7 Postlaryngectomy Respiratory System and Speech Breathing

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The biological function of the respiratory system is to maintain stable blood gas values during changing homeostatic demands (e.g., changing posture, walking, exercising, sleeping, increased cognitive load, speaking) by exchanging oxygen (O_2) from the air into the blood supply and removing carbon dioxide (CO_2) efficiently (Hugelin, 1986; Shea, 1996; von Euler, 1997; West, 2013). Maintaining this balance requires the seamless integration of automatic and voluntary control systems for respiration. Changes in the upper airway in speakers following a total laryngectomy subsequently alter how clinicians and researchers approach respiration, as well as their production of alaryngeal speech (see Lewis, Chap. 8, and Searl, Chap. 13). Many of these speakers are older, have a past medical history of smoking, and are likely to suffer from some degree of chronic obstructive pulmonary disease (COPD). To compound matters, upper airway changes following laryngectomy likely result in compensatory alterations to breathing. The removal of laryngeal afferent input, greater upper airway resistance, and possible respiratory compromise may affect the flexibility necessary for maintaining homeostasis and for achieving proficient alaryngeal speech (Bohnenkamp, Forrest, Klaben, & Stager, 2012; Bohnenkamp, Forrest, Klaben, & Stager, 2011; Bohnenkamp, Stowell, Hesse, & Wright, 2010; Donnelly, 1991; Fontana, Pantaleo, Lavorini, Mutolo, Polli, & Pistolesi, 1999; Hida, 1999; Lee, Loudon, Jacobson, & Stuebing, 1993; Sant'Ambrogio, Matthew, Fisher, & Sant'Ambrogio, 1983).

Each alaryngeal speech option varies in its reliance on the respiratory system. Speakers who use an electrolarynx (EL) have a decoupled phonatory and respiratory system, whereas speakers who rely on esophageal speech (ES) may be required to have fine control of the respiratory system depending upon the method of esophageal charging for phonation (e.g., inhalation or injection method). Speakers who use tracheoesophageal speech (TE) as their primary mode of communication are most likely to encounter difficulties with speech associated with respiratory changes (see Graville, Palmer & Bolognone, Chap. 11).

The reliance on the respiratory system for speech in all speakers is influenced by many factors. These unavoidable factors include the influence of sensory input, the balance between the voluntary and involuntary control systems, and maintaining the balance of O_2 and CO_2 in the body. These are all manipulated differently by respiratory demand and speech task (e.g., rest breathing, speech, and oral reading). In the case of speakers with a total laryngectomy, the disconnect of the upper airway and removal of the larynx, age-related changes in the respiratory system, and

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smoking-related illness will combine with the aforementioned factors to result in adaptations and compensations to breathing to produce speech. This chapter will provide a background on how respiration and the consequences of total laryngectomy affect breathing in these individuals and how these changes may influence alaryngeal speech.

The Importance of Laryngeal Afference

Total laryngectomy results in the removal of the larynx and superior laryngeal nerve (SLN) of cranial nerve (CN) X (vagus). Subsequently, speakers with a total laryngectomy regardless of past smoking history may experience altered breathing and respiratory patterns. The SLN may influence the timing and firing rate of the respiratory muscles (Fontana et al., 1999), a process which aids in maintaining airway integrity. Airflow, pressure, mucosal temperature, and stretch receptor sensation are altered due to the loss of laryngeal afferent input, which comprises 1/3 of all afferent input from the tracheobronchial tree (Donnelly, 1991; Fontana et al., 1999; Sant'Ambrogio et al., 1983). Though the influence of afferent input may be overstated relative to breath-to-breath control, this afferent input is robust and will terminate any respiratory drive during aspiration in individuals with an intact larynx. In addition, this feedback allows individuals to manipulate air pressure differences within 1–2 cm H_2O during both breathing and speech (Shea, 1996; Davis, Zhang, Winkworth, & Bandler, 1996). In the normal laryngeal system, the posterior cricoarytenoid muscles (PCA), oblique and transverse interarytenoid (IA) muscles, and lateral cricoarytenoid muscles (LCA) are active to valve airflow both during inspiration and during expiration. Specifically, IA and LCA are active during expiration to improve gas exchange by slowing expiratory airflow (Dick, Orem, & Shea, 1997). Ohala (1990), Warren (1996), and Wyke (1983) have stated that this may influence how an individual manipulates the respiratory system for speech.

Automatic and Voluntary Neural Control of Respiration

The neural and blood gas disruptions in speakers with respiratory compromise have received little inquiry by speech-language pathology, though they are likely to affect speakers with a total laryngectomy and are implicated in other communication disorders (Duffy, 1995; Kaneko, Zivanovic, Hajek, & Bradley, 2001; Khedr, Shinaway, Khedr, Aziz Ali, & Awad, 2000; Terao et al., 2001; Wessendorf, Teschler, Wang, Konietzko, & Thilman, 2000). The automatic control of the respiratory system responds to metabolic and blood gas changes by altering air flow resistance and respiratory muscle activity. This is accomplished via central pattern generators within the pons (Hugelin, 1986; von Euler, 1997). An example of an automatic response would be to manipulate the depth or rate of breathing to regulate increased levels of carbon dioxide in the blood. Conversely, the voluntary system is often responsible during increased levels of activity (e.g., during speech and exercise). This active process is accomplished by modulating inspiratory and expiratory durations based on afferent air pressure signals in the tracheobronchial and pulmonary airways (Davis et al., 1996; Garrett & Luschei, 1987; Testerman, 1970). Each control system is dependent upon neural signaling from CN IX and X, which transmit information regarding blood gas levels in the aortic blood flow and the status of the stretch receptors (e.g., respond to lung inflation, vibration, inspiratory/expiratory effort, lung volume changes, degree of expiratory or inspiratory effort, and resistance to airflow) in the lungs (Guz, 1997; Shea, 1996). The stretch receptors are the primary inputs that contribute to the perception of shortness of breath, or what is termed dyspnea (Homma, Obata, Sibuya, & Uchida, 1984).

Involuntary and Voluntary Neural Control of Respiration: Speech

Maintaining appropriate O_2 and CO_2 values differ by activity. Specifically, the demands during speech on that maintenance are greater than that during resting tidal breathing but much less than during exercise. Both demands result in increased CO_2 levels in the blood, which results in stimulation to increase either depths of inspiration, use of a greater amount of vital capacity (VC; i.e., greater inspiratory volumes and expirations into a speaker's functional residual capacity; FRC), or increasing respiratory rate to balance blood gases. These adaptations to demands require an integration of motor intent and sensory feedback wherein the voluntary control system is primarily responsible as it overrides our involuntary respiratory system's automatic control drive to breathe.

CO_2 levels increase for short amounts of time during speech, which requires the use of the voluntary system by speakers to ignore hypercapnia (i.e., increased partial pressures of carbon dioxide in the blood; PCO_2). They must complete the utterance and then return to resting chemostatic values via hyperventilation (Hoit & Lohmeier, 2000). In contrast to the response to CO_2, speakers will ignore reduced O_2 levels in the blood to maintain communication, even during instances when completing rigorous exercise (Bunn & Mead, 1971; Hoit & Lohmeier, 2000; Phillipson, McClean, Sullivan, & Zamel, 1978; Russell, Cerny, & Stathopoulos, 1998; Shea, 1996). Eventually, a speaker can no longer maintain the ability to override the need to breathe, which results in breathing for gas exchange and reducing airflows needed for speech by 55% (Doust & Patrick, 1981).

