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REVIEW ARTICLE

"Hidden in Plain Sight": A Descriptive Review of Laryngeal Vestibule Closure

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Abstract

A major emphasis in the evaluation of swallowing is to identify physiological abnormalities in swallowing that contribute to or explain unsafe swallowing (i.e., ingested material enters the trachea; post-swallow residue in the pharynx). Impairments in laryngeal vestibule closure are widely recognized as one of the major causes of unsafe swallowing, as it is the primary mechanism and first line of defense for preventing material from penetrating the airway during swallowing. However, this complex mechanism is often overlooked and understudied in swallowing research and dysphagia management. The purpose of this review is to promote a better understanding of the mechanism of laryngeal vestibule closure. We discuss where gaps in research exist and propose future directions for incorporating laryngeal vestibule closure as a primary outcome measure in swallowing research. Additionally, we propose that an increased knowledge of the mechanism of laryngeal vestibule closure will increase diagnostic accuracy and optimize dysphagia management for patients with dysphagia.

Keywords Deglutition · Laryngeal vestibule closure · Swallowing · Rehabilitation · Dysphagia

Introduction

The mechanism of swallowing involves reconfiguring the oropharynx from a respiratory tract to a swallowing (alimentary) pathway for a period of less than one second, an event that occurs over 600 times daily [1, 2]. This is a highly complex act requiring sensorimotor integration and coordination with other physiologic functions (i.e., respiration, mastication), as well as rapid and precise coordination of more than 25 muscle pairs and six cranial nerves [3, 4]. The goal of swallowing is to complete this process safely and efficiently in order to maintain adequate nutrition, hydration, and quality of life.

Safe swallowing involves adequately protecting the airway during the swallow to prevent materials, such as food or liquid, from entering the trachea or lungs (i.e., aspiration) [5]. Aspiration is a major concern for individuals with dysphagia (swallowing impairment), especially in neurologic and neurodegenerative diseases, where respiratory infections are a leading cause of death [6]. In fact, the odds for developing pneumonia is 5.6–8.4 times greater for patients with observed aspiration on videofluoroscopy [7, 8]. Patients with poor overall health/dental status and/or are dependent for feeding are especially at risk [9].

Management of dysphagia is a top priority because dysphagia can contribute to reduced quality of life, multiple medical complications (i.e., dehydration, malnutrition, and pneumonia), prolonged hospital admissions, and significant increases in health care costs [10, 11]. Therefore, a major emphasis in the evaluation of dysphagia is to identify physiological abnormalities in swallowing that contribute to or explain unsafe swallowing (i.e., ingested material enters the trachea; post-swallow residue in the pharynx).

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In healthy individuals, airway protection during swallowing involves the closure of the larynx at four levels including adduction of the true vocal folds, approximation of the false vocal folds, epiglottic inversion, and anterior movement of the arytenoids to approximate the base of the inverted epiglottis [12–18]. Although arytenoid adduction is typically initiated first in this sequence, the timing and order of these events can vary depending on factors such as bolus size, volume, and viscosity [12, 16–19]. Despite considerable overlap in the timing of these events, in videofluoroscopic images, the closure of the laryngeal vestibule space has been described as occurring by a compression from "bottom to top." This occurs first at the supraglottic followed by the subepiglottic space in a peristaltic-like motion that can clear the vestibule or squeeze out bolus material that has penetrated the area to avoid aspiration below the vocal folds [12, 20]. However, preventing material from penetrating the airway in the first place is the primary goal in airway protection during swallowing. This is accomplished by arytenoid approximation and epiglottic inversion and is the primary mechanism of **laryngeal vestibule closure (LVC).** LVC is the first line of defense for preventing material from penetrating the airway during swallowing and is distinct from true and false vocal-fold closure, which serves as a secondary defense. The true and false vocal folds prevent aspiration by restricting entry of material into the lower airways and contribute to the forceful ejection of material that has already penetrated the laryngeal vestibule [5, 21]. The anterior border of the laryngeal vestibule is composed of the posterior aspect of the epiglottis. The lateral walls are composed of the thyroid cartilage and aryepiglottic folds, and lastly, the inferior border is the thyroarytenoid muscle [12]. The videofluoroscopic swallowing (VFS) study is the only option for visualizing LVC during swallowing [22]. In videofluoroscopic images, complete LVC is characterized by no airspace in the vestibule given complete contact of the arytenoids to the base of the epiglottis and full epiglottic inversion over the base of the arytenoids [5] (Fig. 1).

