The Role of the Supraglottic Area in Voice Production

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Speech production is essentially the modification of an air stream by a number of different structures along the vocal tract. Traditionally, the airstream produces vibration of the true vocal folds that is modified in amplitude and frequency by characteristics such as vocal fold length, thickness, depth and tension. Then, as a result of vocal fold vibration, both laminar and turbulent air flow through the pharynx and oral cavities, and occasionally through the nasal cavities. The particular sound produced is specified by the shape of the pharyngeal walls, as well as tongue, lip, soft palate and jaw positions. However, it is becoming increasingly clear that a further level of modification of the airstream important to speech production is provided by the supraglottic structures, those structures that lie just above the true vocal folds [1]. Some of these structures have long been known to play an important role in swallowing function and cough. This paper will provide a review of the supraglottic area in voice production, including definitions, anatomy, and their roles in speech production in normal and abnormal voice production.

Definitions

When one considers the term supraglottic, what immediately comes to mind is the anatomical categorization of sites of head and neck cancers. The supraglottic area includes those structures that lie above the true vocal folds (TVF) and below the tongue base. The anatomical structures present in this area are important to speech production lie posterior to the epiglottis. They include: the ventricle; the false vocal folds (FVF); the epiglottis; the arytenoids; the laryngeal aspects of the aryepiglottic folds; and the vestibule. This area is also known as the epilarynx [2,3]. Within this rather small area (approximately 3 cm in length), the air column produced by the vibrating vocal folds can be modified by 2 types of muscular activity; one in the medial-lateral direction and one in the antero-posterior direction. Supraglottic activity in the medial-lateral direction is provided by the compression of ventricular, vestibular or false vocal folds. During phonation, the average gap separating the two false vocal folds for males is 0.613 cm and for females, 0.439 cm [4]. Supraglottic activity in the antero-posterior direction is provided by the compression of the petiole of the epiglottis towards the arytenoid cartilages.

Anatomy

The false vocal folds lie above the true vocal folds, and the space between them is known as the ventricle. The average distance between the two types of folds is 0.53 cm for males and, 0.375 cm for females [4]. Like the true vocal folds, the false vocal folds are organized into 3 layers, the epithelium, the lamina propria and muscle tissue. The epithelium is made up of double-layered ciliated columnar cells, except the uppermost or most superior part, which is covered by stratified squamous cells [1]. Mucous gland cells are also found in the epithelium with excretory ducts that open into the space between the true and false vocal folds [5], producing mucous to lubricate the true vocal folds. Lymph follicles are also present as are the secretion of other proteins and immunoglobulins, suggesting that the FVF are important in the protection of the airway from infectious agents, and thus form part of the laryngeal mucosal immune system [5,6]. The lamina propria is an extracellular matrix of fibrous and interstitial proteins such as collagen and elastin, as well as adipose tissue [1]. The greater amount of adipose tissue compared to that in the true vocal folds results in higher viscosity and lower stiffness [4,7,8]. Chan et al. [1] reported that the male FVF were twice as stiff as the female at a specific tensile strength, likely due to a higher proportion of glandular tissue in the female FVF. The descriptions of the muscular layer seem variable. What seems to be agreed upon is that the thyroarytenoid muscle lies laterally to the false vocal folds, and may be responsible for creating a passive mechanism for false vocal fold adduction. Some muscle fibers within the false folds are oriented in the postero- and anterolateral directions, and are likely the muscles involved in more actively adducting the false vocal folds [9]. Other fibers are oriented in the anteromedial direction, and seem to be isolated fiber bundles scattered within the ventricular fold [9] that may be involved in the downward movement. This may correspond to the ventricularis muscle reported by Kobly et al. [10]. It is clear that the false vocal folds vibrate, either periodically or non-periodically, in phase or out of phase with the true vocal folds, and with or without medial contact [11]. However, because they are less stiff than the true vocal folds, they do not vibrate as well [4]. Innervation of these muscles is thought to be from the external branch of the superior laryngeal nerve [12], as previous work has not demonstrated any lack of movement with unilateral vocal fold paresis. However, more current research does demonstrate lack of movement in some patients with vocal fold paresis [13-15], suggesting some innervation by the recurrent laryngeal nerve. Sensory innervation appears to be from the middle division of the internal branch of the superior laryngeal nerve [16,12].

