an oxygen flow of 8 L-min\(^{-1}\). The mouthpiece was connected to an oral airway intubator that was advanced into the oropharynx during the latter part of the nebulization process. The subjects also gargled 5 mL of 2% lidocaine gel for 1 minute, and 2 mL of 2% lidocaine was injected through the bronchoscope onto the vocal cords, as well as 2 mL into the trachea. The dosage range was 2.5 to 11 mg·kg\(^{-1}\) (mean dose, 5.3 ± 2.1 mg·kg\(^{-1}\)). The mean peak plasma concentration was 0.7 ± 0.4 μg·mL\(^{-1}\), and the highest plasma concentration was 1.6 μg·mL\(^{-1}\). The mean peak concentration occurred 23 minutes following intubation.

- Kirkpatrick et al administered 10 mL of nebulized 4% lidocaine to 10 normal volunteers using a DeVilbiss 646 nebulizer, an oxygen flow of 6 L-min\(^{-1}\), and a mouthpiece during tidal breathing. Blood was sampled at 10, 20, 30, 40, and 50 minutes after the start of nebulization. In three subjects additional samples were obtained at 60, 80, and 120 minutes. The mean peak serum lidocaine level was 0.52 ug·mL\(^{-1}\) and occurred 20 minutes after beginning nebulization. The highest single measurement was 1.05 ug·mL\(^{-1}\).

- Wieczorek et al administered topical lidocaine to a group of 27 obese patients prior to bronchoscopic intubation. Forty milliliters of either 2% lidocaine (14 patients) or 4% lidocaine (13 patients) was administered using a DeVilbiss DV-15-RD atomizer and an oxygen flow of 10 L-min\(^{-1}\). Plasma lidocaine concentrations were measured at intervals from before atomization to 120 minutes following the completion of atomization. Peak plasma concentrations were recorded in the 10 minute samples in both groups. The mean peak level in the 4% group was 6.5 ug·mL\(^{-1}\) (standard error of the mean, 1.0 ug·mL\(^{-1}\)). In the 2% group, the peak level was 2.8 (0.8) ug·mL\(^{-1}\). No clinical signs of lidocaine toxicity were detected.

- Woodruff et al subsequently reported a prospective randomized blinded study from the same center which compared 1% lidocaine with 2% lidocaine using the same atomization technique. Forty milliliters of either 1% or 2% lidocaine was administered. Mean peak plasma lidocaine levels were measured 5 minutes after completion of topicalization and were 3.8 (0.5) ug·mL\(^{-1}\) in the 2% group and 1.4 (0.3) ug·mL\(^{-1}\) in the 1% group. No signs of lidocaine toxicity were detected.

- Gomez et al measured serum lidocaine levels in 29 patients who underwent bronchoscopy and who had between 180 and 400 mg of lidocaine instilled onto the tracheobronchial mucosa. Blood was sampled at intervals between 10 minutes and 2 hours after administration in 9 patients, and from 45 minutes to 2 hours in 20 patients. In the group who had the whole serum concentration curve determined, the average maximum serum concentration was 1.21 ± 0.64 ug·mL\(^{-1}\). The peak concentration occurred at 34.02 ± 10.74 minutes. The average serum level 2 hours after administration was 0.69 ± 0.29 ug·mL\(^{-1}\) and the average serum half-life of lidocaine was 1.35 ± 0.41 hours. The percentage of lidocaine absorbed was calculated to be between 60.88% and 20.89% with an average value of 36.55 ± 13.95%.

- Patterson et al reported the administration of up to 380 mg of lidocaine almost entirely delivered as a 1% solution through the bronchoscope to 21 adult patients. With the exception of one patient, the maximum blood levels of lidocaine recorded were less than 2.48 μg·mL\(^{-1}\). The peak concentration occurred between 5 and 75 minutes following lidocaine administration, usually between 5 and 30 minutes. Considerable individual variation in the maximum concentration measured was noted. One patient with abnormal liver function was found to have a plasma concentration of 18.2 μg·mL\(^{-1}\) but developed no signs of toxicity.

- Xue et al performed a randomized double-blind study which compared 2% and 4% lidocaine administered via the bronchoscope using a spray-at-you-go technique in a group of 52 sedated patients prior to bronchoscopic intubation. The mean dose of lidocaine administered was 3.4 ± 0.6 mg·kg\(^{-1}\) (range 3.2-4 mg·kg\(^{-1}\)) in the 2% group, and 7.1 ± 2.1 mg·kg\(^{-1}\) in the 4% group (range 6.1-8.1 mg·kg\(^{-1}\)). The highest lidocaine concentration measured in the 2% group was 2.0 ug·mL\(^{-1}\), and in the 4% group the highest concentration measured was 3.6 ug·mL\(^{-1}\). Concentrations above 3 ug·mL\(^{-1}\) occurred in only three patients, all in the 4% group. In most patients the peak lidocaine concentration was observed at 20 to 30 minutes after lidocaine administration. Again, significant individual variation in plasma levels was noted.

- Boye and Bredesen administered about 7 mL of 4% lidocaine to nine adult patients prior to bronchoscopy utilizing nebulization, a topical applicator, instillation, and injection through the bronchoscope. Blood was sampled about 8 minutes after the start of anesthesia and after bronchoscopy, about 38 minutes after the start of anesthesia. The mean maximum plasma concentration of lidocaine was 2.7 ug·mL\(^{-1}\). No clinical signs of lidocaine toxicity occurred.

- Bigeleisen et al administered a total of 7 mg·kg\(^{-1}\) of lidocaine to a group of 20 patients who underwent bronchoscopic intubation. Four mg·kg\(^{-1}\) of atomized 4% lidocaine was applied to the nose and nasopharynx, and percutaneous superior laryngeal nerve blocks were performed using 0.5 mg·kg\(^{-1}\) of 1% lidocaine. The patients were then randomized into two groups. Group 1 underwent transtracheal injection of 2 mg·kg\(^{-1}\) of 4% lidocaine, whereas Group 2 received an additional equivalent dose of 4% lidocaine applied to the nasopharynx. Plasma lidocaine levels were measured every 2.5 minutes for 10 minutes after the last dose of lidocaine and then every 5 minutes for an additional 20 minutes. The mean peak lidocaine level in the transtracheal group was 4.06 ug·mL\(^{-1}\) and occurred at 10 minutes. The highest level recorded was 6.04 ug·mL\(^{-1}\). In the nasotracheal group, the mean peak level was 3.16 ug·mL\(^{-1}\) and occurred at 7.5 minutes. The highest level was 6.57 ug·mL\(^{-1}\). No signs of systemic toxicity were detected.

- Loukides et al measured plasma lidocaine levels for up to 2 hours after the start of topicalization in 12 patients who underwent...
fiberoptic bronchoscopy. The lidocaine was administered as a 2% gel to the nose and a 2% solution injected through a laryngeal syringe or the bronchoscope. The mean total dose administered was 622 ± 20 mg (range 500-720 mg). Peak plasma concentrations were observed at 20 minutes in eight patients, at 30 minutes in three patients, and at 60 minutes in one patient. The highest value measured was 2.25 μg·mL⁻¹.¹⁵

- Efthimiou et al measured plasma concentrations of lidocaine in a group of 41 patients who underwent topical airway anesthesia before fiberoptic bronchoscopy. In 32 patients, 14 sprays of 10% lidocaine were administered to the nose and oropharynx followed by 8 mL of 4% lidocaine solution administered via the bronchoscope to the pharynx and vocal cords, and 14 mL of 1% lidocaine to the bronchial tree. In nine patients, 8 mL of 2% lidocaine gel was applied to the nose and the use of the 10% spray was omitted. The average dose of lidocaine administered was 9.3 ± 0.5 mg·kg⁻¹. The average peak plasma concentration was 2.9 ± 0.5 μg·mL⁻¹ and correlated with dose per unit body weight. Two patients had plasma levels above 5 μg·mL⁻¹ but demonstrated no clinical evidence of toxicity. The average time to peak concentration was 42.6 minutes in the aerosol group and 48.4 minutes in the gel group. In a second study of 10 volunteers, plasma concentrations following 4% lidocaine gargle and swallow was compared with 10% oropharyngeal spray. The dose in each group was 6.8 mg·kg⁻¹. The gargle produced a peak concentration of 2.4 μg·mL⁻¹, whereas the spray produced a peak concentration of 1.9 μg·mL⁻¹ at 50 minutes.⁴⁶

- Reasoner et al randomized 40 adult patients undergoing awake fiberoptic intubation and surgery for cervical spine instability into topical and nerve block groups. Up to 20 mL of 4% lidocaine was administered to the topical group via a nebulizer attached to a facemask using a flow rate of 10 L·min⁻¹. Nebulization required about 10 minutes and was followed by cricothyroid puncture and injection of an additional 3 mL of 4% lidocaine. In the nerve block group, airway anesthesia was achieved with 50 mg of 10% lidocaine spray applied to the tongue, bilateral glossopharyngeal nerve block at the palatoglossal fold using 0.5 to 1.0 mL 2% lidocaine, superior laryngeal nerve block using 1 to 2 mL 2% lidocaine, and 3 mL 4% lidocaine injected through the cricothyroid membrane. Arterial blood was sampled for the plasma lidocaine level following administration of local anesthesia, 2 minutes prior to intubation (time zero), and again 10 minutes later. The topical group received 815 ± 208 mg of lidocaine whereas the nerve block group received 349 ± 44 mg. Mean plasma lidocaine levels at time zero were 2.16 ± 1.48 μg·mL⁻¹ in the topical group, and 4.23 ± 1.12 μg·mL⁻¹ in the nerve block group. Ten minutes later, the levels were 3.34 ± 1.87 μg·mL⁻¹ and 4.02 ± 1.02 μg·mL⁻¹, respectively. No plasma sampling was performed after the 10-minute recording. The quality of anesthesia achieved was similar with both the techniques, and intubation was achieved in 3.2 minutes on average. No complications were identified.⁹⁰

- Langmack et al administered topical lidocaine to 51 asthmatic volunteers who underwent research bronchoscopy.⁹¹ Atomized 4% lidocaine was sprayed into the nose and throat, 2 mL of viscous lidocaine was applied to the nose, and 1% lidocaine was administered to the tracheobronchial tree via the bronchoscope. The 2% viscous was excluded from the total dose calculation and the authors considered absorption of intranasal lidocaine to be less than 50%. The mean calculated total dose administered was 8.2 ± 2.0 mg·kg⁻¹ (range 4.3-14.3 mg·kg⁻¹). The venous serum lidocaine concentration was measured at 30 minutes after topical anesthesia was completed (T1) and 30 minutes after the bronchoscopy was completed (T2). The serum lidocaine concentration ranged between 0.1 and 2.90 μg·mL⁻¹ at T1 and between 0.5 and 3.2 μg·mL⁻¹ at T2. The serum lidocaine concentration correlated with the total dose administered, although considerable individual variability among subjects who received the same dose was noted. No signs or symptoms of lidocaine toxicity were detected.⁹²

