Seven Protective Reflexes Every Dentist Should Know

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SEVEN PROTECTIVE REFLEXES EVERY DENTIST SHOULD KNOW

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Learning Objectives: After reading this article, the individual will learn: (1) seven protective reflexes that patients may demonstrate when undergoing dental treatment, and (2) the mechanisms of these reflexes, as well as preventive and management techniques clinicians can use to avoid or address complications caused by these reflexes.

About the Author

Dr. Flores graduated from Lamar University in Beaumont, Tex, with a bachelor’s degree in biology (2001) and a bachelor’s degree in nursing (2004). He worked as a registered nurse (RN) in the emergency, critical care/intensive care unit, behavioral health, neurotrauma, and rehabilitation departments. As an RN, he concurrently attended dental school at the University of Texas Dental Branch in Houston, earning several awards, including the Anesthesiology Safety Practice Award and the Horace Wells Award for Anesthesiology. After graduation, he attended the University of Pittsburgh School of Dental Medicine where he completed his specialty training in dental anesthesia. During his chief residency year, Dr. Flores founded the first predoctoral dental anesthesia society, Dentist Anesthesiologist Club for Students. He has received dual-board certification in dental anesthesia, is a Diplomate in the American Dental Board of Anesthesiology and National Dental Board of Anesthesiology, and is a Fellow in the American Dental Society of Anesthesiology. He served as anesthesiology director for a surgical center in Houston and started a mobile anesthesia and anesthesia consultant practice. He currently serves as the clinic director and director of dental anesthesia for University of New Mexico Medical Group’s Ambulatory Surgical Center and as assistant professor for the advanced education in general dentistry residency. His professional goals are to complete a master’s of healthcare administration and help strengthen dentistry’s transition into the field of oral medicine. He can be reached at jflores77@salud.unm.edu.

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There are certain intrinsic reflexes that are defensive responses the body uses for protection, and these reflexes are often seen as a disruption or obstacle to completing dental care. One or all of these mechanisms may be a reason for a patient’s reluctance to seek general dental care, which can result in declining dental health. While the physiological actions of these reflexes can be executed voluntarily, the functional actions resulting from reflex initiation are involuntary, and it is these autonomic actions that are considered in this article.

PHARYNGEAL REFLEX
The Pharyngeal Reflex—commonly known as the gag reflex—is a reason patients frequently seek deeper levels of sedation for dental treatment. The gag reflex halts swallowing to prevent foreign materials, substances, or large foodstuffs from entering the trachea, pharynx or larynx, thus prompting choking. This reflex is activated when an unwelcome object touches the soft palate in the mouth, posterior one third of the tongue, tonsillar, and surrounding tissues, or the oropharynx. Neurons that innervate the nucleus tractus solitarii (NTS) in the medulla oblongata are responsible for the activation of motor/sensory ninth cranial nerve (CN IX) (also known as the glossopharyngeal nerve), along with accessory nerves, to begin the tongue’s downward positioning and forward thrusting action in preparation to launch the unwanted material.

Though closely related to the Laryngospastic Reflex (to be covered in a later section), the rapid vocal cord closure, or laryngospasm, in the gag reflex differs in that it is not exaggerated or prolonged, only lasting as long as is needed to expel the invading material. This is usually associated with fully awake, nonsedated patients. For patients undergoing procedures in or around the airway, oral cavity structures, soft palate, or posterior tongue (eg, dental procedures), aerosol delivery of 4% lidocaine given during quiet breathing prior to stimulation has been shown to abolish the gag reflex for 15 to 20 minutes.

Management techniques for the gag reflex during dental procedures have ranged from hypnosis to deep levels of anesthesia. A review of the article by Bassi et al, “The Etiology and Management of Gagging: A Review of the Literature,” shows dentists using acupuncture or oral rinses. While moderate success can be obtained, 2 methods have stood out as effective management techniques: (1) deep levels of anesthesia (sedation)/general anesthesia (DS/GA) and (2) behavior modification.