Previous research has indicated that speech is perhaps a more robust voluntary activity than other motor activities. For example, speakers are less likely to complain of the effects of hypercapnia while completing a speech task than while under the same hypercapnic conditions at rest (Phillipson et al., 1978). The voluntary system allows speakers to ignore the hypercapnic stimulus until chemical gas values are much more compromised. However, in contrast to a speech task, Corfield, Roberts, Guz, Murphy, and Adams (1999) reported that individuals in slight hypercapnia demonstrated difficulty manually moving a joystick to track a cursor on a computer screen. Therefore, the complex motor act of speech which includes the integration of language, articulation, phonation, and respiration may not be as vulnerable to the effects of hypercapnia as a simple motor tracking task is to even slight hypercapnia. Alaryngeal speakers with COPD, though compromised, may not demonstrate the chronic effects of hypercapnia due to their ability to overcome the need to breathe to complete the message.

Speech Breathing in Laryngeal Speakers

An explanation of the similarities and differences between the two theories of speech breathing (i.e., the classic; Draper, Ladefoged, & Whitteridge, 1959; contemporary; Hixon, Goldman, & Mead, 1973; Hixon, Mead, & Goldman, 1976) is beyond the scope of this chapter; however, there are numerous aspects of respiratory control in speakers who undergo a total laryngectomy that differ from typical speakers. Both theories reported that speech requires the coordination of active muscular manipulation of the system (e.g., diaphragm, abdominal muscle contraction, external and internal intercostal contraction, etc.) combined with the inherent amount of relaxation forces available to speakers such as those related to tissue elasticity and recoil and gravity (Draper et al., 1959; Hixon et al., 1973, 1976).

The two theories of speech breathing diverge primarily in their explanation of abdominal muscle activity. The classic theory argues that the abdominal musculature is not needed until the ends of utterances, whereas the contemporary theory stated that abdominal activity is needed prior to and throughout speech. The contemporary view posited that simultaneous internal and external intercostal activity during speech combines with relaxation forces to produce speech.

Typical speech requires deeper inspirations and expirations in addition to increased muscle activity in the chest wall in contrast to what is required in quiet tidal breathing (Draper et al., 1959; Hixon et al., 1973, 1976). Speakers rely primarily on rib cage musculature to inspire to greater percent of their vital capacities (%VC) to provide the relaxation forces needed for speech. Inspiration is always active, but tidal breathing at rest relies on passive forces of the rib cage for expiration. The contemporary view reported that the %VC needed for tidal breathing lies in the middle ranges between 45%VC and 36%VC (i.e., approximate resting expiratory level in typical individuals; REL). Speech, in contrast, occurs between 60%VC and 36%VC (Hixon et al., 1973, 1976). Further, speech is terminated at or near REL which improves speech efficiency and reduces effort. Once a speaker speaks past their REL into their FRC, all expiratory force then becomes active or muscular in nature in an effort to overcome the negative forces of inspiration. This all occurs as speech is almost exclusively initiated and terminated at grammatically appropriate sentence of phrase boundaries, which are likely preplanned and influenced by the utterance length and complexity in addition to the balancing of metabolic demands with speech.

Draper and colleagues had earlier argued in the classic theory of speech breathing that abdominal activity was not required until the speaker reaches lower %VC at the ends of utterances and that there was no need for overlap of activity of the inspiratory and expiratory muscles of the rib cage (i.e., external and internal intercostal muscles). In contrast, to produce the pressures needed for speech, Hixon and colleagues had argued in the contemporary theory that constant abdominal activation is present prior to and during speech and that this results in a generally predictable pattern of chest wall configuration for speech (Hixon et al., 1973, 1976). This general speech configuration change consists of expiratory (inward) abdominal movement followed by rib cage expansion (elevation and reduction of the space between ribs). Hixon and colleagues further contradicted the original Draper et al. argument by reporting the co-contraction, or overlap of activity, of the inspiratory and expiratory muscles of the rib cage muscles during speech to allow for quick and subtle changes to air flows and pressures (i.e., net-zero posture). Interestingly, Ladefoged and Loeb (2002) later reported that, in fact, the rectus abdominis is active prior to and throughout speech utterances and there is a general acceptance that abdominal contraction is essential during speech.

Whereas previous researchers have suggested that the abdominal contraction and rib cage configuration prior to speech are a predictable and relatively invariable oppositional speech-specific process in males (90% of the time; Baken & Cavallo, 1981; Baken, Cavallo, & Weissman, 1979; Cavallo & Baken, 1985), others argued there is no standard or predictable process (Hixon, 1988; McFarland & Smith, 1992; Wilder, 1983). The lack of a predictable posturing allows for flexibility in the production of volumes, flows, and pressures for speech depending on utterance demands. Wilder (1983) reported that a predictable response was present in only 32% of typical healthy female speakers, suggesting that prephonatory posturing is likely to differ by sex of the speaker. This provides adaptability, to the variability of speech task and glottal configuration (Iwarsson, 2001; McFarland & Smith, 1992). Taken together, these findings indicate that control of the chest wall for speech breathing is likely to be flexible and adaptive and will vary by sex and task. This suggests that control of the chest wall will be altered in alaryngeal speakers due to the many changes to the upper airway following a total laryngectomy.

Speech Task

It is well documented that speaking task influences breathing behaviors in typical speakers (Goldman-Eisler, 1968; Goldman-Eisler, 1961; Winkworth, Davis, Adams, & Ellis, 1995; Winkworth, Davis, Ellis, & Adams, 1994) and these differences are likely to be exacerbated in alaryngeal speakers. Speakers will regularly hyperventilate on inspiration following the completion of an utterance to balance blood gas values; however, speech research indicates that these inspirations are more likely to be influenced by the length and complexity of an upcoming utterance rather than recovering from a previous utterance (Goldman-Eisler, 1961, 1968). These inspirations that match length of an utterance argue for a preplanning of speech at the level of the respiratory system. This preplanning can differ by task, specifically in relation to oral reading and spontaneous speech. Winkworth et al. (1994) and Winkworth et al. (1995) reported that inspirations are taken at linguistically appropriate boundaries (e.g., clause boundary, sentence) during both spontaneous speech and oral reading. These inspirations are speaker-specific during spontaneous speech but are determined by text during oral reading. The dynamic nature of spontaneous speech may, therefore, necessitate online adjustments by the respiratory system, whereas oral reading requires inspirations taken to match an unknown, upcoming utterance length. When evaluating utterance length and respiratory behaviors, it is important to understand that speakers behave differently when generating spontaneous speech and oral reading. This is especially so in speakers with respiratory compromise, as well as alaryngeal speakers who may further alter their breathing patterns (Lee et al., 1993). When considering the three primary alaryngeal modes, electrolaryngeal, esophageal, and TE speech, TE speakers are the most likely to encounter these issues because of their use of pulmonary air support and their need to overcome the inherent resistance increases in the neoglottis and TE puncture prosthesis. This information is valuable in the assessment of an alaryngeal speaker's breathing patterns because the speech tasks are not interchangeable and are not comparable. These task changes are also influenced by the expected age-related changes in breathing.

Age

Age is an unavoidable influence on the respiratory system. It can be difficult to determine what causes respiratory changes and whether they are an expected process due to aging and/or if they are combined with injury, smoking, and environmental factors. Changes in the respiratory system might be functional, structural, mechanical, or related to ventilation/perfusion/diffusion and nervous system changes (Ayres, 1990; Chan & Welsh, 1998; Janssens, Pache, & Nicod, 1999; Hoit & Hixon, 1987) and, consequently, are likely to affect people who have undergone a total laryngectomy.