- Williams et al measured serum concentrations of lidocaine in 25 participants in a bronchoscopy training course.⁹³ Five milliliters of 4% lidocaine was administered by nebulizer, 2 mL of 5% lidocaine with 0.5% phenylephrine was sprayed into the nose and 4 mL of 10% lidocaine was sprayed into the oropharynx. Four percent lidocaine was also administered via the endoscope. Seventy percent of the nebulizer dose was excluded from the total dose calculation. The average calculated dose administered was 8.8 mg·kg⁻¹ (range 7.3-9.2 mg·kg⁻¹). Serum lidocaine concentrations were measured before topicalization (T0), 20 minutes after topicalization (T20), and then at 10 minutes intervals until 60 minutes after the last dose of lidocaine. The duration of sampling ranged from 100-120 minutes. The highest recorded lidocaine concentration was 4.5 μg·mL⁻¹ and only two subjects experienced peak levels above 3 μg·mL⁻¹. In 8 of the 25 subjects, the highest lidocaine concentration was observed in the final sample, 60 minutes after the last dose of lidocaine was administered. Multiple symptoms of lidocaine toxicity were reported by the subjects including lightheadedness, dysphoria, nausea, and shivering.⁹⁴

- Berger et al performed transnasal fiberoptic bronchoscopy on 21 normal volunteers and 18 patients under topical lidocaine anesthesia administered using a combination of gargle, atomization (spraying), nebulization, gel, and in the 18 patients, endobronchial instillation.⁹⁵ The total mean dose of lidocaine administered to the 18 patients was 2086 mg and to the volunteers 1534 mg. Plasma lidocaine levels were measured in eight patients and six volunteers after gargling, after spraying, after nebulization, and then at 5, 10, 15, 30, and 60 minutes. The mean peak level in the patients was about 3.1 μg·mL⁻¹ and in the volunteers about 1.0 μg·mL⁻¹ as measured from the published graphs. In the patient group the peak concentration occurred at 30 minutes. In the volunteers, the peak occurred after nebulization and this level was sustained until the 30-minute sample.⁹⁶

- Ameer et al administered topical lidocaine for bronchoscopy to 19 adults using a combination of lidocaine gargle, atomized lidocaine, lidocaine jelly, and lidocaine solution injected through the bronchoscope. The time over which lidocaine was administered was 0.79 ± 0.31 hours in 5 young patients and 0.69 ± 0.22 hours in 14 elderly subjects. The total dosage administered was 19.01 ± 1.67 mg·kg⁻¹ in the young adults, and 17.15 ± 2.28 mg·kg⁻¹ in the elderly. The mean maximum plasma concentrations achieved were 3.04 ± 1.27 μg·mL⁻¹ in the young and 2.40 ± 0.92 μg·mL⁻¹ in the elderly. The times
required to reach peak levels were 0.77 ± 0.28 hours in the young and 1.21 ± 0.55 hours in the elderly. No serious drug toxicity occurred.95

Reference has also been made to peak blood levels not occurring until 90 minutes after completion of bronchoscopy94 and occurring 30 to 90 minutes from the start of airway local anesthesia.93 Toxicity associated with lidocaine topically applied to the airway can however occur.

- Martin et al administered 7.14 to 14.77 mg·kg⁻¹ of topical lidocaine (median dose 9.6 mg·kg⁻¹) to 39 volunteers participating in a bronchoscopy training study.95 The airway topicalization included 2% viscous lidocaine gargle, 10% spray, and 2% lidocaine administered through the bronchoscope. Thirty-six of the 39 subjects reported side effects associated with lidocaine topicalization including drowsiness, disorientation, hyperacusis, disinhibition, lightheadedness, visual disturbance, and dysphoria. Tremulousness was clinically evident in three subjects who received 9.7, 10.2, and 13.4 mg·kg⁻¹, respectively, two of whom had a single involuntary limb movement. No serum lidocaine concentrations were measured. No major adverse events occurred.95

- Wu et al reported a grand mal seizure which occurred following the topical application of 300 to 320 mg of lidocaine applied as a 4% spray to the larynx and 10 to 12 mL of 1% viscous lidocaine to the oropharynx and trachea of a 30-year-old, 48-kg woman with renal failure, congestive heart failure, cardiomyopathy, and abnormal liver function tests. The plasma lidocaine level shortly after the seizure was 12 µg·mL⁻¹. They also reviewed seven cases of seizure after the administration of topical lidocaine to mucous membranes of the airway. In each of these seven cases, the topical lidocaine was administered as 2% viscous or 2% to 4% solution.47

- Kotaki et al in 1996 reported a seizure following the application of up to 800 mg of lidocaine as a 2% viscous preparation and 4% solution to the oropharynx. The serum lidocaine concentrations were found to be 11.6 µg·mL⁻¹ and 9.0 µg·mL⁻¹ after 30 and 150 minutes postseizure, respectively. The authors reviewed three cases of seizure in addition to those reviewed by Wu et al associated with topical application of lidocaine to the mucous membranes of the oral cavity and pharynx. In each of these three cases, the lidocaine had been administered as a 4% solution or 2% viscous preparation.46

- In 1996 a healthy 19-year-old female volunteer died as a result of lidocaine toxicity following a bronchoscopy performed as part of a research project.96 The amount of lidocaine administered was not documented in the record of the procedure. Details of the technique of administration, as determined from the references reviewed, were limited to as a spray into the throat, and that 4% lidocaine was administered in the upper airway and 2% in the lower airway. No reference to the use of atomized or nebulized lidocaine or documentation of the subject’s body weight was found in the references reviewed.46,96 The subject was left alone after release from the Medical Center and then found apparently having a seizure about an hour later. Emergency 911 was called and mouth-to-mouth breathing was initiated for apparent apnea. On arrival in the emergency department the subject was in cardiac arrest. The blood lidocaine level measured about 3 hours after the research procedure was 12.9 µg·mL⁻¹. Based on this blood level of lidocaine it was reported that it was “likely that she had received in excess of 1200 mg of lidocaine.” The Medical Examiner’s Office found no evidence of any impairment in the subject’s ability to metabolize or detoxify lidocaine.94

- In 2001 the British Thoracic Society recommended that the dose of lidocaine used for bronchoscopy be limited to 8.2 mg·kg⁻¹ and that extra care be used in the elderly or those with liver or cardiac impairment.97 This maximum dosage recommendation appears to apply to all techniques, and the method of topicalization does not seem to have been taken into consideration. The Thoracic Society of Australia and New Zealand, also in 2001, recommended that the total dose of lidocaine used for bronchoscopy not exceed 4 to 5 mg·kg⁻¹.98

In summary, the maximum safe dose of lidocaine that can be topically applied to the mucous membrane of the airway is difficult to determine and must take into account the method of topicalization employed as well as the time course of administration. Traditional dosage guidelines may be excessively conservative when some or the entire dose is administered by aerosol, based on the available evidence with respect to serum levels and toxicity occurrences. Caution must be exercised however and a precalculated dose should not be exceeded. In clinical practice, the smallest amount of anesthetic sufficient to achieve the desired effect should be used,92 and in general, for awake bronchoscopic intubation, the use of large doses is unnecessary. As always, clinical judgment is required and meticulous attention to detail should be employed when lidocaine is applied to the airway such that effective anesthesia is achieved without producing toxicity.

### 3.3.2.1 Can Topical Lidocaine Anesthesia of the Upper Airway Cause Airway Obstruction?

Several studies have looked at this issue:

- In a study of seven normal subjects, Gal administered 4% lidocaine by ultrasonic aerosol and measured airway responses. After the inhalation of lidocaine, the subjects noted an impaired ability to swallow, and a husky voice suggesting vocal cord paresis. Three of the seven subjects described a sensation of obstruction during deep inspiration, although this was not reflected by significant changes in peak inspiratory flow as recorded in maximum effort flow volume loops. Peak expiratory flow rates were also unchanged. Interestingly, statistically significant increases in maximum inspiratory flow were observed at 60%, 50%, and 40% of forced vital capacity (FVC) following the lidocaine aerosol. The author concluded that the administration of 4% lidocaine by ultrasonic nebulization produced mild bronchodilation and did not adversely affect airway function in normal subjects.99

- Gove et al administered 10 mL of 4% lidocaine by means of an ultrasonic nebulizer attached to a mouthpiece to 33 patients
prior to bronchoscopy. Five of the 33 patients required additional boluses of lidocaine to the bronchial tree during the procedure. Spirometry was recorded in 32 patients. A wide variation in individual response to the nebulized lidocaine was observed. Forced expiratory volume in 1 second (FEV1) varied between −18% and +45%, FVC between −27% and +18%, and peak expiratory flow rate (PEFR) between −41% and +34%. However, no overall effect on airflow was demonstrated. The bronchoconstriction that did occur was not clinically significant and bronchodilator therapy was not required.\textsuperscript{100}

- Kuna et al performed pulmonary function tests (PFTs) on 11 normal subjects before and after topical anesthesia of the airway. The topical anesthesia was achieved using 4% lidocaine spray to the soft palate and posterior oropharynx, an internal approach superior laryngeal nerve block using cotton pledges soaked in 4% lidocaine, and 1.5 mL of 10% cocaine applied by means of a cannula to the epiglottis and vocal cords by direct laryngoscopy. The PFTs consisted of flow volume loops, body box determinations of functional residual capacity (FRC), and airway resistance. The area under the inspiratory curve, peak inspiratory flow (PIF), and forced inspiratory flow (FIF) at 25%, 50%, and 75% of FRC were decreased after airway anesthesia, as was peak expiratory flow. However, the area under the expiratory curve and forced expiratory flow at 25%, 50%, and 75% of FVC were unchanged. The authors noted that the configuration of the flow-volume envelope following topical anesthesia in most subjects demonstrated a plateau or sudden reversible reduction in airflow on inspiration but a relative preservation of gas flow on expiration, and concluded that laryngeal anesthesia can compromise upper airway patency.\textsuperscript{101}

- Listro et al measured specific airway conductance and maximum inspiratory and expiratory flow rates before and at 15, 35, and 45 minutes after topical anesthesia of the upper airway. Anesthesia was achieved using four 10% lidocaine sprays to the oropharynx and hypopharynx, and 2 mL of 4% lidocaine solution instilled twice onto the vocal cords using a laryngeal syringe. Average values of maximum inspiratory flow rate (MIFR) decreased 15 minutes after upper airway anesthesia, but returned to control levels or nearly so at 45 minutes. Transient decreases in flow rates reaching zero flow on some occasions were observed in 13 of 16 subjects during forced inspiratory vital capacity (FIVC) and in 7 of 16 during forced expiratory vital capacity (FEVC) maneuvers. The site of obstruction to air flow was determined in 13 patients using simultaneous measurements of supraglottic pressure, flow rates, and lung volume. In 12 of these 13 patients, the site of obstruction was localized to the glottis, and in one, both supraglottic and glottic obstruction occurred. However, upper airway anesthesia in the absence of maximum forced respiratory maneuvers did not result in a decrease in flow rates. The authors concluded that topical anesthesia of the upper airway induces a glottic obstruction that produces a profound but transient decrease in maximum inspiratory and expiratory gas flow consistent with reflex regulation of upper airway caliber.\textsuperscript{102}

