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LARYNGEAL FUNCTION ASSOCIATED WITH CHANGES IN LUNG VOLUME DURING VOICE AND SPEECH PRODUCTION IN NORMAL SPEAKING WOMEN

by

Claudio F. Milstein

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A Dissertation Submitted to the Faculty of the
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1999
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Claudio F. Milstein entitled Laryngeal Function Associated with Changes in Lung Volume During Voice and Speech Production in Normal Speaking Women and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy.

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I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

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SIGNED: [Signature]
DEDICATION

This work is dedicated to the memory of two men of science, my father Mario Milstein, and my friend and mentor Tom Shipp.
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ABSTRACT

The present study explored possible relations between respiratory and laryngeal function associated with changes in lung volume level during phonation. Respiratory, acoustic, aerodynamic, electroglottographic, and videostroboscopic measures were obtained simultaneously for 14 normal female speakers as they sustained vowels and syllable repetitions throughout the vital capacity. Statistical analyses compared group performances between (a) high and mid lung volumes; and (b) mid and low lung volume levels. Significant differences were found for (1) vertical laryngeal position (VLP); (2) amplitude of vocal fold vibration; (3) minimum flow; (4) degree of glottal closure, and (5) degree of laryngeal compression. Results indicated that during phonation at high lung volumes the larynx displays an overall “more dilated” configuration with a lower position in the neck, larger amplitudes of vocal fold vibration and larger posterior glottal gaps during the closed phase of vocal fold vibration. Conversely, during phonation at low lung volumes the larynx seems to adopt a more constricted configuration, with a more elevated position, smaller amplitudes of vocal fold vibration, more complete glottal closure during the closed phase of vocal fold vibration, and increased degree of compression. Results also indicated that while some vocal function measures displayed different absolute values for sustained vowels as compared to syllable repetitions, the patterns of change were similar for both speech tasks. Individual subject data reflected alternative patterns of laryngeal behavior for achieving the speech tasks. The results were interpreted as evidence that laryngeal function during voice production is clearly influenced by the lung volume at which phonation is produced. Possible underlying physiological mechanisms are discussed. These findings contribute to better understanding of the normal vocal mechanism when phonation is produced outside of the normal mid-range of lung volumes typically used in conversational speech.
CHAPTER I

INTRODUCTION AND STATEMENT OF THE PROBLEM

The interactions between the respiratory and laryngeal systems have been acknowledged for 25 centuries, since Hippocrates, in the 5th century BC, first contemplated the possibility that the lungs played a role in the production of voice. This concept was further developed by Claudius Galen (131-200 AD), who suggested that the lungs and trachea "prepared and pre-arranged the voice for the larynx" (von Laden, 1993). Science has advanced much in the understanding of how these two systems interact. Not only does the respiratory system play an important role in phonation, but the larynx plays an important role in respiration as well.

Because of its active role in respiration, the larynx is considered as part of the respiratory system. Before birth, the larynx regulates the outward flow of amniotic fluid, and it may play a role in pulmonary development (Harding, 1984). Studies in neonates both human and animal, clearly indicate that the larynx plays a crucial role in respiration. During the expiratory phase, there is an increase in laryngeal airway resistance. Neonates have a very compliant chest wall, and as a result, the resting position of the respiratory system is at relatively low lung volumes. The mechanism of increasing laryngeal airway resistance, or "laryngeal braking" preserves lung volume and maintains airway patency by increasing lung volume at end expiration (Fisher, Mortola, Smith, Fox, & Weeks, 1982; Harding, 1984). Movements of the vocal folds coordinated with breathing continue through the life span. They are characterized by abduction of the vocal folds on inspiration, with activation of the posterior cricoarytenoid, and cricothyroid muscles; and adduction on expiration, with lateral cricoarytenoid, and thyroarytenoid muscles activation. These movements regulate glottal aperture and the resulting transglottal airflow. The respiratory activity of the larynx has been shown to be influenced by sleep state (Harding, England, Stradling, Kozar, & Phillipson, 1986; Kuna, Insalaco, &
Woodson, 1988; Kuna, McCarthy, & Smickley, 1993; Kuna, Smickley, & Insalaco, 1990; Marchal, Crance, & Arnould, 1986). Its activity is heightened during hypercapnic and hyperpneic states (Bartlett, 1979; Bartlett, Remmers, & Gautier, 1973; Campbel, Murtagh, & Raber, 1963; Megirian & Sherrey, 1980; Stransky, Szereda-Przestaszewska, & Widdicombe, 1973), and is particularly important in regulation of respiration during high ventilatory output states such as exercising.

Given that laryngeal function is not only essential to prevent foreign particles from entering the lower airways, but also in regulating the flow of air in and out of the lungs during breathing and phonation, it is not unexpected to find shared neural structures and pathways involved in the control of both respiratory and laryngeal muscles. Brainstem motoneuron pools that regulate respiratory patterns and neuronal groups that are considered to be part of the vocalization system are interconnected. Brainstem structures such as the nucleus retroambigualis, and the periaqueductal gray, are believed to play a role in integrating the activity and timing of respiratory, laryngeal and oral muscle activity during voicing (Davis, Zhang, & Bandler, 1993; Davis, Zhang, Winkworth, & Bandler, 1996; Holstege, 1989). Laryngeal activity has also been shown to be strongly influenced by pulmonary reflexes that are associated with lung volume. There is a coordinated set of reflexes mediated by pulmonary stretch receptors that influence not only breathing, but laryngeal function as well. There is evidence that the Hering-Breuer reflex inhibits not only respiratory muscle activity, but respiratory activity of the laryngeal muscles such as the PCA and TA (Bartlett et al., 1973; Fukuda, Sasaki, & Kirchner, 1973; Tully, Brancatisano, Loring, & Engel, 1990; Tully, Brancatisano, Loring, & Engel, 1991). Moreover, it has been shown that when afferent nerve endings in the lungs are stimulated by forced pulmonary deflation, the larynx reacts to this stimulation by increasing electromyographic activity of the intrinsic adductor muscles (Dixon, Szereda-Przestaszewska, Widdicombe, & Wise, 1974; Glogowska, Stransky, & Widdicombe, 1974; Stransky et al., 1973). This phenomenon of increased expiratory laryngeal resistance to airflow elicited by lung reflexes, seems to be very robust and has
been often used by investigators as a means to activate the laryngeal constrictors (Brancatisano, Kelly, Tully, & Engel, 1987).

Although involvement of respiratory function has frequently been suggested as a contributing factor in voice disorders (Aronson, 1990; Wilson, 1987); there is a paucity of empirical data on respiratory function in the voice-disordered population (Sapienza, Stathopoulos, & Brown, 1997). Voice clinicians often subjectively identify or assume the presence of anomalous respiratory behaviors in patients with hyperfunctionally-related disorders. In such cases, clinicians often work on “improving” breathing for voice and speech production. Recently, objective support for the presence of respiratory involvement in hyperfunctional voice disorders was provided by several investigations (Hillman, Holmberg, Perkell, & Kobler, 1997; Sapienza & Stathopoulos, 1994; Sapienza et al., 1997; Sperry, Hillman, & Perkell, 1994). These studies showed evidence that there are significant changes in the speech breathing of women with vocal nodules as compared to matched controls, and that some women with vocal nodules use lung volumes for speech that are below the range that normals typically use, displaying expiratory excursions well below the resting expiratory level (REL). However, much more research is needed to more fully understand the association between vocal hyperfunction and altered speech respiratory behaviors. A particularly important question is whether these alterations in respiratory behaviors developed before (i.e., an etiologic component) or after (i.e., a reactive component) the onset of the laryngeal manifestations of the voice disorder.

Even though the voice appears to be affected during phonation at low lung volumes, there have been very few direct investigations of the interaction (interdependence) between the respiratory and laryngeal systems when phonation is produced outside the range of lung volumes typically used during normal speech production. Studies that investigated phonatory function at different lung volumes suggested that laryngeal function is in fact affected by lung volume during phonation.
(Hoit, Solomon, & Hixon, 1993; Iwarsson, Thomasson, & Sundberg, 1995; Shipp, Morrissey, & Haglund, 1983; Sundberg, Scherer, & Titze, 1993; Zenker, 1964). These changes seem to occur primarily at both lung volume extremes. Previous findings related to laryngeal adjustments that occur during phonation at different lung volumes include: a) changes in vertical laryngeal position (VLP) (Iwarsson et al., 1995; Sundberg et al., 1993; Zenker, 1964); b) changes in voice onset time (VOT) (Hoit et al., 1993); c) changes in electromyographic activity of the cricothyroid muscles (Shipp et al., 1983; Sundberg et al., 1993); and d) changes in subglottal pressures and flow closed quotients (Iwarsson et al., 1995). Although the information provided by these studies is valuable, the data were limited by the fact that comparative measures were often not obtained simultaneously, results regarding cricothyroid muscle activity are conflicting, there were small number of subjects per study, and no study has been systematically replicated. Even though it is difficult to generalize from these studies, some of the laryngeal manifestations reported to occur during phonation at low lung volumes, (i.e. strained phonation, and increased degree of glottal adduction) are usually considered signs of vocal hyperfunction. Consequently, it is possible that phonation at lung volumes below the range of volumes typically used for speech production may elicit signs of laryngeal hyperfunction in normal speakers. Nonetheless, this assumption has not been systematically evaluated, and thus, more research is needed.

In summary, given that: (a) some of the same organs and muscle groups form part of both the respiratory and laryngeal systems (b) the neural control of respiration and voicing may involve shared neural structures and pathways; (c) there is evidence that the larynx is influenced by respiratory reflexes evoked by lung volume; (d) involvement of respiratory function is suspected in hyperfunctional voice disorders; and (e) there is some evidence that laryngeal adjustments are elicited by phonation at high and low lung volumes, the aims of this investigation are to determine the impact on normal laryngeal phonatory function of producing voice and speech at different lung volume levels throughout the vital capacity (VC) range. Of particular interest is determining the extent to which
normal speakers display evidence of vocal hyperfunction at lung volumes below the end expiratory level (EEL).

The specific questions to be answered are: (1) does vertical laryngeal position during phonation change as a function of lung volume? (2) does glottal adduction vary significantly during phonation as a function of lung volume across the entire lung volume range? (3) does the degree of glottal closure during phonation vary significantly as a function of lung volume? (4) is a significant increase in glottal and supraglottal compression elicited by phonation at low lung volumes (i.e. below EEL)? (5) do the data suggest which are the possible mechanisms that may mediate laryngeal adjustments to changes in lung volume?

Based on the findings of previous studies and on pilot data for this investigation, it is predicted that the larynx will undergo the following adjustments when phonating throughout the vital capacity: a lowered vertical laryngeal position during phonation at high lung volumes, and a gradual increase in glottal adduction, glottal closure and supraglottal compression as phonation progresses towards the lower end of the VC. All predictions are formulated relative to normal function.

The specific predictions are:

1) Vertical laryngeal position will be lower at high lung volumes and increase towards the mid-lung volume range as evidenced by a gradually increasing EGG laryngeal-tracking signal.
2) The magnitude of the transglottal flow will decrease towards the low lung volumes.
3) The magnitude of glottal closure will gradually increase towards the low lung volumes resulting in smaller posterior glottal gaps or complete closure of the posterior glottis.
4) The magnitude of minimum flow will gradually decrease because of the gradual increase in glottal closure towards the low lung volumes.

5) There will be an increase in laryngeal compression during phonation at the lower end of the VC, resulting in increased ventricular medialization and/or increased antero-posterior compression.
CHAPTER II.
REVIEW OF THE LITERATURE

This review will summarize available literature on: 1) the respiratory system, focusing on normal physiology and speech breathing; 2) the laryngeal system, focusing on normal phonatory physiology; 3) vocal hyperfunction; and 4) laryngeal dynamics as a function of lung volume.

The Respiratory System

The primary function of respiration is to exchange oxygen and carbon dioxide in order to keep homeostasis during all physiological circumstances (Proctor, 1986b). The regulation of arterial blood gases is accomplished by movement of air to and from gas-exchanging surfaces that result in provision of oxygen to, and removal of carbon dioxide from the cells of the body. The gas exchange is achieved by the mechanical action of the respiratory system, which consists of two cavities, the thorax, and the abdomen with its contents. The rib cage and the pulmonary system (lungs and respiratory airways) form the thorax. Both cavities are separated by a dome-shaped muscular partition called the diaphragm.

In the respiratory physiology literature, the term “chest wall” is used to depict the extrapulmonary parts of the body that participate actively in respiration, i.e., all parts of the respiratory apparatus with the exception of the lungs and airways. Therefore, the chest wall includes the rib cage, the diaphragm, and the abdominal cavity with its contents. From an anatomical standpoint, these structures are independent, separated by elastic walls, although all three are interconnected. From a mechanical point of view, the diaphragm and the abdomen are regarded as a single unit because of the incompressibility of the abdominal contents that lie between the diaphragm and the abdominal walls.
Based on this view of the mechanism, Konno and Mead (1967) proposed a model in which the chest wall is considered as a two part kinematic mechanism with essentially two degrees of freedom of movement, one for each of its constituent parts, the rib cage, and the diaphragm-abdomen. According to the two degrees of freedom model, a change in volume of each of the parts is equal to a change in volume of the whole system (Agostoni & Hyatt, 1986).

The mechanical behavior of the chest wall is dependent upon the interplay of muscular and non-muscular forces of the respiratory system. The interaction between them generates the forces necessary to change the volume of the lungs. The respiratory muscles exert active forces to move the chest wall away from its resting state (Smith & Loring, 1986). The non-muscular or passive opposing forces include: a) the elastic recoil properties of the lung-thorax unit (formed in part by the elastic properties of the lungs and muscle tissues, and in part by the surface tension of the liquid air interface within the alveoli (Comroe, 1965), b) the resistance of airflow by the respiratory airways, and c) the resistance of deformation of the respiratory tissues (Mead & Martin, 1968). These passive opposing forces bring the chest wall back to its point of equilibrium, called the end expiratory level (EEL).

The pressures generated by the respiratory system are highly dependent on lung volume and gas flow (Rodarte & Rehder, 1986). The maximal pressures that can be developed by the respiratory system are a sum of its muscular (contraction of the respiratory muscles) and non-muscular (elastic recoil) pressures. The magnitude and sign of these pressures is dependent upon lung volume. At lung volumes higher than the EEL, the non-muscular forces generate positive pressures, resulting in passive expiration. These pressures are higher at total lung capacity, and decrease gradually as lung volume gets closer to the EEL. At lung volumes lower than the end expiratory level, the passive forces generate negative pressures in the inspiratory direction. These become more negative towards the residual volume, and less negative as lung volume increases toward
the end expiratory level (Hixon & Putnam, 1987). Consequently, the elastic recoil pressures depend on how far the system is from its end expiratory level (Agostoni & Hyatt, 1986). From general muscle mechanics we know that the maximal force that a muscle can generate decreases as the muscle shortens below its optimal length (length-tension relationship), and that there is an optimal velocity of contraction, at which efficiency of power generation is maximal (force-velocity relationship). Therefore, maximum muscular pressures of the respiratory system are dependent on the length, tension, and velocity of contraction of the respiratory muscles. The pressure values during maximum effort are approximately -130 cm H$_2$O for inspiration, and +240 cm H$_2$O for expiration (Agostoni & Hyatt, 1986; Agostoni & Mead, 1964).

**Subdivisions of Lung Volume**

The subdivisions of the gas volume contained in the lungs during several respiratory maneuvers are: a) tidal volume (VT) is the volume of air inspired or expired in a particular breath or the average volume of a series of breaths during quiet breathing; b) inspiratory reserve volume (IRV) is the maximum volume of air that can be inspired from an end-tidal inspiratory level; c) expiratory reserve volume (ERV) is the maximum volume of air that can be expired from the end-expiratory level; d) residual volume (RV) is the volume of air that remains in the lungs and airways after a maximum expiration; e) total lung capacity (TLC) is the volume of air in the lungs and airways after a maximum inspiration; f) vital capacity (VC) is the volume of air that can be forced out after a maximum inspiration (TLC - RV); g) functional residual capacity (FRC) is the volume of air in the lungs and airways at the end expiratory volume; h) inspiratory capacity (IC) is the maximum volume that can be inspired from the end-expiratory level (Hixon & Putnam, 1987; Pappenheimer et al., 1950).
Quiet and Forced Breathing

Inspiration during tidal breathing is accomplished primarily by the action of the diaphragm and inspiratory intercostal muscles. The muscular fibers of the diaphragm have two sources of insertion. The crural or vertebral part of the diaphragm, which attaches to the first three lumbar vertebrae and aponeurotic arches, and the costal part of the diaphragm, which attaches to the inner surfaces and upper margins of the lower six ribs (DeTroyer & Loring, 1986; Mead, 1979). Diaphragmatic contraction causes a flattening of its dome, which descends relative to the costal insertions. When the diaphragm is displaced, its fibers elongate, and the area of apposition (the cylindrical portion of the diaphragm that is apposed to the inner aspect of the rib cage) increases. When the diaphragm contracts, the lung volume increases if the airways are open, or the alveolar pressure decreases if the airways are closed. During diaphragmatic contraction there is an increase in the abdominal pressure, and the abdominal contents are displaced caudally, resulting in an outward push on the anterolateral abdominal wall, shifting the shape of the rib cage (DeTroyer & Loring, 1986). Quiet expiration results from the elastic recoil of the lungs and the chest wall, and expiratory muscle action (Perkins & Kent, 1986). Although quiet expiration is considered mainly as a passive process, the diaphragm, as well as laryngeal muscles are active during the quiet expiratory phase (see section on respiratory related laryngeal function) (Agostoni & Mead, 1964; DeTroyer & Loring, 1986). The process of respiration during tidal or quiet breathing comprises a range between 35% to 51% of the vital capacity (VC), with volume changes ranging between 10% to 16% of the VC in normal individuals.

Forced respiration differs from tidal breathing in the amount of energy expended. In forced respiration either the volume of air or the muscular effort used are greater than in tidal breathing. Forced inspiration is performed with the action of accessory muscles that assist the diaphragm and the external intercostals. Many other muscles can be recruited to assist in forced inspiration, such as the pectoralis major and minor, the
serratus posterior and superior, latissimus dorsi, trapezius, sternocleidomastoid, and the scaleni, although only the latter two show significant respiratory activity in humans (Campbell, 1970). Forced expiration for lung volumes above EEL is accomplished by the recoil forces of the chest wall and lungs together with abdominal and thoracic active muscle forces. Forced expiration below EEL is accomplished by muscle forces alone. In order to reach lung volumes lower than the end expiratory level, accessory muscle force is always required (Draper, Ladefoged, & Whitteridge, 1959). Producing expirations below EEL can be accomplished by two mechanisms, which can act independently or together. One mechanism is an increase of the intra abdominal pressures, which forces the diaphragm in a cephalic direction. The increase in abdominal pressure is accomplished by contraction of the abdominal muscles, which cause an inward displacement of the viscera. These muscles constitute the antero-lateral wall of the abdomen, and include the external oblique, the internal oblique, the rectus abdominus, and transversus abdominus. Their action also pulls down the anterior part of the rib cage. The abdominal muscles are thought to play an important role in inspiration as well (Martin & DeTroyer, 1982). The other mechanism that is used to reach lung volume levels below the end expiratory level is to decrease the antero-posterior or transverse diameters of the rib cage, by caudally displacing the ribs or sternum. Caudal displacement of the ribs and sternum can be accomplished by the action of the internal intercostals, the transversus thoracis, the subcostal muscles, and serratus posterior inferior, the quadratus lumborum, and the latissimus dorsi. Forced inspirations and expirations are used for measuring some of the pulmonary system subdivisions. The respiratory maneuvers that require voluntary maximum efforts include vital capacity (VC), total lung capacity (TLC), and inspiratory and expiratory reserve volumes (IRV and ERV).
Breathing for Phonation

Breathing for speech production is superimposed onto the life sustaining function of the respiratory system. During speech production, the respiratory system generates the aerodynamic forces that drive the vibration of the vocal folds, required to produce the various speech sounds. Major modifications in the vegetative breathing process accompany voice and speech production. For example, during quiet breathing the respiratory cycle is repeated approximately 12 times per minute, with expirations being slightly longer than inspirations (Hixon & Putnam, 1987; Perkins & Kent, 1986). During speech or singing the relative duration of inspirations and expirations change substantially. The proportion of the respiratory cycle used by the inspiratory phase decreases, while the proportion of the expiratory phase increases (Horii & Cooke, 1978; Proctor, 1986b). Initiation of voice for most speech and singing events occurs from lung volumes above the tidal volume range (Bouhuys, Proctor, & Mead, 1966; Bunn & Mead, 1971; Horii & Cooke, 1978), generally starting between 60% and 50% of the VC level (Hixon, Goldman, & Mead, 1973). The percentages of the vital capacity (VC) utilized during normal speech production are larger than those utilized during quiet breathing. While approximately 10 to 15% of the VC is used for quiet breathing, 20 to 25% of the VC is used for conversational speech (Hixon & Putnam, 1987). These percentages may increase (between 40 and 45% VC) when the vocal intensity is increased (Bouhuys et al., 1966; Hixon et al., 1973; Hixon, Mead, & Goldman, 1976).

Even though the air volumes utilized during voice and speech production are relatively larger than those used during the act of quiet breathing, they are generally restricted to the mid-range of the VC (Hixon, 1973). For example, during tidal breathing in an upright position, normal subjects tend to initiate breath groups between 35 to 51% of the vital capacity (VC), with volume changes ranging between 10 to 16% of the VC. During conversational speech and reading events in an upright body position, normal
subjects tend to initiate breath groups between 60 and 50% VC levels, extending over 10 to 20% of the VC, and terminating around a level that approximates the bottom of the tidal breathing cycle. The bottom of the tidal breathing cycle corresponds to the end lung volume (i.e., EEL). Only occasionally do normal speakers phonate below the EEL level. This occurs especially during the production of long utterances (Horii & Cooke, 1978). Increasing intensity of phonation usually increases the proportion of the VC used, but rarely extends beyond the EEL level. Loud phonation typically starts at about 10 to 25% higher lung volumes than the starting volumes for comfortable loudness phonation (Hixon et al., 1973; Hixon et al., 1976), but the lung volume levels at which loud phonations are terminated do not differ from those of comfortable loudness phonation. The lung volume levels and percentages of the VC used during singing generally depend on the amount of voice training that the singer has. Individuals without voice training and non-classical singers typically use a range of lung volumes that slightly exceeds those used during speaking (Hoit, Jenks, Watson, & Cleveland, 1996). Only highly trained classical singers have been reported to use lung volumes that comprise almost the full range of the VC. This generally occurs while meeting the demands of a particularly long phrase (Watson & Hixon, 1985; Watson & Hixon, 1996).