The interaction of passive and active (muscular) forces for speech is altered as age-related changes result in reduced elasticity, thorax stiffening, respiratory muscle weakness, loss of cross-sectional intercostal muscles tissue, and increased use of high lung volumes to create relaxation forces (Brown & Hasser, 1996; Dhar, Shastri, & Lenora, 1976; Kahane, 1980; McKeown, 1965; Pierce & Ebert, 1965; Tolep & Kelsen, 1993). Older speakers have fewer alveoli and fewer capillaries per alveolus which results in a subsequent loss of airway tissue and contributes to the reduction in elasticity. This also may result in changes in gas exchange and air trapping from collapse of the small airways, namely, bronchioles (Janssens et al., 1999). For example, 60-year-old males can expend 20% more energy during tidal breathing than do 20-year-olds (Janssens et al., 1999). Thus, age-related declines in breathing might be exacerbated following a total laryngectomy, especially considering the age of a typical speaker with a laryngectomy.

The physiological response to muscle atrophy secondary to age is that there is a concomitant reduction in the individual's VC (Hoit & Hixon, 1987; Kendall, 2007; Sperry & Klich, 1992). Vital capacities are reduced by up to 1 L in older speakers as a result of calcification of intercostal and vertebral joints (Crapo, 1993; Murray, 1986). Hoit and Hixon (1987) reported that residual volume (i.e., dead space which is unusable during respiration) also increases with age. As a result of the increased muscular effort necessary for successful compensation, older adults may have less physical reserve to deal with illness when it occurs. These age-related differences may make the effects of a disease state more pronounced, especially in older speakers with COPD, as well as creating likely alternations in alaryngeal speakers (Crapo, 1993).

Respiratory Compromise

COPD is characterized by dyspnea (discomfort during breathing), altered O_2 and CO_2 values due to ventilation-perfusion mismatch, excessive secretions, hypertrophy of mucous glands, and narrowing of airways within the lungs (West, 2013). It is a disease that is assumed during life and confirmed only following death. Though COPD is often associated with a past history of smoking, not all who present with COPD have a history of smoking. Speakers with severe COPD may demonstrate breathlessness, shorter utterance lengths, and poor ability to control speaking loudness (Lee et al., 1993). Additionally, speakers with COPD may be hypercapnic at rest (Hida, 1999) and only worsen with increased activity due to a ventilation-perfusion mismatch.

The ventilation-perfusion mismatch is due to the destruction of the walls of the alveoli and the capillary bed, thereby, reducing both blood flow surrounding the alveoli and subsequent gas exchange. The inability for O_2 to enter the arterial blood and for CO_2 to leave the venous blood flow may result in increased levels of CO_2 in the blood. This may be viewed as a maladaptive compensation because COPD speakers are more likely to prefer to function in this mild hypercapnic state versus increasing the work of breathing during inspiration to increase O_2 (West, 2013). Subsequently, speakers with COPD are likely to alter their breathing for speech. An alteration in response to the reduction in relaxation forces would be to increase muscular activity of the rib cage and abdomen to complete gas exchange (Sharp, Goldberg, Druz, Fishman, & Danon, 1977). Examples of these alterations include lip pursing, altering the rate and depth of inspirations, and recruiting extraneous chest wall activity (Lee et al., 1993; Hida, 1999). Speakers with a total laryngectomy are not able to adapt by using lip pursing due to the loss of airflow through the upper airway as a strategy and would be left with chest wall manipulation as a strategy. The chronic hypercapnic state in these speakers could result in a constant level of discomfort during breathing and speech. Typical speakers routinely demonstrate slightly higher CO_2 levels during utterance and subsequently have to recover, or hyperventilate, following speech production to maintain O_2 and CO_2 values (Hoit & Lohmeier, 2000; Russell et al., 1998); speakers with a laryngectomy may experience exacerbated discomfort.

Speakers with COPD have reduced VC, elevated REL, produce fewer syllables per breath group, increase their abdominal activity during both rest and speech, and produce increased expiratory flows (Lee et al., 1993). The result is that speakers with COPD maintain adequate gas exchange by shortening utterances and increasing the number of inspirations. Because of the effects of COPD on reducing elasticity in the respiratory system, speakers with COPD do not benefit by increasing their %VC at initiation. Instead, they produce increased expiratory flows, which results in their speaking well past their REL into FRC. This results in abnormal thoracoabdominal motion, with increased anterior-posterior dimensions of the thorax and paradoxical activity of the abdomen and rib cage during both rest breathing and maximal voluntary ventilation. This subsequently increases the likelihood of increased effort and probably recruitment of accessory muscles of respiration (Sharp et al., 1977) with its own negative consequences on both gas exchange and speech production.

Previous research has attempted to mimic the effects of COPD by placing typical healthy speakers in slightly hypercapnic states. This forces speakers to balance speech demands with their metabolic demands. Under these conditions, they respond by increasing %VC at speech initiation and terminating at increased VC. This is contradictory to how speakers with COPD respond. The lack of elasticity and the physiologic damage caused by COPD preclude these types of adjustments for both speech initiation and its termination. Speakers in a hypercapnic state produce fewer syllables per breath group and use increased chest wall activity. These speakers report difficulty maintaining linguistic boundaries during speech, but the fact that they attempted to maintain this structure indicates that linguistic effects are quite strong, even in high respiratory drive demands during oral reading (Bailey & Hoit, 2002). Based on their findings, Bailey and Hoit posited two models that could influence breathing. The first model is that metabolic needs and linguistic needs alternate during a breathing cycle (e.g., at one moment, metabolic needs predominate, whereas linguistic needs dominate at others). In contrast, the second model suggests the possibility that both speech and metabolic demands are adjusted simultaneously. However, both models acknowledge that linguistic and metabolic demands are important determinants of speech breathing.

Respiratory Changes Following a Total Laryngectomy

There is the likelihood that speakers with a total laryngectomy will present with COPD, with previous authors reporting rates anywhere between 70% and 81% (Ackerstaff, Hilgers, Balm, & Tan, 1998; Ackerstaff, Hilgers, Balm, & Van Zandwijk, 1995; Ackerstaff, Hilgers, Meeuwis, Knegt, & Weenink, 1999; Ackerstaff, Souren, van Zandwijk, Balm, & Hilgers, 1993; Hess, Schwenk, Frank, & Loddenkemper, 1999; Todisco, Maurizi, Paludetti, Dottorini, & Merante, 1984). To worsen matters for these individuals, pulmonary function testing is not likely to be included in standard assessment protocol in head and neck cancer patients and/or speakers with a laryngectomy (Matsuura et al., 1995). One explanation for the lack of testing is that there is inherent difficulty in the measurement of respiratory function for speakers via the tracheostoma; thus, it is clear that a need exists for improved approaches to assessment (Castro, Dedivitis, & Macedo, 2011).

VCs following a total laryngectomy are often less than 100% of their predicted value (Ackerstaff et al., 1998; Usui, 1979). Reports of VC as low as 2.5 L are common in laryngectomized speakers, a volume that is approximately 50% of that expected for adult males (Ackerstaff et al., 1998). Reduced functional expiratory volume, reduced maximum expiratory flow, peak and mean expiratory flows, and reduced residual volume are all expected in the first year following the total laryngectomy (Ackerstaff et al., 1995; Gregor & Hassman, 1984; Hess et al., 1999; Todisco et al., 1984; Togawa, Konno, & Hoshino, 1980; Usui, 1979).

