PHYSIOLOGICAL EFFECTS OF EXPIRATORY MUSCLE STRENGTH TRAINING WITH THE SEDENTARY HEALTHY ELDERLY: PULMONARY, COUGH, SWALLOW, AND SPEECH FUNCTIONS

By

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This document is dedicated to my daughters, Yoonji and Yoonha, and my husband, Heesun Yang.

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With age, physical functions decline which can influence respiratory performance. One of the physical changes is sarcopenia. With sarcopenia, elderly individuals experience reduced muscle mass and strength in the respiratory musculature. Age-related loss of muscle strength in expiratory muscles with reductions in elastic recoil of the lungs and chest wall compliance may compromise the necessary lung pressure for both ventilatory and non-ventilatory activities. This study examined the effects of a 4-week expiratory muscle strength training (EMST) program in healthy but sedentary elderly adults as measured by maximum expiratory pressure (MEP) as well as magnitudes of pulmonary, cough, swallow, and speech functions.

Eighteen healthy sedentary elderly people participated in this study. Sedentary was defined as a person with 24-hours maximum exertion time below 50 in a physical activity scale described within a physical activity questionnaire. Pulmonary measures included maximum expiratory pressure (MEP), maximum inspiratory pressure (MIP), forced

expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), the ratio of FEV_1 to FVC (FEV_1/FVC), and expiratory reserve volume (ERV). Cough measures during capsaicin induced cough included inspiratory phase duration, compression phase duration (CPD), peak expiratory flow rate (PEFR), and post-peak plateau duration, and post-peak plateau integral amplitude (PPPIA). Swallow measures included peak amplitude (PA), duration, and integral amplitude (IA) of submental muscle group activity in surface electromyography (SM-sEMG) during maximal voluntary dry (saliva) swallow, wet swallow (5 cc and 10 cc water), and thin paste swallow (5 cc and 10 cc pudding). Speech measures included aerodynamic measures and acoustic measures including excess lung pressure (P_{EL}) as well as maximum phonation durations (MPDs) at comfortable and loud intensity levels.

Results indicated significant improvements in MEP and MIP, decrease in CPD, increases in PEFR and PPPIA during reflexive coughs produced by capsaicin challenge, PA and IA of SM-sEMG during maximal voluntary dry and 10 cc pudding swallows as well as increase in P_{EL} and MPD at comfortable intensity level.

The utility of EMST as a method of strength training for rehabilitation of respiratory muscle weakness/sarcopenia in sedentary elderly seems to be a viable consideration as a treatment tool, given the positive outcomes of this treatment on multiple physiological functions.

CHAPTER 1
INTRODUCTION AND REVIEW OF THE LITERATURE

With aging, physiological capacities can become greatly limited resulting in increased incidence of disease and disability (Oskvig, 1999). The United States has a population of 280 million, and among them, approximately 12% (33.6 millions) are 65 years and older (U.S. Census Bureau, 2002, 2003). Additionally, our population is growing fast, with the fastest growing group being those over 85 years of age (Oskvig, 1999). Within the next 10 years, the number of people aged 85 and older is estimated to increase by more than 6 million (U.S. Census Bureau, 2003).

"Aging is the irreversible normal changes in a living organism that occur as time passes" (DiGiovanna, 1994, p. 2). Several theories have been postulated to explain the causes and mechanisms of aging from biological, psychological, and cultural perspectives. But no one particular theory can explain the aging process perfectly. While aging is not a disease process but a normal developmental change, almost all age changes reduce a person's ability to maintain healthy survival and a high quality of life (Bowling & Dieppe, 2005; DiGiovanna, 1994; Sarkisian, Hays, & Mangione, 2002; Seeman et al., 1994). Additionally, the aging process is correlated with a very high incidence of diseases. Increased detrimental changes of the body and exposure to harmful factors with aging cause a decline in physical, psychological, and social functions which can increase the susceptibility of diseases.

Particularly, the respiratory system demonstrates significant changes in anatomy and physiology as a function of age. Respiration is a function that is critical for sustaining life but also significantly important for generating the pressure needed to cough, swallow, and speak (Brooks & Faulkner, 1995; Campbell, 2001; Mizuno, 1991). For the clinician, knowledge of age-related changes in the respiratory system is important information since these changes can increase the chance of respiratory disease and aggravate acute or chronic respiratory failure and may influence diagnostic criteria and therapeutic choices (Enright, Kronmal, Higgins, Schenker, & Haponik, 1993; Krumpe, Knudson, Parsons, & Reiser, 1985; Pack & Millman, 1988).

Respiratory System Changes with Age

The main functional changes in the respiratory system with aging are associated with an increase in the lung compliance (i.e., a decrease of the lung elastic recoil: Campbell, 2001; Chan & Welsh, 1998; Knudson, 1991; Mahler, 1983; Niewoehner, Kleinerman, & Liotta, 1975; Pride, 1974; Turner, Mead, & Wohl, 1968) and a decrease in chest wall compliance (i.e., an increase of the chest wall stiffness: Janssens, Pache, & Nicod, 1999; Kahane, 1981; Mahler, 1983; Turner et al., 1968). Decreased lung elasticity is related to the loss of elastic fibers attached within the lungs, dilatation of the alveolar ducts, the fusion of adjacent alveoli, and other changes (Campbell, 2001; Knudson, 1991; Oskvig, 1999). This reduced elastic recoil of the lungs results in increasing residual volume (RV) and decreasing vital capacity (VC). In other words, as the lungs are more distensible with age, more air is trapped into the lungs causing more stale air to remain and less fresh air brought into the lungs with each breath. Increased chest wall stiffness is due to the calcification of intercostal cartilages and other structures within the rib cage and its articulation as well as gradual atrophy and weakened intercostal muscles (Janssens et al., 1999; Turner et al., 1968). If an elderly person has kyphosis (curvature of the spine) or osteoporosis (loss of bony tissue), he/she will have even more significant reduction in chest wall compliance (Turner et al., 1968). This decreased chest wall compliance modifies the curvature of the diaphragm implicating negatively on its mechanical force capabilities; thus the functional residual capacity (FRC) and RV are increased (Janssens et al., 1999). It is known that RV increases by approximately 50% and VC decreases to about 75% between 20 and 70 years of age (Janssens et al., 1999). It is also reported that forced expiratory volume in 1 second (FEV_1) decreases by 14 to 30 mL a year and by 15 to 24 mL a year in nonsmoking men and women after the age of 20, respectively, and after age 65 the rate of declination is even greater (Knudson, Lebowitz, Holberg, & Burrows, 1983; Tockman, 1994). In summary, these changes contribute significantly to an age-related progressive decline of forced vital capacity (FVC), FEV_1, forced expiratory flow (FEF), and expiratory reserve volume (ERV) and an increase in FRC due to rise in RV (Burr, Phillips, & Hurst, 1985; Gibson, Pride, O'Cain, & Quagliato, 1976; Knudson et al., 1983; Schmidt, Dickman, Gardner, & Brough, 1973; Waterer et al., 2001).