The regulation of alveolar pressures for phonation requires a more precise muscular control than for breathing purposes. It is important to note that the driving pressures required for phonation are much lower than the maximum pressures the system is able to exert. The maximum expiratory pressures, produced with a closed airway, can reach up to approximately 240 cm H₂O (Agostoni & Hyatt, 1986), whereas the pressures required to produce speech at conversational levels range between 5 and 15 cm H₂O. Significant alterations in the breathing cycle can occur during singing, particularly when long phrases are sustained. In this case, most of the time of the respiratory cycle is taken by expiration. The expiratory cycle during singing of certain phrases can take up to 95% of the breathing cycle (Proctor, 1980). During singing of these phrases, classically trained singers have been reported to reach the top of their VC during inspiration, start phonation
close to total lung capacity (TLC), and sustain expiratory excursions during phonation to near the residual volume (RV) (Proctor, 1980; Watson & Hixon, 1985; Watson & Hixon, 1996). The ability to sustain phonation throughout the VC at constant intensity requires fine control of subglottal pressures that drive the larynx. The maintenance of constant pressures is dependent upon the interplay between the passive and dynamic forces of the respiratory system (Hixon & Putnam, 1987). These forces vary largely in magnitude depending on lung volume. For example, in order to sustain a tone at a comfortable loudness, the subglottal pressure has to be maintained constant at approximately 7 cm H$_2$O. At high lung volumes, the combined elastic recoil of chest and lungs can produce expiratory forces of 25 to 30 cm H$_2$O. Therefore, the respiratory system needs to oppose the effect of the recoil forces through inspiratory effort, to avoid excessive driving pressures (Bouhuys et al., 1966). As lung volume gets lower, the relaxation pressures decrease gradually towards REL, and build up again but opposite in sign between REL and RV (Agostoni & Mead, 1964). The complex sequence of the interplay of forces during sustained phonation throughout the VC, was described by Hixon as “… the muscular pressure needed is initially opposite in sign to the recoil pressure of the pump, then is the same sign as that of the pump, and finally again opposite in sign to the pump but with the signs of both the muscular pressures and recoil pressures being opposite to their respective signs at high lung volumes.” (c.f. Hixon, 1987, pp. 103). The resultant muscular pressures are equal to the difference between the alveolar pressure desired and the relaxation pressure available at any given lung volume. The respiratory muscles accomplish the wide variety of muscular pressures in a very different manner than that used to accomplish the sole purpose of ventilation. The abdominal musculature has some degree of active tonic contraction during the production of speech (Draper et al., 1959). The result of the contraction of this musculature is a compression of the abdominal contents and a caudal pull on the rib cage. The compressed abdominal viscera push the diaphragm upwards. When the diaphragm is displaced, its fibers elongate, and the area of apposition (the cylindrical portion of the diaphragm that is apposed to the inner aspect of
the rib cage) increases. This action increases the efficiency of the diaphragm as a pressure generator (DeTroyer & Loring, 1986).

The Laryngeal System

The larynx phylogenetically evolves from a valve that protected the lungs of primitive lungfish (Bartlett, 1986). Its primary physiological function is to protect the lungs by preventing the entrance of any harmful substances into the lower airways. Phonation requires modified use of the valve from its biological activities (Perkins & Kent, 1986). Laryngeal functions of protection, respiration and phonation are mediated through several polysinaptic brainstem reflexes.

Protective Function of the Larynx

The primary function of the larynx is to prevent foreign particles from entering the lower airways and lungs. Stimulation of the supraglottal mucosa by means of touch, chemicals or temperature variations may elicit a forceful glottic closure. This protective function closes off the airway at three levels by: (a) forceful adduction at the vocal fold level, (b) adduction of the ventricular folds, and (c) shortening of the aryepiglottic folds. In addition, there is a downward and backward folding of the epiglottis due to laryngeal elevation during swallowing, that serves to redirect the bolus backwards out of the valleculae and towards the upper esophagus (Ardran & Kemp, 1952; Logemann, 1983; Sasaki & Isaacson, 1988). The laryngeal gesture of closing the airway for vegetative purposes is described as sphincteric in nature, and is considered an automatic phase governed by the brainstem. During swallowing these actions occur simultaneously with hyoid and laryngeal elevation, which are raised and brought forward by the action of the
suprahyoid muscles. When the laryngeal sphincter reflex is active, there is: (1) an inhibition of respiratory diaphragmatic activity (Harned, Myracle, & Ferreiro, 1978), (2) an inhibition of posterior cricothyroid muscle activity, which prevents abduction of the vocal folds (Harding & Titchen, 1981), and (3) an enhancement of thyroarytenoid muscle activity, to effect glottal closure (Harding, Johnson, & McMIelland, 1980).

Respiratory Related Laryngeal Function

The larynx acts as a variable resistor, regulating airflow in and out of the lungs. Most variations in laryngeal resistance during breathing and phonation take place at the level of the vocal folds, accomplished by the action of the intrinsic laryngeal musculature. In some circumstances, extrinsic laryngeal mechanisms may also alter laryngeal resistance (Fink & Demarest, 1978).

Because of its active role in respiration, the larynx can be considered as part of the respiratory system. Before birth, the larynx regulates the outward flow of pulmonary liquid, and it may play a role in pulmonary development (Harding, 1984). Studies in animal and human neonates have shown that by increasing laryngeal airway resistance the larynx plays an essential role in respiration (England, Geraldinge, & Stogryn, 1985; Harding et al., 1980). This is accomplished by phasic expiratory activation of the thyroarytenoid (TA) muscle referenced to the diaphragm (Harding, 1984; Harding et al., 1986). Neonates have a very compliant chest wall, and as a result, the resting position of the respiratory system is at relatively low lung volumes. The mechanism of increasing laryngeal airway resistance, or “laryngeal braking” preserves lung volume and maintains airway patency by increasing lung volume at end expiration (Fisher et al., 1982; Harding, 1984).
In adults, movements of the vocal folds during quiet breathing is well coordinated with movements of the respiratory system, and can be easily observed during indirect examination of the larynx. The vocal folds abduct on inspiration and adduct on expiration causing the laryngeal resistance to drop during inspiration and to increase during expiration. Vocal fold movements increase particularly during hypercapnic (increased arterial CO₂ levels) and hyperpneic (more rapid and deeper breathing than normal) states (Bartlett, 1979; Bartlett et al., 1973; Campbel et al., 1963; Megirian & Sherrey, 1980; Stransky et al., 1973). The onset of recurrent laryngeal nerve activity is slightly preceded by the onset of phrenic nerve activity, resulting in abduction of the vocal folds just prior to contraction of the diaphragm (Cohen, 1975; England, Bartlett, & Knuth, 1982; Sherrey & Megirian, 1974). Respiratory activity of the cricothyroid (CT) muscle consists of phasic contraction during inspiration, and residual tonic activity throughout expiration (Horiuchi & Sasaki, 1980; Tully et al., 1991). Although several investigations reported phasic electromyographic activation of the posterior cricoarytenoid (PCA) muscles during inspiration (Kuna et al., 1990; Murkami & Kirchner, 1972; Suzuki & Kirchner, 1969), recordings of adductor laryngeal muscles during expiration are not conclusive. Some authors reported phasic EMG activity in laryngeal adductors (Green & Neil, 1955; Kuna, Insalaco, & Villeponteaux, 1991; Kuna et al., 1988; Sherrey & Megirian, 1974), while others found no adductor activity during quiet breathing (Bartlett, 1979; Bartlett et al., 1973). These latter authors suggested that relaxation of the PCA was responsible for the adductory movements of the vocal folds during quiet expiration. The opening of the airway prior to diaphragmatic contraction avoids inspiration against a partly obstructed airway (Scharf et al., 1978). More recent studies suggested that PCA and CT muscles are active predominantly during inspiration and the lateral cricoarytenoid (LCA) and TA muscles are active predominantly during expiration (Tully et al., 1991). The reflex activation of respiratory related activity by intrinsic laryngeal muscles is neurally controlled by receptors within the tracheobronquial tree and in the upper airway (Sant'Ambrogio, 1982; Sant'Ambrogio, Mathew, Fisher, & Sant'Ambrogio, 1983).
Respiratory function of the larynx is influenced by sleep state. Numerous animal and human findings indicate that sleep and wakefulness states have an important influence on the respiratory activity of the larynx (Harding et al., 1986; Kuna et al., 1988; Kuna et al., 1993; Kuna et al., 1990; Marchal et al., 1986). Patterns of respiratory related activity of intrinsic laryngeal muscles are different between states of wakefulness and non-REM (rapid eye movement) sleep (Megirian & Sherrey, 1987; Megirian & Sherrey, 1980). Recruitment of intrinsic laryngeal muscle activity is higher during quiet sleep than during REM sleep.

Studies that investigated respiratory flow patterns obtained during maximum inspiratory and expiratory effort compared results between normals and individuals with neural impairment of the larynx (e.g., with conditions like unilateral and bilateral vocal fold paralysis). Data showed that flow patterns corresponding to individuals with motor denervation of the larynx were different from normal patterns. In addition, the larynx plays an important role in regulation of respiration during high ventilatory output states such as exercising. During these states, there is an increase of phasic activation of the PCA and a decrease of activation of the laryngeal adductors, which results in increased glottal aperture, thereby facilitating high flow rates of air exchange.

Influence of Respiratory Reflexes Evoked by Volume Changes on Laryngeal Function

Respiratory laryngeal activity is strongly influenced by pulmonary reflexes associated with lung volume. There is a coordinated set of reflexes mediated by pulmonary stretch receptors that influence not only breathing, but laryngeal function as well. These reflexes, often called selbststeuerung reflexes, are triggered by both slowly and rapidly (irritant) adapting stretch receptors. These receptors react in response to
mechanical changes in tension of the airway walls, and provide sensory lung volume information (Coleridge & Coleridge, 1986). The slowly adapting stretch receptors are considered to react to moderate lung inflations and deflations. These receptors are responsible for the Hering-Breuer inflation reflex, which is a respiratory inhibition produced by lung inflation. Increasing discharge of the stretch receptors in inspiration provides an inspiratory off-switch, which inhibits or delays respiratory muscle activity, and their continuing discharge in expiration lengthens the respiratory pause. The inspiratory off-switch produced by input of the slowly adapting stretch-receptor determines tidal volume (VT) and inspiratory time (TI) once a certain threshold volume is reached. There is evidence that the Hering-Breuer reflex inhibits not only respiratory muscle activity, but respiratory activity of the laryngeal muscles such as the PCA and TA as well (Bartlett et al., 1973; Fukuda et al., 1973; Tully et al., 1990; Tully et al., 1991). In addition, these reflexes cause tracheobronchial smooth muscle to relax and thus dilate the airways.

The rapidly adapting stretch receptors are evoked by large and sudden pulmonary inflations and deflations. Their most important function may be to signal the onset of pathophysiological changes in the airways. Lung receptors have been stimulated experimentally in animals in many ways, such as by acute pulmonary congestion, embolization, anaphylaxis, pneumothorax, and intravenous injection of bronchoconstrictor chemicals (Coleridge & Coleridge, 1986; Sellick & Widdicombe, 1970). Stimulation is related to the decrease in lung compliance and increase in airflow resistance, which are experimentally induced. Lung deflation has been shown to stimulate rapidly adapting pulmonary receptors in several species including cats, rabbits, dogs, and guinea pigs. Their activity has not been recorded in humans due to the invasiveness of the experimental procedure. These pulmonary receptors have reflex connections with laryngeal muscles, and their stimulation during pulmonary deflation increases expiratory laryngeal airflow resistance by constriction of the larynx (Brancatisano et al., 1987; Dixon et al., 1974; Glogowska et al., 1974).
Artificially induced expiratory glottic constriction by lung deflation.

The phenomenon of increased laryngeal constriction with lung deflation has been well documented in animals (Dixon et al., 1974; Stransky et al., 1973). It has been shown that when the afferent nerve endings in the lungs (stretch receptors) are stimulated by forced pulmonary inflation and deflation, the larynx reacts to this stimulation. Laryngeal responses that were found during artificial manipulation of lung deflation were glottic narrowing, increased laryngeal airway resistance, and increased EMG activity of laryngeal adductors during the expiratory phase of the breathing cycle (Brancatisano et al., 1987; Dixon et al., 1974). Glogowska and colleagues measured action potentials from single laryngeal motor fibers during inspiratory and expiratory phase in cats, and concluded that expiratory unit discharge correlates with increases in expiratory laryngeal airway resistance (Glogowska et al., 1974). The reflex effect of increased expiratory laryngeal constriction with lung deflation is so robust, that investigators have used lung deflation in their methodology as “the most convenient and effective means of activating the laryngeal constrictors” (Brancatisano et al., 1987 pp., 61).

Even though most of the data come from anesthetized animals, it has been shown that expiratory laryngeal braking associated with the respiratory cycle occurs also under normal physiological conditions. As mentioned earlier in this review, laryngeal control of expiratory flow has been shown to be particularly active in both human and animal neonates (England et al., 1985; Fisher et al., 1982; Harding et al., 1980), adult humans (Brancatisano, Collett, & Engel, 1983b; England et al., 1982), cats (Bartlett et al., 1973), rats (Megirian & Sherrey, 1980), and sheep (Harding et al., 1980). Laryngeal activation during breathing clearly shows the interdependence between the respiratory and laryngeal
systems, and how, to some extent, afferent receptors in the laryngeal airway regulate breathing, and pulmonary receptors regulate laryngeal activity.

Larynx and Phonation

The normal phonatory process requires a precise coordination of a complex series of neuromuscular events which involves a combination of voluntary and reflex control systems that regulate the activity of the laryngeal musculature (Wyke & Kirchner, 1976). According to the myoelastic-aerodynamic theory of voice production (Berg, 1958), the factors necessary for voice production are: 1) the aerodynamic forces of the air that drive the larynx, 2) the adjustments of laryngeal muscles and the elastic properties of the laryngeal tissues, and 3) the aerodynamic coupling between the larynx and the cavities above and below it, and the coupling between the vocal folds. Before vocal fold vibration is initiated, the vocal folds are adducted by approximation of the arytenoid cartilages towards the midline. This action, which positions the vocal folds in a pre-phonatory position is accomplished principally by the interarytenoid and the lateral cricoarytenoid muscles. To effect vocal fold vibration, the airstream coming from the lungs meets the resistance offered by adducted vocal folds, and builds up subglottal pressure. The amount of subglottal pressure required to initiate and sustain vibration is determined by the degree of resistance offered by the vocal folds to the airstream. The amount of resistance can be regulated by altering the tension and the degree of adduction of the laryngeal structures. Once the airflow builds up sufficient pressure to overcome the vocal fold resistance, the vocal folds are pushed apart, letting the airflow through the glottis. Subsequently the vocal folds are pulled together again both by the elastic properties of the tissues and by a suction force (negative pressure) generated by the airflow passing through the glottis, known as the Bernoulli effect. The pulses of air passing through the vibrating glottis generate the glottal airflow, represented by the glottal volume velocity waveform. This time-varying glottal airflow is the sound source that produces the acoustical excitation of the vocal tract, which acts as a filter (Fant, 1983). Vocal fold
vibration depends on interactions among the subglottal pressure, the pressure drop through the glottis, the glottal width, and the tension and stiffness of the vocal folds. The particular set of values of these parameters will determine the characteristics of the resulting output in terms of pattern of vibration, shape and amplitude of the airflow waveform, and acoustic spectrum (Titze, 1994).

**Neurophysiologic Mechanisms of Phonatory Control**

The regulation of phonatory parameters such as vocal pitch and intensity involves changes in length, tension, mass, and stiffness of the vocal folds, combined with changes in lung pressures that drive the larynx. Control of these changes involve several components of the central and peripheral nervous systems (Larson, 1988). Mechanoreceptors at the laryngeal level monitor these changes. Receptors for subglottal pressure, located mostly in the subglottal mucosa, affect the reflexogenic motor signals to the laryngeal muscles. These receptors react to changes in airflow and pressure adjusting the laryngeal musculature accordingly (Campbel et al., 1963). Stretch receptors, located in each of the intrinsic laryngeal muscles, are activated by changes in the length of muscles such as the thyroarytenoid and the cricothyroid (Baken & Novak, 1971). Joint receptors, located in the fibrous capsules of the intercartilaginous joint, sense movements and relative positions of the joints of the laryngeal cartilages, in particular the cricothyroid and the cricoarytenoid joints. The auditory system is involved in monitoring fundamental frequency ($F_o$), through auditory feedback (Sapir, McClean, & Larson, 1981). Wyke (1967) postulated that mechanisms of "pre-phonatory tuning" and "acoustic automonitoring" are involved as well, suggesting auditory governance of voice even before an actual vocalization is produced (Larson, White, Freedland, & Burnett, 1996).
Regulation of Fundamental Frequency

Fundamental frequency ($F_0$) is the primary physical correlate of vocal pitch. $F_0$ is determined by the rate at which the glottal air pulses are generated during vocal fold vibration. Vocal fold vibration rate is primarily determined by the properties (mass and stiffness) of the vocal folds and the magnitude of subglottal pressure, as proposed by the body-cover model (Fujimura, 1981; Hirano, 1974). According to this model, the body of the vocal folds is formed by the muscular tissue of the thyroarytenoid muscle (TA), and the vocal fold cover is comprised of non-muscular tissue corresponding to the epithelium and the superficial layers of the lamina propria (Hirano, 1981).

Changes in the properties of the vocal fold cover depend primarily upon vocal fold length, which in turn depends upon activity of the TA and the cricothyroid (CT). These muscles are antagonistic in relation to length changes of the vocal folds (Fujimura, 1981). The CT muscle adjusts the distance between the thyroid and cricoid cartilages, increasing the length of the vocal folds and passively stretching both the vocal fold cover and body when contracting. In principle, contraction of the TA increases the tension of the body of the vocal folds while slackening the vocal fold cover (Hirano, 1974). But the effect of the contraction of the TA on the vocal fold cover is more complex, depending upon whether the vibrational area is primarily muscular or non-muscular tissue. For example, at high pitches, when the vocal folds are elongated due to the action of the CT and the primary vibrational area is the cover, contraction of the TA shortens the vocal folds and reduces the tension of the cover, resulting in a lowering of the $F_0$. On the other hand, at low pitches when the primary vibrational area is muscular tissue, increase in the contraction of the TA increases the stiffness of the vocal cover, resulting in a rise of $F_0$ (Titze, 1994). There is a clear interaction between the activity of these muscles.
Nonetheless, how this interaction takes place may depend upon a variety of factors such as, the prevailing voice register, the action of opposing contraction of adducting and even abducting muscles that anchor the arytenoid cartilages, as well as other factors.

Another important variable in $F_0$ control is subglottal pressure (Baken, 1987; Hirano, Vennard, & Ohala, 1970; Rothenberg & Mahshie, 1988). Subglottal pressure is usually expressed in cm H$_2$O with respect to the atmospheric pressure. When the chest wall or abdomen are expanded during the inspiratory phase of the respiratory cycle, subglottal pressure becomes lower than atmospheric pressure, so that air flows from the vocal tract into the lungs. During phonation, the subglottal pressure is generally higher than atmospheric pressure, making the air flow from the lungs, passing through the opening between the vocal folds (i.e., there is a transglottal pressure). The magnitude of subglottal pressure combined with the length of the vocal folds determines the amplitude of vibration (Titze, 1989). For a constant vocal fold length, higher subglottal pressures create greater maximum stretch of the vocal folds, which results in higher effective tension. The result of higher effective tension is higher amplitude of vibration with an increase in $F_0$ (Titze, 1989). A 1 cm H$_2$O increment in subglottal pressure results in an increment of 3 to 6 Hz in $F_0$ when the length of the vocal fold remains constant.

Although vocal fold length is primarily regulated by intrinsic laryngeal muscles, they can be passively elongated or shortened by external mechanisms. The extrinsic laryngeal muscles affect the vertical laryngeal position and possibly the tension of the vocal folds (Fink & Demarest, 1978; Zenker, 1964). This is particularly true in individuals with untrained voices, in whom the larynx is lowered to produce low pitches, and raised during sustained phonation to produce higher fundamental frequencies (Shipp, 1975). When the hyoid bone is pulled anteriorly and superiorly by the action of the suprahypoid muscle group, the thyroid cartilage may be tilted forward in relation to the arytenoids. This action increases vocal fold length with a resulting rise in $F_0$, and vice versa. Another external mechanism that may affect vertical laryngeal position, is a
downward pull on the cricoid cartilage by the trachea. This pull has been reported to occur at high lung volumes, when the diaphragm is maximally flattened and its effect may be to shorten the vocal folds, thus lowering $F_0$, provided that the cricothyroid muscle does not compensate by increasing activity (Sundberg, Leanderson, & Euler, 1989; Zenker, 1964). The tracheal pull phenomenon will be discussed in more detail later in this review.

**Regulation of Vocal Intensity**

Vocal intensity is the physical correlate of vocal loudness and it is usually measured in terms of SPL (Baken, 1987). Vocal intensity is primarily determined by the amplitude of the glottal airflow waveform. Regulation of intensity can be accomplished by the interaction of mechanisms at three different levels including changes below the larynx, at the laryngeal level, and above the larynx.

Changes below the larynx comprise adjustments of subglottal pressure (Isshiki, 1964; Ladefoged & McKinney, 1963; Tanaka & Gould, 1983; Titze, 1988). Higher subglottal pressures result in an increase of transglottal airflow, assuming that the glottal resistance is maintained constant. In general, a doubling of the subglottal pressure results in a 9 dB increase in sound pressure level (Bouhuys et al., 1966).

Changes at the laryngeal level depend primarily on the degree of adduction of the laryngeal musculature. The degree of adduction determines the degree of glottal resistance, which is defined as the ratio between subglottal pressure and transglottal airflow. Increased glottal resistance to vocal fold opening results in greater buildup of subglottal pressure.
Changes above the larynx, comprise vocal tract adjustments. The vocal tract acts as a resonator of sound, and changes in its size and shape create a shift in the formant frequencies. When the formant frequencies are adjusted to match with the harmonics of the source, the harmonics that lie within the formants get enhanced, while the harmonics between formants are attenuated. The effect is an overall boost in intensity (Titze, 1994). This form of formant tuning is efficiently used by trained singers as a way to make the sound of their voices audible even when accompanied by a loud orchestra (Sundberg, 1987).

Isshiki proposed that the predominant mechanisms for control of intensity are dependent on pitch. At low pitches glottal resistance, that is, the degree of adduction of the laryngeal musculature, primarily controls vocal intensity. At high pitches, vocal intensity is primarily controlled by the respiratory system, i.e., by changing the degree of flow rate (Isshiki, 1964).

Changes in sound pressure level affect not only the amplitude, but the shape of the glottal volume velocity waveform as well. It has been shown that as intensity increases, the closing slope of the glottal volume velocity waveform becomes sharper, and the duration of the closed phase of the glottal cycle increases (Dromey, Stathopoulos, & Sapienza, 1992; Stathopoulos & Sapienza, 1993a; Stathopoulos & Sapienza, 1993b). The resultant acoustic spectrum of this type of glottal waveform has increased energy in the higher partials region, which results in an overall louder production (Flannagan, 1958; Monsen & Engebretson, 1977). Other parameters of vocal function that have been shown to vary with changes in intensity are maximum flow declination rate (MFDR), laryngeal airway resistance ($R_{law}$), and closed/open quotients. MFDR, a vocal parameter related to the speed of closure of the vocal folds, has been shown to increase with increasing intensity (Colton, 1984; Holmberg et al., 1988; Stathopoulos & Sapienza, 1993a). Laryngeal airway resistance has been shown to increase as intensity increases (Stathopoulos & Sapienza, 1993a). The open quotient of the glottal flow has displaced a
tendency to decrease (Stathopoulos & Sapienza, 1993a) while the closed quotient has tended to increase with increasing loudness (Dromey et al., 1992; Holmberg et al., 1988; Lindqvist, 1970)

**Vertical Laryngeal Position**

Several factors including the breathing cycle, pitch, prolongation of some phonemes, vocal hyperfunction and lung volume have been considered to influence the vertical laryngeal position (VLP) in the neck during phonation. There has been controversy related to whether or not the larynx changes its vertical position with respect to the breathing cycle. The tracheobronchial tree lengthens and shortens with the breathing cycle. There is some evidence that the tone of the extrinsic laryngeal muscles varies rhythmically with breathing to minimize the up and down movements of the larynx, stabilizing the position of the larynx in the neck particularly in hyperpneic states (Bartlett, 1986; Proctor, 1986a). Fink (Fink & Demarest, 1978) presented some evidence that the larynx moves downwards with inspiration, and upwards with expiration. On the other hand, Mitchinson and Yoffey (Mitchinson & Yoffey, 1947) in humans, and Andrew (Andrew, 1955) in rats, did not find consistent laryngeal vertical movements associated with the breathing cycle.