- Beydoun et al measured airway flow resistance in nine healthy volunteers using a random noise forced oscillation technique before and after the application of topical anesthesia to the upper airway.\textsuperscript{103} On two separate occasions, either 100 mg of 5% lidocaine liquid was sprayed into one nostril and onto the mucosa of the pharynx and larynx and then gargled, or 100 mg of lidocaine paste was gargled. Airway flow resistance increased in all but one subject. On average the increase was 81% after lidocaine spray and 68% after lidocaine paste. The increased resistance lasted for 13 ± 3 minutes in the spray group and 12 ± 3 minutes in the paste group. At a separate session no change in resistance was detected 3 minutes after spraying normal saline into the upper airway. The two volunteers who experienced the greatest increase in airway flow resistance subsequently underwent transnasal fiberlaryngoscopy under aqueous topical lidocaine anesthesia. Dramatic changes were observed at the larynx. The vocal cords appeared slack and remained in a semi-closed position. During quiet inspiration, the vocal cords moved medially, producing incomplete obstruction at the glottis. During maximal inspiratory efforts the epiglottis moved toward the vocal cords to produce complete airway obstruction similar to that which occurs during swallowing. The authors concluded that topical lidocaine produces an increase in airway resistance in most normal subjects and that the larynx and epiglottis appeared to be main site of obstruction.\textsuperscript{103}

- Weiss and Parwathan administered lidocaine aerosols to 22 patients with stable asthma and demonstrated an initial decrease of expiratory gas flow of approximately 20% within 5 minutes of aerosol administration. Following this initial response, 12 of 22 patients continued to demonstrate a reduction in measured expiratory gas flow that persisted up to 60 minutes, whereas the remaining 10 patients revealed a significant improvement in expiratory gas flow above baseline.\textsuperscript{104}

- McAlpine and Thomson similarly measured FEV1 in 20 asthmatic patients following the administration of 6 mL of 4% nebulized lidocaine. The maximum percentage change in FEV1 following lidocaine inhalation varied from −42.1% to +28.2%, with a mean of −8.2%. Five of the 20 patients experienced a decrease in FEV1 greater than 15%.\textsuperscript{105}

- Groben et al measured changes in FEV1 in 10 volunteers with mild asthma after topical airway anesthesia with either lidocaine or dyclonine and awake bronchoscopic intubation. The local anesthetic was initially administered by nebulizer and supplemented with a gargle and administration of the anesthetic solution onto the epiglottis via the bronchoscope. Following baseline measurements, FEV1 was measured after saline or salbutamol inhalation, local anesthetic inhalation, intubation, and extubation. No significant difference was found in FEV1 following lidocaine or dyclonine inhalation. Salbutamol inhalation significantly increased FEV1. Following awake bronchoscopic intubation under lidocaine anesthesia, FEV1 decreased 35% and 51% after dyclonine. This decrease in FEV1 was significantly attenuated by salbutamol pretreatment in both groups. Two to five minutes after extubation, FEV1 returned to values close to those obtained following saline or salbutamol administration. No significant difference was found between FEV1 values after extubation as compared to the respective FEV1 baseline.\textsuperscript{106}
• In 2006 Ho et al reported a prospective observational study of the effect of upper airway topical anesthesia on dynamic airflow. Six healthy volunteers, all authors of the study, underwent a series of spirometric measurements before and after topical anesthesia of the upper airway produced by topical lidocaine using an aspiration technique (see Section 3.4.4). Peak inspiratory flow rate (PIFR), forced inspiratory flow (FIF) between 25% and 75% of maximum inhaled volume, FEV1, and FVC were measured before the administration of lidocaine, immediately afterward, and at 10, 20, and 30 minutes afterward. A significant reduction in PIFR and FIF was demonstrated at all time points following the administration of lidocaine. Expiratory flow parameters were not affected. The authors concluded that maximum inspiratory flow is impeded by topical anesthesia of the upper airway and they suggested that caution be exercised in the setting of preexisting airway obstruction.

• Thomson also reported a fall in specific conductance of the upper airway following bupivacaine aerosol administration to asthmatics.

Case reports of airway obstruction following topical anesthesia and instrumentation of the airway have also been published:

• Shaw et al reported a case of respiratory distress following the administration of 10% lidocaine spray to the tongue and oropharynx in the presence of a compromised airway associated with goiter. Air entry could not be maintained despite repositioning the patient onto her left side, jaw thrust, chin lift, and placement of an oral airway. The authors felt that a combination of laryngospasm due to irritation caused by the lidocaine spray, and loss of muscle tone as a result of the local anesthetic action, contributed to the airway obstruction.

• McGuire and El-Beheiry reported complete airway obstruction during attempted awake bronchoscopic intubation under local anesthesia in two patients with unstable cervical spine fractures. Both required surgical airways. Both patients also received sedative agents. One patient developed stridor then complete airway obstruction following introduction of the endoscope after topicalization using 1% lidocaine spray and cricothyroid puncture. The second patient was topicalized using swabs soaked in 4% lidocaine. Insertion of the endoscope was associated with gagging and coughing followed by complete airway obstruction.

• Ho et al reported a case of complete airway obstruction which occurred following the topical administration of 2% lidocaine onto the tongue and pharynx and suctioning in a patient with recurrent neck carcinoma following radiotherapy, who had hoarseness and stridor preoperatively.

Extensive clinical experience with lidocaine has shown it to be an effective topical agent for airway anesthesia and to have a wide margin of safety. However, in the presence of preexisting airway compromise, topical anesthesia and instrumentation of the airway can be associated with complete airway obstruction and in this setting, due consideration must be given to the performance of an awake tracheotomy under local anesthesia.

3.3.3 Tetracaine

Tetracaine (pontocaine), a long-acting amino ester derivative of para-aminobenzoic acid was introduced in 1932, and is still used extensively for spinal anesthesia and topical anesthesia of the eye. Although once widely used for topical anesthesia of the airway, its use for this indication fell into disfavor after reports of toxic reactions including fatalities were published in the 1950s. Of the local anesthetics possessing topical action, dibucaine and tetracaine are the most potent as well as the most toxic. The maximal effective concentration of topical tetracaine is 1%. This concentration has a latent period of 0.6 to 1.1 minutes and a duration of 50.2 to 55.5 minutes. When applied to the tongue, 0.5% tetracaine has a latent period of 1.6 minutes and a duration of action of 18.1 minutes. The latent period for 0.4% tetracaine is 3.8 minutes and the duration of action is 35.8 minutes; for 2% tetracaine, these are 1.1 and 48.6 minutes, respectively. The duration of action on the conjunctiva is approximately twice than that at the tip of the tongue, whereas the duration of action on the lip and palate is intermediate. Tetracaine appears to be superior to other topical anesthetics and this may be due to its ability to anesthetize structures deep to the mucous membrane.

Tetracaine applied to the mucous membranes of the pharynx and trachea is rapidly absorbed into the circulation such that blood levels are almost comparable to those obtained after IV injection. Epinephrine added to the tetracaine does not retard its absorption. In 1951, Weisel and Tella reported a series of 1000 bronchoscopies performed with topical tetracaine. There were 12 minor and 7 severe toxic reactions, including 6 seizures and 1 severe bronchospasm. Loss of consciousness preceded convulsions in two of the cases. Cotton pledges were dipped into a solution of 2% tetracaine and placed successively between the faucial pillars and in each piriform fossa for about 1 minute at each location. Then 1 mL of 2% tetracaine was injected into the trachea using a syringe and a laryngeal cannula. The dose administered was estimated to be ≤0.4 mg in most cases, although measurement was inexact. The toxic reaction occurred following application of the fourth pledget in two patients, and tracheal instillation in five patients, and was heralded by syncope or presyncope. Adriani and Campbell noted 10 fatalities at their institution over a 15-year period caused by topical tetracaine. The maximum safe dosage of tetracaine has never been clearly defined. However, maximum safe doses cited in the literature are said to be 50 mg, 80 mg, or 100 mg in the adults. A maximum dose of 20 mg of tetracaine hydrochloride has also been recommended. Again, when maximum safe doses are considered, the method of administration must also be taken into account. In a 1995 review, topical tetracaine was said to be no longer recommended for topical airway anesthesia because of its narrow margin of safety. Tetracaine (0.45%) administered by atomizer produces excellent intubating conditions; however, the potential for toxicity must be appreciated. Allergic reaction, although rare, is more likely with the ester group of local anesthetics as compared to the amides. As of 2007, a single case of methemoglobinemia had been reported associated with tetracaine. In general, metabolism of local anesthetics with an ester linkage occurs by hydrolysis in plasma and requires plasma...
cholinesterase. The presence of an atypical pseudocholinesterase is associated with decreased metabolism. 34

3.3.4 Does cocaine have a role in providing topical airway anesthesia in current anesthesia practice?

Cocaine, an ester of benzoic acid and a nitrogen base, was first isolated in 1860 and serendipitously discovered to have anesthetic properties. 31,45 It is the only local anesthetic that inhibits reuptake of norepinephrine and thereby produces vasoconstriction, hence its continued popularity for nasal procedures. 37,38,120

The maximum effective concentration of topical cocaine is 20%, and this solution produces an anesthetic effect within 0.3 minutes and has a duration of action of 54.5 minutes. 39 Topical anesthesia produced with 10% cocaine has a latent period of 2 minutes and a duration of action of 31.5 minutes, whereas 4% cocaine has a latent period of 4 minutes and a duration of action of 10.2 minutes. 39 The same degree of blockade is produced with 20% cocaine as with 1% tetracaine. 39 Typically 1% to 10% cocaine is used clinically. 31,69 The vasoconstriction produced by cocaine occurs after a latent period of 5 to 10 minutes. 66

The maximum recommended dose for topical nasal application has been said to be from 1.5 mg·kg⁻¹, 121 to 3.0 mg·kg⁻¹, 141 and 1-3 mg·kg⁻¹; 122 however, toxic reactions have occurred after nasal administration of as little as 20 to 30 mg. 122,123 The use of cocaine has been associated with coronary artery vasoconstriction, increased myocardial demand, 68 and hypertension. 121 Doses as small as 0.4 mg·kg⁻¹ may cause ventricular fibrillation, 123 and fatalities have been reported. 45 Cocaine should be avoided or used cautiously in the presence of hypertension, hyperthyroidism, angina, or in patients taking monoamine oxidase inhibitors (MAOIs). 41 Blood levels of cocaine after topical application to the piriform fossae were similar to levels produced after IV injection. 65,116 Oxymetazoline has been shown to be as effective as cocaine in the prevention of epistaxis caused by nasotracheal intubation 124,125 as has normal saline, 125 phenylephrine/ lidocaine, 121,123 and phenylephrine alone. 115

From the available evidence, the disadvantages associated with the use of cocaine to produce nasal anesthesia for awake intubation appear to outweigh the advantages.

