Welcome to the Spring edition of *The Voice*. This is my first edition as editor and I am very pleased to serve on this wonderful publication. In this issue, we are responding to a reader’s request for thematic content on “breathing.” We encourage reader’s ideas for newsletter themes. Please send the office an email with your ideas (office@voicefoundation.org) and we will put together something informative!

We have used a three-pronged approach for getting at the issue of breathing in this newsletter. First, Drs. Zhaoyan Zhang and David Berry, voice scientists at University of California—Los Angeles, provide a brief tutorial on how breathing and associated airflow contribute to the onset and maintenance of vocal fold vibration for phonation. Next, Dr. Michelle Troche, Speech-Language Pathologist and Clinical Assistant Professor at the University of Florida, describes a method of treating voice disorders by training expiratory muscle strength. Last, Aaron Johnson, Singing Voice Specialist and Pre-doctoral Fellow at the University of Wisconsin—Madison, addresses breathing and breath support for singing.

**Breathing, Airflow and Mechanisms of Phonation Onset**

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According to the classic myoelastic-aerodynamic theory of vocal fold vibration (Van den Berg, 1958), the vocal folds close due to the action of a negative Bernoulli pressure, which is followed by a buildup of subglottal pressure. When the subglottal pressure is sufficiently high, the vocal folds are pushed open and the intraglottal pressure is lowered. The cycle then repeats, which leads to sustained oscillation of the vocal folds.

While this description may seem to provide an adequate explanation for sustained vocal fold vibration during normal phonation, questions quickly arise for other voicing conditions. For example, for a breathy voice (in which complete glottal closure does not occur), would the negative Bernoulli pressure be sufficient to move the vocal folds inward? Or similarly, without complete glottal closure, would the build-up of subglottal pressure be sufficient to move the folds apart? Indeed, under some pathological conditions, phonation fails to occur and the vocal folds are simply blown apart—despite high subglottal pressures and high respiratory effort. Under such conditions, do alternate physical mechanisms exist to better explain the phenomenon of phonation onset?

The myoelastic-aerodynamic theory is correct in identifying the interaction between the vocal folds and the airflow as the underlying mechanism of self-sustained vocal fold vibration. However, as pointed out by Ishizaka (1981) and Titze (1994), the theory is inadequate in explaining how energy is transferred from the airflow to the vocal folds to sustain vibration. According to Bernoulli’s equation, the airflow pressure would always be 90 degrees out of phase with the vocal fold surface velocity, resulting in no net energy transfer from the airflow to the vocal folds over one cycle of vibration. Thus, Bernoulli pressure alone does not provide a mechanism for energy transfer from the airflow to the vocal folds.

The key to a non-zero energy transfer lies in that the vocal folds are not rigid and therefore different portions of the vocal fold surface can vibrate in different phases, i.e., the upper and lower margins of the medial surface do not necessarily move inward and outward together. With a vertical phase difference between the upper and lower margins of the medial surface, the vocal fold often exhibits a wave-like motion, also called the mucosal wave, along the medial surface which propagates onto the superior surface. In his surface wave model of phonation, Titze (1988) showed that the presence of this wave motion caused the intraglottal pressure to be at least partially in phase with the vocal fold surface velocity, resulting in energy transfer from the airflow to the vocal folds, so that the vocal fold vibration could be sustained.

In Titze’s surface wave model, such a wave-like motion was produced by artificially combining an in-phase vibration pattern (the entire medial surface moved inward and outward together) and an out-of-phase vibration pattern (upper and lower portions of the medial surface moved in opposite directions) and forcing these two motions (or modes) to oscillate at the same frequency. In reality, such synchronization of two vibration patterns (modes) to the same frequency is naturally induced by a cross-mode coupling of the glottal airflow, which causes the two modes to approach each other in frequency (Ishizaka, 1981; Zhang et al., 2007). At a threshold subglottal pressure or flow rate, the two modes are synchronized to the same frequency but often with a non-zero phase difference. Such a non-zero phases difference between the two synchronizing modes means that the different portions of the vocal fold surface now may move at different phase, i.e., the upper portion of the medial surface may move inwards as the lower portion of the medial surface moves outwards. Synchronization of two modes (continued on Page 2)
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at different phases causes the intraglottal pressure to have an in-phase component with the vocal fold surface velocity, thus allowing airflow to do work on the vocal folds and transfer energy into the vocal folds (Zhang et al., 2007). Note that this mechanism of mode synchronization does not require a complete glottal closure, with corresponding pressure build-up. A wide glottal opening (as for breathy voice) would reduce but not eliminate this mode synchronization effect.