VLP changes related to variations in pitch has been reported by Shipp and colleagues (Shipp, 1975; Shipp, 1987; Shipp & Izdebsky, 1975; Shipp & McGlone, 1971). They showed that the larynx tends to elevate with higher pitches in untrained singer, and that classically trained male singers tend to keep a low laryngeal position throughout the pitch range (Shipp, 1987). The authors suggested that classical training may counter the influence of pitch on laryngeal elevation. Pabst and Sundberg also reported a correlation between pitch and VLP in classically trained females (Pabst & Sundberg, 1993).
In a study on VLP changes during production of nonsense syllables, Elliot and colleagues reported that prolongations of the /b/ phoneme showed a lower VLP as compared with a sustained vowel (Elliot, Sundberg, & Gramming, 1992), suggesting that phonation of certain phonemes may be another potential factor in laryngeal height changes.

An elevated hyoid bone and laryngeal position are signs that typically accompany vocal hyperfunction (Aronson, 1990), and a common goal of voice therapy for this condition is to achieve a lower laryngeal position during phonation. This is accomplished with different techniques, from digital manipulation of the larynx (Aronson, 1990; Lieberman, 1998), to the inclusion of yawning and sighing as part of therapeutic exercises (Boone & McFarlane, 1994).

Lung volume has been reported as yet another factor that influences changes in VLP. These changes have been described as a lower laryngeal height at high lung volumes, as compared to low lung volumes (Iwarsson & Sundberg, 1997; Iwarsson et al., 1995; Sundberg et al., 1989; Zenker, 1964). Macklin reported the carina, or bifurcation of the trachea, moving caudally as much as 21 mm. at extreme high lung volumes (Macklin, 1925). Zenker proposed that the mechanism that lowers the larynx at high lung volumes was related to the elastic interconnections between the diaphragm and the trachea (Zenker, 1964). During deep inspirations the trachea would be pulled downward as a result of the flattening of the diaphragm. The caudal tracheal movement would exert a pull onto the larynx because of its attachment to the cricothyroid cartilage and ligaments. The descent of the larynx attributable to tracheal traction is referred to as “tracheal pull.” Zenker and Glaminger (Zenker & Glaminger, 1959) measured the magnitude of tracheal pull and reported minimal values that ranged from 20 to 30 g. According to these authors the tracheal pull would be present even in subjects with induced apnea due to anesthesia, suggesting that the tracheal pull creates a background tension force. Zenker (1964) also
suggested that the force that the trachea exerts on the larynx tends to abduct the vocal folds. This assumption was supported by other investigations (Iwarsson & Sundberg, 1997; Iwarsson et al., 1995; Sundberg et al., 1989). Another study on the effects of lung volume on voice onset time (VOT) (Hoit et al., 1993) indirectly addressed this issue. In this study, VOT after the release of voiceless stops was reported to be longer at high lung volumes and shorter at low volumes. The authors suggested that longer VOT’s at high lung volumes were the result of an abductive force exerted onto the vocal folds, presumably attributed to the tracheal pull.

Possible mechanisms that could account for changes in the vertical larynx position in addition to tracheal pull, are tongue position, jaw movement, and the action of the extrinsic laryngeal muscles. These muscles, also called strap muscles, are divided into the infra and supra hyoid groups. The infrahyoid muscle group, which includes the omohyoid, sternothyroid, thyrohyoid, and sternohyoid muscles, is capable of moving the entire laryngotracheal complex downward as much as one whole vertebral body (Tucker, 1987). The suprahidoid muscle group includes the digastric, stylohyoid, geniohyoid, mylohyoid, stylopharyngeus, and the thyrohyoid, which are laryngeal elevators. However, Tucker points out that “... these muscles rarely function alone and that for every agonist there is an antagonist. Thus, relative upward or downward displacement of the laryngotracheal structures is a dynamic activity that is the resultant vector of the arithmetic sums of all muscles active at any given time.” (Tucker, 1987, pp:24)

Glottographic Techniques for Assessment of Laryngeal Function

Several non-invasive techniques are used to monitor vocal fold vibration during phonation. These techniques, referred to as glottographic techniques (Kitzing, 1986) include photoglottography (PGG), derived by transglottal illumination, electroglottography (EGG), flow glottography (FGG), ultrasound laryngography,
ultrahigh-speed photography, and videokymography. Results obtained with these techniques can be objectively quantified. Electroglottography is probably the most widely used as a clinical and research tool, and it is frequently used in combination with flow glottography and acoustic measurements for the study of vocal fold motion.

**Electroglottography**

EGG represents the relative surface area of contact between the vocal folds during the glottal cycle (Baken, 1992). A pair of electrodes placed on the neck on either side of the thyroid cartilage measures electrical impedance changes across the larynx. As the vocal folds vibrate, the current is amplitude modulated. The impedance is greater when the vocal folds are abducted, decreasing as the vocal folds come in contact (Childers & Krishnamurti, 1985). Since it represents contact area, EGG is particularly informative about the closed phase of the vocal fold vibratory cycle. The duty cycle or contact quotient (vocal fold contact time/T) represents the proportion of the cycle during which the folds make contact. Contact quotient may indicate the relative degree of medial compression, or arytenoid pressing, and consequently variations in contact stress. A widening of the contact pulse is associated with increased vocal fold adduction. Closed quotient varies with intralaryngeal adduction levels (Rothenberg & Mahshie, 1988; Scherer, Druker, & Titze, 1988; Scherer & Titze, 1987; Titze, 1990) and it has been shown to vary directly with voice intensity (Orlikoff, 1991). EGG closed quotient has been reported to be the most sensible measure to detect a potential pathogenic voicing pattern such as pressed phonation (Peterson, Barkmeier, Verdolini-Marston, & Hoffman, 1994). There appears to be a linear relationship between EGG closed quotient and assessment of laryngeal adduction from videostroboscopy, with higher values of closed quotient and adduction obtained for pressed voices (Peterson et al., 1994). It was also found to correlate well with posterior glottal gap closing, and with ventricular fold approximation, apparently indicating increased interarytenoid pressure, and vocal fold compression (Scherer, Vail, & Rockwell, 1995). These findings suggest that EGG closed
quotient represents degree of medial fold compression along a hypoadducted to hyperadducted (breathy to pressed phonation) continuum. Holmberg and colleagues (Holmberg, Hillman, Perkell, Guiod, & Goldman, 1995) found a strong correlation between adduction quotients measured from the EGG and airflow signals. Nevertheless, these authors, as well as Peterson et al. (1994) caution that results are dependent on strong and noise-free EGG signals, which are difficult to obtain particularly in subjects with short necks or necks with excessive subcutaneous fatty tissue, which can result in weak and noisy signals.

A multichannel electroglottograph can be used to track the vertical movements of the larynx during phonation. The multichannel technique was initially developed as a way to improve reliability of electroglottography by providing simultaneous EGG recordings from various laryngeal points (Rothenberg, 1992). This system uses multiple electrode pairs that are positioned in the vertical plane, laterally to the thyroid cartilage. When the larynx moves in the vertical plane, it creates an amplitude difference between the signals recorded by each electrode pair. The time varying amplitude difference between the signals can be used to detect upward or downward movement of the larynx relative to its resting position. Changes of as little as 2 mm have been reported with the use of this technique, and it seems to be reliable up to a range of 15mm of vertical laryngeal displacement (Elliot et al., 1992; Rothenberg, 1992).

Multichannel electroglottography has been used by several investigations as a tool to track the vertical laryngeal position (VLP). In a study on VLP changes during production of nonsense syllables (Elliot et al., 1992), it was reported that multichannel EGG has a linear relation with laryngeal height. Pabst and Sundberg used this technique to investigate VLP changes in singers, and reported VLP changes associated with changes in lung volume, pitch, loudness, and vowel production (Pabst & Sundberg, 1993). Iwarsson et al. (1997) reported that high lung volumes were associated with lower VLPs as compared to low lung volume. In an attempt to test the validity of the
technique, Laukkanen and collaborators compared VLP as recorded simultaneously with videofluoroscopic and multichannel EGG signals during various phonatory tasks. Findings showed good correspondence between techniques, suggesting that multichannel EGG signals are a reliable indicator of VLP changes both in terms of timing and change of direction. Calibration of multichannel EGG presents several possible sources of error, such as electrode placement, amount of vocal fold contact, etc. While attempts to obtain quantitative data may be problematic, the authors of the abovementioned studies suggest that multichannel EGG can be used to provide reliable and fairly accurate estimates of VLP changes during controlled phonatory tasks.

Flow Glottography

The glottal airflow waveform is studied with flow glottography (FGG), a technique also known as flow inverse filtering. FGG is a non-invasive method that allows estimation of the vibratory patterns of the vocal folds by removing the influence of vocal tract filtering on the glottal source (Rothenberg, 1973; Rothenberg, 1977). The oral airflow is collected with a circumferentially vented wire-screen pneumotachograph mask, known as the Rothenberg mask. The procedure requires deriving the vocal tract transfer function (formants), and using the inverse of this estimate to design a filter that removes the influence of the vocal tract formants, and yields an estimate of the glottal waveform. The goal is to minimize the formant ripple during the closed phase of the glottal cycle. The correct adjustment of the first formant frequency and bandwidth is critical for obtaining good results. Normal phonation in the modal register typically produces a skewed airflow waveform, with a more gradual slope during the time of increasing flow (positive slope), probably related to a relatively slower opening of the vocal folds within one cycle, and a steeper slope during the time of decreasing flow (negative slope), related to a faster closure of the vocal folds. Measures derived from the glottal volume velocity waveform include:
a) Maximum flow declination rate (MFDR). MFDR represents the closing velocity of the vocal folds. It is extracted from the negative peak amplitude of the differentiated flow glottogram. It serves as an indicator of the excitation of the vocal tract (Fant, 1979; Gauffin & Sundberg, 1989). This measure is believed to reflect the relative magnitude of vocal fold collision forces. It correlates highly with sound pressure level (SPL) (Holmberg, Hillman, Perkell, & Gress, 1994; Perkell, Hillman, & Holmberg, 1994; Sapienza & Stathopoulos, 1994; Stathopoulos & Sapienza, 1993a), in accordance with the acoustic theory that predicts that high intensity voices are a result of high MFDR values (Fant, 1979). Hillman and colleagues (1989) reported higher MFDR values for subjects with voice pathologies such as nodules and polyps. However, Peterson et al. (1994) found that MFDR values change in the same direction for pressed voices as for what they call 'resonant' or therapeutic voices, suggesting that this measure may not be useful to differentiate between pathological and non-pathological voicing patterns. High values of MFDR have been associated with an acoustic source spectrum having more energy in the high frequency regions, and low MFDR values with spectra having less energy in the high frequency regions. This effect on the acoustic spectrum is related to the angle of the waveform between the end of closing and beginning of closed portions of the glottal cycle. A more abrupt vocal fold closure results in a sharper angle of the waveform, which in turn results in a spectrum with more energy in the higher partials, while a more gradual closure of the vocal folds, results in a less sharp angle, which in turn results in a spectrum with less high frequency energy in the high partials (Fant, 1979; Stevens, 1981)

b) AC flow, or peak to peak amplitude of the flow glottogram, is the modulated part of the transglottal airflow. Reports on AC flow suggest that it is determined by the amplitude of vocal fold vibration. It has been found to have a positive relation to SPL, and to increase with intensity (Higgins & Saxman, 1991; Holmberg et al., 1988). AC flow rates have been reported to be higher for males than for females, possibly due to the larger size of male larynges, with larger amplitudes of vocal fold vibrations (Holmberg et al., 1988; Stathopoulos & Sapienza, 1993a). In a study
comparing AC flow values between different voice patterns, Peterson and colleagues have reported higher AC flow values for breathy phonation than for pressed phonation for the vowels /a/ and /i/, suggesting that AC flow reflected higher amplitudes of vocal fold vibration in breathy than in pressed phonation. In this same study however, AC flow did not seem to effectively differentiate between normal and pressed phonation modes (Peterson et al., 1994).

c) Minimum flow, is the un-modulated part of the transglottal airflow. Minimum flow is usually regarded as a measure of the airflow shunted mainly through the posterior glottis, during normal voice production (Rothenberg, 1973, Klatt and Klatt, 1990, Hirano, Yoshida, et al., 1987, Holmberg et al, 1988). Therefore, it would reflect the degree of incomplete glottal closure during the closed portion of the vibratory cycle (Holmberg et al., 1995). However, Hertegard and Gauffin (1995) suggested that MNFL values in the order of 20 to 30 ml/s could be related to vertical movements of the vocal folds during phonation. MNFL has been reported to increase significantly during breathy voice productions. It has also been found to change significantly between normal and soft voice production, but with no change between normal and loud voices. Lack of correlation between this measure with acoustical (Hertegard & Gauffin, 1995), and with adduction quotient measures (Holmberg et al., 1995), has been found. The difficulty in assessing whether a perfectly tight seal between the flow mask and the subject's face has been obtained, may hinder the value of the measure and has lead Holmberg and colleagues to suggest caution be used when interpreting minimum flow data (Holmberg et al., 1995).

d) AC/DC ratio has been reported to be an indirect measure of breathiness (Isshiki, 1981). It has been found to correlate highly with a measure of vocal efficiency (Holmberg et al., 1988), and to increase as loudness levels increase. A similar measure, the minimum flow/peak flow quotient was found to correlate closely with measures of incomplete glottal closure (Hertegard & Gauffin, 1995), and with degree of perceived breathiness (Fritzell, Hammarberg, Gauffin, Karlsson, & Sundberg, 1986; Hertegard & Gauffin, 1995)
e) Flow closed quotient (closed time/T), also called adduction quotient, represents the time the vocal folds are maximally approximated during a glottal cycle. Mean values have been reported to range between 43% and 52% in normal intensity conditions (Holmberg et al., 1994; Sulter & Wit, 1996). Closure duration tends to increase with increasing loudness (Dromey et al., 1992; Holmberg et al., 1988; Lindqvist, 1970). High closed quotients are associated with vocal strain and might cause vocal pathology (Peterson et al., 1994). Iwarsson et al. (1995) found this measure to increase with decreasing lung volumes.

Summary of Normal Respiratory and Laryngeal Function

The production of voice is dependent upon two primary systems: the respiratory and the laryngeal systems. The respiratory system, which mainly functions as the regulator of arterial blood gases, is considered as a two part kinematic mechanism formed by the rib cage and the diaphragm-abdomen. Changes in the volume of these two parts account for changes in the volume of the whole system. Volumetric changes are accomplished through the action of muscular and elastic recoil forces. The respiratory muscles forces actively alter the dimensions of the respiratory system. The elastic recoil forces are passively generated by distinct properties of the lungs and muscle tissues. The interplay of active and passive forces determines the resulting air pressures that the system generates, and the balance between both is highly dependent upon lung volume. Their magnitude is dependent upon how far the system is from its resting expiratory level. The cycle of quiet breathing is accomplished primarily by the action of the diaphragm and the intercostal muscles for inspiration, while quiet expiration results mainly from the elastic recoil of the lungs and chest wall. The volume changes for quiet breathing range between 10 and 16% of the VC in normal adults.
The production of speech requires major modifications of the normal respiratory process. The regulation of pressures for phonation purposes requires a more precise muscular control than for breathing purposes. The fine motor control of the musculature of the respiratory and laryngeal systems requires an exquisite degree of precision in order to sustain phonation at constant pitch and loudness levels. Initiation of most speech events occurs at higher lung volumes than quiet breathing, and larger percentages of the VC are used. The relative duration of inspirations and expirations also change dramatically, with inspiratory phases decreasing and expiratory phases increasing substantially. For most speech activities lung volumes are restricted to the mid-range of the VC. For loud phonation and singing activities, individuals without voice training use a range of lung volumes that slightly exceeds those used during speaking. Only classical opera singers may use lung volumes that comprise almost the full range of the VC.

The laryngeal system’s primary function is protection of the lungs. The closure gesture of the larynx for protective purposes is referred to as the laryngeal sphincteric action. The protective action is mediated through reflexes at the brainstem level. Touch, chemical or thermal stimulation at the level of the supraglottic mucosa may elicit laryngeal closure reflexes with involuntary forceful closure of the larynx. Because of its active involvement during respiration, the larynx can be considered part of the respiratory system. The larynx acts as a variable resistor, regulating airflow in and out of the lungs. This regulation has been shown to be very important at all stages of the life cycle. In neonates, an increase of laryngeal airway resistance during the expiratory phase prevents collapse of the chest wall, preserving lung volume and improving gas exchange. In adults, involvement of intrinsic laryngeal muscles has been reported, from states of quiet breathing, to high ventilatory output states such as exercising. During quiet breathing phasic electromyographic activation of the PCA muscle occurs. During high ventilatory states, there is a higher degree of PCA activation, with a decrement of activation of the laryngeal adductors, which results in increased glottal aperture, which in turn facilitates
high flow rates of air exchange. Laryngeal activation during breathing activities clearly shows the interdependence between the respiratory and laryngeal systems.

Phonation requires a modified use of the system from its biological activities. The production of voice depends on a precise coordination between the voluntary and reflex systems that regulate the activity of the laryngeal musculature, and the regulation of muscular and elastic recoil forces of the respiratory system that generate pressures to drive the larynx. The vibrating vocal folds convert the unmodulated (DC) airflow coming from the lower airways into a modulated (AC) airflow. The pulses of air generated by a vibrating glottis are represented by the glottal volume velocity waveform. This airflow is the actual sound source that produces the acoustical excitation of the vocal tract. The analysis of rate, amplitude and shape of the glottal airflow waveform provides information about vocal source function, such as: a) opening and closing velocities of vocal fold vibration, which permits estimations about relative magnitude of vocal fold collision forces; b) the amount of modulated and unmodulated airflow passing through the glottis, which permits estimations about the degree of glottal closure; c) the relative time the vocal folds are maximally opened or closed within a glottal cycle, which are related to voice register and quality, and may be indicators of breathy or pressed qualities in voice.

The regulation of phonatory parameters such as vocal pitch and intensity involves changes in length, tension, mass, and stiffness of the vocal folds, combined with changes in lung pressures that drive the larynx. Control of these changes involves several components of the central and peripheral nervous systems. Changes in the properties of the vocal fold cover, in the body of the vocal folds and in the magnitude of subglottal pressure are the primary determinants of the $F_0$ output. Changes in degree of vocal fold adduction and in subglottal pressure primarily determine the intensity of phonation. Adjustments of vocal tract positioning can also affect the degree of vocal intensity, although to a lesser degree. Changes in loudness levels of voice production affect not
only the amplitude of the glottal airflow waveform, which increases intensity, but affect
the shape of the waveform as well, which influences the acoustic spectra resulting in
changes in voice quality.

**Vocal Hyperfunction**

Laryngeal hyperfunction refers to excessive or poorly regulated laryngeal and
extra-laryngeal muscular tension. The term “vocal hyperfunction”, introduced by
Froeschels (Froeschels, 1952) refers to “conditions of abuse and/or misuse of the vocal
mechanism due to excessive and/or imbalanced muscular forces” (Hillman et al., 1989,
p.373). Aronson (Aronson, 1990), used the term “musculoskeletal tension disorder” to
describe similar conditions which are typically accompanied by elevated positions of the
hyoid bone and larynx. Morrison and colleagues introduced the term “muscular tension
dysphonia” to refer to voice dysfunction caused by persistent phonation with an abnormal
laryngeal posture (Morrison, Rammage, Gilles, Pullan, & Hamish, 1983). The laryngeal
manifestations of hyperfunction are related to increased mechanical stress and are
described as forceful closure of the arytenoid cartilages at the vocal processes level, and
excessive force in vocal fold approximation. At the supraglottal level, anterior-posterior
constriction and medial lateral constriction characterize it. The anterior-posterior
constriction is generally identified as an anterior displacement of the arytenoid cartilages.
The medial lateral constriction is generally identified by medialization of the ventricular
folds. When visualization of the larynx is attempted with video-endoscopy, these two
signs typically obscure the observable length and width of the vocal folds (Boone &
McFarlane, 1994; Hillman, Holmberg, Perkell, Walsh, & Vaughan, 1989). Vocal
hyperfunction is also associated with an elevated hyoid bone and laryngeal position
Laryngeal hyperfunction is considered as an underlying component in a great number of voice disorders (Boone & McFarlane, 1994; Stemple, 1993). It is present in the majority of dysphonias without organic pathology, and it is considered as a predisposing factor in the development of organic vocal pathology such as nodules, polyps, polypoid corditis, contact ulcers and granulomas (Hillman et al., 1989; Stemple, 1993). Unfortunately, little is known about the underlying mechanism of vocal hyperfunction, or its etiology (Hillman et al., 1989; Hillman, Holmberg, Perkell, Walsh, & Vaughan, 1990; Reed, 1980). Hyperfunctional voices are frequently perceived as having a pressed voice quality and are not infrequently accompanied by dysphonia. The concept of hyperfunction is a guiding principle in therapeutic techniques, where the goal is to reduce the hyperfunctional behavior (Boone & McFarlane, 1994; Stemple, 1984).

Even though the underlying mechanism of vocal hyperfunction, or its etiology are not known (Hillman et al., 1989; Reed, 1980), a concurrent respiratory involvement is generally suspected to be associated with most hyperfunctional voice disorders (Aronson, 1990; Boone & McFarlane, 1994; Green & Neil, 1955; Stemple, 1993). It is believed that in this population the respiratory involvement takes the form of increased respiratory effort (Hillman et al., 1990). Even though abnormal respiratory behaviors seem common in subjects with hyperfunctional voice disorders, it is not known whether they are a predisposing factor or a reaction to the voice disorder.

Constriction of the Laryngeal Vestibule

Constriction of the vestibule of the larynx (supraglottis) is a common sign of vocal hyperfunction and it is probably associated with most hyperfunctional voice disorders. Changes in configuration of the supraglottis during phonation have been studied by videolaryngoscopic methods, using both telescopic and fibrescopic techniques. Supraglottal constriction during phonation has been reported to be present in pathological
voice conditions such as unilateral vocal fold paralysis (Woodson & Murry, 1994); chronic laryngeal fatigue (Eustace, Stemple, & Lee, 1996); Parkinson's disease (Hanson, Gerratt, & Ward, 1984; Smith, Ramig, Dromey, Perez, & Samandari, 1995); and spasmodic dysphonia (references). In the normal voice population, constriction of the supraglottis appears to be almost absent for 91% of males and 99% of females during sustained phonation (Sulter, Schutte, & Miller, 1996). Nonetheless, it appears to be elicited by various vocal tasks, such as voluntary production of pressed voice (Painter, 1986; Scherer et al., 1995); high pitch loud phonation (Painter, 1986), whisper with low and high levels of effort (Painter, 1986; Solomon, McCall, Trosset, & Gray, 1989); vocal exercises in classical singing, such as glissando and messa di voce (Yanagisawa, Estill, Kmucha, & Leder, 1989), and during singing in voice qualities such as falsetto, sob, opera, twang, and belting (Yanagisawa et al., 1989; Yanagisawa, Mambrino, Estill, & Talkin, 1991).

Most studies used visually based perceptual rating scales to evaluate changes in supraglottal configuration from videoendoscopic images, with the exception of Scherer and colleagues (Scherer et al., 1995) and Painter (Painter, 1986), who attempted to measure changes in distance of the supraglottal structures. The terminology used to refer to supraglottal structures and their change in configuration is not consistent across different authors. For example, the reduction of the visible length of the glottis and the antero-posterior length of the vestibule has been referred to as "anterior constriction" (Solomon et al., 1989), "antero-posterior compression" (reference), and "aryepiglottic sphincter narrowing" (Yanagisawa et al., 1989). The rating scales differed greatly across studies. A wide variety of parameters have been examined. While some studies used a two-point rating scale, evaluating overall constriction of the supraglottis (Eustace et al., 1996; Hanson et al., 1984; Sulter et al., 1996), others like Smith et al., (1995), Solomon et al., (1989), and Scherer et al., (1995) included a more detailed examination of the different structures involved. Since most studies depended on subjectively based scaling with different parameters to evaluate, and their tasks were different as well, it is difficult
to compare among them. Regardless of differences in nomenclature and in rating scales, there are two parameters that seem to be the most representative of compensatory adjustments exhibited by supraglottal anatomical structures. These parameters are: 1) shortening of the antero-posterior length of the vestibule, and 2) medialization of the ventricular folds.