The petiole is a short, thin, inferior process on the laryngeal side of the epiglottis that attaches to the thyroid cartilage by the thyroepiglottic ligament [17]. The epiglottis itself is an elastic cartilage rather than a hyaline cartilage, and the collagen fibers in the petiole are arranged in a vertical orientation [18]. The cartilage is covered by epithelium of ciliated columnar cells also with mucous gland cells. Muscle fibers run from the anterolateral surface of the arytenoid cartilage or from the thyroarytenoid muscle to the petiole [18]. Innervation is from the superior division of the internal branch [16] as well as the external branch of the superior laryngeal nerve [19].

Anterior to the epiglottis is the periepiglottic or pre-epiglottic space, which is also considered anatomically as supraglottic. It is bounded anteriorly by the thyrohyoid membrane, thyrohyoid ligaments and the superior parts of the lamina of the thyroid cartilage, superiorly by the hyoepiglottic ligament and membrane, posteriorly by the lingual...
surface of the epiglottis; inferiorly by the thyroepiglottic ligament, and laterally it continues into the adjacent laryngeal tissue [9]. However, as it is not directly in the path of airflow from the lungs, it will not be discussed further in this review.

**Role of Supraglottic Structures on Speech Production Based on Modeling and Cadaveric Studies**

The first area of interest is the study of the influence of the epilarynx on vocal tract acoustics. Using mathematical models, the size of the aperture of the epilarynx is hypothesized to play a role in both the intonation of the vocal tract and the enhancement of certain formant frequencies [2,20,21]. Narrowing of the aperture is thought to result in changes in the location of the formant frequencies, and depending on their interaction with fundamental frequency and harmonics, can result in enhancement of energy and thus a more resonant voice [2]. This narrowing is not related to approximating the FVF, but rather occurs with the FVF retracted laterally and flattened [2]. This is likely to occur by lowering the larynx or moving the tongue forward [2]. However, there is no data from humans to demonstrate this effect. Dollinger et al. [22] reported that this narrowing had little effect on the SPL output, but did lower the subglottal pressure needed to achieve a particular SPL. This was suggested by Titze [2] in a different way. He reported that narrowing the epiglottis may result in lower phonation threshold pressures, which is the minimum subglottic pressure needed to start the vocal folds vibrating [23]. The narrowed epilarynx causes the oscillating supraglottic pressures to be in phase with vocal fold velocity, which lowers that phonation threshold [24].

Several approaches have been used to investigate the influence of approximation or lack of approximation of the FVF. In general, in vitro methods such as mathematical models or animal cadaveric preparations [7,25-31] have been used to determine the effects on phonation. Those studies using mathematical models can examine the effects of fully adducted false vocal folds [7,26,27,29], or simply the presence of false vocal fold tissue [7,25]. The cadaveric studies can examine the effects of the absence of FVF [26,27].

The modeling studies can be summarized as follows. If the FVF are present, but not fully medially adducted or compressed: 1) laryngeal flow resistance is reduced at the glottis because of increased flow [4, 25,28,29]; 2) additional dipole sources of sound are added [28]; 3) oscillation of the vocal folds is favored [7]; 4) the glottal jet is straightened or stabilized (more laminar flow) reducing energy dissipation [29,30] and 5) greater amplitude of vibration of the TVF [25]. If the FVF are medially compressed, TVF vibration may be impeded or damped because of transglaryngeal pressure drop [7].

One of the animal cadaveric studies reported that the loss of both supraglottic structures (FVF and epiglottis) resulted in larger decreases in laryngeal flow resistance due to higher flows and lower pressures, slightly decreased fundamental frequency and a loss of about 3 to 5 dB in sound pressure level [26].

Another of the animal cadaveric studies explored the role of the epiglottis in speech production [27], by comparing measures from the epiglottis in a vertical position to a horizontal one. Vertical position of the epiglottis co-occurred with less false vocal fold coverage of the true vocal folds, resulting in decreased subglottal pressure and increased flow (decreased laryngeal resistance). Finnegan & Alipour [27] also reported that both the epiglottis and the false vocal fold act as resonators, and enhance the acoustic signal. Their absence resulted in increased low frequency noise in the acoustic signal.