3.3.5 How safe and effective is benzocaine?

Benzocaine, an ethyl ester of para-aminobenzoic acid, 126 is a water-soluble ester type local anesthetic that is widely used for topicalization of the airway. 118 It is available as a 20% spray which can deliver between 60 mg 127 and 200 to 295 mg per 1 second spray. 126 Benzocaine is also a component of cetacaine which consists of 14% benzocaine, 2% tetracaine, and 2% butyl aminobenzoate (butamben). 127 The maximum effective concentration of topical benzocaine is 20%, has a latent period of 0.17 minutes, and a duration of action of 4.3 minutes. 39 An onset time of 15 to 30 seconds and a duration of action of 5 to 10 minutes have also been cited. 48 The maximum dose recommended for upper airway anesthesia has been quoted to be 1.5 mg·kg⁻¹, 41 although a dose of 100 mg in the adult has also been cited to be toxic. 118 Benzocaine can produce methemoglobinemia following the administration of as little as 150 to 300 mg in the adult. 128 In a 2007 literature review, 159 episodes of methemoglobinemia associated with the use of benzocaine were identified. 64 Of these, benzocaine had been used alone in 105 episodes. Additional cases have occurred since that time. 129,130 A letter of warning has been issued by the Federal Drug Administration (FDA) in the USA. Methemoglobinemia is potentially fatal 127,131 and treatment with a 1% solution of methylene blue 1 to 2 mg·kg⁻¹ is recommended for methemoglobin levels greater than or equal to 30% or at lower levels if symptoms of hypoxia are present. 132 Normal levels of methemoglobin should be achieved within 20 minutes to 1 hour. 133 However, repeat doses of methylene blue may be necessary. 133 The maximum total dose recommended is 4 mg·kg⁻¹ to 7 mg·kg⁻¹. 130,134 Rebound methemoglobinemia has been reported 2.5 to 20 hours after methylene blue administration. 64,135 In a 2009 review of methemoglobinemia related to local anesthetics, it was recommended that benzocaine should no longer be used. 64 Benzocaine is metabolized by plasma cholinesterase 126 to para-aminobenzoic acid, a highly allergenic molecule 64 and allergic reactions to benzocaine can occur. Given its short duration of action, and its potential for toxicity, the use of benzocaine as a topical anesthetic for airway management seems difficult to justify.

3.3.6 Cetacaine

Cetacaine is a topical anesthetic spray that contains benzocaine 14%, butamben 2%, and tetracaine hydrochloride 2%. According to the manufacturer, the onset time of local anesthesia is 30 to 60 seconds and the duration is typically 30 to 60 minutes. (Cetylite Industries, Inc. Cetacaine Spray product sheet. Cited 2009 Dec 15. Available from: http://www.cetylite.com/cetacaine_spray.html). The manufacturer recommends that cetacaine be applied for approximately 1 second or less for normal anesthesia. A spray in excess of 2 seconds is considered to be contraindicated. The manufacturer states that “dosages should be reduced in the debilitated elderly, acutely ill, and very young patients.” The average expulsion rate from the spray is 200 mg·s⁻¹ at normal temperatures. Each 200 mg of cetacaine contains 28 mg of benzocaine, 4 mg of butamben, and 4 mg of tetracaine. Hypersensitivity reactions can occur and there have been multiple case reports of methemoglobinemia associated with the use of cetacaine. 131,133,136,142

3.3.7 Dyclonine hydrochloride

Dyclonine, a ketone, is a unique local anesthetic agent that was introduced in 1952 and is structurally distinct from the amineethers and aminoamides. 145 It can be used as a 0.5% to 1% solution for topical anesthesia. 31,143 When applied to mucous membranes, the onset time is 2 to 10 minutes and the duration of action is 20 to 30 minutes. Adriani et al noted that dyclonine had limited systemic toxicity but a saturated solution may cause residual numbness that persists for many hours suggesting local injury. 39 One percent dyclonine administered by aerosol has been shown to produce topical airway anesthesia as effective as, and longer lasting than, 4% lidocaine. 106 In a study of 10 volunteers with mild asthma, 4 of the 10 subjects reported much more intense
topical anesthesia following 1% dyclonine inhalation as compared to 4% lidocaine. However, FEV1 decreased to a greater extent in the dyclonine group and the authors concluded that dyclonine must be considered relatively contraindicated in the setting of bronchial hyperreactivity.

Bacon et al reported the use of dyclonine for awake bronchoscopic intubation in a patient with apparent allergy to local anesthetics. The patient gargled and then swallowed 25 mL of 1% dyclonine solution, and 5 mL of 1% dyclonine was then administered by nebulizer. Adequate anesthesia was achieved. Dyclonine has not been widely used, however, for airway anesthesia and is no longer marketed for this purpose in the USA or Canada.

3.4 AIRWAY ANESTHESIA TECHNIQUES

3.4.1 What techniques are available for upper airway anesthesia?

Regional anesthesia of the airway can be achieved using a wide variety of techniques. Each technique requires a meticulous approach, attention to detail, and knowledge of relevant anatomy as well as the pharmacology of the agents employed if an adequate block is to be achieved. The most important prerequisite for a successful awake intubation is adequate regional anesthesia of the airway.

3.4.2 Spray/ointment/gel/EMLA

The posterior third of the tongue, the soft palate, the tonsillar pillars, and the adjacent pharynx can be sequentially anesthetized using commercially available 10% lidocaine spray simply by directing the spray onto the relevant structures. A tongue depressor can be used to gently retract the tongue. The commercially available 10% lidocaine aerosol is fitted with a metered valve that delivers 10 mg of lidocaine as an aerosol with each depression of the pump mechanism. This simple pump mechanism uses manually compressed air as the driving force. Adequate regional anesthesia for awake intubation by direct laryngoscopy can readily be achieved in this manner, and in the emergency setting, time may not permit additional regional techniques. The 10% lidocaine aerosol is marketed in Canada by Odan Laboratories Limited but is not currently available in the USA (see Figure 3-25). Alternatively, 4% lidocaine administered using a mucosal atomization device can be used (Wolf-Tory Medical Incorporated, Salt Lake City, Utah). A curved metal cannula can also be used to inject lidocaine solution into the laryngopharynx and larynx as time and circumstances permit; however, this is not necessary for awake intubation by direct laryngoscopy, and blood levels of lidocaine produced with this technique will probably be higher than those produced by aerosol techniques. Cooperative patients can also gargle 2% to 4% lidocaine in order to achieve topical anesthesia of the posterior tongue and adjacent oropharynx. Residual anesthetic should be expectorated to avoid excessive drug exposure and potential nausea and vomiting. Lidocaine ointment (5%) can be very useful to anesthetize the posterior third of the tongue especially when patients are unable to gargle. Lidocaine gel 2% can also be used. With any of these techniques, time (at least 1 minute to 2 minutes and perhaps as long as 5 minutes) must be allowed for the anesthetic effect to occur.

EMLA cream, a 1:1 eutectic mixture of 2.5% lidocaine and 2.5% prilocaine, has also been used to produce airway anesthesia. Larijani et al applied up to 4 g of EMLA cream to the tongue and pharynx in a series of 20 patients who underwent awake bronchoscopic intubation. The intubation was performed via a Williams airway. The mean time from the application of the EMLA cream to placement of the oral airway was 11 ± 6 minutes. All patients were successfully intubated but all coughed when the scope was passed into the trachea. No toxic plasma levels of lidocaine or prilocaine occurred. A statistically significant increase in methemoglobin levels occurred within 6 hours; however, these levels did not exceed normal values (1.5%). Söhmer et al used 4 mL of EMLA cream applied to the tongue and gargled before performing awake bronchoscopy in 57 patients. In addition, 79.05 ± 14.39 mg of lidocaine was administered through the flexible bronchoscope (FB) for laryngeal anesthesia. Fifty-six of the cases did not require supplemental anesthesia. Bronchoscopic conditions were excellent in 55 cases and good in the remaining 2 cases. The mean time from EMLA application to insertion of the bronchoscope was 5.10 ± 0.45 minutes. EMLA cream applied to the nostril prior to the passage of a flexible bronchoscope provoked rhinorrhea and sneezing that persisted for several hours in 21 of 31 individuals, although the endoscopy was well tolerated. EMLA cream may be an alternative for oropharyngeal topical anesthesia, although experience is limited.

3.4.3 Aerosols

Excellent anesthesia of the airway can be produced by the administration of aerosolized local anesthetic delivered by an atomizer, such as the DeVilbiss RD 15 (see Figure 3-26). The device consists of a glass reservoir, which holds the anesthetic solution, and a nozzle assembly, which can be connected to a high-pressure oxygen source by means of standard oxygen tubing. A small bleed hole...
is cut in the oxygen tubing at a convenient location near its connection to the atomizer. When oxygen flow is delivered into the tubing at about 6.0 to 8.0 L·min⁻¹, occlusion of the bleed hole with a fingertip produces a fine spray of local anesthetic from the atomizer nozzle. When held at the nostril, and coordinated with deep breaths on command (“in through the nose and out through the mouth”), the device can produce profound anesthesia of the airway from the nose to the trachea and beyond in about 5 minutes. The atomized local anesthetic can also be administered through the mouth, although superior gas flow characteristics through the nose may deliver the anesthetic more efficiently to the pharynx, larynx, and trachea. Obstruction of the nasal cavity of course precludes nasal administration. At the author’s institution, the DeVilbiss atomizer has been used routinely for awake intubation for more than two decades with excellent results and no toxic reactions. An excellent block can be achieved with 10 to 12 mL of 3% or 4% lidocaine. Others have recommended up to 20 mL of 0.5% tetracaine or 10 mL of 4% lidocaine with this technique.

In the study by Wieczorek et al in which either 2% or 4% lidocaine was administered by atomizer to a group of obese patients, the authors concluded that atomized lidocaine for awake intubation was efficacious, rapid, and safe. A subsequent study from the same center compared 2% and 1% lidocaine using the same atomization technique and volume of anesthetic. The authors concluded that 1% lidocaine provided measurably inferior airway anesthesia.

Atomization can also be used as a part of a combined technique of airway anesthesia. Aerosolized local anesthetic can also be administered through the mouth using a standard nebulizer attached to a mouthpiece. An oral airway intubation device can be attached to the nebulizer and advanced into the oropharynx as tolerated to deliver the anesthetic to the more distal airway (see Figure 3-27). The authors of the noted report used 4 mL of 4% lidocaine at 8 L·min⁻¹ oxygen flow with this technique and the nebulization required 8 minutes. Additionally, 4 mL of 2% lidocaine was administered through the FB during intubation. Kirkpatrick et al reported that the gag reflex was abolished for a mean time of 32 ± 5.9 minutes in 10 healthy volunteers following the administration of 10 mL of 4% lidocaine using a DeVilbiss 646 nebulizer and mouthpiece. No bronchoscopy or intubation was performed.

Nebulized local anesthetic has also been administered by face-mask using 4 to 20 mL of 4% lidocaine or 6 mg·kg⁻¹ of 10% lidocaine. Nebulization by this technique required 10 to 22 minutes. In a study reported by Kundra et al, 7 of 24 patients required supplemental lidocaine through the fiberoptic bronchoscope after nebulization of 4 mL of 4% lidocaine and administration by mask. This study compared nebulization with a combined regional block (CRB) technique consisting of nasal lidocaine-soaked swabs, superior laryngeal nerve block, and transtracheal injection of lidocaine for awake nasotracheal bronchoscopic intubation. The authors reported that the patients in the CRB group were more comfortable during the procedure. Four of five other reports of awake intubation or bronchoscopy using nebulization by mask also used supplemental anesthesia.

Chinn et al reported that only one of five healthy subjects lost the gag reflex following administration of 10 mL of 4% lidocaine by means of a DeVilbiss 35B nebulizer and Hudson oxygen mask. No bronchoscopy or intubation was performed.

Nebulization has also been used by others as part of a combined technique of airway anesthesia.