Laryngeal Dynamics as a Function of Lung Volume

It has been shown that individuals with pathological conditions use abnormal ranges of lung volumes during speech production. Subjects with conditions such as cystic fibrosis (Sperry, 1993), cervical spine injury (Hoit, Banzett, Brown, & Loring, 1990), and asthma (Loudon, Lee, & Holcomb, 1988), have been shown to speak using higher lung volume ranges than the normal population. Subjects with conditions such as hearing loss (Forner & Hixon, 1977; Whitehead, 1983), and vocal nodules (Hillman et al., 1997; Sapienza & Stathopoulos, 1994; Sperry et al., 1994) have been reported to speak using lower lung volumes.

Following is a review of the different parameters of laryngeal function that have been reported to react to changes in lung volume in normal individuals. Reports on the effects of lung volume on the vertical laryngeal position have been reviewed in an earlier section.

Cricothyroid Electromyographic Activity

Two studies have measured cricothyroid muscle (CT) electromyographic activity as a function of lung volume. Shipp and colleagues (Shipp et al., 1983) reported a marked increase in CT activity at low lung volume extremes in six subjects with both trained and
untrained voices. Their subjects consistently showed increased CT activity at low lung volume levels when compared to high lung volume levels, regardless of fundamental frequency. However, no controlled measures of lung volume were taken, which makes it difficult to assess the percentage of the vital capacity at which CT activity was measured. The authors only reported that subjects were "low" in their lung volume level. Results from Shipp's study are in marked contrast to results reported by Sundberg et al. (Sundberg et al., 1989), who found decreased CT activity at low lung volume levels when compared with high lung volume levels on three classically trained male subjects. The authors reported obtaining measures somewhere below the subjects' functional residual capacity (FRC), but did not specify the percentage of vital capacity at which the measures were taken. Discrepancies between the two studies may be explained in part by factors such as differences in body position, where the subjects used by Shipp et al., were supine, while those used by Sundberg were upright. Other factors may include differences in amounts of anesthesia (no study reported amounts administered), equipment differences, and most importantly perhaps, the fact that CT activity may have been measured at different lung volume levels. Neither study controlled for lung volume. From the description of their methods, it appears that Sundberg and colleagues measured CT activity close to FRC, while Shipp and colleagues seem to have measured it possibly close to the residual volume.

Subglottal Pressure

Iwarsson et al. (Iwarsson et al., 1995) suggested that lower subglottal pressures were associated with lower lung volumes. In this study, subjects with untrained voices showed higher subglottal pressures at high lung volumes than at low lung volumes. Subglottal pressure decreasing with decreasing lung volumes has also been reported in singing without support (Griffin, Woo, Colton, Casper, & Brewer, 1995; Sonninên, Hurme, & Sundberg, 1994). However, these findings are not in accordance with Bouhuys et al. (Bouhuys et al., 1966) who reported rather constant subglottal pressure values
throughout the full range of the vital capacity. Several reasons may account for this discrepancy, such as dissimilar tasks, different instrumentation techniques, and differences in subjects voice training. Bouhuys and colleagues used esophageal balloons as a means to estimate subglottal pressure, their task was sustained vowel phonation, and the majority of their subjects had singing experience. On the other hand, more recent studies used measures of oral pressure to estimate subglottal pressure, a syllable repetition task instead of sustained phonation and subjects had either no voice training (Iwarsson et al., 1995), or sustained the sound with "no support" (Griffin et al., 1995; Sonninen et al., 1994). Furthermore, and probably most important, there is no indication in the Iwarsson et al. study whether SPL was controlled for or not. Findings of the more recent studies suggest that untrained or unsupported voices may not counter the effect of the elastic recoil forces efficiently, resulting in somewhat lower subglottal pressures at the lower end of the vital capacity (Iwarsson et al., 1995).

Voice Quality

A single case report by Hixon and Putnam (Hixon & Putnam, 1987) commented on the relation between voice quality and lung volume. They described a 30-year-old female who produced speech at low lung volumes, far beyond her FRC level, and who spoke with higher lung volume expiratory excursions than normals. Her voice quality at low lung volumes was described as "more authoritative and business-like". When required to phonate at a higher lung volume range than the one in which she usually produced speech, her voice was reported to become less harsh and with increased breathiness.
Glottal Adduction Measures

Glottal compliance, obtained as the ratio of the glottal flow pulse area to subglottal pressure, is believed to represent the degree of glottal adductive forces (Iwarsson et al., 1995). The authors reported reduced glottal compliance at high lung volumes for a constant subglottal pressure and pitch during sustained phonation throughout the VC. Flow closed quotient values were also reported to increase at low lung volumes, showing that levels of glottal adduction increased at low lung volumes, but only for subjects who expanded their abdominal wall during deep inspiration. The authors suggested that changes in these measures were related to specific breathing strategies of individual subjects.

Preliminary Videoendoscopic Data

Preliminary videoendoscopic data was obtained to examine laryngeal configuration changes towards the lower end of the VC. Four females and three males, age range 23 to 55 years participated. All subjects had normal larynges, with no history of voice pathology, and no vocal complaints at the time of examination. One female subject had 7 years of classical voice training. The rest of the subjects had no vocal training and no experience in singing, although all were able to match vocally a target pitch. The task consisted of sustaining the vowel /i/ at a target pitch. Subjects were instructed to start phonation at a comfortable lung volume level and sustain phonation until complete expiration to the reserve volume (RV) level. Subjects were carefully
instructed, and coached during the execution of the task to keep the target pitch, and to maintain phonation as long as they possibly could. Laryngeal visualization during the tasks was performed using a Kay RLS videostroboscopy system, with a rigid 70-degree angle endoscope.

All subjects showed some degree of involvement of supraglottal structures, evidenced by reduction of the diameter of the laryngeal vestibule during phonation towards the low lung volumes. Reduction of the diameter of the vestibule was observed in both the antero-posterior and lateral dimensions. Five subjects exhibited constriction in both dimensions. Two subjects exhibited reduction of the antero-posterior length alone, suggesting differences in individual strategies. The subject with classically trained voice showed antero-posterior constriction alone, that was of lesser degree than the other subjects, and occurred at the very end of her phonation. Furthermore, this subject exhibited a greater separation between her ventricular folds towards the end of the VC. This was the only subject in which this behavior was observed. Both, the very late occurrence of antero-posterior constriction and the separation of her ventricular folds rather than approximation, suggested that her classical training may have impacted on her strategy to adjust for changes in lung volume.

Five subjects (four females and one male) presented an incomplete closure of the posterior glottis during normal phonation. During task performance, the posterior gap was observed to gradually close towards the lower lung volumes. In all five subjects, closing of the posterior glottal gap occurred before constriction of the vestibule was observed, making it the first variable to display a change in relation to decreasing lung volume.

These preliminary data suggested that phonation at low lung volumes elicits an increased degree of glottal closure, and a reduction of the diameter of the vestibule. The supraglottal behaviors exhibited by our subjects during phonation at low lung volumes
resembled signs of vocal hyperfunction. Unfortunately, no lung volume measures were obtained, therefore, no information was obtained about the percentage of the VC at which the adjustments of the supraglottal structures occurred. An additional limitation of this pilot study was the use of transoral endoscopic procedures that could have effected some of the behaviors being observed (Sodersten & Lindestad, 1992).

**Summary of Laryngeal Dynamics as a Function of Lung Volume**

Following is a summary of the previous findings that have been reported with respect to laryngeal adjustments that occur during phonation at different lung volumes:

1) Vertical position of the larynx (VLP) in the neck: The larynx has been reported to descend at high lung volumes, gradually rising as lung volumes get lower. Several researchers suggested that the downward movement of the larynx in the vertical dimension at high lung volumes is caused by flattening of the diaphragm, which in turn pulls down on the trachea and larynx.

2) Voice onset time: Longer onset times have been reported at high lung volumes than at low lung volumes. The authors attributed the longer onset times at high lung volumes to a “tracheal pull”, and the shorter times at low lung volumes to a possible drive of the system to conserve air.

3) Electromyographic activity of the cricothyroid muscles: Two investigations reported opposite results, one found increased and the other decreased CT EMG activity while phonating at low lung volumes. Even though the results are conflicting, it seems apparent that the CT displays changes in levels of activity when phonation is sustained at a constant pitch throughout the vital capacity.

4) Subglottal pressure: There are reports of higher subglottal pressures at high lung volumes as compared with pressures at lower lung volumes.
5) Glottal adduction: levels of glottal adduction have been suggested to increase at low lung volumes, at least for individuals who used a particular respiratory strategy of expanding their abdominal wall during deep inspiration.

6) Voice quality: It has been reported that voice quality becomes strained towards the end of the VC.

7) Supraglottal configuration: There is evidence of increased shortening of the antero-posterior length of the vestibule, and medialization of the ventricular folds towards the end of the VC.

8) Glottal closure: There is evidence of increased glottal closure towards the end of the VC.
CHAPTER III
METHODS

Subjects

Subjects included 14 women with normal voices. The age range was 19 to 34 years, with a mean age of 25 years. The criteria for selection of subjects included: normal articulation, resonance and language ability; normal hearing screened bilaterally at 20 dB HL at 500, 1000, and 2000 Hz (re: ANSI, 1969); no history of respiratory pathology, no history of smoking, and free from allergies or colds on the day of testing. Additional criteria included no history of vocal pathology and no history of formal vocal training in singing. Subjects with professional voice training were excluded because there is evidence that there are differences between trained and untrained voices in vocal function measures (Peppard, Bless, & Milenkovic, 1988; Schutte & Miller, 1983; Shipp, 1987), and in respiratory function measures (Hoit et al., 1996; Watson & Hixon, 1985). Voice training may induce the use of learned physiological strategies to cope with changes in lung volume, masking the effects of natural mechanical respiratory forces on the laryngeal apparatus (Gould & Okamura, 1973).

Tasks

1) syllable repetition task: Subjects were instructed to phonate the syllable /pae/ repeatedly at an utterance rate of 1 per sec, and at comfortable pitch and loudness levels throughout nearly the entire vital capacity range, starting phonation at the highest lung volume possible, and sustaining syllable repetition until they could no longer perform the task, near the reserve volume (RV).
2) sustained vowel: Subjects were instructed to sustain production of an /ae/ vowel at comfortable pitch and loudness levels throughout nearly the entire vital capacity range, starting phonation at the highest lung volume possible, and sustaining phonation until airflow became insufficient to maintain vocal fold vibration.

The vowel /ae/ was chosen because of its high frequency first formant, and the relative frequency separation between its first and second formants, which makes it appropriate for inverse filtering. A high first formant reduces interaction with the fundamental frequency, and clear frequency separation between formants, reduces interaction between them (Miller, 1959).

The syllable /pae/ (voiceless bilabial stop and vowel) was chosen because it enables estimation of subglottal pressure during the lip closure of the /p/ segment and translaryngeal airflow during the vowel segment (Smitheran and Hixon, 1981).

Instrumentation

All physiological signals were collected simultaneously with a sixteen channel computer-based data acquisition system consisting of a 32 channel 12-bit 333kHz Analog to Digital (A/D) converter (Digidata 1200, Axon Instruments Inc.). Signals were amplified and filtered with a signal conditioning amplifier (CyberAmp 380, Axon Instruments Inc.), and monitored in real time during data collection on the system’s acquisition software (AxoScope, Axon Instruments Inc.). All signals were digitized at a sampling frequency of 10 kHz (see figure 3.3).
Respiratory Function

Non invasive measurements of respiratory function were made via the kinematic recording of thoracic and abdominal wall movements, obtained with 2 channel linearized magnetometers (GMG Scientific Inc.). The kinematic method considers the chest wall as a two-part system (rib cage and abdomen) in which each part displaces volume as it moves, and together they displace a volume equal to the volume displacement of the lungs (Konno & Mead, 1967). Consequently, antero-posterior diameter changes of the rib cage and abdomen can be used after calibration to estimate directly their individual volume displacement, as well as the total lung volume changes. Two pairs of magnetometers (one generator and one sensor in each pair) were used to measure rib cage and abdomen antero-posterior diameter changes (Hixon et. al. 1973). The magnetometers were glued directly to the subjects’ skin with a cement that is specially design to adhere appliances to skin surface (Skin-Bond®, Smith & Nephew United, Inc., Florida). The rib cage generator coil was glued anteriorly on to the sternum, at a level above the nipples and below the axilla. The abdominal generator coil was placed either above or below the navel, depending on each subject’s abdominal configuration when in a sitting position. The sensor coils were glued to the back of the torso, at the same axial level of the generator coils.

The magnetometer signals were monitored on a storage oscilloscope (Tektronix 5113), low pass filtered at 200 Hz (4 pole- Bessell filter), amplified, digitized, and recorded on the data acquisition system for further analysis. In addition, signals were monitored on an oscilloscope throughout the experiment, in a motion-motion display, with the ribcage signal in the Y-axis versus the abdominal signal in the X-axis.
Laryngeal Function

Electroglottographic waveforms and tracking of the vertical laryngeal position were obtained with a twin channel Rothenberg electroglottograph (Glottal Enterprises MC2-1). Two pairs of electrodes with a vertical distance of 0.5 cm were attached to a neckband and placed on either side of the subject’s thyroid cartilage. The EGG waveform signal was low passed filtered at 1800 Hz (8 pole- Bessell filter) to prevent digital antialiasing. The laryngeal tracking signal was low pass filtered at 20 Hz (4 pole- Bessell filter).

Oral flow was measured by means of a pneumotachograph attached to a Rothenberg mask (Rothenberg, 1977). This mask has circumferentially placed vents that are covered with a fine-mesh wire screen. The air pressure difference created across the screen by the airflow passing through it, was measured by a differential pressure transducer (Glottal Enterprises) and used to estimate transglottal flow. The output signal of this pressure transducer was low pass filtered at 1400 Hz (4 pole- Bessell filter). The electrical output of the transducer was calibrated in terms of airflow. The mask’s venting screen was heated in order to minimize the effects of condensation.

Intraoral air pressure was measured by means of a second differential pressure transducer (Glottal Enterprises) with a flat frequency response up to about 30 Hz. This transducer was attached to the facemask and connected to a short, thin catheter passed between the subjects lips into the oral cavity. The oral air pressure signal was low pass filtered at 80 Hz (4 pole- Bessell filter).

Sound pressure was sensed with a small mask-mounted Sony ECM-50 PSW electret condenser microphone. Mounting the microphone on the mask allowed for a
constant reproducible distance from the subject’s lips. This signal was low pass filtered at a 4500 Hz (8 pole- Bessell filter).

In addition, the Rothenberg mask was modified by adding ports in the nasal portion of the mask through which a flexible fiberscope could be passed (Kobler, J.B., Hillman, R.E., Zeitels, S.M., & Kuo, J., 1998). Such modification made it possible to acquire videolaryngoscopic data simultaneously with other measures. The added port incorporated an O-ring seal modified from a Tuohy-Borst hemostatic control valve, which provided a tight seal around the endoscope. During the experiment the mask was tightly strapped to the subject’s face. Maintaining an airtight seal around the endoscope and between the mask and the subject’s face was important to prevent air leaks that could introduce errors in glottal aerodynamic measures. Figure 3.1 shows the modified Rothenberg mask and its components. Figures 3.2a and 3.2b show the mask strapped to a subject’s face and the transnasal endoscope inserted through it during examination of one of the subjects.
Figure 3.1 Modified Rothenberg mask showing the straps used to attach the mask to the subject's face, the position of the microphone, and the added ports for the insertion of the flexible transnasal endoscope.
FIGURE 3.2 (a) Close-up of mask strapped to subject's face while the transnasal endoscope is being inserted through the right port into her right nasal passage. 
(b) View of investigator and subject during experimental procedure for simultaneous recording of acoustic, aerodynamic, and video-stroboscopic data.
An important concern was whether or not the instrumentation that was used could have affected the naturalness of the speech being produced. The two pieces of instrumentation that could possibly have interfered with sound production were the facemask and the flexible fiberscope. The facemask used has a measured resistance of 0.30 cm H₂O/liter/second. This value produces an acceptable level of acoustic distortion for most speech measurements (Rothenberg, 1973). Moreover, since the speech tasks in the present study were limited to sustained vowels and syllable repetition, articulation was not expected to be significantly altered by the facemask.

The most invasive procedure in this study was transnasal endoscopy. In order to test for nasal air escape potentially introduced by the placement of the fiberscope through the velopharyngeal port, average oral flow and pressure were measured in two subjects prior to and during the insertion of the scope. No significant differences in airflow rates or pressures were found between these two conditions. Hertegard and colleagues (Hertegard & Gauffin, 1991; Hertegard & Gauffin, 1995) also obtained simultaneous recordings of aerodynamic and videolaryngoscopic data, reporting negative findings of nasal flow during phonation with a fiberscope in place. As an additional precaution, the naturalness of the speech was monitored during the experiments. However, the naturalness of the speech being produced, in particular during phonation at low lung volumes, was expected to be somewhat affected by the nature of the speech tasks themselves, given that subjects were asked (pushed) to function at their lung volume limits.
Laryngeal Imaging

Examination of the larynx was performed using a flexible fiberoptic laryngoscope (Olympus ENF-P 3.5 mm). The endoscope was connected to a stroboscope (Kay Elemetrics) and to video equipment (CCD-camera, Panasonic AG-1960 ProLine S-VHS video-recorder, and Panasonic BTS901Y monitor). The fiberscope was inserted through the O-ring valve of the facemask, and into the subject's nasal cavity. The tip of the fiberscope was placed below the level of the epiglottal rim at the beginning of vocalizations and held constant relative to the mask throughout each task.

The stroboscope system was placed outside of the sound treated room in which data were collected, to eliminate the high level of noise that it produces from the acoustic recordings. This was accomplished by extending the fiberoptic light cable of the flexible transnasal endoscope.

Synchronization of Video and Physiological Signals

The physiologic signals and the videoendoscopic images were time synchronized with a 690 Time Code Reader/Generator (Shintron). The video signal was routed through the time-code generator, which generates a character output with time-code information. This information was imprinted on each video frame. Another output, which codes time in terms of a longitudinal series of square wave pulses, was recorded on one channel of the data acquisition system along with the physiological signals to allow time alignment of video images with these signals. A schematic of the data acquisition system is illustrated in Figure 3.3
FIGURE 3.3. Schematic of data acquisition system
Recording Procedure

Subjects were first given an explanation of the protocol, and then signed a subject’s consent form. A copy of the consent form is included in Appendix 1 (this consent form is part of a larger protocol, thus, it includes explanation of certain procedures that did not form part of the present study). Subjects were paid for their participation in the study. The experiment was conducted inside a sound isolated booth (IAC, Industrial Acoustic Company, Inc., New York). Subjects were familiarized with the instrumentation, and asked to practice the experimental tasks (including respiratory maneuvers and sustaining constant SPL during phonation throughout the VC). A research assistant helped with the placement of the magnetometers, and covered the subject’s torsos with a hospital gown. Throughout the duration of the experiment subjects were seated straight up on an otolaryngologic examination chair. Respiratory calibration maneuvers were obtained first (see section below). After the respiratory calibration was performed, care was taken in monitoring subjects to prevent postural changes that may affect the respiratory kinematic signals. Subsequently, a topical solution consisting of a mixture of 3% lidocaine hydrochloride and 0.25% phenylephrine hydrochloride was sprayed in the subjects’ nostrils to facilitate endoscope insertion and tolerance. Next, the facemask was strapped to the subjects face, and the flexible nasoendoscope was inserted. Two experimenters were inside the booth. One performed the laryngeal examination and coached the subject, while the other ensured that all the experimental criteria were met. A third experimenter was outside the booth, operating the data-acquisition system and monitoring the quality of the signals. Subjects were instructed to keep head, neck, and shoulders as still as possible during the tasks. Prior to the performance of each task, subjects were instructed to reach their EEL level by sighing and relaxing at the end of the expiration, and to hold their breaths by closing the glottis, staying still for a few seconds. The larynx was monitored via endoscopy to ensure that the breath holding was performed with a closed glottis. This was done in an effort to prevent airflow inside of the mask and
obtain a zero flow reference for calibration purposes. Subjects were not required to reach specific SPL and F0 targets, but were instructed to produce the utterance at a comfortable pitch and loudness level, maintaining them constant throughout the entire vocalization. During the performance of the tasks subjects were given feedback on their target loudness level by means of an SPL meter. Tasks were repeated three times to compare subject’s performance across different trials. At the end of the experiment, another set of respiratory calibration maneuvers was obtained to ensure that there was no shift in the magnetometers placement, and that the subjects did not shift position during the tasks performance. The time required for completion of the protocol was approximately 2 ¼ hours.

Calibration Procedures

Calibration for Lung Volume

A series of chest wall maneuvers were conducted at the beginning and at the end of data collection to calibrate the system for lung volume and to obtain the kinematic landmarks corresponding to lung volume change. These included isovolume maneuvers at the end expiratory level (EEL), and vital capacity maneuvers. During these maneuvers the subjects’ nostrils were occluded with a nose-clip to prevent any air leakage through the nares. The isovolume maneuvers were performed by having the subject sigh out until reaching the desired lung volume. Upon reaching this level subjects were asked to close the mouth and slowly displace volume back and forth between the abdomen and the rib cage. During the vital capacity maneuvers subjects were asked to perform a full inspiration, and to expire fully through a mouthpiece coupled to a spirometer. The largest of the three expiratory excursions was considered as the criterion measure of VC. The points of total lung capacity (TLC), and reserve volume (RV) were obtained from the VC maneuver. Vital capacity maneuvers were also used in determining the subjects’ range of
rib cage volumes. The ranges of abdominal volumes were obtained by asking the subjects to displace the abdomen inward maximally and outward maximally while holding their breath at the end expiratory level.

Respiratory signals were calibrated in terms of volume using an 8-liter Collins Survey II spirometer. Spirometer calibration was validated with a 3-liter calibration syringe (CDX Corp.).

Isovolume maneuvers served to establish the functional relationships between the relative motion of the two chest wall parts from which volume-motion relationships were determined. The isovolume maneuvers were used to weight equally the contributions of the rib cage and abdominal signals (Hoit and Hixon, 1987) using custom software written in MATLAB (The Math Works Inc.). These signals were summed and calibrated against the spirometer signal. Respiratory measures were obtained from the calibrated summed signal and expressed as percent vital capacity (%VC).

Vertical Laryngeal Position Calibration

For calibration purposes the EGG electrodes were mounted on a specially designed calibrator. The calibrator has a linear position transducer with a known range of 4 cm. The vertical laryngeal position tracking signal was recorded simultaneously with the calibrator signal (Glottal Enterprises LTC-1). The calibration maneuver was performed by sliding the EGG electrodes up and down the subject’s throat, while the subject phonated an [ae:] vowel at the midrange of lung volumes. Subsequently a linear regression was computed by plotting the EGG tracking output against the output of the calibrator.
A limitation of this technique is that, since it requires phonation, it provides no clear reference with respect to the resting position of the larynx. Therefore, the values attributed to laryngeal height were obtained within each phonatory segment, without reference to a resting laryngeal position. The minimum amplitude of the laryngeal tracking signal during the segment of phonation was assigned the value 0 mm.

**Acoustic and Aerodynamic Calibration**

Sound pressure, air pressure and airflow calibration signals were recorded at the end of each recording session. All calibrations were conducted in the same sound-isolated booth that was used during the experiment. Calibration of the microphone was obtained by recording a calibration signal produced with the tone generator of a Cooper Rand electrolarynx. The sound pressure level of the signal was monitored with a Rion NL-11 sound pressure level meter (on a linear scale, 400-ms averaging time) placed next to the microphone.