Pre-laryngectomy smoking behaviors are most likely to influence respiratory health following the removal of the larynx (Ackerstaff et al., 1995; Gregor & Hassman, 1984; Hess et al., 1999; Todisco et al., 1984; Togawa et al., 1980; Usui, 1979); further, this is the primary predictor of an individual's long-term survival. Interestingly, pulmonary function improves immediately following laryngectomy and will stabilize within the first 5 months post-laryngectomy. However, there is an overall detrimental effect on quality of life and ability to maintain a healthy lifestyle (see Doyle & MacDonald, Chap. 27), with reports indicating that only about 8.5% of patients with a laryngectomy meet the physical activity guidelines of the American Cancer Society (Sammut, Ward, & Patel, 2014). This is likely to occur in speakers who have a past history of smoking, but may not apply to those who are younger and/or were of the small number of those who are laryngectomized as a result of the human papilloma virus (HPV) (see Theurer, Chap. 4).

Changes in pulmonary function are common following removal of the larynx but are not always simply related to premorbid smoking habits. For instance, the removal of the larynx and disconnection of the upper airway via the tracheostoma lead to an increased risk of infection by reducing the ciliary beat in the trachea (Todisco et al., 1984). Bacteria levels in the tracheobronchial tree increase up to 5 months post-laryngectomy and then plateau. This increase in bacteria and resulting infections may be an underlying factor in post-laryngectomy respiratory symptoms (Donnelly, 1991; Todisco et al., 1984). Additionally, the loss of the true vocal folds and larynx with the resultant need to breathe through a tracheostoma impairs resting breathing and tissue oxygenation saturation. However, the use of a humidity and moisture exchanger (HME) will help remedy these issues (see Lewis, Chap. 8).

Alaryngeal speakers are presented with a number of developmental and acquired physical changes that will affect speech. The effects of aging, respiratory compromise, and the loss of laryngeal sensation may theoretically influence breathing in speakers following a total laryngectomy, particularly the breath-to-breath control or timing of the firing of the muscles needed for forced expiration. In addition, the lack of airflow, pressure, mucosal temperature, and stretch receptor sensation due to loss of laryngeal input might alter breathing and gas exchange in these speakers. The likelihood that these speakers will suffer from COPD indicates that gas exchange and discomfort during speech may be present. Speakers who suffer from COPD are likely to manipulate the respiratory system differently than what has been reported in typical speakers placed in a hypercapnic state. Alaryngeal speakers have to overcome these respiratory influences and changes for speech. Specifically, TE speakers are the most likely to have to overcome upper airway and changes due to respiratory compromise, in addition to overcoming the increased resistance in the voice prosthesis and PE segment (see Childes, Palmer & Fried-Oken, Chap. 15). However, EL speakers and ES speakers must also adapt to these changes.

Speech Breathing in Alaryngeal Speakers

The three most common types of alaryngeal speech differ in the demands placed on the respiratory system. Speech using the electrolarynx does not require pulmonary air support, and there is a subsequent decoupling of respiratory and phonatory systems. Because of the nature of the phonatory source in EL speakers, there is no demand to maintain similar lung volumes at initiations and termination of speech as laryngeal speakers. Additionally, there is no linguistic or physiological demand to take inspirations at appropriate locations during speech or oral reading; however, the respiratory system is influenced and manipulated throughout speech in these speakers. Esophageal speech, in contrast, does not require pulmonary air support to vibrate the PE segment, but rather air is injected or inhaled into the esophagus for subsequent alaryngeal phonation which may require manipulation of the chest wall (DiCarlo, Amster, & Herer, 1955; Isshiki & Snidecor, 1965). The effects of a total laryngectomy on alaryngeal speech are most likely to be demonstrated in TE speech. The ability to rely on pulmonary air support is the perceived advantage over EL and ES speech (Robbins, Fisher, Blom, & Singer, 1984); however, the placement of a voice prosthesis can increase resistance to airflow by up to three times that of the larynx, with resistances as high as 7.5 times that of laryngeal speakers when including the pharyngoesophageal segment (Weinberg, Horii, Blom, & Singer, 1982; Weinberg & Moon, 1982; Weinberg & Moon, 1986). This changes how speakers manipulate lung volumes as well as rib cage and abdominal configurations to overcome the anecdotal reports of effortful speech.

Electrolaryngeal Speakers

Pulmonary support is not required for electrolaryngeal speech; however, different chest wall configurations in these speakers can help shed light on the effects of a laryngectomy on respiration and alaryngeal speech. EL speakers' lung volumes at initiation and termination of speech during both spontaneous speech and oral reading are similar to that of tidal breathing, and their %VC are similar to what has been previously reported in laryngeal speakers as optimal for speech ((60–36%VC); Hixon et al., 1973, Hixon et al., 1976). The reports of similar %VCs provided in Table 7.1 are misleading in that people with a total laryngectomy are likely to demonstrate elevated REL (~45%VC; Bohnenkamp et al., 2012; Bohnenkamp et al., 2011; Bohnenkamp et al., 2010; Todisco et al., 1984; Togawa et al., 1980; Usui, 1979). Subsequently, interpretation of the termination lung volumes that are similar to typical speakers is likely due to EL speakers speaking past REL and into their FRC, similar to what has been reported in COPD speakers (see Table 7.1). EL speakers, if driven by metabolic demands, would likely choose to terminate expiration closer to 45%VC in contrast to 36%VC (Hixon et al., 1973, 1976).

Though not necessary, the rib cage and abdomen both expand during inspiration and contract

Table 7.1 Summary of mean percentage and standard deviation (SD) of vital capacity at initiation and termination during speech, oral reading, and tidal breathing

Speech mode	Speech	Oral reading	Tidal breathing
EL[1]			
Initiation	60.58 (6.34)	55.49 (7.52)	59.18 (6.51)
Termination	37.39 (7.98)	41.81 (5.95)	44.91 (9.56)[a]
TE[2,3]			
Initiation	67.51 (12.01)	72.74 (14.59)	61.51 (14.23)
	53.00 (8.00)	54.00 (7.00)	53.00 (7.00)[a]
Termination	36.36 (9.88)	36.37 (9.61)	46.16 (8.52)
	35.00 (10.00)	39.00 (8.00)	39.00 (8.00)[a]

Sources: Adapted from [1]Bohnenkamp et al. (2010); [2]Bohnenkamp et al. (2011); [3]Ward et al. (2007)
[a]Represents resting expiratory level

during expiration during speech (Bohnenkamp et al., 2010; Stepp, Heaton, & Hillman, 2008). This is interesting because of the contradictions and lack of consensus in previous literature as to whether speakers have a predictable posturing behavior prior to speech (Baken & Cavallo, 1981; Baken et al., 1979; Cavallo & Baken, 1985; Hixon, 1988; McFarland & Smith, 1992; Wilder, 1983). In the first 4 months following surgery, EL speakers demonstrate chest wall posturing similar to that of typical speakers (Stepp et al., 2008).

Over time, EL speakers will posture their chest wall less than half of the time during speech and oral reading tasks, and, likely to improve efficiency, any chest wall movement associated with speech more closely resembles tidal breathing or resembles a decoupling of abdominal contraction (Bohnenkamp et al., 2010; Stepp et al., 2008). Typical kinematic behaviors are firmly established throughout adulthood; however, the lack of posturing of the abdominal wall prior to speech indicates that perhaps an EL speaker's ventilatory control during speech is less likely to follow typical movements in the absence of respiratory demand. Their chest wall movements are similar to what would be demonstrated during tidal breathing and speech and have little effect on effort in these speakers (Table 7.2).