This complex mechanism relies on precise movements of several key structures including the tongue, pharynx, larynx, and hyoid bone to achieve complete closure of the laryngeal vestibule. These include (1) arytenoid adduction and arytenoid approximation to the base of the epiglottis and (2) epiglottic inversion which is achieved by (3) tongue base retraction, (4) hyolaryngeal excursion, and (5) pharyngeal constriction. In order to fully understand this mechanism, it is necessary to understand which component(s) are *critical* for achieving complete closure. Currently, it is unclear if each component is equally valuable, or if one holds more importance for the function to happen. The contribution of multiple structures increases the complexity of LVC, yet given the importance of this mechanism, they may serve as multiple fail-safes, wherein if one structure is impaired or perturbed, other structures might compensate to ensure adequate airway protection during swallowing [23].

In dysphagia research, there is an overwhelming appreciation for consequences of aspiration, yet the physiologic mechanisms responsible for aspiration, namely LVC, are not well understood. While the impact of bolus properties (i.e., consistency, taste, volume) and therapeutic maneuvers on swallowing physiology have been well documented in the literature, few report how these influence LVC in particular. The purpose of this paper is to promote a better understanding of the mechanism of LVC that is needed for better dysphagia treatment. We will first provide a thorough description of the components of normal LVC, followed by abnormal LVC and behavioral modifications. Secondly, we will discuss where gaps in research exist and propose future directions for incorporating LVC as a primary outcome measure in swallowing research.

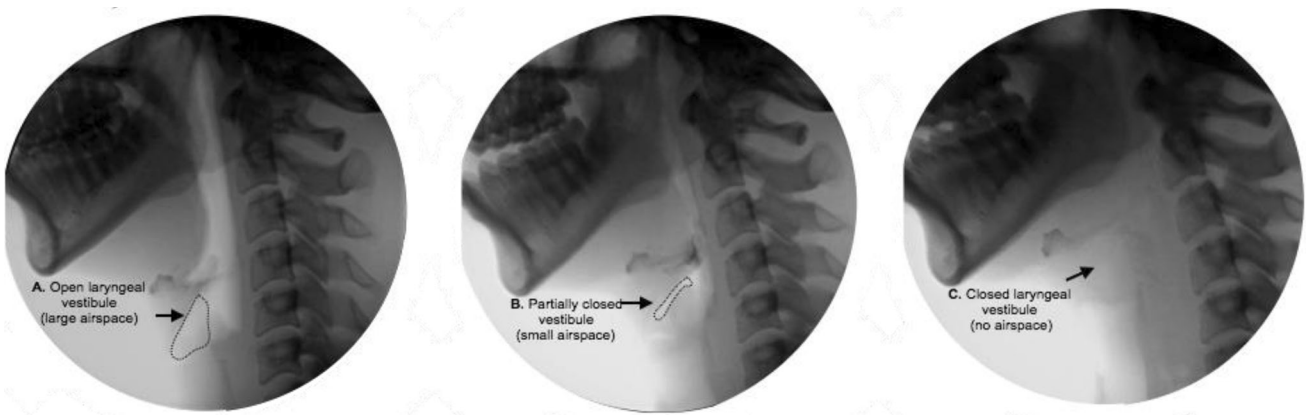

Fig. 1 Videofluoroscopic images of laryngeal vestibule closure. **a** Open laryngeal vestibule at rest; **b** partially closed laryngeal vestibule during swallow; **c** closed laryngeal vestibule at the height of the swallow. Dotted line represents airspace in laryngeal vestibule

Components of Laryngeal Vestibule Closure

Arytenoid Adduction and Anterior Movement

Anterior tilting of the arytenoid cartilages has been suggested as one of the most important contributors to closure of the laryngeal vestibule [24]. The aryepiglottic and lateral cricoarytenoid muscles move the arytenoids anteriorly, accounting for one-half to one-third of closure [5]. Despite attachment to the posterior lamina of the cricoid cartilage, arytenoid movement is not a biomechanical effect of laryngeal elevation and is under separate, active neuromuscular control [5, 19].