The flow transducer and mask were calibrated with a series of 6 known dc flow levels, ranging from 0.067 to 0.417 liters per second, produced with a compressed air supply and monitored on a tri-flat flow meter tube (Fischer and Porter Co.). The flow source was coupled to the facemask via a custom-made mold. The air pressure was calibrated with a series of six pressure levels, from 0 to 24 cmH₂O, generated with a syringe and monitored with a U-tube water manometer. The data extracted from the calibration signals were used to derive scale factors in order to convert signal levels to actual values in dB (SPL) units, ml/s units, and cmH₂O units.
Derived Signals and Measures

The utterance duration was measured from the microphone signal using cursors in the Axoscope software (Figure 3.4)
FIGURE 3.4. Example of signals acquired simultaneously. Start and finish represent the onset and offset of phonation measured from the microphone signal.
Estimates of lung volume were obtained from the summed rib cage and abdominal respiratory signals. Signals were measured at three lung volume levels, which were superimposed onto the vital capacity that was established separately prior to phonation. The condition high LV was obtained at 80% VC, the condition mid LV was obtained at 40% VC, and the condition low LV was obtained at 20% VC, or the closest % VC that corresponded to a mid-vowel portion for the /pae/ syllables. Measures were obtained at mid-vowel portions, to minimize possible effects of articulation. Figure 3.5 shows an example in which the LV measures were obtained at 83.5%, 37.5%, and 18.5% VC, which corresponded to the mid-vowel portions closest to 80%, 40%, and 20% VC.
FIGURE 3.5. Lines marked as a, b, and c, correspond to the sections of the signals at which the measures were obtained. These lines correspond to the conditions high, mid, and low lung volumes at 83.5%, 37.5%, and 18.5% VC respectively. Data was analyzed at the mid-vowel portions closest to 80%, 40%, and 20% VC. Note how in this case phonation started at 95% VC and was sustained to approximately the end of VC.
Inverse filtering was performed using custom made software. The software enables inverse filtering based on the frequencies and bandwidths of the first and second formants. The criterion used for adjusting the filters was the minimization of ripple in the closed phase cycle of the flow glottogram. Analysis of the syllable repetition segments was performed from the middle portion of the selected vowels in an effort to avoid traces of articulatory movement. The following measures were derived from inverse filtered estimates of the glottal airflow waveform:

a) Peak airflow (l/sec), defined as the maximum flow from zero baseline.

b) AC flow (modulated flow), defined as the difference between peak and minimum flow.

c) minimum flow (l/sec), defined as the amount of flow from baseline to the minimum flow during the flat part of the closed phase.

Measures of intraoral air pressure during /p/ production were made to estimate subglottal air pressure following the method developed by Smitheran and Hixon (1981). From now on, estimates of intraoral air pressure will be referred to as subglottal pressure.

An estimate of the relative vertical laryngeal position (VLP) was obtained from the calibrated vertical tracking signal (twin channel Rothenberg electroglottograph).

Videostroboscopic data

A visual-perceptual study using a paired comparisons paradigm was conducted to analyze the videostroboscopic data. Perceptual evaluations were obtained from five judges who had extensive experience in videostroboscopic evaluation. Judges were uninformed about the purpose of the study. The judges were first required to rate presence or absence of incomplete posterior glottal closure for each image of the pair. In
cases of incomplete glottal closure, they then indicated which of the two images displayed a larger posterior glottal gap. Finally, judges indicated which of the two images had a greater degree of laryngeal compression. The criteria given to the judges to characterize laryngeal compression were: a) Excessive arytenoid adduction (arytenoid squeezing); b) shortening of the antero-posterior glottal length (antero-posterior compression); c) shortening of the ventricular fold distance (ventricular compression); d) shortening of the aryepiglottic folds, with epiglottal retraction; and e) overall changes in shape of laryngeal vestibule. Judges could take as much time as they wanted to rate each paired comparison. Each judge completed the evaluation of images two times in random order to test intra-judge reliability.

For each subject trial three frames showing maximal glottal closure were digitized corresponding as closely as possible to the time points at which the high, mid and low lung volume aerodynamic, acoustic and EGG measures were obtained. The disparity between the time when the video image was acquired and the time when the waveform data was measured could have ranged from 0 to .75 seconds, depending on the phase of the glottal cycle relative to the time points at which the lung volume signal reached the 80, 40, and 20% vital capacity levels. 5 utterances (2 sustained vowels and 3 syllable repetitions) had to be excluded because of poor visibility of the vocal folds. Therefore, 79 utterances were used ([14 normal subjects x 3 repetitions x 2 tasks] minus 5 excluded).

The three images from each subject trial were used to create two pairs of images for comparisons. One pair compared images corresponding to the high and mid lung volume levels, and the other pair compared the images for the mid and low lung volume levels. These pairings yielded a total of 158 comparisons (79 utterances x 2), which were presented to the judges in random order via computer. Figure 3.6 and 3.7 show examples of pairs of images that were obtained from two subjects performing the sustained vowel task.
FIGURE 3.6. Examples of two image pairs from a subject's trial of a sustained vowel. Pair (a) compares high and mid lung volume level conditions, while pair (b) compares low and mid lung volume levels. Note the large posterior glottal gap evident at high lung volumes, which is not longer seen at mid lung volumes, and the severe antero-posterior supraglottal compression seen at low lung volume levels.
FIGURE 3.7 Examples of two image pairs from a subject's trial of a sustained vowel. Pair (a) compares high and mid lung volume level conditions, while pair (b) compares low and mid lung volume levels. Note the posterior glottal gap evident at high lung volumes, which reduced at mid lung volumes, and the difference between the degree of inter-arytenoid squeezing between the mid and low lung volume conditions.
Data Analysis

In order to verify the reliability of the measures obtained during the production of sustained vowel and syllable repetition tasks, twenty percent of the data (corresponding to three subjects) were reanalyzed by the principal investigator. The inter-judge data were subjected to a Pearson correlation coefficient analysis. A p value of <.01 was chosen for significance testing.

Statistical analysis of the data was performed using SYSTAT 7.0 (SPSS Inc.). A detailed evaluation of results for individual subjects was the first stage of data analysis. Analysis of data for individual subjects primarily involved plotting and visual inspection of individual measures. Plots were made of the raw data to compare differences (changes) in data values across measurement points. For this initial inspection, respiratory measurement points were obtained at 5% VC intervals, starting at the highest lung volume at which the task was initiated, throughout the end of phonation. The rest of the signals were plotted against %VC attempting to identify patterns in the way the sets of measures changed throughout the task performance. All data (measured at 5% VC intervals) corresponding to the dependent variables VLP, AC flow, and minimum flow were investigated with a best fit function. This was done in order to examine patterns of change in the dependent variables as a continuous function of the independent variable. The predictor variable in the curve-fitting regression was lung volume, allowing the examination of patterns of change in the dependent variables as lung volume changed from high to low. The formula used for the curve fitting analysis was: $y = ax + b$, where: $y =$ value of dependent variable; $x =$ lung volume; $a =$ slope; and $b =$ intercept. The curve fitting analysis was based on individual trials. A conservative probability level of .01 was chosen for significance testing.
Group-based comparisons were subsequently carried out. The primary goal was to determine the extent to which the dependent variables were significantly altered when lung volume was manipulated. For group comparisons, performance under three experimental conditions was evaluated: high, mid, and low lung volume. These conditions refer to percentages of the VC at which the measures were obtained. The condition “high lung volume” was obtained at 80% VC, “mid lung volume” was obtained at “40% VC, and “low lung volume” at 20% VC, or the closest mid vowel location that corresponded to the target %VC level. Group-based analyses were performed initially for individual variables. Summary statistics included means, standard deviations, and ranges for all dependent variables across the three lung volume conditions.

The data were subjected to a univariate repeated measures analysis of variance for each dependent measure across the lung volume condition. The grouping factor “Subject” (14 subjects) was the between-subject factor, and task (sustained vowel vs. syllable repetition) and lung volume level (high vs. mid vs. low LV) the within-subject factors. The variable Pressure was subjected to a repeated measures ANOVA with one between subjects factor (subject), and only one within subjects factor (lung volume level), because pressure data was solely obtained during repeated syllables task. Contrast coefficients were calculated between the levels of the independent variable lung volume. This allowed comparisons to be made between each of the three lung volume levels. Repeated univariate analyses are often associated with problems like loss of statistical power and the increased probability of finding false significance, therefore, a conservative probability level of .01 was used for significance testing.

Given that SPL correlates with a number of laryngeal function measures (Holmberg et al., 1988; Klatt & Klatt, 1990; Stevens & Hanson, 1995), a Pearson r correlation was performed to determine whether there was a significant relationship between SPL and the other dependent variables. If a significant correlation was found, the data for that dependent variable was adjusted in an effort to correct for the potential
influence of SPL variation. A multiple regression was computed for each subject using all levels of lung volume and their associated SPL values. Then, using the regression equation, the predicted value of the dependent variable was computed for each of the three lung volume level conditions and their associated SPL values. Finally, the predicted values of the dependent variable, taking into account both lung volume and SPL values, were entered into the ANOVA with three lung volumes and two tasks as treatments.
CHAPTER IV
RESULTS

Descriptive and inferential statistics were used to analyze the acoustic, aerodynamic, electroglottographic and videoendoscopic data. Means and standard deviations were calculated for each lung volume condition across the three trials of each task for individual subjects. The trial means were used to calculate group means and standard deviations for all measures at each lung volume condition.

Reliability

Intra-judge reliability for measures obtained during sustained vowel, and syllable repetition tasks was calculated via Pearson correlation coefficients. 20 percent of the data (corresponding to 3 subjects) were re-measured. Pearson correlation coefficients ranged from 0.845 to 0.994 (\( \bar{X} = 0.958 \) and \( \text{sd} = 0.063 \)) for sustained vowels, and ranged from 0.798 to 0.987 (\( \bar{X} = 0.949 \) and \( \text{sd} = 0.048 \)) for syllable repetition tasks. All were highly significant (p-values < 0.0031).

Group Data
Utterance Duration

Descriptive statistics for utterance duration showed that the utterance duration averaged across subjects was 18.62, \( \text{sd} = 4.32 \), ranging between 11.44 and 26.29 for sustained vowels, and 16.45 seconds, \( \text{sd} = 4.28 \), with a range of 8.36-25.21 for syllable repetitions.
A t test for paired comparisons \((n = 14)\) compared the mean phonation time for sustained vowel \(/ae/\) task with the mean phonation time during a syllable repetition \(/pa\overline{e}/\) task. This test was found to be statistically significant, \(t = 4.575, p < 0.001\), verifying that subjects produced longer utterances when sustaining a vowel \((\overline{X} = 18.619 \text{ sec.})\) than when they were producing a syllable train \((\overline{X} = 16.451 \text{ sec.})\).

**Acoustic Measures**

**Fundamental Frequency**

The fundamental frequency \((F_0)\) averaged across subjects and lung volume conditions was 232.38 Hz, \(sd = 29.56\), with a range of 203-291 Hz. The average values for \(F_0\) are shown in Table 4.1. \(F_0\) values were subjected to a repeated measures analysis of variance with three levels of lung volume (high, mid, and low), and two levels of task (sustained vowel and syllable repetition). None of the main effects of lung volume \((F = .32, p = .72)\) and task \((F = 1.19, p = .91)\), or the lung volume by task interaction \((F = 1.14, p = .33)\) were found to be statistically significant, suggesting that subjects maintained a constant pitch level across lung volume levels for production of both sustained \(/ae/\)'s and \(/pa\overline{e}/\) syllable strings (see Figure 4.1 and Table 4.2).
<table>
<thead>
<tr>
<th>Lung Vol</th>
<th>Task</th>
<th>F0 (Hz)</th>
<th>SPL (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>ae</td>
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<td>53-87</td>
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<tr>
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<tr>
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<td></td>
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<tr>
<td>Range</td>
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<tr>
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<td>232.63</td>
<td>79.16</td>
</tr>
<tr>
<td>SD</td>
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<td>9.65</td>
</tr>
<tr>
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<td>188-295</td>
<td>53-89.7</td>
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<tr>
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<th>F ratio</th>
<th>p</th>
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<td>0.324</td>
<td>0.724</td>
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<tr>
<td></td>
<td>Task</td>
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<td>0.284</td>
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<td>1.14</td>
<td>0.33</td>
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<tr>
<td></td>
<td>Subjects</td>
<td>13</td>
<td>55.33</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Subjects x Lungvol</td>
<td>26</td>
<td>3.31</td>
<td>&lt; 0.001*</td>
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<tr>
<td></td>
<td>Subjects x Task</td>
<td>13</td>
<td>0.95</td>
<td>0.54</td>
</tr>
</tbody>
</table>

| SPL (dB) | Lung Vol            | 2  | 7.003    | 0.002* |
|          | Task                | 1  | 71.216   | < 0.001* |
|          | Lung Vol x Task     | 2  | 5.22     | 0.008* |
|          | Subjects            | 26 | 4.23     | < 0.001* |
|          | Subjects x Task     | 13 | 10.06    | < 0.001* |
|          | Subjects x Lungvol x Task | 26 | 1.46 | 0.11 |

Note. * p < .01

**TABLE 4.2.** Analysis of variance summary table for acoustic variables for main effects of Lung Volume, Task, and Subjects, and interaction effects for Lung Volume x Task, Subjects x Lung Volume, Subjects x Task, and Subjects x Lung Volume x Task.
Acoustic Measures

Figure 4.1. Mean values and standard errors for the acoustic measures of fundamental frequency (F0) and sound pressure level (SPL) across subjects for the three lung volume levels. Lung volume 1 = high; lung volume 2 = mid; lung volume 3 = low. Graphs to the left correspond to repetitions of /pæ/ syllables, and graphs to the right correspond to sustained /æ/ vowels.
Sound Pressure Level

The average values for SPL are shown in Table 4.1. Subjects were instructed to maintain SPL constant during phonatory tasks and were provided with visual feedback via an SPL meter. Analysis of individual data revealed that whereas a majority of subjects were able to maintain SPL constant throughout their speech tasks, some showed larger than +/- 1 dB SPL variations during task performance. During the syllable repetition task subjects S1 and S8 showed an average decrease in SPL of 5.25, and 2.64 dB respectively, while subjects S2 and S11 showed average increases in SPL values of 2.54, and 3.08 dB respectively. During the sustained vowel speech task, a decrease in SPL was displayed by subjects S1 ($\bar{X} = 5.83$); S3 ($\bar{X} = 2.55$); S4 ($\bar{X} = 4.65$); S9 ($\bar{X} = 2.92$); S12 ($\bar{X} = 3.08$); and S13 ($\bar{X} = 2.69$); whereas S2 showed an average increase in SPL of 2.42 dB.

Repeated measures analysis of variance for SPL showed that lung volume was statistically significant ($F = 7.003, p = 0.002$). Polynomial contrasts between lung volume levels revealed that average SPL values at the high ($\bar{X} = 78.67$ dB.), mid ($\bar{X} = 78.07$ dB.), and low ($\bar{X} = 77.80$ dB.) lung volume were all significantly different from one another ($p > .01$). The main effect of task was also significant ($F = 71.21, p < 0.001$) with repetition of the /pæ/ syllable strings ($\bar{X} = 79.30$ dB.) being produced significantly louder than the sustained /æ/ vowel ($\bar{X} = 77.05$ dB). There was a significant interaction between lung volume and task, ($F = 5.22, p = .008$), meaning changes in SPL across lung volume were task dependent, with sustained vowels being associated with larger changes in SPL than syllable repetitions. See Table 4.2.

The subject main effect was statistically significant ($F = 169.67, p < 0.001$), as was the subject by lung volume and subject by task interactions. The 3-way interaction of lung volume-by-task-by-subject was not significant ($F = 1.46, p = .11$). Thus, the nature
of changes in SPL across lung volume varied depending on which task was produced, but that relationship did not vary significantly across subjects.

Overall group based Pearson product moment correlation coefficients between SPL and AC flow ($r = .30$), and between SPL and Pressure ($r = .01$) proved to be non-significant.

Differences in individual subject performances may explain the fact that even though group based correlation coefficients were non significant, the results from the repeated measures ANOVA showed a small but significant SPL change across lung volume levels. Therefore, given that the ANOVA showed significant differences, and the clear observation that not all subjects were able to maintain constant SPL levels, it was decided to also carry out an analysis in which both AC flow and Pressure values were adjusted for SPL variability in an effort to account for the potential influence of SPL on these measures, even though group based correlations between these variables were non significant.

**Electroglottographic Measure**

**Vertical Laryngeal Position (VLP)**

Vertical laryngeal excursion data was obtained for 13 subjects. In one subject the VLP signals were erratic, probably due to instrumentation problems at the time of recording, and therefore, were not included in the analysis.

The vertical laryngeal displacement ranged from 4.03 mm (subject 14) to 32.35 mm (subject 8), with an average displacement across subjects of 13.14 mm (sd. 6.37). Average VLP values are shown in Table 4.3.
<table>
<thead>
<tr>
<th>Lung Volumes</th>
<th>Task</th>
<th>VLP (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ae</td>
<td>Mean 3.38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 6.14</td>
</tr>
<tr>
<td>High LV</td>
<td>Range 0-27</td>
<td></td>
</tr>
<tr>
<td></td>
<td>pae</td>
<td>Mean 3.03</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 5.94</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 0-24</td>
</tr>
<tr>
<td></td>
<td>ae</td>
<td>Mean 10.07</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 4.62</td>
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<tr>
<td>Mid LV</td>
<td>Range 2.9-20</td>
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</tr>
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<td></td>
<td>pae</td>
<td>Mean 10.73</td>
</tr>
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<td></td>
<td></td>
<td>SD 6.04</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 1.4-30</td>
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<tr>
<td></td>
<td>ae</td>
<td>Mean 10.93</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 5.15</td>
</tr>
<tr>
<td>Low LV</td>
<td>Range 0-21.8</td>
<td></td>
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<tr>
<td></td>
<td>pae</td>
<td>Mean 10.76</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD 5.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range 0.7-23</td>
</tr>
</tbody>
</table>

**TABLE 4.3.** Means, standard deviations, and ranges of EGG measure of vertical laryngeal position (VLP) for the three lung volume conditions by task. N = 14.

Plotting and visual inspection of individual trials revealed that a gradual increase in the VLP signal as lung volumes decreased was the most often occurring pattern of vertical laryngeal displacement. The data were investigated with a best fit function, in which the predictor variable in the curve-fitting regression was lung volume, allowing for the examination of patterns of VLP change as lung volume progressed from high to low. The curve fitting analysis was based on individual trials. The most frequently occurring
pattern was best characterized by a linear trend which accounted for 78.2% of the trials (61 of 78 total trials). For the remaining data, 14.1% (11 trials) were best fit with a quadratic function (p < .01), while the remaining 7.7% of the tokens displayed a more random pattern of change, which could not be characterized with a mathematical function. For the 61 trials that showed a good linear fit, the correlation coefficients ranged from $r=.704$ to $r=.998$ (p < .01), with a mean $r=.912$ (sd.= .079). The average VLP slope ranged from -.322 to -.015 mm/%VC, with a mean slope of -.141 (sd.= .072). Intercepts of the slopes ranged from .05 to 31.15 mm, with an average intercept of 14.32 (sd.= 6.873). Figure 4.2 shows the type of VLP signals that characterized the most often occurring pattern of laryngeal displacement observed in the study, with a linear goodness of fit: a gradual rise in VLP as lung volumes decreased.

Although the gradual increase in VLP during phonation between the high and mid lung volumes was the most typical pattern of VLP change, it was not universal. Some subjects showed a different vertical laryngeal displacement pattern than the rest of the group, as voice was sustained from high to low lung volumes. For example, subject S7’s VLP signal consistently decreased as lung volumes decreased, exhibiting a positive exponential slope in all 6 trials (8%). Coincidentally, this subject is the one who exhibited the largest vertical displacements (mean= 28.58 mm; sd=5.58). Figure 4.3 shows an example of subject S7’s performance on a sustained vowel.
FIGURE 4.2. Examples of typical traces of the vertical laryngeal tracking signal during production of a sustained /ae/ vowel (a), and a syllable string (repetition of /pae/); (b). The vertical tracking signal is shown on top of each graph, the acoustic signal on the bottom.
FIGURE 4.3. Example of vertical laryngeal tracking trace (top) for subject S7, which exhibited a descending VLP signal as lung volumes decreased. Bottom trace corresponds to the acoustic signal.
Subject S3 is one of four subjects who exhibited a different laryngeal displacement pattern, with a quadratic function providing the best fit (p < .01). In these cases, the larynx was observed to initially move upwards during phonation between high and mid lung volumes, and then move down towards the low lung volumes. (This "upwards-downwards" behavior was observed consistently in all 6 utterances for subject S3, 3 utterances from subject S5, 1 from S1, and 1 from S9). Figure 4.4 shows this pattern of VLP trace, which accounted for 14.1% of the total (11 tokens).
FIGURE 4.4. Example of the vertical laryngeal traces of subject S3. The vertical laryngeal trace is shown on top and the acoustic signal on the bottom. Both graphs correspond to production of sustained vowels. Note how the vertical laryngeal trace initially increases, and then drops towards the lower end of the vital capacity.
Qualitative observations on the vertical laryngeal position signal.

The direction of change in the VLP traces was validated via comparisons with videostroboscopic recordings, i.e., with the endoscope held in a constant position. Laryngeal elevation was verified by the images becoming larger, and vice versa. Therefore, consistent with previous reports (Laukkanen, Takalo, Vilkman, Nummenranta, & Lipponen, 1997), it appears that the direction of vertical laryngeal movement can be extracted from the EGG’s VLP trace. However, due to possible calibration inaccuracies, this approach may not always be reliable for measuring the absolute magnitudes of vertical laryngeal displacement.

Figure 4.5 shows an example of how videostroboscopic images were used to validate direction of VLP change. The top graph shows VLP trace (top) and acoustic signal (bottom) of a sustained vowel production. Note the VLP trace gradually increasing with progression of phonation. The image in the bottom shows a composite of 9 images from the video segment obtained simultaneously with the signals in the top graph. These images depict a superior view of the larynx during a typical maximum utterance duration task. During the task performance, the tip of the transnasal endoscope was held constant, therefore, the observed increase in size of the laryngeal structures as phonation progressed has to be a result of the larynx moving upwards. This is particularly evident in the size of the arytenoid-corniculate complex, and the aryepiglottic folds.
FIGURE 4.5. Top graph shows VLP trace (top) and acoustic signal (bottom) during a sustained vowel production. Note VLP trace gradually increasing with the progression of phonation. Bottom shows a composite of 9 images obtained simultaneously with the top graph signals. The lower right corner of each image shows the percentage of the vital capacity at the time the image was obtained. As the images progress to the right and down, %VC decreases. Note how the arytenoid-corniculate structures progressively increase in size.
Another example of the validation of the observed VLP change of direction by videostroboscopic images can be seen in Figure 4.6. Note the upwards-downwards direction of change in the VLP trace (upper graph), and how laryngeal images get larger, and subsequently smaller, as phonation progresses to the right and down from the upper left image.
FIGURE 4.6. Top graph shows a VLP trace on top and acoustic signal on bottom during a sustained vowel task. Note the upwards-downwards direction of change in the VLP signal. Lower graph shows a composite of 9 images obtained simultaneously. Phonation progresses to the right and down, starting on the upper left image. Note how laryngeal structures appear to get progressively larger, and then subsequently smaller as phonation progresses to the end of the vital capacity.
Group based results

For group comparisons, performance under three experimental conditions was evaluated: high, mid, and low lung volumes. For this purpose only three data points of each trial (obtained at the lung volume levels corresponding to 80 %, 40 %, and 20 % VC) were used. These data were subjected to a univariate repeated measures analysis of variance. Results of the repeated measures analysis of variance for VLP (n = 13) showed that lung volume effect was statistically significant (F = 603.10, p = < 0.001), while task effect was not. There was no significant interaction between lung volume and task (Table 4.4). Polynomial contrasts showed that the average VLP values at high (X̄ = 3.21 mm.), mid (X̄ = 10.40 mm.), and low (X̄ = 10.85 mm.) lung volume levels were all significantly different from one another. The lowest VLP values were obtained at the high lung volume condition, with higher values for both mid and low lung volumes. The largest difference between VLP mean values was observed at the higher end of the VC, where subjects showed a marked increase in laryngeal height as lung volumes decreased from high to mid. The change in average VLP between the mid and low lung volume conditions was not nearly as large. These results suggest that the larynx was positioned lower in the neck at high lung volume levels, gradually raising as lung volumes got lower, with the largest displacement occurring between high and mid lung volumes, and a less marked VLP change between the mid and low lung volume levels (Figure 4.7).
<table>
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<th>df</th>
<th>F ratio</th>
<th>p</th>
</tr>
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<td>603.10</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td>Task</td>
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<td>0.01</td>
<td>0.91</td>
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<td>0.30</td>
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<td>Subjects x Lungvol</td>
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<td>Subjects x Task</td>
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<td>0.001*</td>
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<td>Subjects x Lungvolume x Task</td>
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<td>1.74</td>
<td>0.04</td>
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</table>

Note. * p < .01

**TABLE 4.4.** Analysis of variance summary table for EGG vertical laryngeal position (VLP) variable for main effects of Lung Volume, Task, and Subjects, and interaction effects for Lung Volume x Task, Subjects x Lung Volume, Subjects x Task, and Subjects x Lung Volume x Task.
Electroglottographic measure

Figure 4.7. Mean values and standard errors for vertical laryngeal position across subjects for the three lung volume levels. Lung volume 1 = high; lung volume 2 = mid; lung volume 3 = low. Left graph corresponds to repetitions of /pae/ syllables, right graph correspond to sustained /ae/ vowels.