Respiratory muscle function also significantly decreases with age (Berry, Vitalo, Larson, Patel, & Kim, 1996; Brooks & Faulkner, 1995; Chan & Welsh, 1998; Chen & Kuo, 1989; Enright, Kronmal, Manolio, Schenker, & Hyatt, 1994; Janssens et al., 1999). Because of the difficulty in accurately quantifying the age-related changes to respiratory muscles, specific changes in either morphological or functional properties of respiratory muscles with aging have not been reported extensively (Brooks & Faulkner, 1995). Most of the deficits of the respiratory muscle, composed mainly of skeletal muscle much like the upper and lower limbs, are estimated by measures of the deficits that occur in the limbs (Powers & Howley, 2001; Tolep & Kelsen, 1993). The most common change in skeletal muscles with aging is muscle fiber atrophy, especially with a disproportionate atrophy of the fast-twitch fibers (i.e., type II fibers). Type II fibers are responsible for fast and powerful movements.

Respiratory Muscle Atrophy and Strength in the Elderly

Skeletal muscle atrophy (i.e., a reduction in skeletal muscle mass) causes a reduction in muscle strength and power, which is referred to as sarcopenia (Doherty, Vandervoort, & Brown, 1993; Greenlund & Nair, 2003; Roubenoff, 2000). Sarcopenia, first coined by Rosenberg (1989), is highly prevalent in the elderly population. Generally, the prevalence of sarcopenia in general skeletal muscles ranges from 6 to 30% in persons over the age of 60 years (Baumgartner et al., 1998; Melton et al., 2000; Tanko, Movsesyan, Mouritzen, Christiansen, & Svendsen, 2002), and varies depending on measurement, definition, and participant selection as well as the gender of the individual. Furthermore, some studies have postulated that sarcopenia increases more than 50% after 80 years of age (Baumgartner et al., 1998; Iannuzzi-Sucich, Prestwood, & Kenny, 2002). Generally, prevalence rates are much higher in men than in women since testosterone produced by the testes and adrenal glands are greatly reduced in elderly men (Iannuzzi-Sucich et al., 2002; Melton et al., 2000). Testosterone contributes to the build up of skeletal muscle mass influencing strength and function of skeletal muscles. Even though the possible causes of sarcopenia are not clearly known, major contributing factors are evident, including decreased physical activity, altered neuromuscular function (e.g., less motor units innervating muscle), and inadequate nutrition, as well as changes in molecular status (e.g., mitochondrial volume and activity) and anabolic hormonal status (e.g., testosterone, dehydroepiandrosterone, growth hormone, insulin growth factor-I) with age (Morley, Baumgartner, Roubenoff, Mayer, & Nair, 2001).

Characterized by decreases in muscle mass (cross-sectional area) and a decrease in the number and size of muscle fibers (Melton et al., 2000), sarcopenia results in a skeletal muscle cross-sectional area decrease by 20% to 40% between the ages of 20 and 60 years (Doherty, Vandervoort, Taylor, & Brown, 1993; Lexell, Taylor, & Sjostrom, 1988; Overend, Cunningham, Paterson, & Lefcoe, 1992; Young, Stokes, & Crowe, 1985). By age 80, muscle mass is dramatically reduced by up to one-half of the total muscle mass (Lexell et al., 1988). Most muscle atrophy and reductions in the number and size of muscle fibers with age are explained by either age-related physical inability or neuromuscular changes that include a decreased number of motor units, changes in neuromuscular junctions, and loss of peripheral motor neurons (Booth & Weeden, 1993).

Skeletal muscles, in general, consist of several different muscle fiber types of which the characteristics are determined by the properties of the motor units innervating them. The type of fibers in skeletal muscles is mostly composed of type I and type II. Type I, slow oxidative (slow-twitch) fibers are innervated by slow fatigue resistant motor units, and type II (fast-twitch) fibers are subcategorized into type IIa (fast oxidative-glycolytic) fibers innervated by fast fatigue resistant motor units and type IIb (fast glycolytic) fibers innervated by fast fatigable motor units (Doherty, 2003).

Age-related atrophy is predominantly shown in type II fibers (Booth & Weeden, 1993; Brown & Hasser, 1996; Lexell et al., 1988; Morley et al., 2001; Proctor, Balagopal, & Nair, 1998; Tolep & Kelsen, 1993). Type I fibers are also decreased in number and size; however, the extent of their reduction is much less than that of type II fibers, particularly type IIa (Proctor et al., 1998). Previous studies demonstrate that the mean area of type II fibers in individuals age 70 years decreases from 20 to 50% (Doherty, Vandervoort, Taylor et al., 1993; Lexell et al., 1988) and the percentage of type II fibers relative to total muscle fibers also decreases by 40% in the elderly aged 60 years and above (Larsson, 1983; Lexell, Downham, Larsson, Bruhn, & Morsing, 1995). The decrease in the proportion of type II fibers can be explained by either a direct loss in the total number of type II fibers due to decreases in muscle protein synthesis or the conversion from type II to type I fibers due to selective denervation (Booth & Weeden, 1993; Doherty, Vandervoort, Taylor et al., 1993; Tolep & Kelsen, 1993). With aging, progressive loss of motor neurons in the spinal cord results in denervation of fast-twitch fibers along with reinnervation of these fibers by axonal sprouting from adjacent slow-twitch motor neurons (Brooks & Faulkner, 1995). Age-related skeletal muscle atrophy results in the loss of muscle size and strength (Powers & Howley, 2001).

Muscle strength is defined as the maximum force generation capacity and is divided into isometric (static) and dynamic (including isokinetic) muscle strength (Macaluso & De Vito, 2004). Isometric strength is the maximum force when there is no change in muscle length, while dynamic strength is the maximum force generated from actions and accounts for the maximum power which is the product of force and speed of muscle contraction when movement exists (Macaluso & De Vito, 2004). Several studies have shown that muscle strength of both the isometric and dynamic types declines with aging. Isometric muscle strength decreases by 20% to 40% in elderly individuals after age 60 (Larsson, Grimby, & Karlsson, 1979; Murray, Gardner, Mollinger, & Sepic, 1980; Young, Stokes, & Crowe, 1984; Young et al., 1985) and maximally up to 76% (Hakkinen & Hakkinen, 1991; Overend, Cunningham, Kramer, Lefcoe, & Paterson, 1992). In addition, losses in dynamic muscle strength have been reported with an almost 50 to 60%

loss of isokinetic strength in limb muscles between the ages of 30 and 80 (Frontera, Hughes, Lutz, & Evans, 1991; Murray et al., 1980). Changes in proportion of fiber types may also explain a reduction of tension and velocity of contraction and relaxation compared with those of young muscles (Narici, Bordini, & Cerretelli, 1991; Roos, Rice, Connelly, & Vandervoort, 1999) which can reduce power of the skeletal muscles. Sarcopenia of the respiratory muscles also occurs decreasing their potential strength. Chen and Kuo (1989) indicated that respiratory muscle strength and endurance decreases by approximately 20% by the age of 70.