Arytenoid adduction and arytenoid anterior movement, when combined with epiglottic inversion, allows for contraction of the aryepiglottic muscle which leads to adduction of the aryepiglottic folds in a posterodorsal direction [12]. This further aids in airway protection by forming the lateral walls of the laryngeal vestibule and tightens the laryngeal inlet [25]. The aryepiglottic folds are mucous membranes encompassing ligamentous and muscular fibers that attach anteriorly to the lateral edges of the epiglottis and wrap around posteriorly to attach to the arytenoids. They play an important role in airway protection by directing the swallowed bolus around the laryngeal inlet toward the upper esophagus. These lateral folds act as "walls" allowing the bolus to pass between the aryepiglottic folds and the lateral pharyngeal wall. Contained within the aryepiglottic folds are the corniculate and cuneiform cartilages, which add stiffness to the folds for further protection from airway invasion [21, 26, 27]. While approximation of the arytenoids to the base of the epiglottis is easily visualized during swallowing in videofluoroscopic images, we are unable to visualize the medial movement of the arytenoids in the lateral plane.

Epiglottic Inversion

During normal swallowing, epiglottic inversion contributes to LVC by contacting the arytenoids, covering the laryngeal inlet, and diverting the bolus laterally away from the laryngeal vestibule toward the upper esophageal sphincter (bolus typically splits into two pieces to flow down and around the airway) [26]. Inversion of the epiglottis is primarily a passive movement that has been described as a two-step procedure, [1] moving from an upright position to a horizontal plane, then [2] moving from the horizontal position to its fully inverted position [28]. Although epiglottic inversion also occurs with a very small bolus (i.e., saliva swallow), a larger bolus can provide additional weight upon the epiglottis to promote inversion; however, there are no data to confirm this phenomenon. Studies have also reported size and shape differences of the epiglottis related to body mass index (BMI) and severe obstructive sleep apnea (OSA); however, there is no evidence to support these differences impact swallowing function and/or airway protection [29, 30]. Epiglottic elevation, tongue base retraction, and pharyngeal constriction primarily facilitate epiglottic inversion in order to achieve LVC. Secondarily, the aryepiglottic muscle is a paired intrinsic laryngeal muscle that contributes to approximating the epiglottis to the arytenoid. Epiglottic elevation occurs because it is part of the larynx, which is elevating as a unit. ***Laryngeal elevation*** is accomplished by contraction of the longitudinal pharyngeal muscles, which include the salpingopharyngeus, palatopharyngeus, and stylopharyngeus as well as the thyrohyoid muscle. When laryngeal elevation occurs, the epiglottis is closer to the base of tongue, which will impinge upon the epiglottis to move it to a horizontal position [31] (Fig. 2). ***Tongue Base Retraction***, or posterior propulsion of the tongue, contributes to closure of the laryngeal vestibule by facilitating posterior and downward movement of the epiglottis to a horizontal position (Fig. 2). This movement is achieved by dual contraction of the styloglossus and hyoglossus muscles, moving the tongue posteriorly to meet the pharyngeal wall. Posterior movement enables the passive epiglottis to invert horizontally [31]. The final movement of the epiglottis includes movement from the horizontal position to its fully inverted position with the epiglottis tip contacting the arytenoid base, which is accomplished by ***pharyngeal constriction***. When the longitudinal pharyngeal muscles contract, in addition to laryngeal elevation, they shorten and elevate the pharynx. The pharyngeal constrictors reduce the lumen of the pharynx. The sequential contraction of the pharyngeal constrictor muscles creates a "stripping wave" that moves inferiorly to the pharyngeal–esophageal sphincter [32]. Contraction of the pharyngeal constrictors provides compression on the tip of the epiglottis to further aid its inversion [31, 33].

The role of ***hyoid elevation***, in isolation, on LVC has been debated. Based on visual observations from videofluoroscopic studies, early reports attributed epiglottic inversion to anterior hyoid movement [5, 12, 34], laryngeal elevation, [5, 35], and tongue base retraction [5, 35]. However, there was disagreement regarding which swallowing events were responsible for each step of epiglottic inversion noted above. More recently, studies have shown that while hyoid movement may correlate with epiglottic movement, it is not necessary for inversion. For example, placement of surface electrical stimulation on the supra- and infrahyoid muscles results in significant hyolaryngeal descent and restricted range of motion (peak elevation) during swallowing [36]. When providing continuous surface electrical stimulation during swallowing tasks, despite