Phonation onset occurs as two modes of the vocal folds are synchronized by the glottal flow. The resulting vocal fold vibration pattern depends critically upon the characteristics of the two modes that are synchronized. This means that vocal fold vibration and therefore voice quality may be varied by changing the vibrational characteristics of the underlying modes which are synchronized. Changes in these modes are usually induced by changes in the geometry or stiffness of the layered vocal fold structure. For example, by changing the body-cover stiffness ratio, Zhang (2009) show that a series of vocal fold vibration patterns with different sound production efficiency could be produced.

In humans, different combinations of biomechanical properties of the multiple vocal fold layers can be achieved through neural stimulation of laryngeal muscles, through which we are able to produce many varieties of voice, minimize the respiratory effort required to initiate and sustain phonation, and thus optimize voice production efficiency.


Expiratory Muscle Strength Training for the Treatment of Persons with Voice Disorders

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The respiratory system is often the primary therapeutic target in traditional rehabilitation of voice disorders given that appropriate respiratory drive, defined in large part by adequate subglottal pressure, is essential to phonation (e.g., Hixon, 1987). Often times the elastic recoil forces of the lung-thorax unit are not sufficient to complete the act of voicing, speaking, or singing; therefore, the development of appropriate subglottal pressures for these tasks requires work by the expiratory muscles. These driving pressures are necessary for the maintenance of good vocal quality, appropriate sound pressure levels (loudness), and increased sound durations (e.g., Ishihiki, 1964, Scherer, 1995, Scherer, Vail, & Guo, 1995). The “breathing exercises” used in voice therapy often have as their goal an awareness of breathing, reductions in muscle tension, and improved “respiratory drive” for speech and voice. The latter can be difficult to achieve given that most respiratory or breathing exercises provide no “physiological load” to the muscles. In other words, the respiratory muscles are not forced to work more than their usual. Techniques such as breathing through pursed lips or maximum inhalations and exhalations are examples of this. Recently, strength training paradigms for respiratory muscles has garnered significant clinical and research interest. Our research group has been particularly interested in the effects of expiratory muscle strength training (EMST) on voice, speech, swallow, and cough function. The rationale for utilizing EMST for the treatment of voice disorders is based on two guiding principles: 1) overload and 2) transference.

The principle of overload suggests that in order to produce a peripheral or central change to the neuromuscular system, the system must work harder than it usually does. To achieve this end, we utilize an EMST device (Figure 1) which houses a calibrated, one-way, spring-loaded valve to mechanically overload the expiratory and submental muscles (Wheeler, Chiara, & Sapienza, 2007). The valve blocks the flow of air until sufficient expiratory pressure is produced. Once opened, air is allowed to flow through the device. The physiological load on the targeted muscles can be increased or decreased depending on the device setting. Traditionally we have set the device to 75% of a person’s maximum expiratory pressure (MEP) largely mimicking the training protocols with limb muscles (Powers & Howley, 2004). The treatment paradigm lasts five weeks, with participants completing 25 breaths a day (5 sets of 5 repetitions) five days per week. We call this the “power of 5”. During the five weeks the device is reset as changes in expiratory muscle strength take place.

Another guiding principle for the use of EMST is that of transference. This is the idea that changes can occur to systems which are not specifically targeted through training. Therefore, the effects of training with an EMST device should not only result in improvements to tasks similar to taking a breath through the EMST device. Instead, increased maximum expiratory pressures or MEPs secondary to training with the EMST device should result in higher-pressure support for voice resulting in improved sound quality, sound pressure level, and sound durations. In a healthy person, increased maximum expiratory pressures may not result in perceivable changes to voice, but in cases of laryngeal pathology or dysphonia increased expiratory pressures may help

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