The respiratory muscles include the inspiratory and expiratory muscle groups. The diaphragm, internal intercostals of the parasternal region, external intercostals, and other accessory muscles mainly constitute the inspiratory muscles. The lateral internal intercostals, external obliques, internal obliques, transverse abdominis, rectus abdominis, serratus posterior inferior, and quadratus lumborum constitute the expiratory muscles (Mizuno, 1991). These muscles not only act as the major pump for ventilation, but also play a role in non-ventilatory activities such as coughing, sneezing, valsalva maneuver, talking, singing, vomiting, swallowing, and other functions that are accompanied by expiratory effort.

A decrease in respiratory muscle strength with aging can deteriorate ventilatory as well as non-ventilatory functions (Burzynski, 1987; Mizuno, 1991). During expiration at rest, the passive elastic recoil of the lungs is typically used to generate expiratory force/pressure. However, the expiratory muscles must contract to produce the necessary lung pressure during non-ventilatory activities (Burzynski, 1987) and contract below FRC (Zeleznik, 2003). Mizuno (1991) reported that the mean fiber cross-sectional area of expiratory internal intercostal muscles decreases by approximately 7% to 20% at about 50 years of age because of a reduction of both type I and type II fibers, predominantly type II fibers. However, these changes are not observed in the diaphragm (Mizuno, 1991). Other studies observing changes in the respiratory muscles demonstrated no or less change in muscle mass and no change in muscle fiber types in diaphragmatic muscle and inspiratory external intercostal muscles with aging (Caskey, Zerhouni, Fishman, & Rahmouni, 1989; Krumpe et al., 1985; Polkey et al., 1997; Tolep, Higgins, Muza, Criner, & Kelsen, 1995), suggesting the expiratory muscles are more affected by the aging process than the inspiratory muscles.

Declining lung and chest wall functions, whether due to aging or disease, would require more muscular effort in both expiratory and inspiratory phases. In an early study of lung and chest wall compliance, Turner et al. (1968) examined changes in lung elasticity as a function of age. Their findings concur with more recent reviews of lung elastic recoil and chest wall compliance by Janssens, Pache, and Nicod (1999) that showed decreased chest wall compliance and decreased static elastic recoil of the lungs with aging. With decreases in chest wall compliance and lung elasticity, respiratory muscles will be required to work more to move the chest wall during breathing and doing other non-ventilatory tasks. Chen and Kuo (1989) also reported that 70% of the total elastic work of breathing is required at age 70 years compared with 40% of the requirement for a 20-year-old. Thus, strengthening respiratory muscles should help minimize the physical changes associated with the loss of lungs and chest wall compliance as a function of age, since respiratory muscle contraction is necessary for moving the chest wall and the lungs.

Muscle Strength and Sedentary Lifestyle in the Elderly

It is well known that muscle atrophy results from muscle disuse, which can be caused by immobilization or by the reduced loading of a muscle that are closely related to a sedentary lifestyle (Powers & Howley, 2001; Rolland et al., 2004). Particularly, a reduction in the strength and power of skeletal muscles as a function of age is closely related with a decreasing physical activity with sedentary lifestyle (Mizuno, 1991; Rolland et al., 2004; Taylor et al., 2004). Considerably, the prevalence of elderly individuals with a sedentary lifestyle is increasing (DiPietro, 2001). It is estimated that around 10% of elderly individuals participate in regular exercise, but that more than 50% of the population over 65 years of age has a sedentary lifestyle (Pollock, Lowenthal, Graves, & Carroll, 1992; Taylor et al., 2004). Inactivity, due to the lack of physical exercise, accelerates the changes in musculoskeletal structures and, thus, speeds-up the aging process (Campbell, Sheets, & Strong, 1999). Many of the changes in the musculoskeletal system result more from disuse than from simple aging. Further, decreases in muscular strength and power in the respiratory musculature accompanied with sedentary lifestyle in the elderly may accelerate reductions in the ventilatory and non-ventilatory functional capacities.

Measurement of Respiratory Muscle Strength

While directly measuring the number and size of muscle fibers might be useful to assess respiratory muscle strength related to muscle mass, doing so would require invasive procedures to directly measure the morphology of the respiratory muscles in vivo. Direct measurement of the force output of the human respiratory muscles is also impractical (Tolep & Kelsen, 1993). Therefore, the morphological changes that occur in respiratory skeletal muscles with aging have been studied in rodents or other animals

(Kelly et al., 1991; Maltin, Duncan, & Wilson, 1985; Powers et al., 1994; Powers, Lawler, Criswell, Lieu, & Dodd, 1992; Tolep & Kelsen, 1993; Zhang & Kelsen, 1990). Available data on the morphological aspects of human respiratory muscles with aging come largely from the results of Mizuno's postmortem study (Mizuno, 1991).

Another, less invasive way to measure the strength of the overall respiratory muscles is by testing the function of respiratory muscles using indexes, such as maximum inspiratory pressures (MIPs) and maximum expiratory pressures (MEPs) (Berry et al., 1996; Black & Hyatt, 1969; Bruschi et al., 1992; Chen & Kuo, 1989; Enright et al., 1994; Karvonen, Saarelainen, & Nieminen, 1994; McConnell & Copestake, 1999; McElvaney et al., 1989; Ringqvist, 1966). These measures provide an indirect way of examining maximum strength of the respiratory muscles. Researchers use MIPs to measure inspiratory muscle strength at the level of either FRC or RV and MEPs to measure expiratory muscle strength at the level of total lung capacity.

Ringqvist's study (1966) which investigates the ventilatory capacity and respiratory forces in healthy individuals aged 18 to 83 years, many others have investigated the relationship between age and MIPs or MEPs. Black and Hyatt (1969) measured respiratory muscle strength in participants from 20 to 86 years of age. They observed that respiratory muscle strength declines at a rate between 0.25 to 0.79 cm H_2O a year for MIP and between 1.14 to 2.33 cm H_2O a year for MEP in both men and women, respectively. Enright et al. (1994) also found similar age-related decrements in both MIP and MEP with a rate of decline in MIP at about 1 cm H_2O a year and that for MEP about 2 to 3 cm H_2O a year for those between 65 to 85 years of age. Results from other studies indicate no statistically significant negative relationship between age and MIP and MEP due to other variances, such as the number of participants or body surface area; however, some degree of decreased MEPs were found, especially in men over the age of 55 years (Bruschi et al., 1992; McElvaney et al., 1989). Based on these findings it appears that respiratory muscle strength reduces with aging. Furthermore, these data suggest that strength of the expiratory muscles is more reduced than the strength of the inspiratory muscles with aging.

Table 1-1 summarizes studies supporting the decline of MEP levels in both men and women as a function of age. All studies demonstrated higher MEPs in men than in women since MEPs are related to height (Ringqvist, 1966) and men are typically of greater height than women. Obvious differences were found in the MEPs across the studies in Table 1-1. Some factors that may be related to the differences obtained in MEPs across the studies follow. First, the participants in the Chen and Kuo study (1989) were Asian and physically of smaller stature than those in the Ringqvist (1966) and the Black and Hyatt (1969) studies, which included Caucasian participants. Second, the study of Chen and Kuo was done 20 years after the Ringqvist and the Black and Hyatt studies. Differences in the types of pressure transducers, their sensitivity, and other measurement protocol issues could certainly contribute to the database variations.