DS/GA by a trained dental anesthesia provider is an effective technique for preventing the gag reflex. The patient is chemically but safely brought into a decreased responsive state, thereby lowering the responsiveness to the nerve stimulation needed to initiate the gagging motion. DS/GA is most beneficial with patients who have a severe or highly disruptive gag reflex. DS/GA is quick to administer and most dental work can be completed in one visit.
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For those providers who do not have access to a dental anesthesiologist, one of the more widely useful techniques is behavior modification. While DS/GA can be a short-term solution, behavior modification can be a long-term solution and is most useful for slight to moderate gaggers. Behavior modification is based on the fact that the act of gagging is multifactorial, and triggers such as sight or smell of equipment or materials are controlled and adapted. Behavior modification attempts to identify the dental trigger event and adapt the 5 anatomical intraoral areas known to be “trigger zones” to accept pressure from dental manipulation. These trigger zones are palatoglossal and palatopharyngeal folds, base of tongue, palate, uvula, and posterior pharynx.4 The drawback to successful behavior modification is that dental treatment must now be performed during a number of office visits with reinforcement of the acclimatizing technique at each appointment.

Cough Reflex
As with the gag reflex, the NTS nerve clusters in the medulla also facilitate the cough reflex, but with increased neuronal transmission involvement from the vagal afferent nerves (CN X), which are more sensitive to mechanical, chemical, and thermal stimulation. In the oral cavity, pharynx, and larynx, it is the area of distribution of the glossopharyngeal (CN IX) and trigeminal (CN V) nerves that lend sensory and motor support to the cough reflex action. Stretch receptors, or Rapidly Adapting Receptors, in the conducting airways and lungs can enhance or reduce the magnitude of the coughing event.4,5 The protective purpose of the cough reflex is to halt and reverse the inhalation of aerosol, particulate, or gaseous irritants when the tracheal, laryngeal, or bronchial mucosa is stimulated.4,6

Interestingly, the cough reflex can be initiated by both external stimulation (invasion of noxious stimuli) or by internal stimulation (mucus secretions moving up the bronchial tract via ciliary movements and bronchial muscle contractions). When a large enough bolus of secretions has made its way high enough up the larynx, the cough reflex is initiated to begin the secretion expulsion process.7 The cough reflex can be restrictive to dental treatments, especially in long-term smokers, where secretion clearance must now be performed during a number of office visits with reinforcement of the acclimatizing technique at each appointment.

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Bronchospastic Reflex
The Bronchospastic Reflex, also known as reflex bronchoconstriction or an asthma attack, is related to the cough reflex in that it can be a progression from failure of the cough reflex to adequately expel inspired harmful irritants. Like the cough reflex, the Bronchospastic Reflex starts with mucosal stimulation in the larynx, trachea, and bronchioles.13,14 Unlike the cough reflex, which causes airway hyperexpansion in preparation for a violent exhalation force and uses mucous as a carrier mechanism to further expel unwanted materials, the Bronchospastic Reflex causes airway constriction and increased mucus production as a protective coating to decrease the severity of airway insult to the pulmonary tissues. The purpose of the constriction during a bronchospasm is a sort of “hunker down” mentality of the pulmonary tissues to protect them as they ride out the irritant storm.15,16 A bronchospasm in response to anesthesia equipment irritation is essentially an asthma attack, so dental work during acute exacerbation events should be avoided, and pretreatment with rapid-acting beta2-agonist therapy, long-acting beta2-agonist therapy, and corticosteroids will abate the reflex.17

In a study by Groeben et al, lidocaine significantly reduced bronchoconstriction of respiratory smooth muscle cells and caused reflex suppression most notably with inhalation administration via nebulizer. Inhalation administration of lidocaine also yielded a much higher, longer-lasting concentration of active drug in the airway tissues and negligible increases in blood
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plasma lidocaine levels, therefore decreasing the possibility of subsequent cardiac arrhythmias. Lidocaine also blocks the vagus pathway (CN X); this along with direct relaxation of the smooth muscle makes nebulized lidocaine an effective mechanism to stop the Bronchospastic Reflex.\(^{18}\)

Management of the Bronchospastic Reflex (or asthma attack) almost always involves rescue medications and starts at the preoperative or screening appointment.\(^{19,20}\) A good history and physical examination should inform the operating dentist that the patient may have a medical condition prone to bronchospasm, such as asthma.\(^{20,21}\) As an attack begins, the clinician must first stop the procedure, clear the mouth of all dental materials, position the patient properly (usually upright), and prepare to administer a bronchodilator either via inhaler or nebulizing mask. For severe bronchospasms, subcutaneous, intramuscular, or intravenous epinephrine may be needed. Proper training is imperative for the doctor in these situations to ensure proper dosing. Corticosteroids and histamine blockers may also be given but are not first-line rescue drugs for a patient facing life-threatening pulmonary constriction.\(^{20,22,23}\)