### 3.4.4 Local anesthetic aspiration

Local anesthesia of the airway can also be achieved using an aspiration technique. In this technique, lidocaine solution is simply dripped onto the dorsum of the tongue of a supine patient during tongue traction. The swallowing reflex is initially stimulated, but lidocaine subsequently pools in the posterior pharynx and is
aspirated into the trachea.\textsuperscript{152} Gargling with two consecutive 5-mL aliquots of 2\% lidocaine can decrease the intensity of this swallowing reflex.\textsuperscript{152} Chung et al instilled the lesser of 0.2 mL·kg\textsuperscript{-1} or 20 mL of 1.5\% lidocaine following the gargle as described above and reported satisfactory bronchoscopic intubating conditions in 39 patients, although mild coughing or gagging did occur with the scope in the trachea in 10 patients, and with the tube in the trachea in another 21 patients. Supplemental local anesthesia was not required. Eighteen patients were intubated orally, using a Williams airway intubator, whereas 21 were intubated nasally. Gauze packing soaked in 1.5 mL of 5\% lidocaine was used for nasal anesthesia. The time required for intubation varied from 1 to 10 minutes (median time 3.25 minutes).\textsuperscript{152} By comparison, intubation after a DeVilbiss technique can usually be accomplished in about 30 seconds.

### 3.4.5 Nasal anesthesia

Local anesthesia of the nasal cavity can be achieved by a variety of methods. Nebulized lidocaine can be administered by facemask and the patient instructed to breathe through the nose.\textsuperscript{72,74} Lidocaine or tetracaine administered with a DeVilbiss atomizer can also be very effective in achieving topical anesthesia of the nasal cavity. Alternatively, long cotton-tipped applicators or pledgets, held in bayonet forceps and soaked in 4\% lidocaine or 4\% cocaine, can be introduced into the selected nostril.\textsuperscript{118} One applicator can be inserted parallel to the anterior border of the nasal cavity along the septum until it reaches the anterior end of the cribiform plate at a depth of about 5 cm (see Figure 3-28).\textsuperscript{153} The local anesthetic-soaked applicator can then be left in place for 5 to 15 minutes to produce a transmucosal block of the anterior ethmoidal nerve.\textsuperscript{118,153} A second applicator can be inserted at an angle of about 20 to 45 degrees to the floor of the nose until bony resistance is felt at a depth of about 6 to 7 cm.\textsuperscript{153} In this location, the tip of the applicator is adjacent to the sphenopalatine ganglion located deep to the nasal mucosa and similarly can be left in contact with the mucosa for 5 to 15 minutes to produce a transmucosal block of the sphenopalatine nerves.\textsuperscript{118,153} Nasal anesthesia has also been produced using sprays from a multiorificed cannula,\textsuperscript{123} a 20-gauge angiocatheter,\textsuperscript{118} an epidural catheter,\textsuperscript{154} 10\% lidocaine aerosol, and lidocaine gel.

A vasoconstrictor is frequently applied topically to the nasal mucosa in an effort to prevent the epistaxis that can occur with nasotracheal intubation following nasal spraying with 0.05\% oxymetazoline, 10\% cocaine, or normal saline.\textsuperscript{125} Similarly, Gross et al found no significant difference among groups pretreated with 4\% cocaine, 3\% lidocaine with 0.25\% phenylephrine, or 0.25\% phenylephrine.\textsuperscript{126} Mitchell et al compared 5\% cocaine, 4\% lidocaine/0.5\% phenylephrine, and normal saline and again found no significant difference in the prevention of epistaxis.\textsuperscript{155} Latorre et al compared 10\% cocaine with 3\% lidocaine/0.25\% phenylephrine and found no difference.\textsuperscript{127} Katz et al found lidocaine 4\% with epinephrine 1:100000 to be less effective than 0.05\% oxymetazoline but no difference between 10\% cocaine and oxymetazoline, or cocaine and lidocaine with epinephrine.\textsuperscript{124}

Thus, the efficacy of the practice of administering vasoconstrictors to prevent epistaxis associated with nasotracheal intubation is doubtful and cocaine would appear to offer no significant advantage over oxymetazoline or phenylephrine.

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**FIGURE 3-28.** Placement of cotton-tipped applicators to contact the anterior ethmoidal nerve (A) and the sphenopalatine ganglion and nerves (B). (Reproduced, with permission, from Murphy TM. Somatic blockade of the head and neck. In: Cousins MJ, Bridenbaugh PO, eds. Neural Blockade in Clinical Anesthesia and Management of Pain. 3rd ed. Philadelphia, PA: Lippincott-Raven Publishers; 1998:489-574.)
3.4.6 Translaryngeal anesthesia

Injection of local anesthetic through the cricothyroid membrane was described in the 1920s, and use of this technique to facilitate endotracheal intubation was described in 1949.26,156 A 21- to 23-gauge needle can be passed posteriorly in the midline immediately cephalad to the cricoid cartilage to enter the larynx (see Figure 3-29).26,38,157 Alternatively, a 20-gauge angiocatheter can be used.115 Directing the needle caudally will direct it away from the vocal cords which are located 1.3 cm cephalad from the transverse plane at the midpoint of the cricothyroid membrane.26 The correct intraluminal position of the needle can be confirmed by the aspiration of air.26,118 Then, 0.5 to 2.0 mL of 4% lidocaine,157,158 3 mL of 4% lidocaine,90 4 mL of 2% to 4% lidocaine,118 or 2 to 3 mL of 2% lidocaine,159 can be injected either at end exhalation150 or inhalation.118 Translaryngeal injection predominantly produces anesthesia of the infraglottic mucosa.72 The cough precipitated by the injection facilitates the spread of the anesthetic which has been shown to reach the superior aspect of the true cords in 95% of cases.61 The sensory blockade above the glottis is dependent on the magnitude of the cough response.72 If the goal is to spread anesthetic into the larynx and pharynx then injection at end inspiration seems most logical. Four milliliter of 2.5% cocaine149 or 3 mL of 4% cocaine158 have also been used for translaryngeal anesthesia. Tetracaine has also been used in the past; however, severe reactions have been reported with the injection of tetracaine solution into the larynx.115 Serum lidocaine levels following injection of 5 mg·kg⁻¹ of a 10% solution into the larynx via cricothyroid puncture have been found to be in the antiarrhythmic therapeutic range at a mean time of 5.1 ± 3.2 minutes.162

Contraindications to cricothyroid puncture include coagulopathy, local pathology, and an inability to clearly identify the cricothyroid membrane due to obscured landmarks as in the morbidly obese.26,115 Relative contraindications include those circumstances in which vigorous cough could be deleterious, such as raised intracranial pressure or intraocular pressure, open eye injury, or unstable cervical spine injuries.118 The use of translaryngeal anesthesia in the presence of a full stomach is controversial.68 Complications of laryngeal anesthesia including laryngospasm and soft tissue infection have been rarely reported.26,157 Potential complications include bleeding, subcutaneous emphysema, pneumomediastinum, pneumothorax, vocal cord damage, and esophageal perforation.138 A review of 17,500 cricothyroid punctures revealed only 8 complications: 2 laryngospasms, 2 broken needles, and 4 soft tissues infections of the neck.26 In a series of 286 emergency department nasal intubations using translaryngeal anesthesia, Danzl and Thomas reported only 1 complication due to the cricothyroid membrane puncture, a case of superficial cellulitis.157

Bigeleisen et al compared the local anesthesia achieved with and without a transtracheal component in a group of 20 patients who underwent bronchoscopic intubation.88 Atomized lidocaine was administered to all patients and superior laryngeal nerve blocks were performed prior to randomization into a transtracheal and nasopharyngeal groups. Transtracheal injection of 2 mg·kg⁻¹ of 4% lidocaine was performed in group 1 and in group 2 the same dose of lidocaine was applied topically to the nasopharynx. The authors reported that the patients appeared equally comfortable in both groups and that there was no qualitative difference in the ease of intubation between the groups.89

3.4.7 Spray-as-you-go

Lidocaine administered through the bronchoscope is commonly used during diagnostic bronchoscopy.163 This technique can also be used for awake bronchoscopic intubation and can be combined with other methods of local anesthetic administration.13,32,54,72,91,92,95,149 However, the time required to produce maximal local anesthesia after bronchoscopic instillation may be as long as 5 minutes, and this technique may provoke unnecessary reflex glottic closure and cough.

In the study reported by Xue et al safety and efficacy of 2% and 4% lidocaine administered by a spray-as-you-go technique for bronchoscopic intubation were compared.35 The patients were sedated and an oral airway and a jaw lift were used. Nineteen of 26 patients in the 2% group and 17 of 26 in the 4% group exhibited a change in facial expression or grimace when the endotracheal tube was advanced into the trachea. The time impinged at the level of the glottis in 5 of 26 patients in the 2% group and 7 of 26 in the 4% group and required tube rotation. Slight and moderate coughing occurred in 54% and 15%, respectively, of those in the 2% group, as compared to 50% and 12%, respectively in the 4% group. Intubation times were 30.2 ± 9.8 seconds in the 2% group and 29.3 ± 10.1 seconds in the 4% group. Overall 61.5% to 73.1% of patients displayed grimacing or coughing responses during intubation. The authors reported intubating conditions to be excellent or acceptable in all patients, although a lack of complete anesthesia was reported in both groups.35

Webb et al compared transcricoid injection of lidocaine with the spray-as-you-go technique in a group of 62 patients who underwent transnasal diagnostic bronchoscopy.164 The authors concluded that the transcricoid method was more effective than the spray-as-you-go technique in that, despite a lower dose of lidocaine, the bronchoscopy required less time to complete, the cough rate was lower, and the procedure was at least as acceptable to the patients in the transcricoid group.164

Graham et al compared transtracheal injection of cocaine, cocaine injected through the bronchoscope, and lidocaine administered by
nebulizer in a group of 53 patients who underwent flexible bronchoscopy.\textsuperscript{149} The patients in the transtracheal group coughed less, experienced less stridor, and required less supplemental lidocaine. The authors concluded that transtracheal injection was the superior technique.

Sethi et al compared the spray-as-you-go technique, transtracheal injection, and nebulization in a group of 60 patients who underwent bronchoscopic transnasal intubation prior to elective surgery.\textsuperscript{150} The nasal passages of all patients were lubricated with 2 mL of 2\% lidocaine jelly, four sprays of 4\% lidocaine were applied to the posterior pharynx and the patients gargled the excess solution. The patients were then randomized into a transtracheal group (A) who were given 4 mL of 4\% lidocaine, a spray-as-you-go group (B) who were given 2 mL of 4\% lidocaine into the larynx and 2 mL into the trachea through the bronchoscope, and a nebulization group (C) who were given 4 mL of 4\% lidocaine starting 20 minutes before the procedure. Additional aliquots of 2\% lidocaine were administered as required. The patients’ VAS for symptoms and the endoscopist’s unblinded assessment showed a preference for the spray-as-you-go technique. The cough count in Group B was 12, as compared to 18 in Group A, and 20 in Group C. Stridor occurred in 2 of 20 patients in Group B, as compared to 3 of 20 in Group A, and 10 of 20 in Group C. The best intubating conditions occurred in 16 patients in Group B, 10 in Group A, and 6 in Group C. Eight patients in Group B required a mean extra lidocaine dose of 60 mg, as compared to 2 patients (20 mg) in Group A and 12 patients (120 mg) in Group C. The data suggests that all three groups in this study had incomplete anesthesia.\textsuperscript{150}

Spray as you go can also be used as part of a combined technique.