Table 7.2 Summary of mean percentage of maximal rib cage (%RC) and maximal abdominal (%Ab) use and standard deviation (SD) during speech, oral reading, and tidal breathing

Speech mode	Speech	Oral reading	Tidal breathing
EL[1]			
%RC	32.12 (16.83)	22.99 (15.80)	26.79 (16.42)
%Ab	22.51 (9.12)	16.02 (11.24)	12.87 (7.77)
TE[2]			
%RC	31.44 (16.72)	39.76 (16.30)	33.43 (22.09)
%Ab	33.40 (11.06)	36.95 (21.70)	12.51 (9.77)

Sources: Adapted from [1]Bohnenkamp et al. (2010); [2]Bohnenkamp et al. (2012)

Further support for this decoupling in EL speakers is demonstrated in the timing of their activation of the electrolarynx with speech inspirations. Please see Table 7.3. Stepp et al. (2008) reported that EL speakers are less likely to activate the electrolarynx at the onset of expiration prior to speech. This is in contrast to that of laryngeal speakers who initiate speech on expiration. Instead, Stepp et al. reported that EL speakers are more likely to initiate the electrolarynx before peak inspiration and the onset of expiration 8–64% of the time during counting tasks and 10–44% during oral reading of the Rainbow Passage. There were individual differences by participant, but half of their EL speakers inspired during speech over 30% of the time. They were significantly more likely to inspire during speech production than ES and TE speakers. In addition, inspirations during speech production increased with increased time post-laryngectomy with one participant increasing their inspiration during speech from 19% at 4 months post-laryngectomy to 64% at 12 months. This is similar to the findings of Bohnenkamp et al. (2010) who reported that their electrolaryngeal speakers activated the

Table 7.3 Summary of mean percentage of inspirations at appropriate locations during speech and oral reading and standard deviation (SD) in EL and TE speakers

Speech mode	Speech	Oral reading
EL[1]		
All breaths	56.66 (19.76)	41.75 (12.00)
Only appropriate locations	82.03 (11.89)	80.41 (26.45)
EL pauses match inspirations	59.68 (22.47)	46.04 (26.82)
EL pauses match linguistically appropriate locations	50.29 (22.17)	36.58 (15.18)
TE[2]		
All breaths	79.30 (7.19)	77.63 (16.33)
Consistency across readings		63.67 (17.67)
Typical speakers		
All breaths	67.00 (N/A)[3]	90.00 (N/A)[4]
Consistency across readings		88.75 (6.96)[4]

Sources: Adapted from [1]Bohnenkamp et al. (2010); [2]Bohnenkamp et al. (2012); [3]Winkworth et al. (1995); Winkworth et al. (1994)

Table 7.4 Summary of mean and standard deviation (SD) of temporal measures of speech and oral reading in alaryngeal speakers compared to previous reports in typical laryngeal speakers

Speech mode	Speech	Oral reading
EL[1]		
Syllables/breath	7.45 (3.30)	8.10 (3.12)
Utterance length(s)	2.30 (1.24)	2.17 (0.97)
TE[2]		
Syllables/breath	11.27 (3.31)	12.98 (5.34)
Utterance length(s)	2.60 (1.00)	2.97 (1.06)
Typical		
Syllables/breath (50 yo)[3]	18.20 (5.68)	
Syllables/breath (75 yo)[3]	12.54 (2.41)	
Syllables/breath (~66 yo)[4]		17.50 (3.66)
Utterance length (s)	3.84 (2.05)[5]	3.36 (1.39)[6]

Sources: Adapted from [1]Bohnenkamp et al. (2010); [2]Bohnenkamp et al. (2012); [3]Hoit and Hixon (1987); [4]Solomon and Hixon (1993); [5]Winkworth et al. (1995); [6]Winkworth et al. (1994)

electrolarynx prior to onset of expiration 62% of the time during spontaneous speech (range = 16–83%) and 58% of the time during reading (range = 10–96%). Similar to Stepp et al., as time post-laryngectomy increased, EL speakers were more likely to inspire during speech production (Doyle, 2005). In the absence of the physiological need for expiration for speech production, EL speakers appear to rely on a speech breathing pattern that more closely resembles tidal breathing (see Nagle, Chap. 9). This might prove more comfortable or efficient and demonstrates the likelihood that they decouple speech demands from respiratory system control.

EL speakers are similar to laryngeal speakers in that they inspire at grammatically appropriate locations during spontaneous speech (Winkworth et al., 1995). This is interesting, considering that these speakers can mark grammatical boundaries using the on-off control of the electrolarynx versus inspirations and inspiratory pauses; however, they do not maintain this similarity during oral reading tasks. Bohnenkamp et al. (2010) reported that EL speakers were much less likely to inspire at grammatically appropriate locations during oral reading, which might indicate that oral reading requires less cognitive effort along with a lack of the physiological need to mark pauses. EL speakers do not have the limitations of having to balance linguistic demands of oral reading and speech and could demonstrate longer utterances than laryngeal and TE speakers; however, they produce utterance lengths shorter than what has been reported in typical and TE speakers (Ward et al., 2007; Winkworth et al., 1994; Winkworth et al., 1995). Please see Table 7.4. As previously discussed, speech breathing is a complex activity in laryngeal speakers; however, EL speakers demonstrate a complexity of a different sort in that they decouple their control of the respiratory system and demonstrate variable and unpredictable responses to loss of a physiological phonatory source.

Esophageal Speakers

Esophageal speech does not require pulmonary air support to vibrate the PE segment, but rather, air is injected or inhaled into the esophagus for alaryngeal phonation (see Doyle & Finchem, Chap. 10). There is little research related to the speech breathing behaviors of ES speakers. ES speakers do coordinate their inspiratory movements with both injection and inhalation methods of esophageal insufflation (DiCarlo et al., 1955). DiCarlo et al. investigated how ES speakers manipulated the respiratory system for speech during tidal breathing and while oral reading. The ES speakers used greater rib cage and abdominal movement during tidal breathing, which the authors suggested were likely due to the effects of COPD. In general, ES speakers used inspiratory and expiratory chest wall movements similar to laryngeal speakers during speech, though the amplitudes of chest wall movement during speech in ES speakers were less than seen in laryngeal speakers. The esophageal speakers who were rated as most intelligible used chest wall movements that were most similar to those of typical speakers, whereas poor speakers demonstrated dyscoordination of the inspiratory and injection activity with both the inspiratory and expiratory phases. As would be expected, all ES speakers demonstrated shorter utterance lengths than laryngeal speakers.

Tracheoesophageal Speakers

The effects of a total laryngectomy on respiration in alaryngeal speech are most likely to be demonstrated in TE speech. The ability to use pulmonary air for TE speech is the perceived advantage over EL and ES speech to overcome the anecdotal reports of effortful speech. Highly intelligible (90%) TE speakers initiate and terminate spontaneous speech similar to laryngeal speakers (Bohnenkamp et al., 2012; Bohnenkamp et al., 2011; Ward et al., 2007). The termination of speech near 36%VC in TE speech is misleading because speakers who undergo a laryngectomy have elevated REL (i.e., approximately 9% higher). Bohnenkamp et al. (2011) reported that speakers terminated speech at levels 10% lower than their REL (35–45%VC) and exclusively spoke well below REL to complete utterances. Ward et al. did not state whether their speakers consistently spoke past REL. Whether these behaviors would be similar in alaryngeal speakers with low intelligibility warrants investigation.