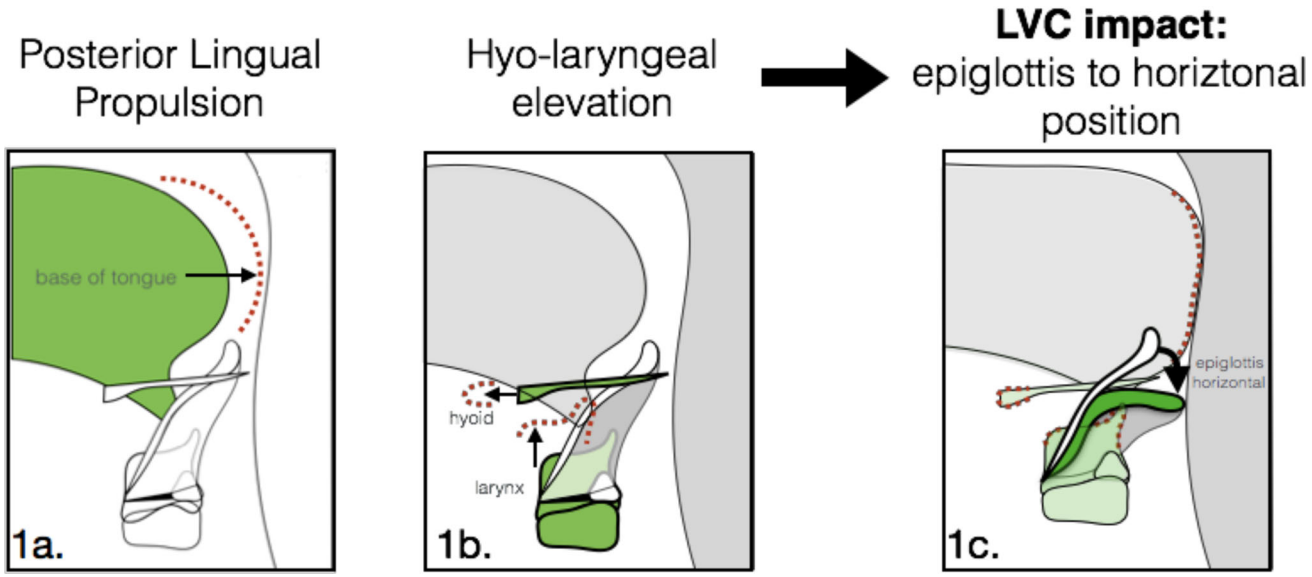

Fig. 2 Posterior lingual propulsion and hyolaryngeal elevation facilities movement of the epiglottis to horizontal position

reduced hyoid excursion, LVC timing was unchanged [36, 37]. In a computational analysis of swallowing mechanics, Pearson, Taylor, Blair, and Martin-Harris [31] showed that the two-step movement of the epiglottis is primarily achieved by laryngeal elevation and tongue base retraction, not hyoid movement. They suggested that although hyoid excursion might correlate with epiglottic movement, hyoid movement alone does not generate either stage of epiglottic inversion when laryngeal elevation or tongue base retraction is impaired [31].

Evaluation of Laryngeal Vestibule Closure

LVC can only be visualized using videofluoroscopy in order to judge complete contact of the arytenoid to the base of the epiglottis, and full epiglottic inversion over the base of the arytenoids. Evaluation of LVC includes judgments made about the [1] **range of motion** and [2] **timing** of closure. The range of motion involves judging the amount of closure by visualizing the amount of airspace obliterated within the vestibule at the height of the swallow. Another way to evaluate LVC is to quantify the timing, such as the duration of closure and the duration to closure [38].

The penetration–aspiration scale (PAS) is a tool used to describe the depth of airway invasion and patient response to penetration or aspiration [39]. The PAS is not useful for evaluating LVC because the PAS does not determine the physiologic impairment that causes airway invasion. Furthermore, physiologic impairments that cause airway invasion before or after the swallow (i.e., delayed swallow resulting in aspiration before the swallow, or UES dysfunction resulting in aspiration after the swallow was complete) would be distinct from normal or disordered LVC, an event that should only be judged during the swallow.

Range of Motion

Amount of LVC

The most commonly used measure for judging the amount of LVC is the Modified Barium Swallow Impairment Profile (MBSImP) [40]. The MBSImP is a clinical tool used to identify and distinguish the type and severity of physiologic swallowing impairments to standardize assessment. The MBSImP includes 17 different swallowing impairments. LVC is scored based on the presence or absence of contrast material or air in the laryngeal inlet, judged at the height of the swallow. Complete LVC is defined as no air or contrast in the laryngeal vestibule, incomplete closure is characterized by a narrow column of air or contrast in the laryngeal vestibule, and no closure is characterized by a wide column of air or contrast in the laryngeal vestibule [40].