**Role of Supraglottic Structures in Normal Voice Production**

The size of the epilarynx and its effect on phonation has primarily been described in the singing literature. Yanagisawa et al. [32] described videovideodoscopic observations of singers producing different types of vocal qualities. The three loudest qualities, twang, belting and opera, especially at higher pitches [21], all demonstrated narrowing of the epilarynx primarily in the anterior-posterior direction (i.e. compression of petiole towards arytenoids). This observation was first reported by Garcia in 1855. Yanagisawa et al. [32] further attributed the presence of energy around the bandwidth of 3 kHz (or the singer’s formant) to this narrowed epilaryngeal configuration. Sundberg [33] also reported that this singer’s formant occurred as a result of the clustering the third, fourth and fifth formants, when the cross-sectional area of the pharynx is 6 times greater than the cross-sectional area of the epilarynx. Titze [2] reported that the narrowing of the epilarynx also helps to match the impedance between the glottis and the vocal tract, resulting in the lowering of the subglottic pressure needed to keep vocal fold oscillation going. The techniques of resonant voice therapy are thought to result in the epilaryngeal narrowing [2].

Based on the cadaveric and modeling work, the presence of FVF promotes more efficient airflow, but the full compression of the FVF towards midline is not associated with efficient voicing. There are degrees of compression by the FVF, from partial covering of the view of the true vocal folds to complete covering. Given that compression results in non-efficient voicing, one would not expect supraglottic activity in an individual with no voice complaints and normal vocal quality. In this vein, at least 4 groups of researchers have assessed still images from endoscopic examinations to determine if FVF or AP compression occurs in the larynx of individuals with no voice complaints and normal vocal quality. Their findings will be detailed subsequently. Should supraglottic activity be found to be present, then one needs to consider the degree of compression, from just slightly covering the TVF to completely covering them. If the degree of compression is only slight, then perhaps it is not enough to interfere with normal voice quality. Next, if supraglottic activity is found in persons with normal voice quality, one needs to consider whether a particular type of speech task or phonetic context corresponds to that activity. Two major types of activity can be present, “static” and “dynamic” [34]. “Static” compression implies that the degree of compression is constant throughout phonation. At rest, there might be no compression, but when phonation starts, the supraglottic structures move into position, and remain there until they return to their original location when the vocal folds are no longer vibrating. “Dynamic” compression implies that the degree of compression changes throughout a particular speech task, perhaps depending on the phonetic context. A subset of “dynamic” compression would be a slight compression occurring at the initiation of the speaking task. Also, the presence of supraglottic activity may differ depending on gender. Finally, one needs to determine if supraglottic activity is present only if a particular type of scope is used to record the examination. A recent study by Nospes et al. [35] found that less supraglottic compression was found during phonation recorded using flexible fiberoptic scope than the rigid 70 or 90 degree endoscope. If a particular type of scope is used to record the examination. A recent study by Nospes et al. [35] found that less supraglottic compression was found during phonation recorded using flexible fiberoptic scope than the rigid 70 or 90 degree endoscope. If supraglottic activity is found using the rigid endoscope, but not using the flexible endoscope, then we might be looking at an artifact.

The first studies of supraglottal activity were by Casper et al. [36] using a flexible fiberscope and Pemberton et al. [37] using both rigid and flexible endoscopes. Both reported that supraglottal activity could
be present in individuals with no voice complaints and normal vocal quality. Stager et al. [34] also examined the incidence of static and dynamic supraglottic activity in controls for a number of speaking tasks using a flexible fiberscope. Results suggested that the controls demonstrated both static FVF and static AP compression. Since static FVF compression was found in controls, the next step was to look at the degree of compression. Two different approaches were used to quantify the degree of FVF and AP compression (i.e. how much the FVF covered the TVF) for control subjects [24,38]. In both studies, a range of degrees of compression was found, but the FVF or AP compression did not often completely cover and obscure the view of the TVF.