**LARYNGOSPASTIC REFLEX**

While the gag reflex is often initiated more anatomically superior and in the oral cavity, the protective Laryngospastic Reflex is most often initiated posteriorly in the endolarynx at the vocal cord level and initiates a much more violent, exaggerated, prolonged, and life-threatening closure response. The superior laryngeal nerve mediates the vocal cord closure during laryngospasm events.\(^{24}\) In severe, true laryngospasm events, closure may involve the entire epiglottic body, obscuring the true cords, completely blocking airflow, and is a key difference in glottal closure that is seen during the gag reflex.\(^{25}\) In a dental setting, this is most often seen concurrently in a sedated patient where poor surgical field control has allowed saliva, water irrigation, blood, or other surgical debris to slip down the throat. Though its activation is airway protective, in most instances the Laryngospastic Reflex in the sedated patient needs intervention on the part of the dental provider to be stopped in order to regain proper oxygen levels to the patient.\(^{20,21,26}\) This is in opposition to the glottal closure seen in the gag reflex, which usually involves a conscious patient and the closure is self-limiting to the noxious event. Another key difference between the 2 reflexes is the purpose of the 2 cord closures. Whereas vocal cord closure during the gag reflex is in preparation for forceful exhalation to launch debris away from the cords, the Laryngospastic Reflex’s purpose is to halt debris progression into the trachea, but may not necessarily be followed by forceful exhalation.\(^{1,4,5,15}\) Persistence of the Laryngospastic Reflex without intervention may result in hypoxemia, hypercapnia, and body systems injury.\(^{20,23,26}\)

Interestingly, patients undergoing increasing levels of sedatives during a dental procedure will experience reduced airway protective reflexes, with the exception of the Laryngospastic Reflex, with concurrent breathing cessation, or apnea.\(^{27}\) In a study conducted by Tagaito et al, when all other protective airway mechanisms are diminished, the laryngospasm replaces these diminished reflexes as the primary protector.\(^{27}\) This reflex can be abolished, but the sedative concentrations needed to do so will require the patient’s airway to be expertly managed. Glottal spasms (incomplete laryngospasms) are actually a common occurrence in nonsedate persons and can be similar to true laryngospasm.\(^{25}\) A person who swallows a drink too quickly while holding a conversation or inhaling concurrently has experienced the Laryngospastic Reflex followed by the cough reflex. The first reflex halts the liquid’s intrusion into the trachea, and the latter reflex quickly expels the liquid away from the glottal opening.\(^{21-25}\) Recovery is most often very rapid, followed by the person stating, “Oh, it went down the wrong pipe.” However, in a sedated patient, laryngospasms can be a life-threatening occurrence. As stated earlier, laryngospasms increase when other reflexes are blunted. Combine this with the fact that a dental provider is performing surgical procedures in a shared space and the risk of complications increases.\(^{27}\)

The management for a laryngospasm is first to clear the airway of any operative materials, next suction the airway—preferably with a Yankauer suction catheter—to reach the glottal opening, perform a head-tilt chin lift, and apply a bag valve mask using positive pressure ventilation (PPV) with 100% oxygen to force increased concentrations of oxygen into the airway and break the vocal cords open. Effective PPV may require the placement of an advanced airway device.\(^{20,23,26}\) These maneuvers are not second nature and require training and practice to be successful in a rescue situation. If these maneuvers fail, pharmacological intervention may be needed and should only be administered by proficiently trained individuals.\(^{20,23,25,26}\)

**SWALLOW REFLEX**

In opposition to the Laryngospastic Reflex’s more urgent, brutish action for protection, the *swallow reflex*—also referred to as the Palatal Reflex or Deglutition Reflex—is one of the more gracefully fluid protective reflexes. This reflex involves both sensory and motor inputs via the glossopharyngeal (CN IX) and
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vagus (CN X), nerves with significant activity when the superior laryngeal branch of the vagus is stimulated. Coordination of the tongue, pharynx, and epiglottis will direct foodstuffs and saliva into the esophagus. Movement of material toward the pharynx, above or adjacent to the vocal cords. The neuronal pathway for this reflex is complex, involving the trigeminal (CN V), facial (CN VII), glossopharyngeal (CN IX), vagus (CN X), accessory (CN XI), and hypoglossal (CN XII) nerves. With a complex synchronization, the cough reflex, the vomit reflex, and breathing are momentarily halted while the swallow reflex is in action. Disordered swallowing is most seen in patients with special needs and can be a disruption.