The MBSImP has great utility for standardized quantification of swallowing impairment and is especially useful for clinicians reporting functional outcomes for patients. However, quantification of LVC based solely on the amount of closure using a 3-part categorical scale (complete, incomplete, or none) using MBSImP guidelines can be limiting and lacks specificity when trying to quantify changes in LVC. This is because there are numerous airway protection patterns considered "incomplete" when considering the relationship between the epiglottis and arytenoids. Additionally, judging only the amount of

laryngeal closure discounts timing of LVC, such as how long the laryngeal vestibule stays closed, and how quickly the laryngeal vestibule closes, which are important for maintaining airway safety [41].

Timing

Adequate timing of laryngeal closure is essential for maintaining airway protection during the swallow [41]. *Duration* of laryngeal vestibule closure is a measure of how long the laryngeal vestibule maintains complete closure. It is measured (usually in milliseconds) by calculating the time between the first frame of laryngeal vestibule closure and the first frame of laryngeal vestibule re-opening. *Duration to* laryngeal vestibule closure is a measure of how quickly the laryngeal vestibule closes once the swallow is initiated, and is measured by calculating the time between the first frame of hyoid burst and the first frame of laryngeal vestibule closure.

Incorporating timing measures into the evaluation of LVC allows for the inclusion of objective outcome measures that can be compared to published, normative data. Additionally, timing of LVC is measured on a continuous scale (milliseconds) and has the potential to capture subtle yet significant mechanistic changes in response to treatment or sensory input. However, there are limitations preventing the frequent use of timing of swallowing events in standard clinical practice. Timing analysis requires access to frame-by-frame review of videofluoroscopic images and some clinicians may not have the ability to record videofluoroscopy for secondary review due to lack of equipment or limited access to archived materials. However, given the rapid nature of swallowing, reviewing swallowing studies in slow motion is often essential; thus, poor access can be a significant barrier to best practice. We believe that access to and frequent use of appropriate imaging techniques for clinicians should be standard care. Underutilization of these techniques is a barrier to clinicians' ability to accurately diagnose and treat LVC [42]. Another limitation is that proper evaluation of swallowing events during videofluoroscopy requires a minimum temporal resolution of 30 frames per second. Poor access can include recording at reduced frame rates (i.e., 7 or 15 frames per second) which can be inadequate for capturing LVC timing [43]. Furthermore, timing calculations done with lower frame rates will be inaccurate if compared to normative values published in the literature that are based on frame rates of 30 frames per second.

Normal Variability

Duration of Laryngeal Vestibule Closure

A systematic review of temporal variability in swallowing found 14 publications that reported means and standard deviation (SD) or standard error of the mean (SEM) for the duration of LVC in healthy, normal swallows. Mean values for LVC duration ranged from 310 to 1070 ms with 95% CIs ranging from [0.26 to 0.36] to [0.97 to 1.17] respectively, reflecting the large variability for this measure [44]. A more recent study reported mean and standard deviation for duration of LVC in 1051 swallows for six different consistencies from healthy young and older adults [45]. This study explicitly defined complete LVC as full epiglottic inversion and complete contact of arytenoids to the base of the epiglottis and thus, duration of LVC as the time between the first frame of laryngeal closure and the first frame of laryngeal re-opening. Mean values for the duration of LVC ranged from 466 (SD 131) msec for a mixed consistency bolus to 603 (SD 272.5) msec for a thin liquid bolus [45] (Table 1).

