Ludwig's Angina: Anesthetic Management

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Ludwig's angina (LA) is a gangrenous cellulitis of the neck that spreads via continuity of the fascial planes. Treatment of LA includes aggressive antibiotic therapy as well as surgical drainage in many cases. The most common cause of infection is odontogenic and can be due to both aerobic and anaerobic bacteria. Signs and symptoms of LA include bilateral cervical swelling, dysphagia, drooling, neck tenderness, elevation and posterior distension of the tongue, restricted neck movement, trismus, dyspnea, and stridor, which can result in difficult airway management. Proper preoperative assessment of patients with LA should include identifying features that may cause difficulties with mask ventilation, direct laryngoscopy, and intubation. Alternative methods of ventilation should be considered and immediately accessible, including a plan on how and when they would be used in the event that a patient cannot be mask ventilated or intubated. Marking external anatomical airway landmarks prior to manipulating the airway can save vital time if an emergent airway becomes necessary.

Key Words: Ludwig's angina; General anesthesia; Preoperative assessment; Difficult airway algorithm.

First described in 1836 by the German surgeon, Friedrich Wilhelm von Ludwig, Ludwig's angina (LA) was discovered to be a gangrenous cellulitis in the soft tissues of the neck.^{1,2} Angina comes from the Latin word *angere*, which means "to strangle."¹ The classical description includes (a) the infection is bilateral, (b) both the submandibular and sublingual spaces are involved as well as the submental space, (c) the infection is a rapidly spreading indurated cellulitis without abscess formation or lymphatic involvement, and (d) the infection begins in the floor of the mouth.¹ Presentation of LA can occur at any age but is most commonly seen in middle age; the infection has a 2:1 propensity for males.³ Those with lower socioeconomic background are at a higher risk because of a potential lack of dental and primary care.⁴ Odontogenic infection (especially second and third mandibular molars), peritonsillar abscess, and mandibular fracture are the most common comorbidities associated with LA.² Predisposing factors that increase the risk of developing LA include poor dental hygiene (contributing to approximately 75–95% of cases), malnutrition, intravenous drug use, diabetes mellitus, acquired immune deficiency syndrome, immunosuppres-

sion, and systemic lupus erythematosus.¹ Signs and symptoms of LA include malaise, dysphagia, drooling, bilateral cervical swelling, neck tenderness, dysphonia, elevation, posterior displacement and swelling of the tongue, pain in the floor of the mouth, sore throat, restricted neck movement, and stridor.² These signs and symptoms suggest impending airway obstruction.¹ During the past century, in at-risk populations, the incidence of developing LA has decreased from 60% to 10% through the use of antibiotics and improved oral hygiene.^{2,5}

ANATOMY

Knowledge of head and neck anatomy is important for understanding how the spread of infection occurs. The neck is composed of 2 layers of fascia: the superficial and deep, which are in communication with one another. The deep layer is composed of 3 separate layers: the general investing layer of deep cervical fascia (superficial), the pretracheal fascia (middle), and the prevertebral fascia (deep; see Figure 1).⁶ The superficial layer of the deep cervical fascia encircles all of the muscles of the neck, extending posteriorly to attach to the spinous process of the vertebrae, superiorly to the hyoid bone and submandibular gland, and along the mandible posteriorly to the parotid gland. The pretracheal layer extends from the hyoid bone to the thorax and encloses the trachea, thyroid, and esophagus. It is

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Figure 1. Fascial layers of the neck: 1, investing layer of deep cervical fascia; 2, pretracheal fascia; 3, carotid sheath; 4, superficial fascia; 5, prevertebral fascia.²⁰

contiguous with the buccopharyngeal space inferiorly.⁶ The buccopharyngeal space is constructed of loose connective tissue, which allows for infection to spread easily to the retropharyngeal space. Communication between the retropharyngeal space and the “danger space,” which lies immediately posterior, can serve as a conduit for spread of infection from the pharyngeal region to the mediastinum reaching the thoracic cavity.^{2,7} Generally, the danger space is usually indistinguishable from the retropharyngeal space, but with infection, it can become distended as the space fills with fluid or pus. The deep layer encompasses the prevertebral muscles and forms the floor of the posterior triangle of the neck; it spans from the base of the skull to the endothoracic fascia of the thorax.