Aerodynamic Measures

Subglottal Air Pressure

Table 4.5 displays group based means, standard deviations and ranges for subglottal pressure values. Mean values ranged between 7.31 and 8.04 cmH$_2$O across lung volume levels. Also shown in table 4.5 are the means, standard deviations and ranges for the “SPL adjusted” pressure data. Mean values ranged between 7.33 and 8.04 cmH$_2$O across lung volume levels.
A repeated measures analysis of variance for pressure (Table 4.6) showed that the main effect of lung volume was statistically significant ($F = 9.64, p < .001$). Contrasts showed that while there was not a significant difference between the mean pressure values at high ($\bar{X} = 8.04 \text{ cmH}_2\text{O}$) and mid ($\bar{X} = 7.87 \text{ cmH}_2\text{O}$) lung volumes, the mean pressure values at mid ($\bar{X} = 7.87 \text{ cmH}_2\text{O}$) and low ($\bar{X} = 7.31 \text{ cmH}_2\text{O}$) lung volumes significantly differed from each other (Figure 4.8).

ANOVA results for the pressure values adjusted for SPL variability (Table 4.6) indicated that the main effect of lung volume was statistically significant ($F = 58.08, p < .001$). Polynomial contrasts showed that while there was not a significant difference between the mean pressure values at high ($\bar{X} = 8.03 \text{ cmH}_2\text{O}$) and mid ($\bar{X} = 7.55 \text{ cmH}_2\text{O}$) lung volumes, the mean pressure values at mid ($\bar{X} = 7.55 \text{ cmH}_2\text{O}$) and low ($\bar{X} = 7.33 \text{ cmH}_2\text{O}$) lung volumes significantly differed from each other. Results for both adjusted and non-adjusted pressure data suggest that pressure values towards the lower end of VC were slightly lower than those produced at high and mid lung volumes.
<table>
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<th>Task</th>
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<th>Press (adj) *</th>
<th>AC</th>
<th>AC (adj) *</th>
<th>MNFL</th>
</tr>
</thead>
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<tr>
<td>ae</td>
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<td>-</td>
<td>-</td>
<td>213.02</td>
<td>220.87</td>
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<td></td>
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<td>104-440</td>
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<td>8.03</td>
<td>230.9</td>
<td>232.9</td>
<td>159.04</td>
</tr>
<tr>
<td></td>
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<td>76-301</td>
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<tr>
<td>ae</td>
<td>Mean</td>
<td>-</td>
<td>-</td>
<td>191.71</td>
<td>188.28</td>
<td>141.23</td>
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<td>-</td>
<td>-</td>
<td>76.93</td>
<td>76.31</td>
<td>74.9</td>
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<tr>
<td>Mid LV</td>
<td>Range</td>
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<td>-</td>
<td>93-396</td>
<td>93.9-389</td>
<td>60-409</td>
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<td>7.55</td>
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</tr>
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<td>SD</td>
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<td>2.25</td>
<td>67.52</td>
<td>66.34</td>
<td>66.73</td>
</tr>
<tr>
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<tr>
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<td>-</td>
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<tr>
<td></td>
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<td>-</td>
<td>-</td>
<td>73.19</td>
<td>66.17</td>
<td>34.96</td>
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<tr>
<td>Low LV</td>
<td>Range</td>
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<td>-</td>
<td>78-364</td>
<td>89-352</td>
<td>21-177</td>
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<td>Mean</td>
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<td>7.33</td>
<td>183.88</td>
<td>181.8</td>
<td>103.21</td>
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<td>SD</td>
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<td>56.19</td>
<td>53.18</td>
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<td>4-12.7</td>
<td>111-341</td>
<td>120-336</td>
<td>39-253</td>
</tr>
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</table>

**TABLE 4.5.** Means, standard deviations, and ranges of aerodynamic measures for the three lung volume conditions by task. N = 14. Pressure measured in cmH₂O. Flow measured in ml/s.

* These are adjusted for SPL.
<table>
<thead>
<tr>
<th>Measure</th>
<th>Term</th>
<th>df</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
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<td>9.64</td>
<td>&lt; 0.001*</td>
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<tr>
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<td>Subjects</td>
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<td>Lung Vol x Subjects</td>
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<td>0.007*</td>
</tr>
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<td>58.08</td>
<td>&lt; 0.001*</td>
</tr>
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<td>Subjects</td>
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<td>133.51</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Lung Vol x Subjects</td>
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<td>14.54</td>
<td>&lt; 0.001*</td>
</tr>
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<td>Lung Vol</td>
<td>2</td>
<td>150.70</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Task</td>
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<td>14.62</td>
<td>0.001*</td>
</tr>
<tr>
<td></td>
<td>Lung Vol x Task</td>
<td>2</td>
<td>1.04</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Subjects</td>
<td>13</td>
<td>33.90</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
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<td>8.82</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Subjects x Task</td>
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<td>1.63</td>
<td>0.135</td>
</tr>
<tr>
<td></td>
<td>Subjects x Lungvolume x Task</td>
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<td>1.79</td>
<td>0.034</td>
</tr>
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<td>Ac (adj)*</td>
<td>Lung Vol</td>
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<td>331.38</td>
<td>&lt; 0.001*</td>
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<tr>
<td></td>
<td>Task</td>
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<td>20.33</td>
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</tr>
<tr>
<td></td>
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<td>0.68</td>
<td>0.50</td>
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<tr>
<td></td>
<td>Subjects</td>
<td>13</td>
<td>78.68</td>
<td>&lt; 0.001*</td>
</tr>
<tr>
<td></td>
<td>Subjects x Lungvol</td>
<td>26</td>
<td>13.95</td>
<td>&lt; 0.001*</td>
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Note. * p < .01

**TABLE 4.6.** Analysis of variance summary table for aerodynamic variables for main effects of Lung Volume, Task, and Subjects, and interaction effects for Lung Volume x Task, Subjects x Lung Volume, Subjects x Task, and Subjects x Lung Volume x Task. Pressure measured in cmH2O. Flow measured in ml/s.

* (adj) = Values adjusted for SPL variability.
Aerodynamic Measures

**Pressure**

![Pressure Graph]

**AC Flow**

![AC Flow Graph]

**Minimum Flow**

![Minimum Flow Graph]

**MNFL**

![MNFL Graph]
Figure 4.8. Mean values and standard errors for the aerodynamic measures of pressure, AC flow, and minimum flow across subjects for the three lung volume levels. Lung volume 1 = high; lung volume 2 = mid; lung volume 3 = low. Graphs to the left correspond to repetitions of /paε/ syllables, and graphs to the right correspond to sustained /ae/ vowels. Pressure and AC flow data were adjusted for SPL changes.

AC Flow

Table 4.5 displays group based means, standard deviations and ranges for AC flow values. Mean values ranged between 160.81 and 230.9 ml/s across lung volume level condition. Table 4.5 also shows the means, standard deviations and ranges for the “SPL adjusted” AC flow data. Mean values ranged between 164.41 and 232.9 ml/s across lung volume levels. Results revealed that the average total decrease in “adjusted AC flow” from high (measured at 80% VC) to low lung volumes (measured at 20% VC) was 51.1 ml/s for syllable strings, and 56.45 ml/s for sustained vowels.

Analysis of individual data revealed that a gradual decrease in Ac flow as lung volumes decreased was the most often occurring pattern. Data points obtained throughout the VC, at 5% VC increments were investigated with a best fit function, in which the predictor variable in the curve-fitting regression was lung volume. This allowed for the examination of patterns of change in the flow measures as lung volume progressed from high to low. The curve fitting analysis was based on individual trials.

Results revealed that out of a total of 84 trials, 72 trials (86%) were better characterized by a linear pattern of change. From the remaining data, six trials (7%), all of which corresponded to the same subject (Subject 1) showed that a quadratic function provided the best fit, while the remaining 7% displayed a random pattern of change which could not be characterized with a mathematical function. The correlation
coefficients for the linear trend for AC flow ranged between .7 and 1.0, with an average of $r = .919$ (sd. = .087). Mean Ac flow slopes were equal to .938 ml/s/%VC (sd. = .429), with an average intercept of 141.39 ml/s. Figure 4.9 exemplifies the most common occurring AC flow pattern of change.

![Graphs showing AC flow and minimum flow values](image)

**FIGURE 4.9** Graphs show AC flow (x) and minimum flow (o) values in milliliters per second, as a function of percentage of vital capacity. The data corresponds to subject S6, and it exemplifies the most often occurring flow patterns, a gradual decrease in AC flow and minimum flow as vital capacity decreases. Left graph represents production of a syllable train. Right graph represents production of a sustained vowel.

Analysis of variance results for both adjusted and unadjusted data are shown in Table 4.6. The repeated measures analysis of variance for the non-adjusted AC flow values showed that lung volume main effects were statistically significant ($F = 150.70$, $p < .001$). Results of polynomial contrasts between lung volume levels showed
that the average AC flow values at high ($\bar{X} = 221.96$ ml/s), mid ($\bar{X} = 204.11$ ml/s), and low ($\bar{X} = 172.34$ ml/s) lung volume levels were all significantly different from one another. Across both tasks, the highest AC flow values were obtained for the high lung volume condition, intermediate values were obtained for the mid lung volume condition, and the lowest values were obtained for the low lung volume condition. The main effect of task was also significant ($F = 20.33, p < 0.001$), suggesting that the repetition of the /pae/ syllable strings ($\bar{X} = 206.04$ ml/s) was produced with significantly higher AC flow values than the sustained /ae/ vowels ($\bar{X} = 191.19$ ml/s). There was no significant interaction between lung volume and task ($F = 1.04, p = .35$).

Results for the “SPL adjusted” AC data indicated that both main effects for lung volume and task were statistically significant ($F = 331.38, p < .001$). Contrasts between lung volume levels showed that the average AC flow values at high ($\bar{X} = 226.88$ ml/s), mid ($\bar{X} = 195.85$), and low ($\bar{X} = 173.11$ ml/s) lung volume levels were all significantly different from one another. Across both tasks, the highest AC flow values were obtained for the high lung volume condition, intermediate values were obtained for the mid lung volume condition, and the lowest values were obtained for the low lung volume condition. The main effect of task was also significant ($F = 20.33, p < 0.001$), suggesting that the repetition of the /pae/ syllable strings ($\bar{X} = 206.04$ ml/s) was produced with significantly higher AC flow values than the sustained /ae/ vowels ($\bar{X} = 191.19$ ml/s). There was no significant interaction between lung volume and task ($F = .68, p = .5$).

Given that the SPL adjustment did not influence the results of the analysis of variance, with both adjusted and unadjusted data yielding similar significant ANOVA main effects, only the adjusted data will be discussed from here on.
Minimum Flow (MNFL)

Minimum flow values averaged across subjects ranged between 93.14 and 162.48 ml/s across lung volume level conditions. Means, standard deviations and range values for minimum flow are shown in Table 4.5.

Analysis of individual trials revealed that the most often occurring pattern was a gradual decrease in minimum flow from high to low lung volumes. Curve fitting regression results based on individual trials revealed that out of a total of 84 trials, 52 (62%) displayed a linear pattern of change. The correlation coefficients for the linear trend ranged from \( r = 0.70 \) to \( r = 0.986 \), with a mean correlation coefficient of \( r = 0.871 \) (sd. = 0.08). The mean slope was found to be 1.358 ml/s/%VC (sd. = 1.206), with an average intercept of 86.61 ml/s (sd. = 104.07). It is evident from the slope and intercept standard deviation values that there was large MNFL variability across subjects. Figure 4.9 shows examples where MNFL decreased in a linear fashion with decreasing lung volumes.

Although this was the most occurring pattern, analysis of individual data showed that in some instances, subjects exhibited a more complex pattern of minimum flow change. Six subjects showed a variation in the way minimum flow changed with changes in lung volume (26 trials, 31%). Figure 4.10 is an example of this variation in minimum flow change, showing data for subject S5, in which minimum flow first increased, peaking around the mid lung volumes, and then decreased as VC got lower. Three subjects (S4, S5, and S11) showed this pattern of minimum flow change consistently. The other three subjects exhibited primarily the average group trend, with this different minimum flow pattern of change in one or two tokens each. A quadratic function provided the best fit for these 26 trials, while the remaining 6 trials (7%) displayed a random pattern of change (p < .01).
FIGURE 4.10. Graphs show AC flow (x) and minimum flow (o) values in milliliters per second, as a function of percentage of vital capacity. The data corresponds to subject S5, and it exemplifies a variation of minimum flow pattern, with minimum flow first increasing and then decreasing as voice is sustained throughout the vital capacity. Left graph represents production of a syllable train. Right graph represents production of a sustained vowel.

Visual inspection of the videostroboscopic data corresponding to these trials revealed that in some cases the closure patterns of the posterior glottis followed the minimum flow pattern, with a gradual opening followed by gradual closing of the posterior glottis with decreasing lung volumes. Figure 4.11 shows a composite of 9 laryngeal images obtained during a sustained vowel task produced by subject S13. Note the complete glottal closure observed at 80% and 70% VC. At 55% we can observe more separation between the arytenoid edges (less arytenoid squeezing), and a posterior glottis slightly open. Between 50% and 40% VC there is a clear posterior glottal gap, which
closes towards the 30% VC, with a resulting closed glottis towards the end of the vital capacity.
Figure 4.11 Composite of 9 laryngeal images obtained during a sustained vowel task. Note the posterior glottis is initially completely closed. Subsequently a posterior gap develops, as can be seen between 55% and 40% of the vital capacity. The posterior glottis is closed once again between 30% and the end of the vital capacity.
Analysis of group data revealed that the average total minimum flow decrease from high (measured at 80% VC) to low lung volumes (measured at 20% VC) was 55.83 ml/s for repeated syllables, and 82.85 ml/s for sustained vowels. A repeated measures analysis of variance for minimum flow showed that lung volume was statistically significant (F = 182.2, p = < .001), as shown in Table 4.6. Polynomial contrasts between lung volume levels showed that all three means were statistically significantly different from each other. Across both tasks, the highest average minimum flow values were obtained for the high lung volume condition (X = 162.48 ml/s), intermediate values were obtained for the mid lung volume condition (X = 139.81), and the lowest values were obtained for the low lung volume condition (X = 93.14 ml/s). See Figure 4.8. In minimum flow there was clearly a large lung volume effect in both tasks, but a significant interaction between lung volume and task obscured the task effect. This means that the task effect depended on the level of lung volume. The mean minimum flows for sustained vowels were higher than the mean minimum flows for repeated syllables at high and mid lung volume levels, but this relation was reversed at low lung volumes, where subjects showed higher minimum flows during syllable repetition than during sustained vowels. For syllable strings there was an average decrease of 20.66 ml/s between the high and mid lung volumes, and of 35.17 ml/s between the mid and low lung volumes, with a total drop of 55.83 ml/s. Average decreases in flow for sustained vowel were measured at 26.11 ml/s for the high-mid contrast, 46.67 ml/s for the mid-low contrast, with a total minimum flow decrease of 72.80 ml/s across lung volume conditions. On average, there was a progressive minimum flow reduction with decreasing lung volumes. The minimum flow decrease was larger between the mid to low lung volumes, than between the high to mid lung volumes (Figure 4.8).
Videostroboscopic evaluation

Spearman correlation coefficients (for non-parametric data) were used for calculation of intra and interjudge reliability of the visual-perceptual data.

Glottal Gap ratings:

Intrajudge reliability scores ranged between $r = .735$ and $r = .962$, with a mean correlation coefficient of $.872$ ($sd = 0.057$). Interjudge reliability measured by correlations between all judges ranged between $r = .64$ and $r = .855$, with a mean $= .766$ ($sd = 0.011$).

Results for comparisons of the posterior glottal gap size for the high vs. mid lung volume conditions are summarized in Figure 4.12. In 71.93% of the comparisons, the high lung volume condition was judged to have a larger gap than for mid lung volume condition. In 12.28% of the comparisons, the mid lung volume condition was judged to have larger glottal gaps than the high lung volume condition, and 15.79% of the comparisons were rated as having the same posterior gap size in both conditions.

When comparing gap size between the mid and low lung volume level conditions, 85.29% of the comparisons indicated larger gap sizes for the mid lung volumes, 5.88% of the ratings indicated larger gap sizes for the low lung volumes, and 8.82% of the comparisons were rated as having the same gap size between both lung volume conditions (Figure 4.13). For both lung volume conditions, all tokens rated as “same” showed no glottal gap (complete closure). Results also indicated that while 32.91% of the tokens corresponding to high lung volumes showed complete glottal closure, the percentage of ratings of complete glottal closure got increasingly higher for the other two
lung volume conditions; 55.69% for the mid lung volume levels, and 79.75% for the low lung volume level condition (see Figure 4.14).

These results indicate that the majority of the subjects (67.09%) exhibited a posterior glottal gap at high lung volumes. Furthermore, there was a reduction of gap size with decreasing lung volumes. The posterior gap tended to either decrease in size or close completely from high to mid lung volume levels, as indicated by smaller gap sizes in the mid lung volume condition as compared to high, and by a lower percentage of tokens (44.3%) showing incomplete closure in the mid volume condition. The same relation was found between the mid and low lung volume levels, where the gap size was either further reduced (85.29%) or the glottis was closed (79.75%) for most of the tokens at the low lung volume level (Figure 4.14).

**FIGURE 4.12.** Comparison of degree of glottal closure between the high and mid lung volume level contrast.
FIGURE 4.13. Comparison of degree of glottal closure between the mid and low lung volume level contrast.

FIGURE 4.14 Percentage of tokens that were rated as having complete or incomplete glottal closure for each of the three lung volume conditions.
Laryngeal hyperfunction ratings:

Most inter-rater reliability correlation coefficients related to laryngeal hyperfunction ratings were slightly above chance level. The data were therefore re-examined to evaluate whether or not there was better/acceptable inter-rater agreement in a smaller subset of the data. It was decided to use only tokens in which there was at least 70% agreement in judges ratings. All 10 ratings for each token were used (5 judges X 2 sessions), and samples that had less than 7 equal ratings were discarded. 45 samples (28.5%) were eliminated with this procedure, leaving 71.5% of the tokens that met the reliability criteria.

Of the subset of tokens meeting the reliability criteria, results from comparisons between high and mid lung volumes showed that 38.18% of the tokens were judged to have more laryngeal compression at the high lung volumes, 45.45% as having more compression at the mid lung volumes, and the remaining 16.36% were rated as having the same degree of compression for both lung volumes (Figure 4.15). Results from comparisons of laryngeal compression for the mid versus low lung volumes revealed that while only 18.96% of the samples were judged to have more laryngeal compression at the mid lung volumes, a much higher percentage (68.96%) was judged to have more laryngeal compression at the low lung volumes. The remaining 12.07% of the samples were judged to have the same degree of laryngeal compression for both lung volume conditions (Figure 4.16).
FIGURE 4.15. Comparison of degree of laryngeal compression between the high and mid lung volume contrasts.

FIGURE 4.16. Comparison of degree of laryngeal compression between the mid and low lung volume contrast.
Inter-subject variability

Z scores were calculated to evaluate the variation of measures for individual subjects relative to the distribution of measures for the entire group. Table 4.7 presents the mean Z scores of all dependent variables for each subject for each lung volume level. Data shows that in general, Z scores were smaller than +/- 2, meaning that individual speakers did not exceed 2 standard deviations relative to the normative group mean values. The exceptions were: 1) subject 7 who showed pressures and minimum flows at high and mid lung volumes that exceeded +2 standard deviations; 2) subjects 8 and 15 who exhibited SPL values that fell –2 standard deviations at all lung volume levels; 3) subject 12 who exhibited F0 at all lung volume levels that fell below –2 standard deviations; and 4) subject 9 who showed vertical laryngeal displacements at high and mid lung volumes that exceeded +2 standard deviations.
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<td>-0.775</td>
<td>0.161</td>
<td>0.217</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>-0.137</td>
<td>-2.529</td>
<td>-0.107</td>
<td>-0.524</td>
<td>0.078</td>
<td>0.101</td>
</tr>
</tbody>
</table>
TABLE 4.7. Mean Z scores for each subject at three lung volume levels for all dependent variables. Lung volume 1 = high, 2 = mid, 3 = low. Values that exceed 2 standard deviations relative to the normative group mean values appear in boldface. * These are adjusted for SPL.

Summary of results for group data

1) SPL

When compared to mid lung volumes, subjects produced significantly higher SPL during high lung volumes, and significantly lower SPL during low lung volumes. Syllable repetitions were produced at significantly higher SPL than sustained vowels. These results, even though statistically significant, represent a small absolute magnitude of change.

2) VLP

When compared to mid lung volumes, subjects showed a significantly lower vertical laryngeal position at high lung volumes, and a significantly higher vertical laryngeal position at low lung volumes. The largest difference was found to occur between the high and mid volume levels. There were no significant task-related differences in VLP changes across lung volume.

3) Subglottal Pressure

Subjects did not display significant changes in subglottal pressure between the high and mid lung volume levels, but did show a slight drop in pressure at low lung volume that was statistically significant.

4) AC Flow

When compared to mid lung volumes, subjects produced significantly higher AC flows at high lung volumes, and significantly lower AC flow at low lung volumes. AC
flow levels were significantly higher during syllable repetition than during phonation of sustained vowels.

5) Minimum Flow

When compared to mid lung volumes, subjects produced significantly higher MNFL levels at high lung volumes, and significantly lower MNFL levels at low lung volumes when compared with mid lung volumes for both speech tasks. There was a significant task effect that showed that there was a larger MNFL drop for sustained vowels than for syllable trains, when phonation was sustained between the mid and low lung volume levels.

6) Glottal Gap

When compared to phonation at mid lung volumes, subjects showed a significantly larger posterior glottal gap during high lung volumes, and a significantly smaller gap and/or a higher incidence of complete glottal closure at low lung volumes. Overall, stroboscopic observations have indicated a reduction of gap size, and/or complete glottal closure with decreasing lung volumes.

7) Degree of laryngeal compression

When compared to phonation at high and mid lung volumes, subjects showed an increased degree of laryngeal compression at low lung volume levels.