Swallowing in the dental setting is usually not troublesome and is easily managed. Of most dental supplies that slip down the airway, up to 60% end up caught in the valleculae of the oropharynx, above or adjacent to the vocal cords. However, in the event a patient has disordered swallowing, the swallow reflex can be elicited with oral stimulation. Disordered swallowing is most seen in patients with special needs and can be a disruption. Hypersalivation can be managed through vigilant suctioning or antisialogogues such as low-dose glycopyrrolate. Management can be achieved through DS/GA with a very experienced provider to achieve levels deep enough to abolish the reflex. Nonpharmacological management can be achieved with the aid of physical devices, such as bite blocks or oral molts, high Fowler’s (beach) position (head of the chair raised 30° to 90°) of the dental chair—thus reducing the force on the base of the tongue during retraction—and attentive suctioning of oral secretions.

VOMIT REFLEX

Like the Laryngospastic Reflex, the vomit reflex is characteristically violent in nature. An expulsive force is used to quickly move noxious material away from airway structures or from damaging gastric mucosa. Vomiting, or regurgitation, can be voluntary or involuntary depending on a patient’s medical conditions. The involuntary reaction during dental treatment will be discussed in this article, but not postoperative nausea and vomiting. The vomit reflex can be viewed as an extended consequence of the gag reflex’s failure or hyperactivity due to signals carried from the vagus (CN X) from the chemoreceptor trigger zone. As an unwanted intrusion escapes past the first 2 reflexes, it is either immediately expelled along with gastric contents or enters the esophagus via swallow reflex actions where it is expelled along with gastric contents. The vomit reflex begins with diaphragmatic contractions and progresses rapidly to laryngeal elevation and distention in preparation to clear a path for the vomitus. As the larynx clears a path, the pharyngeal muscles intrinsically relax and provide esophagus via swallow reflex actions where it is expelled along with gastric contents.

In most cases, the vomit reflex closely follows the gag reflex. Therefore, managing vomiting means limiting gagging. Physiologically, vomiting is primarily activated with aggressive stimulation of the phrenic (CN III, IV, and V), vagal (CN X), and accessory sympathetic nerves and is a reflex innate at birth, but has the capacity for modification of intensity and trigger. In patients with special needs, the vomiting reflex can be a defense mechanism to ward off dentists. These patients require special care to acclimate to the dental environment and providers. If a patient begins to vomit in the dental setting, providers may need to help sit the patient forward and lean him or her over to optimize the reflex, allowing gravity or dental suction devices to aid in ensuring that during the first recovery breath, aspiration does not occur.

ESOPHAGOGLOTTAL CLOSURE REFLEX

This last reflex is the most recent protective reflex being studied and is thought to play a role in preventing refluxed stomach contents from damaging the airway. The Esophagoglottal Closure Reflex (ECR) is elicited during the proximal esophageal distention that occurs during reflux events. As the name implies, a proximal esophageal distention of 10- to 60-mL volume causes closure of the glottal opening (vocal cords) to prevent aspiration of gastric contents. Larger volumes cause concurrent upper esophageal
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sphincter (UES) dilation and belching along with glottal anterior movement to sweep materials away from the glottal opening. These larger volumes of refluxate cause anterior movement of the hyoid bone, which in turn recruits a wider range of tongue, pharyngeal, and laryngeal movements to move refluxate up and away from the respiratory opening. While proximal esophageal distention has a direct relationship and activation of glottal closure, UES dilation to initiate a belching event does not directly activate glottal closure.43-44

ECR should not be confused with gastroesophageal reflux disease (GERD). GERD is the medical condition resulting from an abnormally weakened lower esophageal sphincter (LES) that results in chronic mucosal damage to the esophagus due to escaping stomach contents. ECR is completely autonomic, mediated by the vagus (CN X), and is a normal physiological means of the airway protecting itself when refluxed contents are detected.44 GERD and ECR are related in that when GERD is occurring, ECR will initiate to protect the subglottal portions of the airway.43-44 (Management of the dentition for patients with GERD will not be addressed in this article.) Focus for management will be of the reflex mechanism during dental procedures. ECR may result in a transitory cessation of breathing or forceful coughing, may involve vomitus expelled into the oral cavity during technique-sensitive dental procedures, and may result in the patient expressing retrosternal burning pain during the reflux episode.43-44

Management starts with taking a complete medical history to determine a patient’s likelihood of reflux occurrence, elevation of the dental chair into a beach position (30° to 90°), different types of suction tips available (Yankauer is the most useful), scheduling appointments during times when reflux possibility is at its lowest, and avoiding products with mint, coffee, and onions, as all 3 have been shown to relax the LES.48,49 Extensive and lengthy dental procedures may need to be broken up into smaller appointments. Requesting that patients see their medical care provider for reflux treatment is wise.