The next question was whether any of the supraglottic activity was related to phonetic context. Stager et al. [30] reported task differences for FVF compression, but not for AP compression. As well as at the initiation of speech, dynamic FVF compression was found for speech samples that included glottal stops. Glottal stops are usually inserted between a word ending in a vowel sound and the next word when it also begins with a vowel sound. For example, “we eat” would have a glottal stop inserted between the two words, but “my dog” would not, even though both words consist of sounds that are all voiced. It is assumed that the insertion of the glottal stop adds to the clarity of how speech is perceived [39], and so has been termed clarity of laryngeal articulation. This appears to be a normal function of the FVF.

Stager et al. [40] also assessed gender differences for incidence of FVF and AP compression using still images from a flexible fiberscope examination, given that many of the voice disorders presumed to be associated with supraglottic activity are found primarily in females [41]. Results suggested that static FVF and AP compression were present at a significantly greater incidence in males than females. FVF activity associated with speech initiation was significantly more present in females than males, Nemetz et al. [42] also examined gender differences in FVF compression using still images from a rigid endoscope examination with controls. Specific anatomical points were selected on each image to determine the FVF configuration, and they were rated as showing a convex, concave or linear shape. Results suggested that the concave configuration was the most common. Based on the figures, both convex and linear configurations appear to be related to compression of the FVF, with convex being more compressed than linear. No differences between genders were reported for the incidence of concave configurations in the control subjects. Differences were found between genders for the linear configuration, such that males had a higher incidence, supporting the results from Stager et al. [40]. However, differences were found between genders for the convex configuration, such that females had a higher incidence, results that do not support the Stager et al. [40] findings.

In summary, supraglottic activity was present in individuals with no voice complaints, no structural abnormalities and normal voice quality, despite the fact that the modeling and cadaveric studies suggest that more efficient vocal production is based on no FVF or AP compression. It was also clear that the type of scope used for the recording of the images did not make a difference, as supraglottic activity was found using both types of scope. No studies, however, quantified of the degree of compression in the same individual from images recorded from each type of scope. However, the explanation for the presence of supraglottic activity in voice production in controls is unclear. One might argue that supraglottic compression may be a precursor to the development of voice disorders, such that it may need to be present for a certain length of time before voice quality is noticeably affected [43]. One might also argue that recovering from an injury or an illness to the larynx might involve the use of supraglottic activity to help compensate, and then when the injury or illness has resolved, its use has become habitual [44].

Before moving on to supraglottic activity in voice-disordered speakers, it should be pointed out that another use of the FVF by individuals with normal voice quality, no vocal complaints and healthy larynges is to produce musical tones. In other words, certain types of singing involve the vibration of the FVF, which is as critical to the sound being produced as the vibration of the TVF underneath. The list of these include: Asian throat singing [45-48], Mediterranean traditional polyphony [49], and rock singing [50]. The vibratory gestures can be periodic or aperiodic; in phase with vocal fold vibration, or out of phase; and with or without medial contact of the FVF [11].

Use of Supraglottic Structures in Voice-Disorder Speakers

There are certain voice disorders in which excessive supraglottic activity is known to be present. This section discusses the findings on supraglottic activity for these voice disorders.

Hyperfunctional voice disorders

Boone [51] suggested that supraglottic activity, and more particularly FVF compression, is one of the most common laryngeal manifestations of hyperfunction. Based on the presence of either FVF compression or AP compression, several authors have identified types of muscle tension dysphonias [52,53] muscle misuse disorders [54] or vocal hyperfunction [55]. Before continuing with this discussion, some clarification of the term “hyperfunction” is needed.

Hillman et al. [43] suggested that vocal hyperfunction refers to “conditions of abuse and/or misuse of the vocal mechanism due to excessive and/or imbalanced muscular forces”. I would add to that definition that these conditions must result in voice quality that is not normal. In my opinion, the term “hyperfunction” should not be used if an individual’s voice quality is within normal limits. These disorders are often termed “functional” because they are thought to reflect the individual’s poor use of the laryngeal musculature, although the term functional has also been used as a synonym for psychogenic voice disorders. To some, the specific term hyperfunctional voice disorder (some using the term functional for this specific group eg [19,52,56]) means a disorder in which the conditions of excessive and/or imbalanced muscular forces result in poor voice quality but no visible findings on endoscopic (either rigid or flexible) or neurologic examination. In some cases, these excessive and/or imbalanced muscular forces can produce vocal fold lesions such as nodules, contact ulcers, hemorrhages, or polyps. In some cases, these excessive and/or imbalanced muscular forces seem to affect intrinsic and extrinsic laryngeal muscles, producing a tight, strangled sound similar to adductor spasmodic dysphonia, called muscle tension dysphonia [54] or primary muscle tension dysphonia [57]. Finally, some use the term secondary muscle tension dysphonia to refer to excessive and/or imbalanced muscular forces that compensate for some underlying glottal insufficiency [57].