Duration to Laryngeal Vestibule Closure

The aforementioned systematic review included an in-depth review of 46 publications which reported temporal swallowing data, yet none of these publications reported data on how quickly the laryngeal vestibule closes following hyoid burst (duration to LVC) [44]. There may be

Table 1 Normative data for timing of LVC

Timing measure	Ultra-thin liquid	Barium ice chips	Room temp pudding	Frozen pudding	Ultra-thin + chocolate chips
Duration of LVC[a]	603.7 (± 272.53)	498.43 (± 137.28)	516.02 (± 123.23)	508.86 (± 123.32)	466.52 (± 131.45)
Duration to LVC[b]	95.27 (± 53.7)	118.5 (± 40.5)	107.42 (± 37.4)	115.566 (± 45.6)	91.806 (± 51.5)

Means and standard deviations of each consistency for each LVC timing measure based on a sample of 1051 swallows from healthy young and older adults [45]

[a]Duration of laryngeal vestibule closure (dLVC): time between first frame of laryngeal vestibule closure and the first frame of laryngeal vestibule re-opening

[b]Duration to laryngeal vestibule closure (dtLVC): time between first frame of hyoid burst and first frame of laryngeal vestibule closure

only two studies to date that have reported data for the duration to LVC in healthy, adult swallows [38, 45]. These studies operationally defined duration to LVC as the time between the first frame of hyoid burst and the first frame of LVC, which again included full epiglottic inversion and complete arytenoid contact as a prerequisite for complete LVC. Guedes, Azola, Macrae, Sunday, Mejia, Vose, and Humbert [38] reported means and 95% confidence intervals for the duration to LVC for 5 ml and 10 ml thin liquid water and barium natural swallows in 69 healthy adult participants. Mean values for the duration to LVC ranged from 160 to 210 ms with 95% CIs ranging from [140, 170] to [180, 230], respectively. Humbert, Sunday, Karagiorgos, Vose, Gould, Greene, Tolar, and Rivet [45] also reported mean values for the duration to LVC ranging from 95 (SD 53.7) msec for thin liquid to 115.566 (SD 45.6) msec for frozen pudding (Table 1).

Sources of Normal Variability

Patient and Stimulus Factors

It has been established that swallowing physiology can be influenced by several types of patient and stimulus factors. Tactile, chemical, and thermal sensory stimuli such as changes in bolus volume, consistency, and temperature have been shown to modify the timing of swallowing [46–48]. Other factors, such as mode of delivery and age can also influence swallowing timing and kinematics, thus increasing variability in normal swallowing [44, 49, 50]. However, many of these studies reported changes in swallow initiation (or swallowing response time), pharyngeal transit time, or duration of esophageal opening, yet few reported changes in the timing of laryngeal closure, thus limiting our understanding of how external factors influence the mechanism of LVC [16, 51–54]. Studies that have reported factors that influence LVC timing have shown that duration of LVC increases with increasing bolus volume and increases with the time the bolus remains in the pharynx, yet none of these studies reported a change in duration to LVC [52, 55–60].

Normal variability in patterns of laryngeal closure also varies according to the mode of delivery. In healthy individuals using sequential straw swallows, some exhibit a pattern of maintaining LVC throughout sequential swallows, while others demonstrate hyolaryngeal descent and opening of the laryngeal vestibule between sequential swallows, thus alternating a closed–open position while coordinating respiration with sequential swallows [61, 62]. While these data support the notion that LVC is highly responsive to sensory input during swallowing, further studies are needed to explore additional changes in stimulus factors as well as patient factors, which may influence the timing of LVC, especially duration to LVC where little information has been reported.

Volitional Manipulation of LVC

While LVC can be modified by changes in sensory inputs, it can also be modified volitionally. For example, Hind, Nicosia, Roecker, Carnes, and Robbins [63] showed that duration of LVC was significantly longer for effortful swallows compared to non-effortful swallows. Also, alterations in head positioning such as chin down swallowing result in longer duration of LVC due to closure occurring earlier in the swallow and re-opening that occurs later [64, 65]. Humbert and colleagues demonstrated that LVC could be directly manipulated when healthy adults and individuals with dysphagia due to stroke are instructed to volitionally prolong closure [64, 66]. Furthermore, utilizing videofluoroscopy to provide kinematic visual biofeedback improves accuracy in training volitional prolongation of LVC [67]. Although participants were not explicitly instructed to close their airway faster, healthy adults had a statistically significant decrease in duration to LVC when learning the volitional laryngeal vestibule (vLVC) swallowing maneuver [38]. These data are significant because by instructing participants to prolong the duration of LVC, they were able to demonstrate volitional control over a predominantly brainstem-mediated swallowing event given the control mechanisms of LVC are considered reflexive in nature when it occurs in the context of airway protection during swallowing [69–71]. However, by implementing direct volitional control, manipulating this event has enormous rehabilitation potential [72]. These are the first, and only, studies that report direct, volitional manipulation of the laryngeal vestibule.