The middle range of lung volumes (60–35%VC) reported in typical speakers is thought to be the optimal configuration for speech; however, TE speakers have reduced passive forces available to them for speech due to the elevated REL. As such, TE speakers use breathing behaviors that are closely related to what has been reported in older speakers, as well as speakers with COPD (Hoit & Hixon, 1987; Lee et al., 1993). TE speakers terminate speech at low lung volumes into FRC versus their inspiring to higher %VC because of the increased effort involved. TE speakers seem to prefer to overcome PE segment and TE puncture voice prosthesis resistance by increasing muscular effort at the ends of utterances, as opposed to increasing inspirations at the beginning. In addition, these speakers may not initiate speech at %VC as a way to control for comfortable loudness (see Table 7.1).

If diagnosed with COPD, TE speakers are likely to use more rib cage activity during oral reading than is used for spontaneous speech (Bohnenkamp et al., 2012). In fact, TE speakers with COPD used 49% of their maximal rib cage movement while reading orally, whereas they relied on only 34% of maximal rib cage movement for speech. This agrees with reports that respiratory difficulties associated with COPD include the loss of elasticity in the respiratory system resulting in the need for increased muscle activity. The use of a greater amount of rib cage activity during expiration is contradictory to the physiologically efficient use by healthy older speakers of taking deeper inspirations for speech.

Abdominal activity during speech in tracheoesophageal speakers is similar during both oral reading (37% of maximum) and speech (33% or maximum), and both instances are considerably higher than previous reports of 7–10% of maximal abdominal movement in laryngeal speakers

(Hoit & Hixon, 1987). TE speakers do contract the abdominal wall prior to phonation nearly 100% of the time. This is likely due to the need to maintain adequate pressures in the trachea needed to force both the TE puncture voice prosthesis and PE segment open for comfortable and efficient speech. However, there is the probability that these active compensations may to lead to fatigue, even if increased effort in the abdominal wall is the most efficient and optimal configuration of the chest wall for TE speakers (see Table 7.2).

The Relationship of Speech Intelligibility and Temporal Measures of Speech

TE speakers produce similar rates of speech compared to typical speakers and are likely to take inspirations at grammatically appropriate locations during speech and oral reading. Though TE speakers' speaking rate is comparable to laryngeal speakers' rates, TE speakers produce fewer syllables per breath group and produce utterances that are approximately two-thirds as long as what would be expected in laryngeal speakers (Bohnenkamp et al., 2012; Bohnenkamp et al., 2011). Please see Tables 7.3 and 7.4. It has been argued that intelligibility in TE speakers could be acoustic-related, indicating a lack of power in the phonatory source that results in reduced formant frequencies (D'Alatri, Bussu, Scarano, Paludetti, & Marchese, 2012). But there is also the likelihood that intelligibility is influenced by the speakers' shorter utterance length regardless of their ability to use grammatically appropriate inspiration patterns. One explanation may be that TE speakers may address concerns regarding intelligibility by shortening utterances and inspiring at times which are grammatically appropriate during speech. Oral reading requires TE speakers to rely on punctuation to mark inspiratory locations and pauses. Subsequently, TE speakers will inspire to a perceived appropriate %VC and terminate speech at lower %VC to complete the utterance. The alternative would be that these speakers are forced to reduce the number of syllables produced per breath group to balance metabolic demands. Therefore, TE speakers appear to preplan their utterances to make use of the amount of air available to them. This is not the case during oral reading, wherein the grammatical structure is specified by the passage's linguistic construction. TE speakers must adapt their respiratory control to the structure of the utterance. This is accomplished primarily by initiating speech at a high lung volume and speaking into FRC, with the only other alternative to take inspirations when physiologically necessary (Bohnenkamp et al., 2012).

Conclusions

The demands placed on alaryngeal speakers differ by their mode of communication. EL speakers, who have the least need for respiratory control for speech, continue to manipulate the system as if timing for speech. However, this speech-related behavior appears to change with increasing post-laryngectomy time as speakers begin decoupling respiration from speech to most efficiently produce speech with the least demand on the system. It is likely that ES speakers continue to manipulate the chest wall similar to laryngeal speakers, depending upon their method of charging air into the esophagus. There is, however, a dearth of research in this population which indicates that much more can be done. TE speech production might be very demanding on the respiratory system, even in intelligible speakers. Alaryngeal speakers' use of greater lung volumes during speech and oral reading and consistently speaking into FRC influences their ability to place grammatically appropriate inspirations during speech and oral reading. Targeting utterance length and specifying locations of inspirations may be viable therapy goals for these speakers.

Finally, TE speech is viewed very favorably in that it allows speakers to use pulmonary air support and subsequently produce longer utterances which may improve their communication effectiveness. However, the literature also indicates that TE speech is effortful and that EL and ES speech remain viable approaches to speech rehabilitation, especially in those who suffer a severely compromised respiratory system. It is, therefore, essential that clinicians understand that there is a physiological cost associated with speech breathing in all three alaryngeal speech modes. Complex control of the respiratory system in typical healthy laryngeal speakers is influenced by numerous predictable factors such as age and speech task. The remarkable communication challenges faced by alaryngeal speakers (often with respiratory compromise such as that related to COPD) and their ability to communicate effectively, regardless of speech mode, speak to a robust physiological system that is very adaptive to change and can maintain functionality under severely increased physical and communicative challenges.

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References

Ackerstaff, A. H., Hilgers, F. J., Balm, A. J., & Tan, I. B. (1998). Long-term compliance of laryngectomized patients with a specialized pulmonary rehabilitation device: Provox stomafilter. *Laryngoscope, 108*, 257–260.

Ackerstaff, A. H., Hilgers, F. J., Balm, A. J., & Van Zandwijk, N. (1995). Long-term pulmonary function after total laryngectomy. *Clinical Otolaryngology and Allied Sciences, 20*, 547–551.

Ackerstaff, A. H., Hilgers, F. J., Meeuwis, C. A., Knegt, P. P. M., & Weenink, C. (1999). Pulmonary function pre- and post-total laryngectomy. *Clinical Otolaryngology, 24*, 491–494.

Ackerstaff, A. H., Souren, T., van Zandwijk, N., Balm, A. J., & Hilgers, F. J. (1993). Improvements in the assessment of pulmonary function in laryngectomized patients. *Laryngoscope, 103*, 1391–1394.

Ayres, J. G. (1990). Late onset asthma. *British Medical Journal, 300*, 1602–1603.

Bailey, E. F., & Hoit, J. D. (2002). Speaking and breathing in high respiratory drive. *Journal of Speech, Language and Hearing Research, 45*, 89–99.

Baken, R. J., & Cavallo, S. A. (1981). Prephonatory chest wall posturing. *Folia Phoniatrica, 33*, 193–203.

Baken, R. J., Cavallo, S. A., & Weissman, K. L. (1979). Chest wall movements prior to phonation. *Journal of Speech and Hearing Research, 22*, 862–871.

Bohnenkamp, T. A., Forrest, K. M., Klaben, B. K., & Stager, J. M. (2011). Lung volumes used during speech breathing in tracheoesophageal speakers. *Annals of Otology, Rhinology, & Laryngology, 120*, 550–558.

Bohnenkamp, T. A., Forrest, K. M., Klaben, B. K., & Stager, J. M. (2012). Chest wall kinematics during speech breathing in tracheoesophageal speakers. *Annals of Otology, Rhinology, & Laryngology, 121*, 28–37.