Inspection of individual subject data showed that some subjects displayed patterns of laryngeal behavior that differed from the average performance of the group.
CHAPTER V
DISCUSSION

Primary Findings

This study attempted to explore possible relationships between changes in lung volume levels and laryngeal function during phonation. The main question asked was whether or not laryngeal function is affected when phonation is produced outside of the mid range of lung volumes. More specifically, the purpose of the study was to determine whether vertical laryngeal position, glottal adduction, glottal closure, and laryngeal compression are altered as phonation is sustained throughout the full range of lung volumes.

The results of this work indicate that there are systematic alterations in laryngeal activity associated with changes in lung volume when phonation is sustained throughout the vital capacity. Specifically, subjects showed a lower vertical laryngeal position, increased amplitude of vibration of the vocal folds, and increased minimum flows with larger posterior glottal gaps at high lung volumes as compared to mid lung volumes. Conversely, during phonation at low lung volumes, subjects exhibited higher vertical laryngeal position, decreased amplitude of vocal fold vibration, decreased minimum flows with increased glottal closure, and increased degree of laryngeal compression as compared to mid lung volumes. The largest laryngeal excursions in the vertical plane occurred between the high and mid lung volume levels, while the larger changes in glottal adduction, glottal closure, and laryngeal compression occurred between the mid and low lung volume levels. These results suggest that during phonation at high lung volume levels, the larynx is positioned lower in the neck, displaying an overall "more dilated" configuration, with larger amplitudes of vocal fold excursion, and posterior glottal gaps during the closed phase of vocal fold vibration. Conversely, during phonation at low lung volume levels, the larynx seems to adopt a more constricted configuration,
with an elevated position, smaller amplitudes of vocal fold vibration, more complete
glottal closure during the closed phase of vibration, and increased compression of
laryngeal structures.

Large intersubject variation was found in the present study for several variables
within each lung volume level condition. This finding is in agreement with previous
reports of large intersubject variation in aerodynamic measures such as AC flows and
subglottal pressures (Holmberg et al., 1994; Holmberg et al., 1988; Shutte, 1981).

The following discussion will first examine each of the variables individually in
terms of how present findings compare with what has been reported in the literature,
followed by a discussion of possible underlying mechanisms that mediate respiratory and
laryngeal functions. This investigation generally confirms findings from previous studies
related to changes in vertical laryngeal position, subglottal pressures and transglottal
flow. In addition, the present study adds new information regarding amplitude of
vibration of the vocal folds, degree of glottal adduction, degree of glottal closure, and
laryngeal compression, as the larynx adjusts to changes in lung volume when phonation
is maintained throughout the vital capacity. This novel information allows for a more
comprehensive appreciation of the relationship between the respiratory and laryngeal
systems, and further understanding of normal laryngeal phonatory physiology when
phonation is produced outside of the normal range of lung volumes.

Duration of maximum sustained vowels

The mean durations for maximum sustained phonations of vowels that were
obtained in this study (18.62 seconds) are in general agreement with previously reported
data. In a summary from previous research, Hirano reported that maximum times ranged
from 17.4 to 25.7 seconds in adult females (Hirano, Koike, & Leden, 1968). Colton and
Casper have reported that the average maximum duration times for females is approximately 15 seconds (Colton & Casper, 1990). Kent and colleagues reported that the duration of maximum phonation is affected by factors such as age, sex, stature, and particularly vital capacity, and laryngeal efficiency in terms of expenditure of transglottal flow (Kent, Kent, & Rosenbek, 1987). The authors also point out the importance of practice and proper instruction given to the subjects on this measure, concluding that three trials may be sufficient when proper instruction and modeling is provided. In the present study, subjects were given careful instruction prior to and during the experiment, and practiced the task repeatedly prior to data collection.

Speech task effects

Data from the present study showed that sound pressure level, AC flow, and minimum flow were speech task dependent. When comparing the results across the two types of speech tasks, it was found that the maximum durations of the syllable repetition tasks were shorter (shorter on average by 2.17 seconds), slightly louder, and with slightly higher AC flows and minimum flows, than sustained vowels. The difference in duration between tasks may be related to the fact that some airflow is spent during the aspirated part of the stop plosive /p/. Even though the absolute magnitude of change for dependent variables was different for the two speech tasks, the direction of the changes across lung volumes was the same. These results suggest that the general nature of laryngeal phonatory adjustments to changes in lung volume are not significantly influenced by the addition of articulatory gestures.

Previous studies have found differences in vocal function parameters between syllable repetition and sustained vowel phonation (Higgins & Saxman, 1993; Holmberg et al., 1995). However, results from studies that have examined airflow data across speech tasks are inconsistent. Higgings and Saxman reported that both young and elderly
speakers with normal voices showed higher glottal airflows during a sustained vowel task, as opposed to a syllable repetition task (Higgins & Saxman, 1993). Higher average airflows have also been demonstrated for the same vowel when it is produced in a consonant-vowel-consonant context as opposed to when it is produced in isolation (Isshiki & Ringel, 1964). On the contrary, Sapienza and Stathopoulos (1995) found no significant AC flow differences when comparing results from vowel and syllable tasks (Sapienza & Stathopoulos, 1995). Results from the present study indicated higher AC flows for syllable repetition, with the values for syllables being on average 21.92 ml/s greater. Regardless of the statistical significance, the actual numerical differences are quite small. These differences are not likely to be significant from a perceptual or clinical standpoint.

With respect to changes in fundamental frequency, the present results agree with reports from Higgins and Saxman (1993), who found no F0 differences between speech tasks in a group of young female subjects. However, this is not in agreement with Sapienza and Stathopoulos (1995), who reported significantly lower F0 for a syllable repetition task as compared to a sustained vowel task for two different subject groups (normals and vocal nodules).

Regarding SPL variation, Sapienza and Stathopoulos (1995) found no significant differences in SPL between sustained vowels and syllable repetition tasks, although they did find that a reading passage was produced on average 2.5 dB louder than sustained vowels (Sapienza & Stathopoulos, 1995). In the present study, comparisons were made between SPL at mid vowel portion in the syllable repetition task, and the SPL of a sustained vowel, measured at similar lung volume locations. A significant SPL difference across speech tasks was found, with SPL being on average 2.5 dB higher for syllable repetition than for sustained vowels. It would appear that the vowels produced in the syllable repetition task got a boost in SPL, probably due to the stop consonant context.
In summary, results of this study indicate that even though the absolute magnitudes of some variables are different, the direction of change of the observed laryngeal adjustments to changes in lung volume is the same for both speech tasks. These findings suggest that when phonation is sustained throughout the full range of lung volumes, the phonetic context in which phonation is produced does not seem to influence the basic underlying pattern of laryngeal adjustments to lung volume changes.

**Sound Pressure Level**

Even though results from statistical analyses showed that there were significant average SPL changes across lung volume conditions for group data, the magnitude of these changes was small (less than .3 dB for syllable repetitions and less than 1.5 dB for sustained vowels). However, as noted earlier, individual subjects showed larger SPL variations. These changes are discussed on an individual basis.

**Subglottal Air Pressure**

Subglottal air pressure represents the aerodynamic driving force of the vocal folds for phonation (Fant, 1982). Data for normal speakers at comfortable pitch and loudness levels has been reported to be between 5 and 10 cmH₂O (Higgins & Saxman, 1991; Holmberg et al., 1995; Holmberg et al., 1988; Netsell, Lotz, DuChane, & Barlow, 1991). Data from the present study are in general agreement with these previously reported values, with group based averages of 8.04, 7.87, and 7.31 cmH₂O for lung volume conditions from high to low respectively. All but one subject had mean estimates of subglottal pressure that were within the normal range reported in the literature for normal women. The remaining subject (S6) had higher than normal estimated subglottal air...
pressures in all lung volume conditions. She exhibited mean subglottal pressure values of 16.39, 13.71, and 12.6 cmH2O for lung volumes high, mid, and low respectively, with $Z$ scores (standard deviations away from the group mean) of 3.07, 2.09, and 1.68 for the same lung volume conditions. These subglottal pressure values did not result in higher than normal sound pressure level values ($Z$ scores of 0.4 for all lung volume conditions). It would appear that the higher pressures were not producing higher SPL output, therefore some other laryngeal adjustments may have occurred. These values may have reflected a larger than normal incomplete glottal closure (large posterior gap). This was confirmed by aerodynamic findings of high minimum flow magnitudes. This subject showed the highest minimum flow values in the group at high and mid lung volume levels (means of 319, 338, and 117 ml/s, and $Z$ scores of 2.90, 3.18, and 0.06 for high, mid, and low lung volume conditions respectively). Accordingly, videostroboscopic findings showed a large posterior glottal gap that extended into the musculo-membranous glottis at high and mid lung volume levels, and a completely closed glottis at low lung volumes. Regardless of these findings, her voice quality was perceived as “normal” by the experimenters.

Group based statistical evaluation of the subglottal air pressure data indicated that subjects were able to maintain constant subglottal pressure levels during phonation throughout the high to mid lung volume range, but that there was a slight but significant decrease towards the low lung volumes (an average drop of .56 cmH2O). These results are in agreement with previous reports (Griffin et al., 1995; Iwarsson et al., 1995; Sonninen et al., 1994), which suggested that lower lung volumes were associated with lower subglottal pressures. However these findings are not in accordance with Bouhuys and colleagues who reported rather constant subglottal pressure values throughout the full range of the vital capacity during the same phonatory tasks (Bouhuys et al., 1966). One possible explanation for this discrepancy is differences in voice training. In Bouhuys’ study most subjects were experienced singers with extensive voice training. Subjects from the other studies, had either no voice training, or, as in the case of Sonninen and Griffin’s studies, subjects were singers, but were instructed to sustain vocalizations.
without “support.” The present study supports the hypothesis suggested by Iwarsson and colleagues, in which untrained or unsupported voices may not counter the effect of the elastic recoil forces efficiently, resulting in lower subglottal pressures when sustaining voice at low lung volume levels (Iwarsson et al., 1995).

**Vertical Laryngeal Position (VLP)**

Results for the present study showed significant changes in VLP between lung volume levels. VLP values indicated that subjects showed a lower laryngeal position in the neck while phonating at high lung volumes, and that the laryngeal position gradually rose as lung volumes got lower, in both sustained phonation and syllable repetition tasks. These data are in agreement with previous reports of a lower laryngeal height at high lung volumes, as compared to low lung volumes (Sundberg et al., 1989; Zenker, 1964), and with reports from Iwarsson and colleagues (Iwarsson & Sundberg, 1997; Iwarsson et al., 1995) who showed a similar direction of laryngeal displacement with lung deflation during phonation of repeated syllables.

Group data showed that on average, the largest laryngeal elevation occurred when phonation was sustained from high to mid lung volumes. Group data also showed that with phonation changing from mid to low lung volumes, there was laryngeal elevation as well, but of a much lesser degree than with phonation between the high and mid lung volumes. Inspection of individual data showed that some subjects exhibited a different pattern of laryngeal vertical displacement than the group. These subjects exhibited an initial increase in laryngeal height during phonation between the high and mid lung volumes, but followed by a subsequent decrease in laryngeal height when phonation progressed between the mid and low lung volumes. This type of “upwards-downwards” movement was observed in 14% of the total tokens analyzed.
AC Flow Effects

AC flow values are considered to reflect amplitude of vocal fold vibration. There is a broad range of AC flow values reported in the literature for adult females during normal loudness phonation ranges. Holmberg and colleagues have reported AC flow ranging from 90 to 250 ml/s, with a mean of 140 ml/s (Holmberg et al., 1988; Holmberg, Hillman, & Perkell, 1989), where Sutler and colleagues have reported mean values of 260 ml/s for untrained females at normal loudness levels (Sutler & Wit, 1996). Even though in the present study, AC flow dropped across lung volumes, mean values for the three lung volume level conditions fall within the range of normal values reported by other researchers.

AC flow is generally considered to reflect amplitude of vibration of the vocal folds, and decreased AC flow values are suggestive of increased vocal fold stiffness, with more tightly approximated vocal folds (Hillman et al., 1990; Sapienza & Stathopoulos, 1995; Sapienza & Stathopoulos, 1994). As the vocal folds become more tightly approximated, the vocal fold vibration amplitude decreases, exhibiting a longer closed phase, and resulting in small amplitude of the airflow passing through the glottis (Gauffin & Sundberg, 1989). Increased vocal fold tightness may also have an effect on vocal quality, producing “pressed” voices. Peterson and colleagues have reported decreased AC flow values for “pressed” voices when compared with “breathy voices” (Peterson et al., 1994). Results from the present study seem to be in agreement with these report in the sense that findings of lower AC values, decreased minimum flows with increased glottal closure, and increased laryngeal compression, are all features consistent with pressed phonation.
Minimum Flow Effects

Minimum flow measures are generally considered to reflect airflow shunted mainly through the posterior glottis, during normal voice production (Rothenberg, 1973, Klatt and Klatt, 1990, Hirano, Yoshida, et al., 1987, Holmberg et al, 1988). Even though small changes in MNFL could be related to vertical movements of the vocal folds during phonation, these changes are on the order of 20 to 30 ml/s (Hertegard and Gauffin, 1995). Higher MNFL values are likely to come from air leakage through the glottis during the closed phase of the glottal cycle.

Even though minimum flow decreased across lung volume conditions, mean minimum flow values obtained in the present study at all lung volume levels were in agreement with values reported elsewhere (Holmberg et al., 1988; Holmberg et al., 1989; Sapienza & Dutka, 1996; Sapienza & Stathopoulos, 1995; Stathopoulos & Sapienza, 1993a). The presence of a minimum flow offset component during normal phonation is a common occurrence, and has been reported in the literature (Holmberg et al., 1988; Rothenberg, 1977; Stathopoulos & Sapienza, 1993a; Sundberg et al., 1989). All of our subjects showed a minimum flow component throughout most of their vocalizations. However, present results revealed that on average, minimum flow decreased with decreasing lung volumes. Moreover, it was shown that on average, the largest decrease in minimum flow occurred when phonation was sustained from mid to low lung volumes.

The present data suggest that minimum flow changes were caused by variations in the degree of separation between the focal folds at the level of the posterior glottis. Present results from videostroboscopic examination showed that the size of the posterior glottal gap decreased with decreasing lung volumes. There seemed to exist a good correlation between minimum flow measures and visual observations of vocal fold closure, in the sense that the highest minimum flow values and the largest posterior glottal gaps were measured at high lung volumes, the lower minimum flows and the
smaller gaps were measured at low lung volumes, with intermediate values of both parameters at mid lung volume levels. Moreover, analysis of individual data showed that in some trials in which the minimum flow initially increased, and then decreased across decreasing lung volumes, a corresponding pattern of glottal closure was often observed from the videostroboscopic data.

However, there were several trials in which variation in minimum flow values were not correlated with observations of changes in degree of glottal closure. For example, there were some instances in which, even though visual ratings showed complete glottal closure between lung volume conditions, minimum flow data showed large variations between the same lung volume conditions. In other words, while in some cases, minimum flow changes were corroborated via stroboscopic examination, this did not hold for all tokens. One explanation for this discrepancy, is possibly the fact that videoendoscopic examination of the larynx provides only a superior view of the vocal folds. The extent to which it is possible to observe the posterior glottis with this technique is always dependent on several factors such as positioning of the tip of the scope relative to the posterior glottis, individual anatomical variations of the glottal introitus, and individual variations in “posturing” of the laryngeal structures. For example, anterior hooding of the arytenoid-corniculate complex will obscure a superior view of the posterior glottis. In such cases, incomplete closure at the posterior glottal level may be hidden from view, and on visual inspection mistakenly judged to be completely closed. Therefore, it appears that measurement of minimum flow is more reliable than visual inspection of the larynx when an accurate evaluation of glottal closure is needed.

Visual Ratings

It has been shown that incomplete closure of the posterior glottis during phonation is a common finding in normal subjects, particularly in females. A high incidence of
posterior glottal gaps has been reported in females with normal voices (Biever & Bless, 1989; Bless, Biever, & Shaik, 1986; Linville, 1992; Peppard et al., 1988; Sodersten & Lindestad, 1990). Present findings showed that 12 (86%) out of 14 subjects exhibited a posterior glottal gap (at either high or mid lung volumes). Only two subjects (14%) consistently showed complete glottal closure throughout performance of the speech tasks.

There is evidence in the literature that glottal closure may be related to intensity level. Higher intensities have been reported to be associated with increased glottal closure (Sodersten, Hertegard, & Hammarberg, 1994; Sodersten & Lindestad, 1992). However, there are no pre-existing data on the relation between glottal closure and lung volume level. Results from the present study have indicated a reduction of chink size with decreasing lung volumes, suggesting that there is a strong positive correlation between degree of glottal closure and the lung volume level at which phonation is produced.

As pointed out by Koufman, “the normal biomechanical configuration for effortless phonation comprises glottal closure with contact between the free edges of the vocal folds, without involvement of the supraglottic structures” (Koufman et al. 1996). The present study assessed involvement of supraglottic structures, in order to determine presence or absence of laryngeal hyperfunction. Laryngeal hyperfunction refers to excessive or poorly regulated laryngeal and extra-laryngeal muscular tension. Its etiological mechanism is poorly understood. At the supraglottal level, it is characterized by the following: degree of arytenoid adduction or squeezing, antero-posterior compression, representing a shortening of the antero-posterior glottal length, ventricular compression, or shortening of the distance between the ventricular folds, shortening of the aryepiglottic folds, with epiglottal retraction, and overall changes in the shape of laryngeal vestibule. When visualization of the larynx is attempted with video-endoscopy, these signs typically obscure the observable length and width of the vocal folds.

In the present study, an increase in laryngeal hyperfunction was considered as a
more “effortful” voice production. The comparison of degree of hyperfunction between high and mid lung volume levels did not show a clear increase in hyperfunction in either lung volume condition. But the comparison between the mid and low lung volume levels showed a clear difference between these two conditions, with almost 70% of the tokens corresponding to low lung volumes rated as having more laryngeal hyperfunction than the mid lung volumes. These results suggest that there is an increased amount of laryngeal hyperfunction when phonation is produced towards the low lung volume levels, possibly resulting in a more effortful phonation.

Explanatory considerations

Having discussed the findings for the individual parameters examined in this study, it is important to consider the parameters together and to consider possible mechanisms to explain the combined findings. Although the present study was not designed to discriminate underlying mechanisms that mediate respiratory and laryngeal functions, it is nonetheless valuable to consider them for the development for future research.

To reiterate, there was global evidence of laryngeal dilation at high lung volumes, compared to a mid volume “control” situation. In contrast, low lung volumes were characterized by global evidence of laryngeal constriction, as compared to the mid lung volumes. Possible explanations for the high lung volume findings are discussed next, followed by speculations regarding the findings for low lung volumes.

Possible explanations for the high lung volume effects
Vertical laryngeal position, AC flows, minimum flows, glottal closure, and measures of laryngeal constriction indicated a general laryngeal dilation at high lung volumes in comparison to mid lung volumes. Together, the findings are consistent with those reported by other authors related to a diaphragmatic or tracheal pull. This has been discussed in previous sections. Briefly, it has been proposed that the flattening of the diaphragm during maximal inspirations, exerts a downward pull on to the tracheobronchial tree, which in turn distends the larynx caudally (Zenker, 1964; Zenker & Glaninger, 1959). Vocal fold abduction, then, may be an extension of this same gesture (Zenker, 1964; Zenker & Glaninger, 1959).

Mechanism for vertical laryngeal displacement

The possible mechanisms that can affect vertical laryngeal position are:

(a) action of the strap muscles: the infrahyoid muscle group (omohyoid, sternothyroid, thyrohyoid, and sternohyoid muscles), which together are capable of moving the entire laryngotracheal complex downward as much as one whole vertebral body (Tucker, 1987); and the suprahyoid muscle group (digastric, stylohyoid, geniohyoid, mylohyoid, stylopharyngeus, and thyrohyoid muscles) which are all elevators of the larynx. Tucker pointed out that these muscles “rarely function alone...therefore, a relative upward or downward displacement of the laryngotracheal structures is a dynamic activity that is the resultant vector of the arithmetic sums of all muscles active at any given time.” (Tucker, 1987, pp:24)

(b) tongue position: tongue protrusion affects the position of the hyoid bone, pulling it forward and upwards, and consequently, elevating the larynx.

(c) jaw movement: downward jaw movement can lower the tongue and hyoid bone, consequently lowering the larynx.

(d) tracheal pull: as previously noted, due to the elastic interconnections between the diaphragm and the tracheobronchial tree, when the diaphragm contracts and flattens during maximal inhalations, it may pull downwards on the laryngotracheal complex. As
the diaphragm raises with decreasing lung volumes, the downward pull would gradually be released, allowing the larynx to return to a higher position.

Given the type and direction of vertical laryngeal movements observed in the present study, in particular between the high and mid lung volumes, it is more likely for these displacements to be accounted for by either strap muscle action or tracheal pull.

The most typical vertical laryngeal displacement observed in this study was a gradual increase in laryngeal height when phonation progressed from high to mid lung volumes. This type of laryngeal movement is more likely to have been accounted for by a diaphragmatic or tracheal pull, than by any of the other three mechanisms. Tongue position was maintained constant during both speech tasks, since the vowel during task production remained unchanged (this was confirmed perceptually). Therefore, tongue position is not likely to have accounted for the type of VLP movements observed. Jaw movement during articulation of the syllable trains could have occurred, but it is unlikely that it could account for the gradual increase in laryngeal displacement. If articulatory jaw movements during a syllable train production were responsible for vertically displacing the larynx, the type of displacement observed would have been an “up and down” type of pattern produced with the articulation of each syllable. Therefore, jaw movement is also an unlikely candidate. Even though this study was not designed to determine which are the underlying mechanisms that would account for VLP movements, we can hypothesize that a passive mechanical pull makes more sense biomechanically than active extrinsic laryngeal musculature contraction. There seems to be no logical reason why the strap muscles would consistently pull the larynx downwards with large inspirations, and release the pull gradually as lung volumes get smaller. Since high lung volumes are usually achieved with a lower diaphragmatic position, this would result in a lower laryngeal position associated with the high lung volumes. As lung volumes decrease, the larynx would return to a higher position in the neck, in the face of a decreasing downward pull. Therefore, the present data supports the hypothesis that the
mechanism responsible for the observed shifts in laryngeal height between the high and mid lung volume levels, is a tracheal, or diaphragmatic pull. This hypothesis was also supported by previous reports (Hoit et al., 1993; Iwarsson & Sundberg, 1997; Iwarsson et al., 1995; Macklin, 1925; Sundberg et al., 1989; Zenker, 1964).

According to Zenker, the downward pull on the laryngeal structures generates a mechanical force that tends to abduct the vocal folds widening the glottis (Zenker, 1964). This theory was supported by an investigation from Hoit and colleagues (Hoit et al., 1993), in which they studied the relation between lung volume and voice onset times (VOT). They found that voice onset times were longer at high lung volumes than at low lung volumes, suggesting that phonation at high lung volumes was associated with an abductory force onto the vocal folds. Present data showed that at higher lung volumes, both AC flow and minimum flow values were higher than at low lung volumes, suggesting that the vocal folds were more abducted and with a larger degree of incomplete closure. If indeed the lower laryngeal position observed at high lung volumes tends to widen the glottis, then, the present findings of increased AC flow, increased minimum flow and larger posterior glottal gaps would be consistent with a less adducted glottis. In sum, the present data are consistent with a hypothesis regarding causal biomechanical relations between high lung volumes and laryngeal excursion and dilation.