Since MIPs and MEPs are used to reflect an individual's respiratory muscle strength, these indices can be used to study the relationship between strength and ventilatory and non-ventilatory functions. Recent studies support that MEPs are the most appropriate indices for quantifying respiratory muscle strength in the elderly (Berry et al., 1996; Chen & Kuo, 1989; Enright et al., 1994; Karvonen et al., 1994; McConnell & Copestake, 1999).

Table 1-1. Normal maximum expiratory pressure (MEP) values with age.

Age range	Ringqvist[1]		Black & Hyatt[2]		Chen & Kuo[3]		Enright et al.[4]		Berry et al.[5]	
	Men (*n*)	Women (*n*)	Men (*n*)	Women (*n*) [*]	Men (*n*)	Women (*n*)	Men (*n*)	Women (*n*)	Men (*n*)	Women (*n*)
18-29	247 ± 41 (37)	170 ± 29 (33)			141.2 ± 8.8 (20)	97.9 ± 5.4 (20)				
30-39	248 ± 38 (12)	163 ± 29 (8)			136.6 ± 8.9 (20) [†]	92.8 ± 4.2 (20) [†]				
40-49	253 ± 52 (15)	178 ± 33 (12)								
50-59	252 ± 32 (13)	157 ± 28 (12)	218 ± 74 (5)	145 ± 40 (8)	133.6 ± 8.9 (20) [‡]	88.4 ± 6.2 (20) [‡]				
60-64	209 ± 49 (16) [§]	157 ± 27 (17) [§]	209 ± 74 (3)	140 ± 40 (4)	117.4 ± 7.4 (20) [¶]	75.1 ± 5.1 (20) [¶]				
65-69			197 ± 74 (7)	135 ± 40 (6)			188 (113)	125 (176)	190 ± 55 (44) [**]	125 ± 36 (57) [**]
70-74	200 ± 42 (13)	165 ± 29 (10)	185 ± 74 (10)	128 ± 40 (10)			179 (105)	121 (119)		
75-79							161 (59)	102 (85)		
80-84							142 (43)	84 (34)		
85+							131 (9)	94 (13)		

Note: MEP value or mean MEP ± standard deviation (in cm H_2O) included.
[1]Ringqvist, T. (1966). The ventilatory capacity in healthy subjects. An analysis of causal factors with special reference to the respiratory forces. Scand J Clin Lab Invest Suppl, 88, 67.
[2]Black, L.F. & Hyatt, R.E. (1969). Maximal respiratory pressures: Normal values and relationship to age and sex. Am Rev Respir Dis. 99(5), 698–99.
[3]Chen, H.I. & Kuo, C.S. (1989). Relationship between respiratory muscle function and age, sex, and other factors. J Appl Physiol, 66(2), 945.
[4]Enright, P.L., Kronmal, R.A., Manolio, T.A., Schenker, M.B., & Hyatt, R.E. (1994). Respiratory muscle strength in the elderly. Correlates and reference values. Cardiovascular Health Study Research Group. Am J Respir Crit Care Med, 149(2 Pt 1), 432.
[5]Berry, J.K., Vitalo, C.A., Larson, J.L., Patel, M., & Kim, M.J. (1996). Respiratory muscle strength in older adults. Nurs Res., 45(3), 155. Linear regression for men, MEPs = 360 − 2.47 × age; for women MEPs = 242 −1.75 × age.
[*]Number of participants in each group in this column was not defined in the original paper, so it was estimated number of participants from the figure shown in published manuscript.
[†]Range of age = 31–45.
[‡]Range of age = 46–60.
[§]Range of age = 60–69.
[¶]Range of age = 61–75.
[**]Range of age = 65 and older.

Ability to generate maximal expiratory force plays a critical role for non-ventilatory tasks, such as cough, swallow, and speech (Enright et al., 1994; Karvonen et al., 1994), which are important functions with which elderly patients demonstrate problems, particularly those post-stroke or with other neuromuscular diseases such as Parkinson's disease, spinal cord injury, or multiple sclerosis (Chiara, 2003; Kang et al., 2005; Saleem, 2005).

Respiratory Muscle Strength Training in the Elderly

Given the age-related declines in respiratory muscle strength, a mechanism for training the muscles might be beneficial and actually aid and/or prevent a certain degree of muscle wasting (Powers, Coombes, & Demirel, 1997). Progressive resistance training of skeletal muscles has resulted in significant improvements in limb muscle strength in the young, elderly, and even in the frail elderly (Bemben & Murphy, 2001; Charette et al., 1991; Hakkinen, Kallinen et al., 1998; Lexell, Robertsson, & Stenstrom, 1992; Pyka, Lindenberger, Charette, & Marcus, 1994). Elderly individuals enrolled in strength training programs demonstrate increased muscle strength and endurance of lower extremity muscles, similar to what is observed for young people (Fiatarone et al., 1990; Fiatarone et al., 1994).

Strength training is associated with a combination of both central (neural) and peripheral (muscle mass) adaptations. After a person completes a few days to a few weeks of strength training, a rapid improvement of muscle strength is noticed without hypertrophy. This rapid improvement relates to neural adaptations, including increases in the number of motor neurons and recruitment of motor units to agonist muscles, an increased discharge rate of motor units to agonist muscles, decreases in antagonist coactivation, or fiber type transitions from type IIb to type IIa fibers, which are associated with the acquisitions in muscle strength observed in the early stage of training (Carolan & Cafarelli, 1992; Hakkinen, Newton et al., 1998; Patten, Kamen, & Rowland, 2001;

Powers & Howley, 2001; Staron et al., 1990). The result of neural adaptations has been consistently demonstrated across studies in elderly and young participants. However, the exact mechanisms of central adaptations are not clearly understood. Preliminary studies provide indirect evidence of neural adaptations from measuring maximal voluntary neural activation recorded on surface electromyography (Hakkinen, Alen, Kallinen, Newton, & Kraemer, 2000; Hakkinen, Kraemer, Newton, & Alen, 2001) as well as motor unit discharge rate using an indwelling electrode (Leong, Kamen, Patten, & Burke, 1999; Patten et al., 2001) in both short- and long-term muscle strength training programs. In these studies, neural activities and maximal motor unit discharge rates of trained muscles were significantly increased with maximal voluntary contraction after muscle strength training.

The peripheral adaptations are related to muscle hypertrophy and increased contractile capacity and occur in later stages (generally, after 6 weeks) of muscle strength training programs (Baker, 2003; Fleck & Kraemer, 1997; Goto et al., 2004; Hakkinen, 1989). Researchers have shown that strength training promotes an increase in muscle protein synthesis, resulting in muscle hypertrophy, an increase in muscle strength with an overload stimulus (Frontera et al., 2003; Yarasheski, Zachwieja, Campbell, & Bier, 1995), and a cross-sectional area increase in both type I and II single muscle fibers of skeletal muscles of the elderly (Trappe et al., 2000). In addition, satellite cells, which are important for muscle fiber regeneration and hypertrophy, are also increased in their proportion and activities following strength trainings (Roth et al., 2001).