Bohnenkamp, T. A., Stowell, T., Hesse, J., & Wright, S. (2010). Speech breathing in speakers who use an electrolarynx. *Journal of Communication Disorders, 43*, 199–211.

Brown, M., & Hasser, E. (1996). Complexity of age-related change in skeletal muscle. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 51*, 117–123.

Bunn, J. C., & Mead, J. (1971). Control of ventilation during speech. *Journal of Applied Physiology, 31*, 870–872.

Castro, M. A., Dedivitis, R. A., & Macedo, A. G. (2011). Evaluation of a method for assessing pulmonary function in laryngectomees. *Acta Otorhinolaryngologica Italica, 31*, 243–247.

Cavallo, S. A., & Baken, R. J. (1985). Prephonatory laryngeal and chest wall dynamics. *Journal of Speech and Hearing Research, 28*, 79–87.

Chan, E. D., & Welsh, C. H. (1998). Geriatric respiratory medicine. *Chest, 114*, 1704–1733.

Corfield, D. R., Roberts, C. A., Guz, A., Murphy, K., & Adams, L. (1999). Modulation of the corticospinal control of ventilation by changes in reflex drive. *Journal of Applied Physiology, 87*, 1923–1930.

Crapo, R. O. (1993). The aging lung. In D. A. Mahler (Ed.), *Pulmonary disease in the elderly patient* (Vol. 63, pp. 1–21). New York, NY: Marcel Dekker.

D'Alatri, L., Bussu, F., Scarano, E., Paludetti, G., & Marchese, M. R. (2012). Objective and subjective assessment of tracheoesophageal prosthesis voice outcome. *Journal of Voice, 26*, 607–613. https://doi.org/10.1016/j.jvoice.2011.08.013

Davis, P. J., Zhang, S. P., Winkworth, A., & Bandler, R. (1996). Neural control of vocalization: Respiratory and emotional influences. *Journal of Voice, 10*, 23–38.

Dhar, S., Shastri, S. R., & Lenora, R. A. K. (1976). Aging and the respiratory system. *Medical Clinics of North America, 60*, 1121–1139.

DiCarlo, L. M., Amster, W. W., & Herer, G. R. (1955). *Speech after laryngectomy*. Syracuse, NY: Syracuse University Press.

Dick, T. E., Orem, J. M., & Shea, S. A. (1997). Behavioral control of breathing. In R. G. Crystal, J. B. West, E. R. Weibel, & P. J. Barnes (Eds.), *The lung: Scientific foundations* (2nd ed., pp. 1821–1837). Baltimore, MD: Lippincott-Raven.

Donnelly, D. F. (1991). Laryngeal reflexes: Integrated and cellular aspects. In G. G. Haddad & J. P. Farber (Eds.), *Developmental neurobiology of breathing* (Vol. 53, pp. 271–278). New York, NY: Marcel-Dekker.

Doust, J. H., & Patrick, J. M. (1981). The limitation of exercise ventilation during speech. *Respiration Physiology, 46*, 137–147.

Doyle, P. C. (2005). Clinical procedures for training use of the electronic artificial larynx. In P. C. Doyle & R. L. Keith (Eds.), *Rehabilitation following treatment for head and neck cancer: Voice, speech, and swallowing*. Austin, TX: Pro-Ed Publishers.

Draper, M. H., Ladefoged, P., & Whitteridge, D. (1959). Respiratory muscles in speech. *Journal of Speech and Hearing Research, 2*, 16–27.

Duffy, J. (1995). *Motor speech disorders: Substrates, differential diagnosis, and management*. New York, NY: Mosby.

Fontana, G. A., Pantaleo, T., Lavorini, F., Mutolo, D., Polli, G., & Pistolesi, M. (1999). Coughing in laryngectomized patients. *American Journal of Respiratory and Critical Care Medicine, 160*, 1578–1584.

Garrett, J. D., & Luschei, E. S. (1987). Subglottic pressure modulation during evoked phonation in the anesthetized cat. In T. Baer, C. Sasaki, & K. Harris (Eds.), *Laryngeal function in phonation and respiration* (pp. 139–153). Boston, MA: College Hill Press.

Goldman-Eisler, F. (1961). Continuity of speech utterance, its determinants and its significance. *Language and Speech, 4*, 220–231.

Goldman-Eisler, F. (1968). *Psycholinguistics: Experiments in spontaneous speech*. Ann Arbor, MI: Academic Press.

Gregor, R. T., & Hassman, E. (1984). Respiratory function in post-laryngectomy patients related to stomal size. *Acta Otolaryngologica, 97*, 177–183.

Guz, A. (1997). Brain, breathing and breathlessness. *Respiration Physiology, 109*, 197–204.

Hess, M. M., Schwenk, R. A., Frank, W., & Loddenkemper, R. (1999). Pulmonary function after total laryngectomy. *The Laryngoscope, 109*, 988–994.

Hida, W. (1999). Role of ventilatory drive in asthma and chronic obstructive pulmonary disease. *Current Opinion in Pulmonary Medicine, 5*, 339–343.

Hixon, T. J. (1988). Comment on Cavallo and Baken (1985). *Journal of Speech, Language, and Hearing Research, 31*, 726–728.

Hixon, T. J., Goldman, M. D., & Mead, J. (1973). Kinematics of the chest wall during speech production: Volume displacements of the rib cage, abdomen, and lung. *Journal of Speech and Hearing Research, 16*, 78–115.

Hixon, T. J., Mead, J., & Goldman, M. D. (1976). Dynamics of the chest wall during speech production: Function of the thorax, rib cage, diaphragm, and abdomen. *Journal of Speech and Hearing Research, 19*, 297–356.

Hoit, J. D., & Hixon, T. J. (1987). Age and speech breathing. *Journal of Speech, Language, and Hearing Research, 30*, 351–366.

Hoit, J. D., & Lohmeier, H. L. (2000). Influence of continuous speaking on ventilation. *Journal of Speech, Language, and Hearing Research, 43*, 1240–1251.

Homma, I., Obata, T., Sibuya, M., & Uchida, M. (1984). Gate mechanism in breathlessness caused by chest wall vibration in humans. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 56*, 8–11.

Hugelin, A. (1986). Forebrain and midbrain influences on respiration. In *Handbook of physiology, the respiratory system* (Vol. 2). Bethesda, MD: American Physiological Society.

Isshiki, N., & Snidecor, J. C. (1965). Air intake and usage in esophageal speech. *Acta Otolaryngologica, 59*, 559–574.

Iwarsson, J. (2001). Effects of inhalatory abdominal wall movement on vertical laryngeal position during phonation. *Journal of Voice, 15*(3), 384–394.

Janssens, J. P., Pache, J. C., & Nicod, L. P. (1999). Physiological changes in respiratory function associated with ageing. *European Respiratory Journal, 13*, 197–205.

Kahane, J. C. (1980). Age-related histological changes in the human male and female laryngeal cartilages: Biological and functional implications. In V. Lawrence (Ed.), *Transcripts of the ninth symposium: Care of the professional voice*. New York, NY: The Voice Foundation.

Kaneko, Y., Zivanovic, V., Hajek, V., & Bradley, T. D. (2001, November 11). *Sleep apnea in stroke patients predicts worse functional status and longer hospitalization*. Paper presented at: Annual Meeting of the American Heart Association, Anaheim, CA.

Kendall, K. (2007). Presbyphonia: A review. *Current Opinion in Otolaryngology & Head and Neck Surgery, 15*, 137–140.