### 3.4.8 Glossopharyngeal nerve block

In the majority of individuals, the application of topical anesthesia to the mucosa of the oropharynx is sufficient to abolish the gag reflex. However, in the presence of a very pronounced gag reflex or excess secretions, glossopharyngeal nerve block may be a reasonable alternative approach. Submucosal pressure receptors in the posterior third of the tongue may also be involved in the gag reflex\textsuperscript{70,118,165-168} and are not felt to be susceptible to topically applied local anesthetics.\textsuperscript{118,168}

The glossopharyngeal nerve can be blocked using a posterior approach as it runs about 1 cm deep to the mucosa behind the midpoint of the palatopharyngeal fold (see Figure 3-30).\textsuperscript{70,118,120,165,167-169} A 23-gauge angled tonsillar needle with 1 cm exposed shaft at the tip can be inserted 0.5 cm behind the midpoint of the palatopharyngeal fold, directed laterally and slightly posteriorly to a depth of about 1 cm.\textsuperscript{165,169} Following a negative aspiration test, 2 mL of 2\% lidocaine\textsuperscript{127} or 3 to 5 mL of 1\% lidocaine\textsuperscript{167,169} can be injected. Mouth opening must be sufficient to permit visualization of the palatopharyngeal fold (posterior tonsillar pillar).\textsuperscript{70} Adequate topical anesthesia of the tongue and adjacent pharyngeal mucosa is necessary to permit exposure of the tonsillar pillar with a tongue blade or laryngoscope.\textsuperscript{70,118} Barton and Williams reported a series of 130 patients who underwent glossopharyngeal nerve block for bronchoscopic procedures or tonsillectomy with no complications, and elimination of the gag reflex in all but three patients.\textsuperscript{165,127} Cooper and Watson similarly reported a series of 893 patients who underwent bronchoscopy or tonsillectomy using glossopharyngeal block.\textsuperscript{169} Again no complications were reported. Onset time of the block has been noted to be about 1 minute\textsuperscript{169} and the duration of the block to be 45 to 60 minutes.\textsuperscript{166} Demeester and Skinner performed glossopharyngeal nerve block on 500 patients who underwent bronchoscopic procedures.\textsuperscript{167} Superior laryngeal nerve blocks were also performed and supplemental anesthetic was administered through the bronchoscope. An inadequate block occurred in 10 patients. Blood was aspirated in six patients requiring needle repositioning and four additional patients complained of headache thought to have been due to partial intra-arterial injection of the local anesthetic. Two patients had a seizure during the endoscopy and five developed an arrhythmia following the block. The overall complication rate secondary to the glossopharyngeal nerve block was reported to be 2\%.\textsuperscript{167} Complications in addition to those noted above include local infection and hematoma formation.\textsuperscript{118} Contraindications include coagulopathy and local pathology. This posterior approach, glossopharyngeal nerve block, is not widely used and may be impractical in the setting of difficult intubation.

Alternatively, the lingual branch of the glossopharyngeal nerve can be blocked as it runs deep to the mucosa of the palatoglossal fold (anterior tonsillar pillar) (see Figure 3-31).\textsuperscript{118,166,168,170} Although the lingual branch of the nerve supplying the posterior third of the tongue is blocked primarily, in some cases retrograde submucosal tracking of the local anesthetic has been shown to occur, with blockade of the pharyngeal and tonsillar branches.\textsuperscript{118,171} A 22- to 27-gauge needle is inserted in the floor of the mouth, 0.5 cm lateral to the lateral aspect of the base of the tongue at the palatoglossal fold (see Figure 3-31).\textsuperscript{118,168,170} The needle is inserted to a depth of about 0.5 cm,\textsuperscript{166,169} and following a negative aspiration test, 2 mL of 2\% lidocaine\textsuperscript{166,169} or 2 to 5 mL of 1\% lidocaine\textsuperscript{118,170} can be injected. If blood is aspirated, the needle should be redirected medially.\textsuperscript{118} If air is aspirated, the needle has passed through the palatoglossal fold to enter the oropharynx and should be withdrawn until no air is aspirated.\textsuperscript{118} Woods and Landers reported...
34 anterior approach glossopharyngeal nerve blocks and noted a duration of action of 15 to 20 minutes with plain lidocaine and 60 minutes with lidocaine and epinephrine. The blocks were performed with a minimum of patient discomfort. The gag reflex was not completely obliterated in “a number of patients.” Sitzman et al reported a prospective, randomized, single-blinded crossover study of airway anesthesia for direct laryngoscopy on 11 anesthesiologist volunteers which compared 2% viscous lidocaine swish and gargle (S&G), S&G combined with 10% lidocaine spray, and S&G combined with bilateral anterior glossopharyngeal nerve blocks. There was no significant difference between the S&G/spray and S&G/block groups with respect to discomfort during direct laryngoscopy; however, the S&G group did experience significantly more discomfort than the other two groups. A trend toward less coughing and gagging with S&G/spray compared with S&G/block was noted, although the difference was not significant. Oropharyngeal discomfort lasting 24 hours or more occurred in 91% of the participants in the block group, and four participants had discomfort lasting more than 3 days. The study was stopped due to this oropharyngeal discomfort. The study used 5 mL of 1% plain lidocaine bilaterally, and the authors suggest that the discomfort may have been related to the volume of solution injected. Contraindications include coagulopathy and local pathology. Potential complications include intra-arterial injection, patient discomfort, hematoma formation, and anatomic distortion. In addition, local anesthetic injected into the floor of the mouth anterior to the palatoglossal fold may produce bilateral hypoglossal nerve block and impair the ability to swallow. The block is considered to be acceptable in the presence of a full stomach.

3.4.9 Superior laryngeal nerve block

The internal branch of the superior laryngeal nerve can be blocked as it runs just deep to the mucosa of the piriform fossa using Kraus or Jackson forceps to hold a cotton pledget soaked in 4% lidocaine against the mucosa for about 1 minute (see Figure 3-32). Keeping the lidocaine-soaked pledget in contact with the mucosa of the piriform fossa for 5 minutes has also been recommended but in the author’s experience, this is not necessary.

Alternatively, this block can be performed using an external approach to the superior laryngeal nerve as it penetrates the thyrohyoid membrane just below the greater cornu of the hyoid bone. With the patient supine and the head extended, the hyoid can be palpated as a freely mobile bony structure cephalad from the thyroid cartilage (see Figures 3-33 and 3-34). The hyoid can be fixed between the operator’s index finger and thumb and displaced manually toward the side to be blocked. A 21- to 25-gauge needle can be passed medially in the frontal plane through the skin to contact the hyoid at or...
Following a negative aspiration, 2 to 3 mL of 2% lidocaine can then be injected. If blood is aspirated, the needle may have entered the superior laryngeal artery or vein or the carotid artery, and in this circumstance it should be withdrawn and redirected anteriorly. If air is aspirated, the pharyngeal lumen has been entered and the needle must be withdrawn until no air is aspirated prior to injection. Entry into the laryngopharynx has been used as an integral part of the technique, although this is not necessary. If the hyoid bone cannot be identified by palpation or if palpation produces undue patient discomfort, the thyroid cartilage can be used as a landmark. The needle is then walked cephalad to reach and perforate the thyrohyoid membrane. Alternatively, the thyrohyoid membrane itself can be identified by palpation with the index finger immediately cephalad to the lateral aspect of the thyroid cartilage. The carotid pulse can be felt posteriorly (see Figure 3-35). The needle can then be passed medially anterior to the fingertip. The feeling of resistance changes as the needle punctures the membrane and is relied on to indicate proper depth. The needle may also be advanced to contact the thyroid cartilage as a depth guide and then walked cephalad.

The onset time for the block is 5 to 10 minutes and the duration of action is at least 90 minutes and may be as long as 4 to 6 hours when 2% lidocaine is used. Complications include intra-arterial injection, hematoma (reported incidence 1.4%), unintended pharyngeal perforation, hypotension, and bradycardia. Contraindications include local pathology, coagulopathy, and poor anatomic landmarks. The block has also been said to be contraindicated in patients at risk of aspiration.
### 3.5 OTHER CONSIDERATIONS

#### 3.5.1 Is regional anesthesia of the airway contraindicated in the presence of a full stomach?

Local anesthesia of the larynx and trachea obtunds protective airway reflexes and may predispose to aspiration. However, local anesthesia of the airway has been used in circumstances associated with increased risk for aspiration without aspiration actually occurring. Thomas reported a series of 25 patients who were intubated awake, 21 of whom had a full stomach. Topical anesthesia was administered to all 25 and bilateral superior laryngeal nerve blocks were performed on 21 patients. Nine patients were given translaryngeal injections. No incident of aspiration occurred. Duncan reported 12 patients who underwent awake intubation for emergency surgery under regional anesthesia of the airway including transtracheal injection and recorded no incident of aspiration. Kopman et al reported 55 awake intubations in patients with full stomachs under topical anesthesia to the nose, mouth, pharynx, and larynx. There was no evidence of aspiration in any of the patients. Danzl and Thomas performed 286 emergency nasotracheal intubations under transtracheal anesthesia and reported no aspirations. Meschino et al reported a series of 165 patients with cervical spine fracture who underwent awake intubation. Sixty-four patients required emergent endotracheal intubation and “a regional block or local anesthesia of the larynx” was administered in 137 patients. No evidence of aspiration of gastric contents was documented during intubation. Ovassapian et al performed 114 awake bronchoscopic intubations on patients with a full stomach risk under regional anesthesia of the airway including transtracheal injection or injection into the larynx and trachea through the flexible bronchoscope. Again no aspirations occurred.

The risk of aspiration during awake intubation under regional anesthesia of the airway must be weighed against the risks associated with other airway management modalities. As always, good clinical judgment and a common sense approach are mandatory.

#### 3.5.2 Are antisialagogues helpful or even essential in awake bronchoscopic intubation?

Secretions in the airway interpose a mechanical barrier between the mucosa and topically applied local anesthetics, dilute the anesthetic solution, and wash it away from the intended site of action. Antisialagogues are therefore invaluable adjuncts to facilitate awake bronchoscopic intubation. Atropine and glycopyrrolate are both effective, although glycopyrrolate is the more potent drying agent. Scopolamine has also been used as an antisialagogue; however, it produces sedation as well as amnesia and can produce delirium.

These antimuscarinic drugs dry the airway by decreasing the production of secretions. They must therefore be administered far enough in advance of planned airway manipulation to allow eradication of secretions that have already accumulated, as well as to permit the drug to exert its antisialagogic effect. Following IV administration of glycopyrrolate to volunteers, dryness of the mouth was noted 7 minutes after injection and a significant drying effect was observed after 15 minutes. Following IM administration, glycopyrrolate has an onset of action of 20 to 40 minutes and the peak effect occurs at 30 to 45 minutes. Inhibition of salivary secretions persists for up to 7 hours after parental administration of glycopyrrolate and is dose related. The appropriate dose of IM glycopyrrolate when used as a drying agent is 0.2 to 0.4 mg in the adult. The corresponding dose in children is about 10 μg·kg⁻¹. Glycopyrrolate can also be given subcutaneously and is dose related. After IM administration, doses of glycopyrrolate sufficient to produce a 75% inhibition of salivation produced only minimal heart rate changes, although an increase in heart rate can occur after IV administration. Glycopyrrolate is a quaternary ammonium compound and does not cross the normal blood–brain barrier.