We have 4 different rating scales that include diagnostic criteria based solely on whether the examiner observes FVF compression and/or AP compression in the absence of any other visible findings [52-55]. On the other hand, we make diagnoses based upon visible findings from the endoscopic examination (eg nodules), which are thought to result from hyperfunctional use (i.e. excessive and/or imbalanced muscular forces) of the larynx. FVF compression and AP compression
are assumed to be present because they are considered signs of hyperfunctional voice use. This assumption needed to be tested. For any sign to be diagnostic, it needs to be demonstrated that it occurs when someone has a voice disorder and does not occur when someone does not have a voice disorder. Since we already know that individuals with normal voice quality and no evidence of structural abnormality do exhibit some supraglottic activity, we know that simply whether or not FVF and/or AP compression are present is not diagnostic. However, other factors such as degree of compression or occurrence in other phonetic contexts compared to normals might be diagnostic. Thus, the next sections examine the studies that have compared FVF and AP compression in individuals with normal vocal quality to those with hyperfunctional voice disorders.

First, we consider studies comparing controls to those with poor voice quality and no visible findings on endoscopic (either rigid or flexible) or neurologic examination. Sama et al. [56] examined the sensitivity and specificity of 12 clinical features, 6 from Van Lawrence’s fiberoptic features of vocal hyperfunction, and the 6 from Morrison & Ramage’s classification of muscle tension dysphonia. A speech-language pathologist and an otolaryngologist rated fiberoptic video recordings with no audio signal. No significant differences were found in the prevalence of these 12 features between groups. Approximately 50% of the control subjects were rated as abnormal. None of the features demonstrated the degree of sensitivity or specificity to make it a good diagnostic indicator of a voice disorder. Behrman et al. [24] used quantitative measures of both types of supraglottic activity to compare subject groups. The control subjects demonstrated medial FVF compression, but no significant difference in the amount of compression was found between groups. For AP compression, they reported that voice-disordered patients had a significantly greater amount than the controls, but cautioned that an overlap existed in the ranges of measures from the two groups, so AP compression may not be diagnostic of a voice disorder.

Only one unpublished study has examined quantitative differences in FVF and AP compression just between controls and nodule patients (Stager, Horst & Bielamowicz, unpublished). The results of this study found a significant difference between these two groups as far as the amount of AP compression, with the nodules patients demonstrating greater AP compression than the controls. As reported in the Behrman et al. [24] study, an overlap in the ranges of the measures from the two groups was found. No significant differences were found for the amount of FVF compression between groups.

Three other studies have examined several types of patients with poor voice quality, including those with lesions and those with no visible findings on endoscopic examination. Stager et al. [34] studied the incidence of FVF and AP compression using images from flexible fiberoptic examinations of several different speech tasks in controls, those with poor voice quality and no visible findings on endoscopic exams, and those with vocal fold nodules. For static FVF and AP compression, controls had the lowest incidence, followed by those with nodules followed by those with no visible endoscopic findings. All three groups demonstrated a similar incidence of dynamic FVF compression, especially on speaking tasks involving glottal stops. Stager et al. [38] quantified the amount of supraglottic activity in images from flexible fiberoptic examinations comparing controls to those with nodules and those with vocal fatigue (another group with no visible findings, but not necessarily considered hyperfunctional). They reported no significant differences between groups in the amount of normalized static or dynamic FVF compression, but those with voice disorders demonstrated significantly greater normalized static A-P compression than the normal controls. Nemetz et al. [42] also examined gender differences in FVF compression using a rigid endoscope with controls and those with voice disorders, including nodules, vocal fold paresis, granulomas and cysts or other minor structural alterations. They rated the configurations of the FVF as concave, linear and convex based on anatomic points. Results suggested that the concave configuration of the FVF was the most common for both control and voice disordered subjects. In the dysphonic group, males were significantly more likely to have convex configuration, and the females, significantly more likely to have a linear configuration. Based on the figures in the paper, both convex and linear configurations appear to be related to compression of the FVF, with convex being more compressed than linear.