Interest in the potential of a training program to increase the strength and/or endurance of respiratory muscles in an elderly population has increased in the last few years (Tolep & Kelsen, 1993). Leith and Bradley (1976) were one of the first to attempt to train respiratory muscles by performing strength and endurance training to target specific ventilatory muscle groups. Four participants with mean age of 27 years were trained 5 days a week for 5 weeks maintaining CO_2 levels at a specific level, which is called as voluntary isocapnic hyperpnea (McConnell & Romer, 2004a). The training required extensive equipment to monitor the CO_2 levels and this training consumed a relatively large amount of time and highly depended on a subject motivation (McConnell & Romer, 2004a).

Consequently, other respiratory muscle strength training programs were developed to overstep the limitation of Leith and Bradley's complex equipment requirement. Commonly executed respiratory muscle strength training programs were flow-dependent resistance training and flow-independent pressure-threshold training. Resistance training does not depend on the lung pressure generated by respiratory muscles but depends on the airflow, which travels through a variable diameter orifice of the training device (McConnell & Romer, 2004a). The limitation of this training is that respiratory pressure developed during the training varies with flow and the orifice size. Therefore, during this training, breathing pattern should be monitored carefully. McConell and Romer (2004a) also mentioned that this training is a relatively time consuming and physically demanding. In contrast, pressure-threshold training of respiratory muscles has good reliability and is relatively easy to use (Baker, 2003; McConnell & Romer, 2004a). This involves a participant performing a certain number of repetitions a week with a specific number of exercise sets of training in each day. This training requires that an individual produce a certain amount of lung pressure to open a one-way valve on the training device so that air from the lungs flows through the device. It was also postulated that pressure-threshold training would result in greater training effect than resistance training because it requires a higher level of force to meet the load presented at a specific level compared to resistance training and allows the clinicians to set the training pressure-threshold depending on participants maximal pressure regardless of their breathing pattern or respiratory flow (Baker, 2003; Martin, Davenport, Franceschi, & Harman, 2002).

Most training paradigms with the elderly have used inspiratory resistive or inspiratory threshold loading (de Bruin, de Bruin, Lees, & Pride, 1993; Harver, Mahler, & Daubenspeck, 1989; Hsiao, Wu, Wu, & Wang, 2003; Larson, Kim, Sharp, & Larson, 1988; McConnell & Romer, 2004b; Olgiati, Girr, Hugi, & Haegi, 1989; Sturdy et al., 2003; Tolep & Kelsen, 1993; Weiner, Magadle, Beckerman, Weiner, & Berar-Yanay, 2004; Wiens, Reimer, & Guyn, 1999). Many of these studies have focused especially on inspiratory muscle strength training (IMST) in elderly patients with pulmonary disease (e.g., asthma, chronic obstructive pulmonary disease) or those with respiratory muscle weakness (e.g., multiple sclerosis, Parkinson's disease) with expectations to improve ventilatory capacity. However, interest in expiratory muscle strength training (EMST) has developed more recently, particularly for improving non-ventilatory functions such as cough, swallow, and speech.

Evidence that EMST increases expiratory muscle strength is evident from other studies of healthy adults (Baker, 2003; O'Kroy & Coast, 1993; Suzuki, Sato, & Okubo, 1995), high school band students (Sapienza, Davenport, & Martin, 2002), hypotonic children (Cerny, Panzarella, & Stathopoulus, 1997), high-risk performers (Hoffman-Ruddy, 2001), patients with chronic obstructive pulmonary (Weiner, Magadle, Beckerman, Weiner, & Berar-Yanay, 2003a), multiple sclerosis (Chiara, 2003; Gosselink, Kovacs, Ketelaer, Carton, & Decramer, 2000; Smeltzer, Lavietes, & Cook, 1996), Parkinson's disease (Saleem, 2005; Saleem, Sapienza, & Okun, 2005), and myasthenia gravis (Weiner et al., 1998). These studies demonstrated that EMST is effective in increasing the strength of expiratory muscles resulting in augmenting expiratory driving pressure which is utilized for cough, swallow, or speech (Table 1-2). Little outcome data are available on respiratory muscle strength training in the healthy elderly in either an inspiratory or expiratory direction, and use of respiratory muscle strength training may be beneficial for prevention or treatment of normal age-related respiratory muscular weakness (Tolep & Kelsen, 1993). Watsford, Murphy, Pine, and Coutts (2004) trained 26 older female participants (mean age of 64.4 years) with 8 weeks of 12 respiratory muscle training sessions with both IMST and EMST using a commercially available training device (Powerlung™ , PowerLung Inc., Houston, Texas, USA). They obtained significant increases in maximum voluntary ventilation, MIPs, MEPs, and other performance assessments such as time-to-rate of perceived exertion 15 walking test. However, this study only documented healthy elderly female performance and there was no detailed explanation regarding whether respiratory muscle training was aimed to improve expiratory muscle strength or inspiratory muscle strength. In addition, the outcome measures were associated only with ventilatory capacities. To date, no study has examined the effect of EMST on expiratory muscle strength in healthy elderly males and females and other studies of EMST on expiratory muscle strength have attempted to train elderly persons with diseases.

Table 1-2. Summary of expiratory muscle strength training (EMST) studies.

Study	Participants	*N*	Training program	Training (wks)	Training Load	MEP gain (%)	Significance Level	Functional Improvement
O'Kroy & Coast[1]	Healthy adults	6	RT	4	32% of MEP	NS	NS	Not applicable
Suzuki et al.[2]	Healthy adults	6	PT	4	30% of MEP	25	$p < 0.01$	Not applicable
Cerny et al.[3]	Hypotonic children	9	RT	6	2.5-7.5 cm H_2O	69	$p < 0.001$	Speech
Smeltzer et al.[4]	Multiple Sclerosis	10	PT	12	Not reported	37	No testing completed	Cough (subjective report)
Gosselink et al.[5]	Multiple Sclerosis	9	PT	12	60% of MEP	35	NS	Cough (subjective report)
Hoffman-Ruddy[6]	High risk performers	8	PT	4	75% of MEP	84	No testing completed	Speech
Sapienza et al.[7]	High school band students	26	PT	2	75% of MEP	47	$p < 0.001$	Not applicable
Baker[8]	Healthy young adults	32	PT	4-8	75% of MEP	29-50	$p < 0.05$	Speech & cough
Chiara[9]	Multiple Sclerosis	17	PT	8	40-80% of MEP	40	$p < 0.05$	Speech
Wingate et al.[10]	Professional voice users	18	PT	5	75% of MEP	77	$p < 0.001$	Speech
Saleem[11]	Parkinson Disease	10	PT	4	75% of MEP	22-37	$p < 0.001$	Cough & swallow