These layers can also be thought of in their relation to the hyoid bone. The pretracheal space is found solely inferior to the hyoid bone. The prevertebral and retropharyngeal spaces span the entire length of the neck. Spaces that lie entirely above the hyoid bone include the submental, sublingual, submandibular, and lateral pharyngeal spaces.⁸ The borders of the submental space are the hyoid bone inferiorly, the midline medially, and the anterior belly of the digastric laterally. The sublingual space is bound inferiorly by the mylohyoid muscle and anteriorly by the mandible (see Figure 2). The submandibular space is defined superiorly by the body of the mandible, medially by the anterior belly of the digastric, and posteriorly by the posterior belly of the digastric. The submental, sublingual, and submandibular spaces freely communicate with one another. The lateral pharyngeal space (or

parapharyngeal space) is deep to the submandibular space and anterior to the retropharyngeal space. In addition, these spaces can be designated relative to the boundaries enclosed by the muscles of mastication such as submasseteric, pterygomandibular, temporal or infratemporal, and buccal spaces. Collectively, they are known as the masticator spaces.

PATHOPHYSIOLOGY

A typical LA infection originates in the floor of the mouth and rapidly spreads throughout the submandibular space, resulting in elevation and posterior displacement of the tongue.⁹ This may lead to blockage of the oropharynx.^{10–12} From there, spread of infection occurs to the neck via continuity of the spaces between fascial layers and not through lymphatics. This presents clinically as a “bull neck”.^{1,7} Occasionally, crepitus in the neck can be appreciated due to gas formed in these spaces by anaerobic and aerobic bacteria.^{4,12} Common bacteria causing LA include *Streptococcus viridans*, *Staphylococcus aureus*, *B-hemolytic streptococcus* species, *Staphylococcus epidermidis*, *Bacteroid* genus, *Fusobacterium nucleatum*, *Peptostreptococcus*, and *Enterobacter aerogenes*.^{3,13} Greater than 50% of diabetic patients with LA present with *Klebsiella pneumoniae*. Treatment with intravenous antibiotics is necessary, and incision and drainage is often indicated. The regimen of choice is intravenous Penicillin G with metronidazole or similar effective against β -lactamase-producing anaerobic flora.^{2,13} Failure to recognize and properly treat LA may result in a compromised airway and lead to mortality.¹ Additional treatment may include intravenous dexamethasone (eg, 10 mg every 8 hours) for 48 hours to reduce edema helping to preserve airway integrity.^{5,14} Forensic reports of mortality from LA have shown pus accumulation around the thyroid cartilage, soft-tissue edema in the tongue-larynx-trachea regions, and edema of the epiglottis, true and false vocal cords, and aryepiglottic folds. The edema, in addition to the external compression secondary to inflammation and induration, may cause a narrowing in the laryngeal cavity and result in airway obstruction.^{1,13}

PREOPERATIVE ASSESSMENT

Proper preoperative assessment is key in the identification of a possible difficult airway. A difficult airway can be described as one in which an experienced anesthesiologist has a difficult time with mask ventilation, intubation, or both.^{15,16} Difficult mask ventilation is

Figure 2. Pathways for spread of odontogenic infections. (A) Coronal section at first molars: a, maxillary antrum; b, nasal cavity; c, palatal plate; d, sublingual space (above mylohyoid muscle); e, submandibular space; f, intraoral presentation with infection spreading through the buccal plates inside the attachment of the buccinator muscle; and g, extraoral presentation to buccal space with infection spreading through the buccal plates outside the attachment of the buccinator

the inability to provide adequate ventilation because of one or more of the following problems: inadequate seal, excessive gas leak, or excessive resistance to gas flow.¹⁶ When performing a preoperative airway assessment, multiple features should be evaluated, as one feature alone is not sufficient enough to suggest a difficult airway. The preoperative examination should begin with the patient's overall appearance, identifying any issues that may cause difficulties with mask ventilation.¹⁶ In patients with LA, difficult mask ventilation may be anticipated because the swelling under the neck is often found to be "woody," "calloused," and tense.¹⁷ A mouth opening of less than 4 cm, which is very common in LA patients, has been associated with difficult intubation, as well as the presence of loose teeth, dentures, or prominent anterior teeth.¹⁶ The Mallampati classification yields information about mouth opening and tongue size relative to the pharyngeal space. The classes are as follows (see Figure 3):

- Class I: soft palate, uvula, fauces, and tonsillar pillars are visible
- Class II: soft palate, uvula, and fauces are visible
- Class III: soft palate and base of uvula are visible
- Class IV: hard palate is visible