Following the IM administration of atropine, inhibition of salivation is seen within 30 minutes, the peak effect is seen at 1.0 to 1.6 hours, and the duration of action is 4 hours. The heart rate increases 5 to 40 minutes after the IM administration of atropine and peaks within 20 to 60 minutes. At doses necessary to produce a 75% inhibition of salivation, atropine increases the heart rate by more than 15%. When used as an antisialagogue, 0.2 to 0.8 mg of atropine can be given IM or subcutaneously 30 to 60 minutes before airway manipulation. Rarely at low doses, atropine can exert a parasympathomimetic effect and produce a bradycardia. Atropine readily crosses the blood–brain barrier and can produce a CNS effect.

In a prospective, randomized, double-blind study of 37 surgical patients, the drying effect of glycopyrrolate 0.2 mg IM administered 90 minutes preoperatively was compared to 0.2 mg IV given 10 minutes preoperatively, to 2 mg given by mouth 90 minutes preoperatively, and to placebo. No significant difference was found in the patient’s sensation of mouth dryness 10 minutes after IV injection of glycopyrrolate or placebo, and no difference between groups in the anesthetist’s perception of dryness of the airway following intubation by direct laryngoscopy. Ten minutes may not be sufficient time to permit significant drying to occur after IV injection as the effect has been shown to increase for up to 15 minutes. The peak effect of IM glycopyrrolate occurs at 30 to 45 minutes following injection and 90 minutes following injection may not have been the optimal time for observation. Furthermore, the inhibition of salivation is dose related and 0.2 mg is at the low end of the dose range recommended in the adult. Direct laryngoscopy is also a different stimulus as compared to awake bronchoscopic intubation under local anesthesia. Cowl et al reported a double-blind, placebo-controlled study of 217 patients who underwent bronchoscopy and intubation under local anesthesia and sedation. Patients
were randomly allocated to receive atropine 0.01 mg·kg⁻¹ IM, glycopyrrolate 0.005 mg·kg⁻¹ IM, or saline placebo 2 mL IM, 15 to 45 minutes preoperatively. The time of administration in each patient was not recorded however, and the number of patients who received glycopyrrolate 15 minutes before the procedure is unknown. The operators noted no significant difference in antisialagogic effect or cough suppression for either atropine or glycopyrrolate as compared to placebo. However, the patients reported significantly higher visual analog scores for secretion control with glycopyrrolate. Roffe et al randomly allocated 190 consecutive patients undergoing bronchoscopy employing local anesthesia and sedation to receive either IM atropine 0.6 mg, IM glycopyrrolate 0.3 mg, or no antisialagogue 30 minutes preoperatively. Troublesome coughing was less frequent in the glycopyrrolate group as was patient movement. Mouth dryness was most common with glycopyrrolate but overall assessment of discomfort was similar in all three groups. In 2009, Malik et al reported a double-blind, placebo-controlled study of 1000 patients who underwent diagnostic bronchoscopy under local anesthesia and sedation. Patients were randomly assigned to receive atropine 0.01 mg·kg⁻¹, glycopyrrolate 0.005 mg·kg⁻¹, or 2 mL normal saline IM 20 to 40 minutes before the procedure. The time of administration in each patient was not reported. Nebulized 4% lidocaine was administered and 5 mL of 2% lidocaine was instilled into the trachea via the bronchoscope. Additional boluses were administered via the bronchoscope as required. Neither the total dose of lidocaine administered nor any differences in doses among the groups were reported. The nasotracheal approach was used in approximately 95% of patients but any differences in approach among the groups were not reported. The authors reported that the patient and bronchoscopist’s visual analogue scores for airway secretions, cough, and discomfort were lower in the patients treated with antimuscarinics compared with placebo, but only reached statistical significance in the bronchoscopist’s score for secretions. In the atropine group changes in the blood pressure and heart rate were statistically significant as compared to glycopyrrolate and placebo. These changes were however clinically modest. There was no significant difference in the occurrence of major adverse events among the groups. Brookman et al randomized 80 adult patients undergoing elective dental extraction to receive 0.4 mg hyoscine hydrobromide PO, 0.4 mg hyoscine hydrobromide IM, 0.4 mg glycopyrrolate IM, or placebo 60 minutes preoperatively. All patients underwent nasal bronchoscopic intubation. The clarity of the visual field was noted to be significantly improved in all three antimuscarinic groups.

The results of these studies are somewhat difficult to extrapolate to the awake bronchoscopic intubation setting. In the author’s experience, drying agents are immensely helpful in facilitating awake bronchoscopic intubation. Glycopyrrolate 0.4 mg IM given to the adult 30 minutes before airway manipulation will provide optimum conditions for topical anesthesia of the airway in the vast majority of patients. Inflammatory conditions of the airway can be associated with excess secretion production and antisialagogues may be less effective in this setting. In the absence of pathology at the site of injection, superior laryngeal nerve block, or glossopharyngeal nerve blocks may be more effective in this circumstance; however, intubation under topical anesthesia alone can usually be achieved. The nasal approach requires less suppression of the gag reflex and may be the more appropriate route in the presence of excess secretions.

### 3.5.3 Is sedation useful in facilitating awake intubation?

If adequate local anesthesia of the airway can be achieved, awake intubation can be rapidly and easily accomplished without the use of sedation. Regional anesthesia of the airway can be achieved with minimal patient discomfort, although stimulation of the gag and cough reflexes can be unpleasant. The emphasis should however be placed on the development of regional anesthesia skills, rather than on the use of sedation in an attempt to compensate for poor airway anesthesia. As time and circumstances permit, sedation can be used to further minimize discomfort, produce amnesia, and attenuate recall, although the need for amnesia or amnesia is reduced or eliminated if airway anesthesia can be rapidly and skillfully achieved. In the presence of airway compromise or respiratory distress, any additional impairment of the level of consciousness can lead to deterioration in the clinical situation. The goal of sedation during awake intubation is a calm and cooperative patient who remains crisply responsive to command. During awake bronchoscopic intubation, the ability of the patient to breathe deeply on command improves visualization of the airway structures, moves the epiglottis anteriorly out of the path of the advancing bronchoscope and endotracheal tube, and by producing maximum abduction of the vocal cords facilitates glottic cannulation. Sedation can therefore make bronchoscopic intubation more difficult.

Sedation to facilitate awake intubation can be achieved using a variety of drugs. In general, the minimum amount of sedation required for anxiolysis should be used and the dose carefully titrated to effect.

Midazolam administered in increments of 0.25 to 0.5 mg to the adult produces anxiolysis and amnesia, and titration to a suitable end point without losing patient cooperation is usually achievable, although the ability of the patient to briskly respond to command may be impaired. The onset time is 1 to 3 minutes and the duration of action is about 2 hours. Fentanyl can provide sedation, analgesia, and euphoria, can attenuate laryngeal reflexes, and has an antitussive effect. Respiratory depression can also occur, as can bradycardia. When used in combination, a synergism between fentanyl and midazolam occurs which potentiates the effects of both drugs.

Droperidol, a butyrophenone that produces a state of quiescence with reduced motor activity and indifference to one’s surroundings, also has been used to facilitate awake intubation. However, about 10% of individuals exposed to droperidol experience a feeling of mental restlessness and agitation, so-called...
dysphoria, and prolongation of the QT interval can occur even at low doses. Ketamine can produce excessive secretions, disorientation, hallucinations, and mild respiratory depression and is not commonly used to facilitate awake intubation.

In the emergency situation, when judicious chemical restraint is required to permit airway management of combative and intoxicated patients, haloperidol, also a butyrophenone, can be immensely helpful. Intravenous doses of 2 to 10 mg in the adult can be carefully titrated to effect. Ketamine may be particularly useful in the uncooperative patient without IV access who requires chemical restraint.

Recently, the use of remifentanil for awake bronchoscopic intubation as a single agent and in combination with propofol or midazolam has been reported.

- Reusche and Egan reported a case of Ludwig’s angina in which awake nasotracheal intubation was performed using a remifentanil infusion at 0.05 to 0.175 μg·kg⁻¹·min⁻¹. The patient was also given glycopyrrolate 0.2 mg, droperidol 0.625 mg, and midazolam 2.0 mg IV. Four milliliters of 4% lidocaine was administered by nebulizer, the right naris was swabbed with 4% cocaine, and 2 mL of 4% lidocaine was sprayed on the vocal cords through the bronchoscope. The patient was intubated without gagging, bucking, or coughing.

- Johnson et al reported the use of a remifentanil bolus of 3.2 μg·kg⁻¹ in addition to 26 μg·kg⁻¹ of midazolam to perform direct laryngoscopy on a patient who was predicted to be a difficult intubation on physical examination. The patient remained conscious and followed commands throughout the direct laryngoscopy and subsequent intubation, appeared to tolerate the procedure well, and had no recall of the event. No local anesthetic was used.

- Puchner et al reported the use of a remifentanil infusion to facilitate awake nasotracheal intubation of a patient with odontogenic, facial, and cervical infection. The nose was anesthetized with 4% lidocaine spray, and 2 mL of 4% lidocaine was sprayed through the bronchoscope onto the vocal cords and subglottic area. The patient remained conscious, calm, and cooperative. No reflex glottic closure was observed. Remifentanil infusion was subsequently compared to a combination of fentanyl and midazolam during awake bronchoscopic intubation in 74 patients. Remifentanil was administered in dosages of 0.1 to 0.5 μg·kg⁻¹·min⁻¹. Both groups received PO midazolam 1 hour preoperatively. Four percent lidocaine spray was administered to the nose, and 4 mL of 4% lidocaine was sprayed through the bronchoscope onto supraglottic and subglottic areas. Patients in the remifentanil group had a significantly reduced response to the nasal passage of the tube and less cough as the larynx was intubated. The investigators felt that remifentanil suppressed laryngeal reflexes significantly better than fentanyl and midazolam and improved intubating conditions.

- Machata et al compared a remifentanil 0.75 μg·kg⁻¹ bolus, followed by 0.075 μg·kg⁻¹·min⁻¹ with a 1.5 μg·kg⁻¹ bolus followed by 0.15 μg·kg⁻¹·min⁻¹ for awake intubation in 24 patients. All patients were premedicated with midazolam 0.05 mg·kg⁻¹ and glycopyrrolate 0.2 mg IV. The nostril was anesthetized using 2% lidocaine gel coating on nasopharyngeal tubes. The supraglottic region was sprayed with 5 mL of 2% lidocaine and 2 mL was instilled onto the vocal cords through the working channel of the bronchoscope. No respiratory rate less than eight breaths per minute occurred, no patient recalled pain, and all patients remained cooperative. Intubating conditions were adequate in all patients and comparable between the groups. Both regimens blunted airway reflexes sufficiently.

- Xu et al determined that when combined with midazolam 0.1 mg·kg⁻¹ IV and topical anesthesia of the airway, the ED₉₅ of remifentanil for successful awake direct laryngoscopy and intubation was a bolus of 0.62 μg·kg⁻¹ followed by an infusion of 0.062 μg·kg⁻¹·min⁻¹.

- Mingo et al reported the use of remifentanil for awake bronchoscopic nasotracheal intubation in a group of 24 patients scheduled for elective surgery. Topical anesthesia was limited to the nasal mucosa. The remifentanil infusion was started at 0.3 μg·kg⁻¹·min⁻¹ and titrated between 0.2 and 0.5 μg·kg⁻¹·min⁻¹ to produce adequate sedation defined as “falling asleep if unstimulated but immediately responsive to command.” Intubation difficulty as rated by the endoscopist and an observing anesthetist was reported, respectively, to be easy in 17 and 15 of 24 patients, and moderate in 5/24 and 7/24. Both raters reported the intubation as difficult in two patients. Coughing during intubation occurred in six patients and grimacing in six. Ten patients recalled the procedure but only one would not repeat it if it were considered necessary. The authors concluded that intubation had most likely been made possible by the suppression of airway reflexes and the intense analgesia produced by remifentanil. Although the results of this study cannot be readily extrapolated to the setting of acute airway compromise, attenuation of airway reflexes by remifentanil in this emergency setting may be beneficial.