One study [58] has looked at FVF compression in patients with primary muscle tension dysphonia, and it was in the context of a comparison between these two patients and those with spasmodic dysphonia. They concluded that those with primary muscle tension dysphonia demonstrated relatively sustained FVF compression throughout the speech sample, regardless of the phonetic context. However, their conclusion was that endoscopy alone does not provide a reliable diagnosis of primary muscle tension dysphonia.

**Ventricular dysphonia**

Ventricular dysphonia is described as a voicing disorder in which the FVF are used as a vibratory source either in addition to, or instead of the TVF. As reported above, the FVF are not structurally the same as the TVF, they are less stiff and more elastic than the TVF, so they do not produce a normal voice quality when they vibrate. Static FVF compression may also be present when the vocal folds are at rest [59,60]. This diagnosis can easily be made based on endoscopic examination alone [60].

**Spasmodic dysphonia**

In the study by Leonard & Kendall [58], comparing supraglottic activity in psychogenic (muscle tension dysphonia) versus spasmodic dysphonia using flexible fiberscope, FVF compression was found in 67% of the spasmodic dysphonia patients, but it was episodic in nature, depending on the speech task and type of spasmodic dysphonia (abductor vs adductor). This study did not examine the presence of AP compression.

**Vocal fold paresis**

In the above mentioned disorders, the TVF are for the most part vibrating and meeting medially. In this particular group of patients, function of one or both vocal folds is impaired because of neurological injury. In our clinical experience, FVF compression can be present at rest, unlike any other voice disordered population. During phonation, in patients with a unilateral paresis, the paralyzed vocal fold was associated with greater static FVF compression compared to the paralyzed side or either vocal fold of the controls. In patients with bilateral paresis, FVF compression was found for both vocal folds [13]. Traditionally, the use of supraglottic activity in this patient population was to compensate for the lack of glottal closure [13,15]. Some support for this notion comes from a study by Bielamowicz et al. [39]. They reported that greater FVF compression was identified in unilateral paresis patients with normal laryngeal function measures than those with abnormal measures. This would support the notion that the supraglottic activity may indeed be compensating for the lack of TVF closure.
In the studies of normal voice, dynamic FVF compression was found for speech samples that contained glottal stops, and this was termed laryngeal clarity. Leydon et al. [39] reported that listeners were significantly more likely to report good clarity for repeated /i/, repeated /isi/ and repeated /izi/ syllables for controls than patients with vocal fold paresis. In our clinical experience, we have found that poor laryngeal clarity is diagnostic of laryngeal nerve injury. However, good laryngeal clarity is not diagnostic of not having laryngeal nerve injury. Individuals with documented neural injury by laryngeal electromyographic examination can have good laryngeal clarity. This could be explained in two ways. It may be that laryngeal clarity had recovered by the time of evaluation, even though other functional measures had not. It may be that laryngeal clarity was never affected by the nerve injury. This impairment of laryngeal clarity in patients with paresis also suggests some innervation by the recurrent laryngeal nerve to those muscles responsible for producing dynamic FVF compression.

Finally, Roy et al. [61] reported that following a lidocaine block of the external superior laryngeal nerve (ESLN), 60% of participants with normal voice demonstrated a deviation of the petiole of the epiglottis to the side of the cricothyroid muscle weakness during an upward glissando produced at normal volume. This was followed up by a series of case reports on patients with denervation of the ESLN either separately, or in combination with other nerve injury [62]. This deviation of the petiole was found in those with ESLN denervation, but was not found in a single patient with only recurrent laryngeal nerve injury. These findings suggest that this deviation of the petiole during high-pitched voice production may be diagnostic for unilateral ESLN denervation.