Abnormal Laryngeal Vestibule Closure

Impairments in laryngeal closure are recognized as one of the major causes of aspiration [6]. If the duration of laryngeal closure is absent, too short, or if closure is delayed, this can lead to the unwanted entrance of food or liquid into the airway [73, 74]. Several studies have highlighted the relationship between impairments of LVC and aspiration. Park, Kim, Ko, and McCullough [75] showed reduced duration and delayed laryngeal closure are significantly associated with aspiration that occurs during the swallow in post-stroke patients with dysphagia. Similarly, decreased duration of LVC was a primary impairment for predicting aspiration in patients following stroke [41]. In a study of neurological older patients, duration to LVC was double that of healthy subjects resulting in aspiration during the swallow [76, 77]. In a cohort of patients studied

with head and neck cancer who received radiation focused on the geniohyoid muscle, it was noted that absent or reduced duration of LVC or prolonged duration to LVC was also significantly associated with increased aspiration [39, 78]. Cabib, Ortega, Kumru, Palomeras, Vilardell, Alvarez-Berdugo, Muriana, Rofes, Terre, Mearin, and Clave [6] summarize these findings by describing prolonged time to laryngeal closure as the key abnormality in unsafe swallowing in neurological patients. However, these studies lack specificity in regards to which component of LVC is delayed, absent, or impaired (i.e., epiglottic inversion due to impaired tongue, pharynx, or laryngeal elevation? Arytenoid movement?). This is important because therapy targets for tongue base retraction might differ greatly than those that target laryngeal elevation or pharyngeal constriction. Identifying the specific impaired component of LVC could lead to more effective treatment to improve swallowing airway protection.

Future Research

Despite widespread appreciation in the literature for the consequences associated with impaired LVC, this complex mechanism is in plain sight in videofluoroscopic evaluations but is overlooked and understudied in swallowing research and dysphagia management. While many studies report the influence of various therapies, maneuvers, stimuli, and patient factors swallowing physiology, many omit laryngeal vestibule closure as a primary outcome measure, thus limiting our understanding of how external factors influence this primary airway protection mechanism. Furthermore, our understanding of which components of LVC are most salient, or critical, is also limited. However, much can be learned about mechanisms when directly manipulating or perturbing the components of laryngeal closure. We already know that if you perturb the hyoid bone during electrical stimulation tasks, a healthy individual can compensate (i.e., laryngeal closure remains unchanged) [37]. By examining and manipulating the swallowing events that contribute to laryngeal closure, we will better understand which components are necessary versus complementary. Currently, many studies already report impairments in these events; however, few report the consequences to LVC if present. Thus, more perturbation studies that examine laryngeal motor control are warranted.

Another limitation is that considerable attention has been paid to structures and muscle groups that may not directly contribute to LVC (i.e., hyoid excursion, submental muscles) despite evidence to the contrary [31, 36, 79]. This might be explained by the ease of submental and hyoid bone measurement. While movements of the larynx and pharynx are difficult to visualize and measure using videofluoroscopy, the hyoid bone is prominent and that submental muscles are larger and accessible.

Currently, much of what we know about the mechanism of laryngeal closure is based on subjective videofluoroscopic observations or binary measures of laryngeal closure (i.e., complete vs. incomplete). Additionally, many studies report bolus outcomes (penetration, aspiration) as a measure of airway protection, neglecting the physiology responsible for airway invasion and disregarding when airway invasion occurs (i.e., before, during, or after the swallow). However, as Kendall [80] reports, incorporating quantitative timing measures improves diagnostic accuracy and sensitivity when evaluating the accuracy of LVC. LVC timing measures might detect subtle changes that may not be otherwise apparent in crude, subjective measures [48, 80]. Increased knowledge of the mechanism of LVC will optimize dysphagia management by promoting the development of successful rehabilitation strategies that directly target LVC. This will lead to increased diagnostic accuracy and well-targeted treatment plans to rehabilitate particular muscle groups known to directly target LVC and thus may improve swallowing safety in dysphagic patients. Ultimately, improving LVC with swallowing rehabilitation may help to decrease morbidity and mortality in patients with poor airway protection.

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Compliance with Ethical Standards

Conflict of interest Alicia K. Vose, MA CCC-SLP, declares that she has no conflict of interest. Ianessa Humbert, PhD CCC-SLP, declares that she has no conflict of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants included in the study.

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