This classification scheme has shown good discriminatory power for difficult direct laryngoscopy and intubation but poor power to predict difficult ventilation.¹⁶ Regardless, most patients with LA will be Mallampati 3 or 4, such that difficult intubation and mask ventilation should be anticipated. Thyromental distance is used to assess the mandibular space and tissue compliance to determine the amount the tongue can be displaced during direct laryngoscopy.¹⁶ Measurements from the chin to the thyroid cartilage of less than 3 cm may be used to predict a difficult intubation.^{16,18} Neck range of motion with normal flexion and extension allow for optimal head manipulation to enhance the glottic view during direct laryngoscopy. It is not uncommon for patients with LA to have decreased cervical range of motion. Decreased cervical spine motion has been identified as an independent risk factor for difficult mask ventilation, direct laryngoscopy, and intubation (see Table 1 and Table 2).¹⁶ Additional airway risk factors include a high arched palate, previous head and neck radiation therapy, severe rheumatoid arthritis, and a history of

← muscle. (B) Lingual aspect of the mandible: a, tooth apices above the mylohyoid muscle with spread of infection into sublingual space; b, tooth apices below the mylohyoid muscle (mandibular third molars) with spread of infection into submandibular space.²

Figure 3. Mallampati views. (A) Class I: soft palate, uvula, fauces, and tonsillar pillars are visible. (B) Class II: soft palate, uvula, and fauces are visible. (C) Class III: soft palate and base of uvula are visible. (D) Class IV: hard palate is visible.

previous difficult intubation. Review of preoperative computed tomography scans with the treating surgeon can aid in determining the extent of pharyngeal and hypopharyngeal swelling.

AIRWAY MANAGEMENT

The American Society of Anesthesiologists (ASA) Closed Claims database reveals that airway events account for 34% of claims. Difficult intubation was identified as the most common cause of morbidity since the 1990s.⁴ With deep neck space infections, mortality most often occurs due to hypoxia or asphyxia. Infection can also cause trismus, laryngeal edema, and limited neck mobility and may contribute to difficult patient management.² Computed tomography scanning is generally indicated preoperatively to investigate potential airway problems and mediastinal involvement.^{2,4}

There is no universal agreement on the proper approach to airway management in patients with LA

because of the wide variety of presentation and mitigating factors. Three of the most commonly used intubation techniques for patients with LA include elective tracheostomy, awake blind nasal intubation, and flexible fiber-optic nasal or oral intubation.^{4,12} Standard direct laryngoscopy will often fail because of firmness in the floor of the mouth and the posterior displacement of the tongue.¹⁹ If a slow inhalation induction prior to intubation is considered, the use of sevoflurane will allow for rapid control of anesthetic depth, cardiovascular stability, and maintenance of spontaneous breathing, but airway obstruction can still suddenly occur. If an awake nasal fiber-optic intubation is to be performed, having the patient seated upright with the head in the sniffing position is optimal because this will maximally open the pharyngeal airway to allow for the passage of the nasal endotracheal tube. This also allows for a more accurate assessment of the airway when planning for extubation.¹⁹ While benzodiazepine sedation may aid in patient comfort and provide some level of amnesia for awake intubation, it must be noted that sponta-

Table 1. Disease States Associated With Difficult Airway Management

<i>Congenital</i>	<i>Acquired</i>
Pierre-Robin syndrome	Morbid obesity
Treacher-Collins syndrome	Acromegaly
Goldenhar’s syndrome	Infections involving the airway (Ludwig’s angina)
Mucopolysaccharidoses	Rheumatoid arthritis
Achondroplasia	Ankylosing spondylitis
Micrognathia	Tumors involving the airway
Down’s syndrome	Trauma

Table 2. Predictors of Difficult Intubation

Mallampati class
Head and neck movement
Mouth opening
Thyromental distance
Sternomental distance
Upper lip bite test
Prominent “buck” teeth
Interincisor distance
Mandible luxation

neously breathing LA patients with significant airway compromise may develop airway obstruction subsequent to even mild sedation. Excellent local anesthesia of the airway is important to allow the patient to tolerate awake intubation. Traditional superior laryngeal nerve blocks, intraoral posterior laryngeal nerve blocks, and transtracheal local anesthetic injection for recurrent laryngeal nerve blocks may be difficult in distorted anatomy and even contraindicated when infection has spread to the neck. Topical airway anesthesia may be preferred. However, benzocaine-containing preparations (e.g., Cetacaine) may easily lead to overdose or methemoglobinemia because of the larger doses needed for adequate anesthesia of the large area of soft tissue that is required. Topical lidocaine airway spray or preferably face mask nebulized lidocaine or oral nebulized lidocaine (similar to a “breathing treatment”) may be the best options.