[1]O'Kroy, J.A. & Coast, J.R. (1993). Effects of flow and resistive training on respiratory muscle endurance and strength. Respiration, 60(5), 279–283.
[2]Suzuki, K.S., Sato, M, & Okubo, T. (1995). Expiratory muscle training and sensation of respiratory effort during exercise in normal subjects. Thorax, 50(4), 366–370.
[3]Cerny, F.J., Panzarella, K., & Stathopoulos, E.T. (1997). Expiratory muscles conditioning in hypotonic children with low vocal intensity levels. J Med Speech Lang Pathol, 5, 141–152.
[4]Smeltzer, S.C., Lavietes, M.H., & Cook, S.D. (1996). Expiratory training in multiple sclerosis. Arch Phys Med Rehabil, 77(9), 909–912.
[5]Gosselink, R., Kovacs, L., Ketelaer, P., Carton, H., & Decramer, M. (2000). Respiratory muscle weakness and respiratory muscle training in severely disabled multiple sclerosis patients. Arch Phys Med Rehabil, 81(6), 747–751.
[6]Hoffman-Ruddy, B. (2003). Expiratory pressure threshold training in high-risk performers, Unpublished doctoral dissertation, University of Florida, Florida.
[7]Sapienza, C.M., Davenport, P.W., & Martin, A.D. (2002). Expiratory muscle training increases pressure support in high school band students. J Voice, 16(4), 495–501.
[8]Baker, S.E., Davenport, P., & Sapienza, C. (2005). Examination of training and detraining effects in expiratory muscles. J Speech Lang Hear Res, 48(6), 1325-1333.
[9]Chiara, T. (2003). Expiratory muscle strength training in individuals with multiple sclerosis and health controls. Unpublished doctoral dissertation, University of Florida, Florida.
[10]Wingate, J., Sapienza, C.M., Shrivastav, R., & Brown, W.S. (in press). Treatment outcomes for professional voice users. J Voice.
[11]Saleem, A.F. (2005). Expiratory muscle strength training in patients with idiopathic parkinson's disease: Effects on pulmonary, cough, and swallow function. Unpublished doctoral dissertation, University of Florida, Florida.
MEP = maximum expiratory pressure, N = number of subjects who were trained with EMST program
PT = pressure-threshold training, RT = resistance training, NS = not significant

Expected Outcomes with EMST in the Elderly

Promising results from preliminary studies investigating the effects of expiratory muscle strength in different groups of participants suggest that EMST is able to increase expiratory muscle strength, improve cough function, promote swallow performance, and positively affect speech characteristics, in healthy young and clinical populations (Cerny et al., 1997; de Bruin et al., 1993; Gosselink et al., 2000; Hoffman-Ruddy, 2001; Saleem, 2005; Smeltzer et al., 1996). Hence, one would expect that EMST would improve respiratory function as well as the ability to clear the airway, swallow, and speak in the healthy elderly.

To expect that EMST would improve respiratory function by enhancing expiratory muscle strength in the healthy elderly population is reasonable. Specifically, FEV_1, FVC, and ERV would likely be affected. With age, expiratory force is diminished because of reduced elastic recoil of the lungs, compliance of the chest wall, and expiratory muscle strength. Therefore, FEV_1, FVC, and ERV are decreased in elderly individuals relative to younger counterparts (Waterer et al., 2001). Consequently, RV increases up to 50% and vital capacity decreases by about 75% maximally as adults reach age 70 (Gibson et al., 1976). Strengthening expiratory muscles by EMST would enhance the ability of the elderly to generate more expiratory force and compress the chest wall to a smaller volume as a compensatory mechanism, resulting in an increase in FEV_1, FVC, and ERV. As shown in Table 1-2, MEP levels were increased by a significant amount in healthy young adult participants and clinical populations regardless of the training program, duration of training, and training load.

EMST would also increase MIPs. Previous studies have showed that MIP levels increase following EMST in patients with multiple sclerosis (Chiara, 2003; Gosselink et al., 2000). Gosselink et al. (2000) speculated that the improved inspiratory muscle strength is related to reduced RV caused by a reduction of expiratory lung volume to allow the inspiratory muscles to operate easily with a more advantageous part of their length-tension relationship.

It is anticipated that EMST would increase peak expiratory flow rate during cough production. Cough is a reflexive protective mechanism to clear foreign substances or excessive mucous in the airways to reduce respiratory infection using higher velocities of forced expiratory airflow (Shannon, Bosler, & Lindsey, 1997). Coughing is composed of three consecutive phases: an inspiratory phase, a laryngeal compressive phase, and an expiratory phase (Leith & Bradley, 1976). In the inspiratory phase, once foreign substances or mucous stimulate the peripheral receptors and the stimulus is conducted to the central cough center located in the medulla (Bouros, Siafakas, & Green, 1995), the vocal folds abduct to open the glottis and allow air to fill the lungs. Then, during the laryngeal compression phase, the vocal folds adduct to close the glottis and the expiratory muscles contract to build up high positive intrapleural and intrathoracic pressures as high as 300 mmHg in a very short period of time (less than 200 ms) (Chung, Widdicombe, & Boushey, 2003; McCool, 2006; Irwin et al., 1998). Finally, the air from the intrathoracic airways is expired through a slightly opened glottis by the contraction of expiratory muscles with a velocity as great as 28,000 cm per second (Chung et al., 2003). During the expiratory phase, foreign substances are removed by the generation of the high velocity of expiratory airflow. During these three phases, the interactive activity of various respiratory muscles controls the cough mechanism intricately.

Reduced mucociliary clearance function (Bouros et al., 1995; Puchelle, Zahm, & Bertrand, 1979), decreased sensitivity of pharyngoglottal closure reflex (Shaker et al., 2003), and a diminished laryngeal valving mechanism (Hoit & Hixon, 1987) are the major causes of accumulation of mucous or aspiration in intrathoracic airways in the elderly. This eventually increases the mortality or morbidity of the elderly population from respiratory diseases (Kikawada, Iwamoto, & Takasaki, 2005; Logemann, 1998). To replace these regressed mechanisms, cough plays an important role in expelling foreign materials or secretions (McCool & Leith, 1987). Ineffective cough is also possibly related to reduced expiratory flow rates as well as lengthened laryngeal compression time (McCool & Leith, 1987). McCool and Leith (1987) noted that decreased expiratory peak flow is closely related to decreases in inspiratory or expiratory muscle strength.

Specific methods for increasing cough strength and timing have not been greatly studied to date. Of the limited treatment studies done on cough in patients with respiratory muscle weakness, the approaches rely on physical procedures, such as percussion and shaking, and manually assisted cough or mechanical insufflation and/or exsufflation (Bott & Agent, 2001; Chatwin et al., 2003; Mustfa et al., 2003). The implications are non-trivial for the elderly population. Although not a testable hypothesis in this research it is hoped EMST with the elderly has the potential to decrease or delay the development of respiratory complications by increasing expiratory muscle strength and increasing the ability to voluntarily clear the airway with a strong cough. An improvement in cough function should significantly reduce the occurrence of respiratory infections, thus enhancing the overall health of the elderly. Cough magnitude is directly related to the amount of expiratory driving pressure and expiratory pressure is the direct target of the expiratory training technique (Irwin et al., 1998).