**Possible explanations for the low lung volume effects**

**Behavioral mediation hypothesis**

One possible explanation for the global laryngeal compression seen at low lung volumes is that it represents a behavioral phenomenon. In the present experiment subjects were instructed to maintain constant loudness and pitch levels throughout the vital capacity. Findings revealed that subjects exhibited decreased airflows and subglottal
pressures when maintaining phonation down to the low lung volumes. Several factors could have contributed to these findings: 1) changes in the effectiveness of the respiratory system to maintain constant lung pressures at low lung volumes. As discussed in an earlier section, in order to sustain phonation at low lung volumes, increased respiratory muscle effort is needed. There is evidence that there are differences between trained and untrained voices in vocal function measures (Schutte and Miller, 1983; Peppard, Bless, and Milenkovic, 1988, Shipp, 1987), and in respiratory function measures (Watson and Hixon, 1985, 1996, Hoit et al., 1996). Thus, it is possible that at low lung volumes the respiratory system is at a less efficient biomechanical stage to produce and sustain constant pressures, and speakers with untrained voices may not be able to regulate the laryngeal driving forces as efficiently as at a higher lung volume range. 2) the fact that the intrapulmonary airway passages increase constriction at low lung volumes (Butler et al., 1960), could potentially contribute to a decrease in expiratory airflows.

Therefore, subjects may have implemented a behavioral strategy in where the phonatory changes may have been secondary to articulatory adjustments for subglottal pressure maintenance, in the face of decreasing pulmonary expiratory flows.

**Neural mediation hypotheses**

Alternative or complementary explanations for laryngeal compression at low lung volumes can be based on speculations about neural circuitry that is common to respiratory and laryngeal mechanisms. At least two types of connections are possible. One regards literally shared respiratory and laryngeal circuitry. Animal studies have shown that subcortical structures including the periaqueductal gray matter (PAG) and the nucleus retro-ambigualis (NRA) may play a role in the coordination, activation, and timing of respiratory and laryngeal muscles (Davis et al., 1993; Davis et al., 1996; Holstege, 1989). Thus, it is reasonable to think that hyperfunction in the expiratory muscles, as occurs at low lung volumes, could be accompanied by laryngeal hyperfunction due to a common, high level activation pathway. More evidence related to
a central connection between both systems was provided by Mead and Reid (Mead & Reid, 1988). In a study of respiratory muscle activity during repeated airflow interruptions, the authors found that when the airflow was interrupted at the oral cavity level, there were no synchronous respiratory bursts associated with the airflow interruptions. But in contrast, when the interruptions were produced at the glottic level, significant expiratory muscle and diaphragmatic activity was recorded in synchrony with the glottal stops, suggesting that the airflow interruptions at the glottal level were either neurally programmed or driven reflexively at the laryngeal level. Another study involving human subjects provided further evidence consistent with this hypothesis by showing that activation of intrinsic laryngeal muscles during forced expiration was elicited by respiratory muscle effort (Brancatisano, Collett, & Engel, 1983a). In this study subjects performed active and passive forced expirations (achieved by a rapid passive compression of the chest). Glottic widening was observed during active forced expirations. But when the same expiratory flows were achieved passively, glottic width appeared to decrease. The authors suggested that laryngeal involvement during forced expirations was elicited by expiratory muscle effort alone, independently of chest wall compression and increased airflows (Brancatisano et al., 1983a).

In order to sustain phonation at low lung volumes (particularly below the end expiratory level), the respiratory muscles need to produce increasingly higher muscle forces to counteract the progressive increase in negative recoil forces that accompanies decreasing lung volume (Mead & Martin, 1968). At this point of the expiratory cycle, not only the external intercostals, but the abdominal muscles (Hoit et al., 1983) and possibly the back muscles (Hixon, 1973) are recruited to squeeze the thorax and produce the major active component of lung pressure. Thus, based on the potential shared neural structures and pathways between both systems, and on the evidence that laryngeal muscle activation is associated with respiratory muscle effort, it is possible that the constrictive laryngeal behaviors observed during phonation towards the lower lung volumes in the present
study, are related to the increasing respiratory muscle effort required to maintain phonation at such low lung volumes.

A second type of neural connection between respiratory and laryngeal functions may be reflexive. Animal studies have revealed reflexive relations between pulmonary receptors and laryngeal activation in the sense that both pulmonary inflation and deflation result in activation of laryngeal adductors. Studies have shown that a coordinated set of reflexes mediated by pulmonary stretch receptors influence not only breathing, but laryngeal function as well (Baken & Novak, 1971; Coleridge & Coleridge, 1986; Sellick & Widdicombe, 1970; Stransky et al., 1973). When the afferent nerve endings in the lungs (stretch receptors) are stimulated by forced pulmonary deflation, there is a phasic contraction of laryngeal constrictors (a mechanism known as laryngeal braking), with glottic narrowing, increased laryngeal airway resistance, and increased EMG activity of the laryngeal adductors during the expiratory phase of the breathing cycle. The effect is so robust, that investigators have used lung deflation in their methodology as “the most convenient and effective means of activating the laryngeal constrictors” (Brancatisano et al., 1987 pp., 61). A caution in the interpretation of these data is that the findings were obtained from fully anesthetized or decerebrate animals. It is not clear to what extent similar findings might be seen in awake behaving humans confronted with a speech task (Ludlow & Lou, 1996). Nonetheless, given the robust animal evidence that laryngeal constriction is elicited by pulmonary reflexes associated with lung volume, in particular by rapid lung deflation, it would not be surprising that a similar mechanism could be activated in humans while phonating at the low end of the vital capacity.

Interactive hypothesis

The present data do not rule out any of the hypotheses regarding respiratory laryngeal relations, nor do they explicitly point to any of the individual hypotheses. Specifically designed experiments should be pursued as discussed shortly. In
the meantime, it seems entirely plausible that a combination of both behavioral and neural mechanisms may mediate the changes in laryngeal behavior seen with lung volume changes in the present study. Figure 5.1 presents a model that summarizes the suggested hypotheses. The model is simplified, showing only the factors that were found to be the most predominant, and does not include the variations exhibited by individual subject performances. Also, it reflects findings from both speech tasks used in the experiment (sustained vowels and prolonged syllable repetitions), thus, it may not reflect laryngeal changes that occur during production of conversational speech. Although the behavioral component most certainly played a significant role throughout the performance of the tasks, it is believed that it may have been more predominant towards the lower end of the VC. At lower lung volume levels, subjects may have implemented laryngeal constriction as a behavioral strategy in order to maintain subglottal pressure.
Possible Underlying Mechanisms

Bio-Mechanical Forces
- Elastic interconnections between the diaphragm and the larynx

Reflexive
- Stimulation of laryngeal motoneurons by respiratory muscle afferents (during increased respiratory muscle effort)
- Reflexes activated by pulmonary stretch receptors activate laryngeal motoneurons

Behavioral
- Behavioral strategy in order to prolong phonation maintaining constant pitch and loudness

Physiological Changes
- Pulling down on the larynx
- Abduction of the vocal folds
- Increasing activity of intrinsic laryngeal musculature
- Increasing vocal fold adduction
- Increasing constriction of supraglottal structures
- Activation of laryngeal sphincteric configuration

Effects
- Gradually decreasing
- Gradually increasing

Figure 5.1. Model of possible underlying mechanisms that account for laryngeal adjustments during sustained vowel and prolonged syllable repetition throughout the VC. VLP = vertical laryngeal position, Ps = subglottal air pressure, post. gap = posterior glottal gap.
Individual performances

Individual performances as well as group averages were considered to obtain a representation of vocal function changes associated with changes in lung volume. Analysis of individual data showed that some of the subjects exhibited different patterns of change than the rest of the group, particularly in vertical laryngeal position, glottal closure and minimum flow measures.

As discussed previously, the most often occurring pattern of vertical laryngeal displacement was a lower laryngeal position at high lung volumes, with a gradual rise as lungs deflated during phonation. A different pattern of vertical displacement was observed in four subjects, in which a lower larynx was observed at high lung volumes, with a gradual increase in vertical position as lung volumes decreased to the mid range, followed by a decrease in vertical position as lung volumes continued to decrease to the lower range. An explanation for this “upwards-downwards” variation of VLP displacement is not readily apparent. A possible hypothesis is that there are two mechanisms involved in displacing the larynx vertically. One taking place between the high and mid lung volumes, with laryngeal downward displacement and subsequent rise due to initial diaphragmatic pull and subsequent release of the pull with progression of phonation into the mid lung volumes. The second mechanism, taking place during the progression from the mid towards the low lung volumes, would be an increase in activity of the strap muscles that pull the larynx back down in the face of an overall increase in laryngeal effort.

With regards to minimum flow changes, the most often occurring pattern of change was a gradual decrease in minimum flow as the vital capacity decreased. A different pattern was observed in 6 subjects (consistently in three subjects and in one or two instances in the other three subjects), with an initial increase in minimum flow values
with change from high to mid lung volumes, followed by a decrease in minimum flow with change from the mid to low lung volumes. This minimum flow pattern was validated visually, with observations of a “closed-open-closed” posterior glottis as phonation progressed from high to low lung volumes. A possible explanation to account for this alternative behavior, is that at high lung volumes, the complete glottal closure would prevent too rapid a lung deflation (maybe in an effort to oppose the larger recoil forces from the respiratory system that occur at high lung volumes). When lung volumes decrease to the mid range and the recoil forces from the respiratory system are not so high, the larynx adopts a “more relaxed” phonatory pattern, allowing some airflow through an incompletely closed posterior glottis. Towards the lower lung volumes, and this time presumably in an effort to preserve lung volume and to maintain phonation longer, the posterior gap is closed and some degree of laryngeal compression is exhibited. In this alternative pattern, activation of the intrinsic laryngeal musculature occurs presumably for two different and opposed purposes: at high lung volumes, the strategy would seem to prevent too much airflow to escape through the glottis, and at low lung volumes, the more constricted laryngeal configuration would seem to preserve lung volume.

This alternative pattern may give some insight into a discrepancy of findings reported previously. Shipp and colleagues measured differences in EMG activity between the interarytenoid muscles (laryngeal adductor) and the posterior cricoarytenoid muscles (laryngeal abductors), and reported greater difference at high than at low lung volumes, suggesting greater adduction at high lung volumes (Shipp et al., 1983). Opposite results have been reported by Iwarsson and colleagues (Iwarsson et al., 1995). They have reported smaller adduction forces at high lung volumes as compared to low lung volumes. One possible explanation for this discrepancy, is that Shipp’s subjects may have used the alternative strategy of increased vocal fold adduction at high lung volumes seen in a few of our subjects.
Even though in some cases the minimum flow changes were validated through visual inspection of the videostroboscopic images, this was not possible in all cases. For example, in cases like the one shown in Figure 5.1, while the minimum flow data shows the typical drop from high to low lung volumes, videostroboscopic images show a complete glottis throughout the phonatory task. One explanation would be that there may still have been a posterior opening of the glottis that was not visible from a superior view. Given the anatomical differences between subjects, and the particular positioning of the endoscope, which offers only a superior view, it is possible that visual inspection of the posterior glottis may be obscured by the arytenoid-cuneiform structures. Often times by its natural position, and at other times by anterior hooding of the arytenoids.

Results from this study suggest that the procedure of examining the larynx via endoscopy may not provide accurate information about glottal closure, since it offers only a superior view of the vocal folds. Therefore, minimum flow seems to be a more sensitive measure of degree of glottal closure.

**Lung volume and hyperfunction**

The parameters of vocal function observed during phonation at low lung volumes reflect a larynx with either: (a) a small posterior gap or complete glottal closure, (b) more tightly approximated vocal folds, with (c) less amplitude of vocal fold vibration, and (d) higher than normal degrees of glottal and supraglottal compression. All these factors are consistent with vocal hyperfunction. Hyperfunctional voices are frequently perceived as having a pressed voice quality (Boone & McFarlane, 1994; Stemple, 1984). Even though voice quality was not formally evaluated, it was the subjective impression of the investigator that subject’s voices acquired a more pressed quality towards the lower end of the vital capacity.
It has been shown that there are inefficiencies in speech breathing in individuals with hyperfunctionally related voice disorders, like vocal nodules, (Hillman et al., 1997; Sapienza & Stathopoulos, 1994; Sapienza et al., 1997; Sperry et al., 1994). Specifically, recent studies have shown that some individuals with vocal nodules tend to produce speech at lower lung volumes than subjects with normal voices, often speaking below the end expiratory level (EEL) (Sperry et al., 1994, Hillman, 1997). Hillman and colleagues have stated that a particularly important question to address is whether the aberrant respiratory behaviors exhibited by their nodules subjects developed before or after the onset of the laryngeal manifestation of the voice disorder (Hillman et al., 1997). Results of the present study may provide some insight into this question.

If indeed phonation at low lung volume elicits increased glottal closure, one could hypothesize that speakers with vocal nodules who lose significant airflow due to incomplete glottal closure may speak at lower lung volumes, as a compensatory strategy to increase glottal closure and make phonation more efficient, with less waste of unmodulated air loss. In this hypothetical situation, the use of lower lung volume ranges during speech by nodules subjects would be more likely to have developed after the onset of the vocal nodules.

On the other hand, we could also think of a different and opposite scenario, in which the abnormal respiratory behaviors developed before the onset of the vocal pathology. Results of this study showed that phonation at low lung volumes elicits some degree of hyperfunction-like phonatory behaviors. Given this information, it is possible that producing speech at a lower than normal range of lung volumes could potentially help to trigger hyperfunctional vocal behaviors, thus serving as an etiological factor in the development of vocal pathology (Hillman, 1989, 1990).
Future research

There is need for continued research exploring the inter-relations between the respiratory and laryngeal systems. Based on the results of the present study, the following general directions for future study are suggested: (1) investigation of the effects of lung volume on laryngeal function during phonation in men, (2) comparison of these normative results with those of individuals with hyperfunctional voice disorders, (3) investigation of the underlying mechanisms of laryngeal adjustments associated with changes in lung volume during voice and speech production. Several specific areas of future research are described below:

The overall length of the vocal tract, and the size of the larynx and vocal folds is larger in males than in females. Apart from the size differences, there is evidence that there are gender differences in laryngeal aerodynamics (Holmberg, 1988; Monsen and Engebretson, 1977), differences in glottal configuration (Bless, Biever, and Shaik, 1986; Bless, Hirano, and Feder, 1987), and possibly in the elastic properties and shape of the vocal folds as well (Titze, 1989). Therefore, future research should include investigation of the effects of manipulating lung volume on laryngeal function during voice and speech production in men, in order to compare it with female data, and gain deeper understanding of the present findings.

This study demonstrated a relationship between lung volume and laryngeal function during phonation, and identified various laryngeal adjustments that occur when voice is produced at different lung volume levels. Several hypotheses were proposed to account for the underlying mechanisms responsible for these adjustments. Further research should focus on the investigation of the underlying mechanisms. One of the hypotheses proposed that the increased activation of the laryngeal musculature during phonation at low lung volumes was directly related to increased respiratory muscle effort, that is, laryngeal activation as a reaction to chest wall muscle proprioceptors reflexes. To test whether this is true, a study could be designed in which subjects would be required to
increase expiratory drive during phonation at different lung volumes, particularly at the high lung volume range. Conversely, to test the hypothesis of whether the observed laryngeal effects depend on lung volume independent of chest wall muscle effort, that is, to test whether the larynx reacts to a reflex activation of the pulmonary stretch receptors, passive phonation close to the reserve volume could be investigated. This could be accomplished by applying negative pressures, while subjects phonate attempting to keep subglottal pressures constant.

Findings of this study need to be interpreted with caution, given that the data were obtained on a continuum from high to low lung volumes. It is possible that different ways of manipulating the independent variable (e.g., non-sequential) may provide different information, or may reveal other aspects of the relation between lung volume and phonation. It is also possible that given the particular characteristics of this task, it may have masked or obscured some other effects. Also, the present findings are based on data from only two speaking tasks. These data may not reflect laryngeal adjustments that occur during more complex speech tasks, or during conversational speech. Therefore, future research is necessary to replicate these results under different experimental condition, particularly when the respiratory system is manipulated in a different manner (e.g., with onset of phonation at different lung volume levels), and with speech tasks motorically more complex than the ones used in this investigation.

Conclusion

The present findings provide information about the relationship between laryngeal function and lung volume level during phonation. Data from the present study generally confirm results from other studies of vocal function and lung volume. Based on the present results, it appears that high lung volumes are associated with a lower position of the larynx in the neck. Also, during phonation at high lung volumes, there is increased
amplitude of vocal fold vibration, increased unmodulated transglottal airflows, and larger posterior glottal gaps when compared with phonation at the mid range of lung volumes. Conversely, during phonation at low lung volumes, the larynx appears to be positioned higher in the neck. Also at low lung volumes, the amplitude of vocal fold vibration is decreased, with decreased modulated and unmodulated transglottal airflows, and increased degree of glottal closure. In addition, phonation at low lung volumes is associated with increased laryngeal compression (or hyperfunction) when compared with phonation at mid lung volume levels. Similar lung volume effects on laryngeal function were observed during sustained vowel and syllable repetition tasks. Possible underlying mechanisms to account for the observed effects include behavioral, biomechanical, and neurological factors. Further research is needed to more directly test the viability of these mechanisms in accounting for the observed relationships between lung volume and laryngeal function, and to explore the possible clinical implications that can be attained from the results of this study.
APPENDIX 1:

Copy of letter of approval from the Human Studies Committee
HUMAN STUDIES COMMITTEE

REPORT OF ACTION

PRINCIPAL INVESTIGATOR: Robert E. Hillman, Ph.D.

PROTOCOL TITLE: Objective Assessment of Vocal Hyperfunction

PROTOCOL NUMBER: #92-08-030

This is to certify that the application identified above has been reviewed by the Human Studies Committee appointed to review proposals for clinical research and other investigations involving human subjects. **ANY MODIFICATIONS TO YOUR PROTOCOL OR ANY UNANTICIPATED OR ADVERSE EFFECTS MUST BE PROMPTLY REPORTED TO THE HUMAN STUDIES COMMITTEE.**

DATE OF REVIEW: November 18, 1998

COMMITTEE ACTION: APPROVAL

COMMENTS:

Carolyn Carlson  
Compliance Officer  
Human Studies Committee
APPENDIX 2.

Subject's consent form:
MASSACHUSETTS EYE AND EAR INFIRMARY

INFORMED CONSENT

TITLE: Objective Assessment of Vocal Hyperfunction

INVESTIGATOR: DATE:

DESCRIPTION AND EXPLANATION OF PROCEDURES:

You, , are being asked to participate in a research study which uses a variety of techniques to study various aspects of the human voice. For the purposes of this study, all of these techniques are considered to be experimental. The overall goal of this project is to provide new information concerning the etiology (causation) and treatment of disorders which affect the voice.

This project is being conducted in the Voice and Speech Laboratory at the Massachusetts Eye and Ear Infirmary. In the lab, you will have measuring devices attached to the area around your mouth, nose, neck and chest. With these devices in place, you will be asked to pronounce a number of utterances in a variety of ways (e.g., loud voice, soft voice, high pitch voice, low pitch voice) and read from some prepared text while recordings are made of the outputs from the measuring devices and of your voice.

The following measurements will be made. Air pressure inside your mouth will be measured with a tube that will be placed between your lips and held in position in the middle of your mouth. The outside end of the tube will be attached to a mask that fits over your mouth and nose. The mask is made of rubber, and it has a number of holes drilled in it which are covered by a fine wire mesh. It contains a flow-measuring device. Movements of your vocal cords will be measured using electrodes that are held against your neck by an adjustable band. Other electrodes will be taped to the surface of your neck to monitor the activity of your throat muscles. Measurement of your breathing patterns will be made using two elastic bands that strap around your rib cage and abdomen. In addition, a small metal disk (5.6 mm by 8 mm) may be attached to the front of your neck just above your collar bone using an adhesive that is specially designed for attaching devices to the skin (Skin Bond). This device will monitor the vibrations of your neck as you phonate. All of the above measuring devices are connected to recording equipment. None of these devices interfere with normal function or cause any discomfort.

You will also be given a brief hearing test (screening) through headphones and asked to fill out several forms which gather information about your health, voice use and level of emotional stress.
In addition, your larynx will be examined and photographed. This will involve the placement of a rigid laryngoscope (a tube approximately 5 mm. in diameter) through your mouth and into the back of your throat and/or the passage of a flexible laryngoscope (a tube approximately 3.4 mm. in diameter) up through your nose and down the back of your throat. A camera will be attached to the end of these scopes outside of your body to take pictures of your larynx.

You will receive $35 each time you participate in an assessment session involving the measurements described above. You will probably be asked to participate in only one assessment session, but you could be asked if you can return for additional assessment sessions if it is deemed necessary. Each assessment session will involve from one to two hours of your time, or less.

You may also be asked to undergo the placement of additional devices between your vocal cords and/or into your esophagus (food pipe). This will entail the application of additional topical anesthetic via spraying and/or swabbing of your mouth, throat and nose. A small pressure sensing device on a curved rod will then be passed through your mouth and throat, and positioned between your vocal cords in your larynx. This procedure will be completed in 15 minutes or less. In addition, a small, collapsed balloon will be passed through your nose and into your throat, at which point you will swallow it into your food tube by drinking some water. This additional procedure will be completed in 45 minutes or less. You will be paid an additional $20 to $40 for participating in one or both of these this extra testing procedures.

RISKS AND DISCOMFORTS:

You may experience slight discomfort (irritation) in your nasal passages associated with the passage of the flexible scope. To reduce this discomfort, a light topical anesthetic (solution of 3% lidocaine and 0.25% phenylephrine) will be sprayed into your nose prior to examination with the flexible scope. The anesthetic will normally wear off in approximately 15 to 20 minutes. There is also the possibility that the flexible scope could cause a nosebleed. If this occurs, testing will be suspended and appropriate medical attention will be provided. Placement in your throat of either scope may cause gagging. If this becomes a problem, your throat will be sprayed with the topical anesthetic (10% lidocaine). There is the possibility that you could experience an allergic reaction to the topical anesthetic. If this occurs, testing will be suspended and appropriate medical attention will be provided.

Participation in the additional testing that entails placement of a pressure sensing device between your vocal cords may increase the likelihood of gagging, coughing and/or choking. Also, the introduction of the balloon through your nose could cause increased coughing of choking, or a nosebleed. If any of this occurs, testing will be suspended and appropriate medical attention will be provided as needed.

POTENTIAL BENEFITS:
However, it is expected that the results from this project will assist in better diagnosis and treatment of voice disorders.

There will be no costs to you for any of the services that you receive as part of participating in this study.

CONFIDENTIALITY:

All of the data gathered in this investigation will be held in strict confidence. Any reports or publications will not identify individual participants by name or initials.

RIGHT TO WITHDRAW:

You are not obligated to participate in this study. If you choose not to participate your present or future medical care will not be affected in any way. Also, if you participate, you may withdraw your consent and discontinue participation at any time without affecting your medical care.

COMPENSATION:

In the unlikely event that you should be injured as a direct result of this study, you will be provided with medical treatment. This treatment does not imply any negligence on the part of the Massachusetts Eye and Ear Infirmary or any of the physicians involved. When applicable, the Massachusetts Eye and Ear Infirmary reserves the right to bill third party payers for any emergency services rendered. The Massachusetts Eye and Ear Infirmary does not have any program to provide compensation as a result of any injuries.

RIGHT TO ASK QUESTIONS:

If you have questions regarding this research or your participation in it, either now or at any time in the future, please feel free to ask them. The research team, particularly Dr. Hillman, who can be reached at (617) 573-4050, will be happy to answer any questions you may have. You may obtain further information about your rights as a research subject by calling Carl Finn, Director of Research Administration at the Massachusetts Eye and Ear Infirmary at (617) 573-4080. If any problems arise as a result of your participation in this study, including research-related injuries, please call the principal investigator, Dr. Hillman at (617) 573-4050 immediately.

CONSENT:

I have read the above description of this research study, and I understand it. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I understand that I will receive a copy of this form.
I understand that I am free to withdraw this consent and discontinue participation in the described research study.

I voluntarily consent to my participation in the described research study.

Date  Name of Subject  Signature of Subject

Date  Name of Witness  Signature of Witness

Date  Name of Investigator  Signature of Investigator

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