An otolaryngologist or other surgeon with significant tracheostomy experience should be readily available in the event this method fails and an emergent tracheostomy is deemed necessary. Alternatively, an awake tracheotomy may be used as the primary method for securing the airway and may be preferred when there is significant narrowing of the oropharyngeal airway or significant edema of the floor of the mouth. In this subset of patients, there may be a need for prolonged airway protection until the edema subsides, so tracheostomy may be desired.¹⁹ If intubation attempts are considered with invasive airway access as backup, due to possible distorted neck anatomy, surgical landmarks should ideally be identified and marked prior to intubation attempts.

The safety of extubation after surgery is determined by the preoperative examination, extent of infection and edema, and adequacy of surgical drainage. In cases of extensive preoperative airway edema or where tissue induration and edema are identified rather than a large collection of pus, extubation may be imprudent. Tracheotomy may be considered in these cases.¹⁹ If significant airway edema is suspected, a Cook catheter can be placed through the endotracheal tube (ETT) into the trachea to ensure an atraumatic path for reintubation if necessary. It is recommended that the Cook catheter be left in place for a minimum of 30 minutes after the removal of the ETT.⁴ Alternatively, the ETT cuff may be deflated for at least several minutes prior to extubation. If the patient is able to breathe around the tube with a deflated cuff, the laryngeal edema is most likely not significant enough to compress the lumen of the trachea or otherwise cause soft-tissue obstruction.¹⁶

USE OF THE ASA DIFFICULT AIRWAY ALGORITHM IN PATIENTS WITH LA

It is important to remember that the best time to consider the difficult airway algorithm and possible negative outcomes is in the preoperative period. Preoperatively, when a difficult airway is encountered, it should be determined if intubation is deemed possible. Prior to, and during, intubation attempts, aggressive oxygenation is recommended to allow increased time for adequate patient oxygenation should initial intubation attempts prove unsuccessful. The ASA difficult airway algorithm indicates that when intubation attempts are unsuccessful, the anesthesia provider should first consider calling for help and then return the patient to spontaneous ventilation or awaken the patient, if possible. If face mask ventilation is not adequate, the algorithm recommends that the provider should consider inserting a supraglottic airway, such as a laryngeal mask airway. In the setting of most cases of LA, these devices are likely to fail, even if insertion can be accomplished. If a supraglottic airway is contraindicated or not feasible, as is likely, and ventilation is not adequate, the algorithm suggests a noninvasive emergency airway can be considered, including the use of a rigid bronchoscope (very difficult in this situation), transtracheal jet ventilation (may also be difficult because of neck anatomy distortion and/or infection), or, if the anesthesia provider is experienced in the technique, blind nasal intubation. The use of an esophageal-tracheal combitube can be attempted, but hypopharyngeal edema will likely render it unsuccessful. When emergency noninvasive measures fail, emergency invasive airway access, tracheostomy or cricothyrotomy, must be immediately established (see Figure 4).

SUMMARY

LA is primarily odontogenic in etiology. It spreads via continuity of the fascial planes rather than abscess formation. Treatment of LA includes antibiotics but can require surgical drainage. Signs and symptoms of LA include bilateral cervical swelling, dysphagia, drooling, neck tenderness, elevation, distension and posterior displacement of the tongue, restricted neck movement, trismus, dyspnea, and stridor, all of which can result in difficult airway management. For cases requiring anesthesia, a proper preoperative assessment should be conducted prior to induction and include identifying features that may cause difficulties with mask ventilation, direct laryngoscopy, or intubation.

Figure 4. Difficult airway algorithm.¹⁵

In anticipation of the event that the patient cannot be ventilated or intubated, emergency invasive airway access and other alternative approaches should be immediately available.

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1. Ludwig's angina is classified by all of the following EXCEPT:
 - A. The infection is bilateral
 - B. Both the submandibular and sublingual spaces are involved
 - C. The infection is always odontogenic in nature
 - D. The infection spreads via cellulitis without abscess formation
2. Which of the following are predictors of a difficult intubation?
 - A. Mallampati class
 - B. Mouth opening
 - C. Thyromental distance
 - D. All of the above
3. The most commonly used intubation techniques for LA include:
 - A. Elective tracheostomy
 - B. Awake blind nasal intubation
 - C. Fiber-optic laryngoscopy
 - D. All of the above
4. Greater than 50% of diabetic patients have LA associated with which of the following?
 - A. *Streptococcus viridans*
 - B. *Klebsiella pneumoniae*
 - C. *Staphylococcus aureus*
 - D. *Fusobacterium nucleatum*