Specifically, it is expected that EMST would increase the expiratory strength including peak expiratory flow rate and post-peak plateau during coughing as a result of overcoming high peripheral airway resistance. Peak expiratory flow rate during cough reflects the changes in muscular strength. Post-peak plateau is defined as the sustained expiratory airflow after the peak expiratory flow during a cough and it increase with increasing expiratory driving pressures as expiratory muscular strength is enhanced (Saleem, 2005). In fact, expiratory flow rate during cough is less in the elderly when compared to younger counterparts, which is related to decreased respiratory muscle strength (Babb & Rodarte, 2000), and decrease in lung elasticity (Babb & Rodarte, 2000) as well as an increase in the collapsibility of peripheral airways (Janssens et al., 1999).

In addition, EMST should decrease the laryngeal compression time. Changed afferent inputs in pressure are transferred to the central cough centers (Bouros et al., 1995) and this process should alter efferent outputs to the adductory laryngeal muscles. As a result, laryngeal compression time should decrease (de Bruin et al., 1993). These potential effects would decrease the chance of aspiration and decrease potential of respiratory infection. Baker (2003) and Saleem (2005) found that a 4-week EMST program improved peak expiratory flow and reduced laryngeal compression time during *maximum voluntary cough* in healthy young adults and patients with Parkinson's disease, respectively. In a study of patients with multiple sclerosis, increased expiratory pressure achieved with a 3-month pressure threshold EMST program was effective in increasing cough function, although the reports were subjective (Gosselink et al., 2000). This qualitative effect was also demonstrated in another study of an EMST program with patients with multiple sclerosis (Smeltzer et al., 1996). Ten participants in this study reported diminished choking events post-EMST. Those studies measured the cough characteristics during maximum voluntary cough production.

It is also expected that EMST will improve swallow function. Swallow dysfunction can be a major life-threatening problem in the elderly (Logemann, 1998). Explicit evidence exists regarding age-related changes in structure and physiology of swallowing, resulting in a high risk of swallowing problems in individuals over the age of 60 years. Significantly deteriorated efficiency of all phases of swallowing (oral preparatory, oral transit, pharyngeal, and esophageal phases) has been reported in several studies. These changes include increased duration of the oral stage of swallowing (Jaradeh, 1994; Logemann, 1998), reduced reflexes to trigger laryngeal closure (e.g., pharyngeal reflex, pharyngo-upper esophageal sphincter contractile reflex; McKee, Johnston, McBride, & Primrose, 1998; Ren et al., 2000; Robbins, Hamilton, Lof, & Kempster, 1992; Shaker et al., 2003), reduced laryngeal and hyoid anterior and vertical (superior) movement (i.e., reduced neuromuscular reserve; Logemann et al., 2000; Tracy et al., 1989; Yokoyama, Mitomi, Tetsuka, Tayama, & Niimi, 2000), diminished laryngeal valving capacity (Hoit & Hixon, 1987; Honjo & Isshiki, 1980; Ptacek & Sander, 1966; Titze, 1994), decreased pharyngeal flexibility (i.e., reduced pharyngeal contraction; Logemann et al., 2000), increased duration of the pharyngeal swallow (Jaradeh, 1994; Logemann, 1990; McKee et al., 1998; Tracy et al., 1989), impaired opening of the upper esophageal sphincter (Tracy et al., 1989), increased duration and width of cricopharyngeal opening (Kahrilas & Logemann, 1993; Tracy et al., 1989), and delayed and less efficient esophageal transit and clearance (Mandelstam & Lieber, 1970). Elderly individuals are more likely to aspirate if they have any circumstances of medical conditions, such as neurologic or neuromuscular diseases (Kikawada et al., 2005; Kobayashi, Hoshino, Okayama, Sekizawa, & Sasaki, 1994; Mandelstam & Lieber, 1970; Teramoto, Matsuse, & Ouchi, 1999).

An EMST program should contribute to the reduction of the mechanisms of age-related neuromuscular deterioration in the swallowing structures. Several possible mechanisms are expected to improve the swallow function with an EMST program. As predicted previously, EMST will increase expiratory lung volume and force, resulting in high expiratory airflow. In turn, this would increase the afferent stimulus on the sensory receptors of the tongue and oropharynx, leading to an increase in the activation of the swallow sensory recognition center located in the medulla or lower brainstem (Doty, Richmond, & Storey, 1967; Gross, Atwood, Grayhack, & Shaiman, 2003; Logemann, 1998). After this incoming information is decoded in the nucleus tractus solitarius, the efferent information from the nucleus ambiguous is delivered to motor units participating in oropharyngeal swallow motor pattern (Doty et al., 1967). The increased activity of motor units would improve the efferent motor activities of oropharyngeal, velar, and laryngeal musculatures as well as the speed of oropharyngeal swallow. Consequently, the improved activities and speed of swallowing structures would reduce the general duration of swallowing.

Another possible mechanism for improvement in swallow function could be related to an increase in the hyolaryngeal displacement with increased expiratory force as a result of the EMST program. During the oropharyngeal swallowing phase, the hyoid bone is pulled up which elevates the larynx anteriorly and vertically by the contraction of submental muscle group including the suprahyoid muscles, in other words, laryngeal elevator muscles (Logemann, 1998; Perlman, Palmer, McCulloch, & Vandaele, 1999). This muscle group is composed of the anterior belly of the digastric, mylohyoid, and geniohyoid muscles. Yokoyama et al. (2000) suggested that vertical hyolaryngeal movement causes laryngeal closure so as to protect the lower airway and that anterior hyolaryngeal movement contributes to decreasing the upper-esophageal sphincter pressure to enable a bolus into the upper-esophageal sphincter readily. In turn, anterovertical hyolaryngeal movement associated primarily with the contraction of submental muscles is important for effective and safe passage of a bolus to the esophagus. Hyoid and laryngeal elevations during oropharyngeal swallowing have been commonly observed using submental muscle group activity in surface electromyography (Ding, Larson, Logemann, & Rademaker, 2002; Ertekin et al., 1995; Perlman et al., 1999; Vaiman, Eviatar, & Segal, 2004a, 2004b; Wheeler & Sapienza, 2005). As mentioned previously, elderly individuals have a decrease in hyolaryngeal displacement, which can reduce the laryngeal closure and cause a bolus to escape into other cavities, resulting in high risk of aspiration (Logemann et al., 2000; Yokoyama et al., 2000). However, hyolaryngeal displacement may be increased by forced expiration with EMST. Fink and Demarest (1978) noted that laryngeal displacement is induced by the respiratory cycle, with inspiration associated with downward movement and expiration with upward movement. Particularly, upward laryngeal movement is related to the mechanical contribution of laryngeal elevator muscles. If expiratory force increases, it would enhance the activities and strength of laryngeal elevator muscles, resulting in enhancing hyolaryngeal displacement. With increased hyolaryngeal displacement during swallowing, the glottal closure should be enhanced, thus moving the bolus into the esophagus more easily. This assumption was supported by the study of Wheeler and Sapienza (2005) which compared the submental muscle group activities using surface electromyography during swallow task and respiratory task using EMST device set at 25% and 75% of MEP in 20 young healthy adults. Their study showed that the EMST task produced significantly higher peak amplitude and greater average amplitude of submental muscle group activity compared to either dry swallow or wet swallow tasks. They also observed increased hyoid elevation, while using the EMST device, on videofluoroscopy from one participant. They suggested that EMST enhances the activation of the submental muscle group for swallowing and may impose central and peripheral adaptations during the EMST program.