- Rai et al compared remifentanil and propofol administered as target-controlled infusions in a group of 24 patients who underwent bronchoscopic intubation under topical anesthesia. All patients received 1 to 2 mg of IV midazolam based on weight, and IV glycopyrrolate. They were then randomized into remifentanil and propofol groups and topical anesthesia was administered. Sedation scores were similar in each group. Endoscopy and intubation were more difficult in the propofol group and required more time to complete. Twenty three patients were intubated on the first attempt. One patient in the propofol group required two attempts. The patients in the remifentanil group tolerated the procedure much better as determined by discomfort and postintubation scores. Despite a higher level of recall in the remifentanil group, patient satisfaction was the same in both groups. No adverse events were recorded.

- Remifentanil has also been administered by infusion in combination with low-dose propofol during awake intubation.

The ester structure of remifentanil is unique among fentanyl congeners and results in very rapid metabolism. The peak effect-site concentration of remifentanil occurs within 1 to 2 minutes of bolus injection, and the offset is also rapid. The time necessary to reach a 50% decrease in serum concentration after stopping a continuous infusion at steady state is 4 minutes. The median dose of remifentanil administered over 2 minutes, required to
produce loss of consciousness, has been found to be 12 μg·kg⁻¹·h⁻¹, and at doses ≤5 μg·kg⁻¹ no subjects lost consciousness.193,201 It has been recommended that dosing should be calculated on lean body mass and reduced by as much as 50%-70% in the elderly.193 Remifentanil therefore appears to be very easily titrated. Jhaveri et al noted mild muscle rigidity in 40% and moderate rigidity in an additional 40% of a group of elective surgical patients following the administration of 2 μg·kg⁻¹·h⁻¹ of remifentanil over 2 minutes.201 No severe muscle rigidity was observed at doses less than or equal to 4 μg·kg⁻¹. Wilhelm et al administered remifentanil by infusion to a group of patients undergoing oocyte removal and did not observe muscle rigidity at doses up to 0.4 μg·kg⁻¹·min⁻¹.202

Anecdotally, in the author’s experience remifentanil infusion appears to attenuate the gag reflex as well as laryngeal reflexes, and can facilitate airway anesthesia. It may be particularly useful in patients with hyperactive gag reflexes, and in the presence of excess secretions. Remifentanil may prove to be an exception to the general rule that sedatives cannot or should not be used to compensate for poor regional anesthesia of the airway.

Dexmedetomidine (DEX) is a centrally acting alpha-2-adrenoceptor agonist, which produces sedation, analgesia, anxiolyis, xerostomia, and some degree of amnesia.203-206 The sedation produced by DEX is unique in that patients appear to be asleep but are readily aroused.204 The term “cooperative sedation” has been used191,205 as DEX appears to maintain191,209 or enhance210 patient cooperation, and the ability to follow commands.209 DEX produces minimal191,206,209,211 or no respiratory depression191,203,210 but can produce hypotension, hypertension, tachycardia, and bradycardia.191,203-206 When administered as a continuous IV infusion, however, it is associated with a predictable and stable hemodynamic response.203 Caution is necessary when DEX is administered to patients who are volume depleted, vasoconstricted, or who have severe heart block.203 Contraindications to DEX as listed by Unger include hypovolemia, hypotension, aortic stenosis, idiopathic hypertrophic subaortic stenosis, pulmonary hypertension, and heart block in the absence of a pacemaker.191 The recommended loading dose of DEX is 0.5 to 1.0 μg·kg⁻¹ over 10 to 20 minutes, which can then be followed by a continuous infusion of 0.2 to 0.7 μg·kg⁻¹·h⁻¹.191,193,209,206,209-202 The elimination half-life is 2 hours.203

Avisian et al performed a retrospective review of 19 patients who underwent awake bronchoscopic intubation using DEX for sedation.211 Midazolam and/or fentanyl was also given to all but two patients, and topical anesthesia was utilized. A dose of 1 μg·kg⁻¹ of DEX was administered over 10 to 15 minutes and this was followed by a continuous infusion of 0.2 to 0.7 μg·kg⁻¹·h⁻¹ if required. The intubation was “smooth” in all cases with good patient tolerance and no airway obstruction. Thirteen patients developed hypotension after induction of general anesthesia that was managed with ephedrine or phenylephrine.211

Grant et al reported three cases of awake bronchoscopic intubation in which DEX was used for sedation.206 Local anesthesia was utilized but no other sedatives were administered. Intubating conditions were acceptable in all three patients. No clinically important hypotension or bradycardia was reported. One patient recalled the intubation but was not distressed by that recollection.

Abdulmalik et al reported five cases in which DEX was used as the sole sedative for awake bronchoscopic intubation.209 Topical lidocaine was also utilized. The patients remained responsive to command and were all intubated on the first attempt. Four of the five patients had no recall of the intubation. Hypotension required treatment in two cases. The lowest heart rate reported was 48 bpm.

Bergese et al reported the use of dexmedetomidine sedation in four patients with difficult airways who underwent successful awake bronchoscopic intubation.210 Two patients also received midazolam. Local anesthesia was used in three patients. Two patients were initially uncooperative, but following the administration of DEX were able to follow command, and excellent intubating conditions were achieved. Hemodynamics remained stable during the procedure in all four cases.

Neumann et al and Maroof et al have also reported successful awake bronchoscopic intubations in patients with difficult airways under topical anesthesia and DEX sedation.212,213

Dexmedetomidine infusion in combination with ketamine infusion has also been used for awake bronchoscopic intubation.214 Scher and Gitlin administered a bolus of 1 μg·kg⁻¹ of dexmedetomidine over 10 minutes and followed this with an infusion of 0.7 μg·kg⁻¹·h⁻¹.160 Upon completion of the dexmedetomidine bolus, 15 mg of ketamine was administered as a bolus, and then followed by an infusion of 20 mg·h⁻¹. The patient remained responsive to command and calm. Regional anesthesia of the airway was then performed “in the usual manner,” and bronchoscopic intubation via a bronchoscopic oral airway was performed.214 Intubating conditions were reported to be excellent and included a secretion-free airway. The patient had no recall of the procedure.

Hagberg et al performed a randomized double-blind comparison of remifentanil (R) and dexmedetomidine (DEX) for sedation during awake bronchoscopic intubation in a group of 30 patients.207 All patients were given glycopyrrolate and midazolam and their airways were topicalized with 4% lidocaine. Patients in the remifentanil group were given a bolus of 0.75 μg·kg⁻¹, followed by an infusion of 0.075 μg·kg⁻¹·min⁻¹. Patients in the DEX group received a bolus of 0.4 μg·kg⁻¹ over 10 minutes followed by an infusion of 0.7 μg·kg⁻¹·h⁻¹. All patients were successfully intubated. Thirteen of 17 patients in the R group were intubated on the first attempt, 3 required 2 attempts, and 1 required 3 attempts. In the DEX group, 5 of 13 patients were intubated on the first attempt, 4 required 2 attempts, and 4 required 3 attempts. Minimal hemodynamic instability was observed in both groups. Intraoperative recall was significantly lower in the DEX group.

Dexmedetomidine appears to be very useful as a sedative to facilitate awake bronchoscopic intubation. It is however not available in Canada and the author has no personal experience with this drug. The reports of its use in uncooperative patients, who following the administration of DEX were able to follow command, are particularly intriguing, as is its use in the absence of local anesthesia.

### 3.5.4 How should a patient be prepared psychologically to undergo awake intubation?

Undergoing any medical procedure can be intimidating, anxiety provoking, even frightening, and the practitioner must make every effort to minimize patient anxiety. As time permits in the emergency setting, and routinely in the elective situation, a full...
3.5.5 What technique works well for the average patient?

In the emergency setting, regional anesthesia of the airway for awake intubation by direct laryngoscopy can be readily achieved using 10% lidocaine spray. The spray can be directed onto the posterior third of the tongue, the uvula, and the tonsillar pillars using the malleable, stainless steel nozzle, and a tongue depressor to retract the tongue. Sprays can also be delivered into the piriform fossae; however, spraying into the larynx should be avoided as laryngospasm, cough, and a loss of patient cooperation may be precipitated. An explanation of the procedure should be provided to the patient as time and circumstances permit. In general, the use of sedation should be avoided in the emergency awake intubation and the time required for antisialagogues to provide mucosal drying is not available. If neck movement is permissible, then awake intubation by direct laryngoscopy is most easily performed with the patient in the sitting position.

In the setting of an anticipated difficult intubation in which awake bronchoscopic intubation is planned, profound regional anesthesia of the airway can be achieved using the following technique:

- A full explanation of the procedure should be provided to the patient.
- Glycopyrrolate should be administered IM about 20 to 30 minutes or IV at least 15 minutes prior to planned airway manipulation. The tongue should appear dry prior to airway manipulation.
- If neck movement is allowed, the patient should be in the sitting or semi-sitting position.
- A remifentanil infusion can be initiated at 0.1 to 0.2 ug·kg⁻¹·min⁻¹.
- Five percent lidocaine ointment can be gently applied to the posterior third of the tongue using a tongue depressor. Alternatively, the patient can be asked to gargle 40 to 50 mL of 4% lidocaine solution and expectorate the residual or 10% lidocaine spray can also be directed onto the posterior third of the tongue, uvula, and fauces.
- About 12 mL of aerosolized 3% lidocaine can then be administered optimally via the nose using a DeVilbiss atomizer attached to a high-pressure oxygen source. The spray delivered by the atomizer should be coordinated with respiration, with inhalation through the nose, and exhalation through the mouth. Alternatively, the aerosol can be administered via the mouth.
- Cotton pledges held securely by Jackson forceps can be soaked in 4% lidocaine and then gently advanced over the tongue into the piriform fossa on each side to ensure or supplement laryngeal anesthesia. As the pledge is removed, the mucosa of the oropharynx can be swabbed to confirm the absence of the gag reflex.

Bronchoscopic intubation can then be rapidly achieved as described in Chapter 9.

Awake intubation under topical anesthesia with and without sedation has also been described using various rigid bronchoscopic devices.215-219

3.6 SUMMARY

Awake intubation can be achieved rapidly with minimal patient discomfort. Knowledge of airway anatomy, the medications that can be employed, and techniques of regional anesthesia of the airway are necessary. Manual dexterity, gentleness, and an appropriate bedside manner are also essential.

REFERENCES


SELF-EVALUATION QUESTIONS

3.1. The lower border of the quadrangular ligament forms

A. the true vocal cord
B. the false vocal cord
C. the aryepiglottic ligament
D. the triangular ligament
E. the hyoepiglottic ligament

3.2. The maximum effective concentration of topical lidocaine applied to the tongue is

A. 1%
B. 2%
C. 4%
D. 10%
E. 15%

3.3. Benzocaine

A. is an ester
B. is metabolized to para-aminobenzoic acid
C. can produce methemoglobinemia
D. is an effective topical anesthetic
E. all of the above