Presbylarynges

Given that the traditional view of supraglottic activity is to compensate for lack of glottal closure, another patient population that lacks good glottal closure are those with presbylarynges. Our definition of presbylarynges is a patient over 65 years of age that reports a hoarse voice quality, and has normal neural activity on the laryngeal EMG examination. Bloch & Behrman [63] reported that individuals with presbylarynges had significantly smaller normalized laryngeal outlet measures than control subjects. This outlet measure represented the area of the TVF that could be seen during phonation. Thus, their group of presbylarynges patients did demonstrate more supraglottic activity than a group of controls. Hagen et al. [64] also reported what they considered maladaptive supraglottic hypertension in this group of patients. A study by Stager & Bielamowicz [65] compared patients over 65 with paresis or presbylarynges, as defined by results of the laryngeal EMG examination. Significant differences were found in the proportion of each subgroup exhibiting static FVF compression with more individuals with presbylarynges demonstrating bilateral FVF compression and more individuals with unilateral recurrent nerve paresis demonstrating unilateral FVF compression than expected.

Supraglottic cancer

This final group of patients are actually exactly the opposite of all those discussed above. In this instance, supraglottic structures have been surgically removed because of cancer. With patients who have had FVF removed with preservation of the TVF, we can test some of the findings from the modeling and animal cadaveric studies.

Based on what we know about supraglottic activity, we can predict that these subjects may have: 1) lower flow resistance; 2) higher flows; 3) lower intensity; 4) greater phonation threshold pressures; 5) inability to produce glottal stops; and 6) less resonant vowel productions (because the epilarynx may not be able to be narrowed). In a study by Goodman, Stager & Sadeghi [66] using 5 patients with stage T2/T3 supraglottic squamous cell carcinoma resected via transoral supraglottic laryngectomy (an average of 38 months post surgery), and 6 controls, significant differences were found in vowel resonances, such that patient productions were less resonant than controls because of less tuning between fundamental frequency and the first formant, meaning a greater difference between the two frequencies [20]. Because these patients had structures removed, it was likely that the epilarynx was not able to narrow to assist in resonant production. This supports the literature which has suggested that individuals following this type of surgery report “weak” voices [27]. Perhaps the “weak” refers to poorer resonance rather than to changes in intensity. Stager et al. [67] reported that patients produced significantly fewer glottal stops than expected compared to controls for sentences constructed to elicit glottal stops. However, there was quite a bit of variability between patients in their glottal stop production. For example, one patient with bilateral removal of the false vocal folds did not produce glottal stops, which would support our hypothesis. Another patient had one false vocal fold removed, and yet produced the greatest number of glottal stops. On endoscopic exam, this remaining false vocal fold did compress medially in a dynamic fashion during sentences with glottal stops.

Conclusions

This paper has reviewed the role of the supraglottic area in voice production. More information was related to FVF compression than AP compression. The presence of FVF seems to add efficiency to vocal fold vibration and complete compression reduces the efficiency of voice production. Supraglottic activity (compression of FVF and arytenoids to petiole) is present in individuals with normal voice quality and no visible lesions on endoscopic examinations. Why this activity occurs is not known (except for the activity related to laryngeal clarity). We still do not have a good grasp of the physiology behind these movements and how the FVF are positioned in static compression. It is also not known if having supraglottic activity can progress from normal to poor voice quality over time or is habitual from compensating for a previous injury. Supraglottic activity is not diagnostic for hyperfunctional voice disorders, although it does occur more frequently and the amount of compression is greater than normals. For vocal fold paresis patients, some evidence exists that the FVF compression is compensating for the lack of closure of the TFV, and occurs principally on the side opposite the paresis with unilateral injury. The role of FVF activity in laryngeal articulation was demonstrated in controls, hyperfunctional patients, and paresis patients. The inability to produce glottal stops is diagnostic of underlying laryngeal nerve injury, but the ability to produce glottal stops does not confirm the absence of laryngeal nerve injury. For those who have had the FVF removed, some data suggest that they are not able to produce glottal stops. However, there is much more to learn about the role of supraglottic activity and changes in the epilarynx area and their effects on voice production in both normals and voice disordered individuals.

References