Khedr, E. M., Shinaway, O. E., Khedr, T., Aziz Ali, Y. A., & Awad, E. M. (2000). Assessment of corticodiaphragmatic pathway and pulmonary function in acute ischemic stroke patients. *European Journal of Neurology, 7*, 323–330.

Ladefoged, P., & Loeb, G. (2002). *Preliminary studies on respiratory activity in speech.* Unpublished manuscript, University of California-Los Angeles.

Lee, L., Loudon, R. G., Jacobson, B. H., & Stuebing, R. (1993). Speech breathing in patients with lung disease. *American Review of Respiratory Disease, 147*, 1199–1206.

Matsuura, K., Ebihara, S., Yoshizumi, T., Asai, M., Hayashi, R., Shizuka, T., & Uchiyama, K. (1995). Changes in respiratory function before and after laryngectomy. *Nippon Jibiinkoka Gakkai Kaiho, 98*, 1097–1103.

McFarland, D. H., & Smith, A. (1992). Effects of vocal task and respiratory phase on prephonatory chest wall movements. *Journal of Speech and Hearing Research, 35*, 971–982.

McKeown, F. (1965). *Pathology of the aged*. London: Butterworths.

Murray, J. F. (1986). Aging. In J. F. Murray (Ed.), *The normal lung*. Philadelphia, PA: W. B. Saunders.

Ohala, J. J. (1990). Respiratory activity in speech. In W. J. Hardcastle & A. Marchal (Eds.), *Speech production and speech modelling. Vol. 55. Series D: Behavioural and social science* (pp. 23–53). Boston, MA: Kluwer Academic Publishers.

Phillipson, E. A., McClean, P. A., Sullivan, C. E., & Zamel, N. (1978). Interaction of metabolic and behavioral respiratory control during hypercapnia and speech. *American Review of Respiratory Disease, 117*, 903–909.

Pierce, J. A., & Ebert, R. V. (1965). Fibrous network of the lung and its change with age. *Thorax, 20*, 469–476.

Robbins, J., Fisher, H. B., Blom, E. C., & Singer, M. I. (1984). A comparative acoustic study of normal, esophageal, and tracheoesophageal speech production. *Journal of Speech and Hearing Disorders, 49*, 202–210.

Russell, N. K., Cerny, F. J., & Stathopoulos, E. (1998). Effects of varied vocal intensity on ventilation and energy expenditure in women and men. *Journal of Speech, Language, and Hearing Research, 41*, 239–248.

Sammut, L., Ward, M., & Patel, N. (2014). Physical activity and quality of life in head and neck cancer survivors: A literature review. *International Journal of Sports Medicine, 35*, 794–799.

Sant'Ambrogio, G., Matthew, O. P., Fisher, J. T., & Sant'Ambrogio, F. B. (1983). Laryngeal receptors responding to transmural pressure, airflow, and local muscle activity. *Respiratory Physiology and Neurobiology, 54*, 317–330.

Sharp, J. T., Goldberg, N. B., Druz, W. S., Fishman, H. C., & Danon, J. (1977). Thoracoabdominal motion in chronic obstructive pulmonary disease. *American Review of Respiratory Disease, 115*, 47–56.

Shea, S. A. (1996). Behavioural and arousal-related influences on breathing in humans. *Experimental Physiology, 81*, 1–26.

Solomon, N. P., & Hixon, T. J. (1993). Speech breathing in Parkinson's disease. *Journal of Speech and Hearing Research, 36*, 294–310.

Sperry, E. E., & Klich, R. J. (1992). Speech breathing in senescent and younger women during oral reading. *Journal of Speech and Hearing Research, 35*, 1246–1255.

Stepp, C. E., Heaton, J. T., & Hillman, R. E. (2008). Postlaryngectomy speech respiration patterns. *Annals of Otology, Rhinology & Laryngology, 117*, 557–563.

Terao, Y., Ugawa, Y., Enomoto, H., Furubayashi, T., Shiio, Y., Machii, K., … Kanazawa, I. (2001). Hemispheric lateralization in the cortical motor preparation for human vocalization. *The Journal of Neuroscience, 21*, 1600–1609.

Testerman, R. L. (1970). Modulation of laryngeal activity by pulmonary changes during vocalization in cats. *Experimental Neurology, 29*, 281–297.

Todisco, T., Maurizi, M., Paludetti, G., Dottorini, M., & Merante, F. (1984). Laryngeal cancer: Long-term follow-up of respiratory functions after laryngectomy. *Respiration, 45*, 303–315.

Togawa, K., Konno, A., & Hoshino, T. (1980). A physiologic study on respiratory handicap of the laryngectomized. *Archives of Oto-Rhino-Laryngology, 229*, 69–79.

Tolep, K., & Kelsen, S. G. (1993). Effect of aging on respiratory skeletal muscles. *Clinics in Chest Medicine, 14*, 363–378.

Usui, N. (1979). Ventilatory function in laryngectomized patients. *Auris Nasus Larynx, 6*, 87–96.

von Euler, C. (1997). Neural organization and rhythm generation. In R. G. Crystal, J. B. West, E. R. Weibel, & P. J. Barnes (Eds.), *The lung: Scientific foundations* (2nd ed., pp. 1710–1724). Baltimore, MD: Lippincott-Raven.

Ward, E. C., Hartwig, P., Scott, J., Trickey, M., Cahill, L., & Hancock, K. (2007). Speech breathing patterns during tracheoesophageal speech. *Asia Pacific Journal of Speech, Language, and Hearing, 10*, 33–42.

Warren, D. W. (1996). Regulation of speech aerodynamics. In N. J. Lass (Ed.), *Principles of experimental phonetics* (pp. 46–92). Saint Louis, MO: Mosby-Year Book, Inc.

Weinberg, B., Horii, Y., Blom, E., & Singer, M. (1982). Airway resistance during esophageal phonation. *Journal of Speech and Hearing Disorders, 47*, 194–199.

Weinberg, B., & Moon, J. (1982). Airway resistance characteristics of Blom-Singer tracheoesophageal puncture prostheses. *Journal of Speech and Hearing Disorders, 47*, 441–442.

Weinberg, B., & Moon, J. B. (1986). Impact of tracheoesophageal puncture prosthesis airway resistance on in-vivo phonatory performance. *Journal of Speech and Hearing Disorders, 51*(1), 88–91.

Wessendorf, T. E., Teschler, H., Wang, Y.-M., Konietzko, N., & Thilman, A. F. (2000). Sleep-disordered breathing among patients with first-ever stroke. *Journal of Neurology, 247*, 41–47.

West, J. B. (2013). *Pulmonary pathophysiology: The essentials* (8th ed.). Baltimore, MD: Wolters Kluwer – Lippincott Williams & Wilkins.

Wilder, C. N. (1983). Chest wall preparation for phonation in female speakers. In D. M. Bless & J. H. Abbs (Eds.), *Vocal fold physiology: Contemporary research and clinical issues* (pp. 109–123). San Diego, CA: College Hill Press.

Winkworth, A. L., Davis, P. J., Adams, R. D., & Ellis, E. (1995). Breathing patterns during spontaneous speech. *Journal or Speech and Hearing Research, 38*(1), 124–144.

Winkworth, A. L., Davis, P. J., Ellis, E., & Adams, R. D. (1994). Variability and consistency in speech breathing during reading: Lung volumes, speech intensity, and linguistic factors. *Journal of Speech and Hearing Research, 37*, 535–556.

Wyke, B. (1983). Neuromuscular control systems in voice production. In D. M. Bless & J. H. Abbs (Eds.), *Vocal fold physiology* (pp. 71–76). San Diego, CA: College-Hill.