DISCUSSION

Malfunction of one or all of the protective mechanisms discussed may be the root cause of a patient’s declining oral health. Halitosis, oral lesions, enamel erosion, xerostomia, fungal infections, and sleep apnea are all health conditions that can result from disrupted reflexes. Protective airway reflexes further diminish as patients age due to decreased response from mechanical and chemical receptors located along the laryngeal tissue.50 It is important for general dentists and dental specialists to understand how dysfunction of a patient’s protective reflexes can affect oral health and how normal protective behavior can affect the surgical field during dental procedures. This is especially important for those clinicians who offer sedation concurrently with the dental procedure since sedation, in any form, will blunt these protective reflexes to varying degrees or modify their protective action. Additionally, in children, upper airway protective reflexes are more sensitive to insults that initiate response and the response is of greater severity than in adults with outcomes that lead to apnea and laryngospasm with resulting hypoxemia.51

As anesthesia modalities evolve and patient treatment expectations increase, many practitioners are utilizing combination anesthetic techniques that often involve intravenous drugs with narrow therapeutic indices such as propofol and fentanyl. Hypoventilation and apnea are more likely to occur with combination sedatives. Milgrom et al reported that 63% of patients had at least one apneic episode during dental treatments when sedated with intravenous benzodiazepines and narcotics.52 The prudent practitioner should bear in mind that increases in the administration of sedation medications will cause decreased innate airway protection.

CONCLUSION

Seven intrinsic reflexes that serve as defensive responses for protecting the body are of significant interest to dental professionals. They are the gag reflex, cough reflex, Bronchospastic Reflex, Laryngospastic Reflex, swallow reflex, vomit reflex, and ECR. These reflexes may disrupt dental treatment or even be responsible for patients avoiding dental care. All dental professionals should understand the techniques for prevention and management of these protective reflexes. Also, it should be understood that sedation of patients in any form will blunt these reflexes and affect their protective action.

References

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POST EXAMINATION QUESTIONS

1. Which reflex is activated when an unwelcome object touches the soft palate, posterior one third of the tongue, tonsillar, and surrounding tissues, or oropharynx?
   a. Cough reflex.
   b. Gag reflex.
   c. Bronchospastic reflex.
   d. None of the above.

2. The protective purpose of the cough reflex is to halt and reverse inhalation of aerosol, particulate, or gaseous irritants when the tracheal, laryngeal, or bronchial mucosa is stimulated.
   a. True.
   b. False.

3. General anesthesia (GA) with intubation in a patient with a troublesome cough can lead to bronchospasm. In most cases, GA with intubation in a patient with a troublesome cough can be a relative contraindication in the outpatient setting.
   a. The first statement is true, the second is false.
   b. The first statement is false, the second is true.
   c. Both statements are true.
   d. Both statements are false.

4. Lidocaine blocks the vagus pathway. Nebulized lidocaine is not an effective mechanism to stop the Bronchospastic Reflex.
   a. The first statement is true, the second is false.
   b. The first statement is false, the second is true.
   c. Both statements are true.
   d. Both statements are false.

5. A bronchospasm in response to anesthesia equipment irritation is essentially an asthma attack.
   a. True.
   b. False.

6. When all other protective airway mechanisms are diminished, which reflex replaces these diminished reflexes as the primary protector?
   a. Gag reflex.
   b. Laryngospastic reflex.
   c. Vomit reflex.
   d. Esophagoglottal Closure Reflex.

7. Which of the following is also known as the swallow reflex?
   a. Palatal Reflex.
   b. Deglutition Reflex.
   c. Both a and b.
   d. None of the above.
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8. In most cases, the vomit reflex closely follows the gag reflex. Managing vomiting means limiting gagging.
   a. The first statement is true, the second is false.
   b. The first statement is false, the second is true.
   c. Both statements are true.
   d. Both statements are false.

9. Which reflex is completely autonomic and is a normal physiological means of the airway protecting itself when refluxed stomach contents are detected?
   a. Vomit reflex.
   b. Esophagoglottal Closure Reflex.
   c. Bronchospastic Reflex.
   d. Cough reflex.

10. Milgrom et al reported that _____ of patients had at least one apneic episode during dental treatments when sedated with intravenous benzodiazepines and narcotics.
    a. 25%.
    b. 52%.
    c. 63%.
    d. 76%.
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