Finally, it is predicted that the EMST program will improve speech characteristics. It is well known that the contraction of expiratory muscles is necessary for certain types of speech tasks since it controls the outflow of air in order to speak as well as provides the necessary pressure when elastic recoil forces are not great enough to vibrate the vocal folds (Hixon, 1973). Isshiki (1964) noted that improvements in sound quality, speech intelligibility, duration, and intensity are a function, to some extent, of the degree of expiratory pressure that can be developed. Even though reduced lung volume and pressure, resulting from decreased respiratory muscle strength, thoracic compliance, and elastic lung recoil pressure, do not seem to cause major problems associated with breathing at rest or comfortable effort, the necessary volume and pressure to sustain speech for a long period of time and to perform loud speech or singing cannot be achieved. These tasks require greater lung pressure associated with expiratory muscle force (Hoit & Hixon, 1987). In fact, inadequate lung pressure for speech or singing results in severely decreased vocal intensity and shortened utterance length per breath (Titze, 1994). Particularly, chest wall rigidity and respiratory muscle weakness associated with aging results in compromised lung volumes available for speech (Titze, 1994). Specifically, normal inspiratory volumes cannot be produced by the elderly, thus limiting the available passive recoil pressure for speech and high-effort tasks. When inspiratory volumes are limited and the subglottal pressure demand for particular speech tasks cannot be met (e.g., long durations of speech or loud speech), active expiratory muscles must be recruited to generate the positive airway pressure for these tasks (Burzynski, 1987). When an individual increases expiratory muscle strength, chest wall rigidity may reduce because the individual is able to move the chest wall with greater force (Hoit & Hixon, 1987). This should result in increased speech durations, greater sound pressure level, and improved voice quality, and speech intelligibility.

Previous studies demonstrated that both elderly males and females produce more than 6 dB lower sound pressure level (SPL) in maximum vowel intensities (sound pressure drops by half) than younger counterparts (Morris & Brown, 1994; Ptacek & Sander, 1966; Teles-Magalhaes, Pegoraro-Krook, & Pegoraro, 2000). In addition, a comparative study of young versus elderly male voices indicated a significantly lower vocal intensity in elderly male voices with reduced lung pressure, peak airflow, and open quotient during syllable train production (Hodge, Colton, & Kelley, 2001). Hodge et al. (2001) noted that SPL was greater for young men than for elderly men at all different intensity conditions. In the study, mean lung pressures in the loud condition were 10.82

cm H_2O and 7.96 cm H_2O in the control young group and in the elderly group, respectively. The difference of the lung pressure between the control and elderly groups was statistically significant in this condition. These results suggest that lung pressure is significantly decreased in the elderly group as compared with the young group. Changes in lung pressure are closely associated with changes in SPL. The SPL increases at a rate of 8 to 9 dB when lung pressure is doubled (Hodge et al., 2001). Thus, reduced lung pressure for loud phonation in the elderly compared to the young represents a reduction in lung pressure which may be accompanied with weakness of the expiratory muscles. Additionally, several researchers report reduced vocal fold closure (Honjo & Isshiki, 1980; Linville, 1992; Tanaka, Hirano, & Chijiwa, 1994), histologic and neuromuscular changes in laryngeal muscles (Rodeno, Sanchez-Fernandez, & Rivera-Pomar, 1993), and decreased laryngeal muscle activity with age (Baker, Ramig, Sapir, Luschei, & Smith, 2001; Luschei, Ramig, Baker, & Smith, 1999). Therefore, it was suggested that the changes in lung pressure may be necessary to overcome an age-related changes in laryngeal structure and mechanism to control airflow and air pressure for speech in the elderly (Baker et al., 2001). Furthermore, it has been shown that the number of syllables produced per breath group are reduced with age (Hoit & Hixon, 1987), which is related to the reduced duration of phonation in the elderly. However, these age-related changes in speech might be compensated for by EMST in that it may assist the active expiratory force to positively affect speech characteristics such as increasing expiratory pressure to produce loud phonation or sustain phonation for a long period of time as well as overcoming high laryngeal resistance. In a study of a 4-week EMST program with patients with Parkinson’s disease, significant improvements in the range of vocal loudness during sustained vowel phonation tasks was found (Saleem et al., 2004). It is also known that EMST program for individuals with neurologic impairments improves their speech parameters like vocal intensity. Following a 6-week EMST program, nine children with hypotonia demonstrated significant increases in vocal intensity among participants (Cerny et al., 1997). Furthermore, an 8-week of EMST improved acoustic components of speech including vowel prolongation as well as subjectively reported voice-related quality of life in 17 patients with multiple sclerosis (Chiara, 2003). The participants with multiple sclerosis described an ability to breathe easier and talk louder after EMST program. In a study with high risk performers working in a theme park who were singing along with choreography, expiratory muscle strength and utterances duration per breath significantly increased after a 4-week EMST program (Hoffman-Ruddy, 2001). Additionally, professional voice users with voice problems after the combined treatment of 5 weeks EMST and 6 sessions of traditional voice therapy showed significant improvements in voice handicap scores, voice rating scale scores, subglottal pressure for loud intensity, phonetogram area in both frequency and amplitude, and dynamic range (Wingate, Sapienza, Shrivastav, & Brown, in press). These previous works with the EMST program in the healthy and the clinical populations indicate improvements in pressure support for voice and speech quality post-EMST including increases in vowel and phrase durations, increased SPL, as well as decreased frequency variability, and reductions in breathlessness, and in vocal fatigue.

Statement of the Problem

It is known that EMST has a great impact on increasing expiratory muscle strength in healthy and clinical populations. However, very few have investigated the effect of EMST on healthy elderly population. As described earlier, after the age of 60, people have reduced mass and changed fiber types of expiratory muscles resulting in reduction of muscle strength. This latter age-related changes in skeletal muscles, referred to as sarcopenia, often combined with the sedentary lifestyle in the elderly, leading to a significant reduction in reserve capacity of muscular strength. Reduced physical activity accelerates the changes in respiratory muscle structures with muscle atrophy, which can affect the reduction of particular functions of breathing, cough, swallow, and speech in the elderly.

Therefore, this study will reveal how EMST impact on expiratory muscles which play a major role in breathing, cough, swallow, and speech in the sedentary healthy elderly.

Previous studies have shown that EMST is an effective training paradigm to increase expiratory muscle strength resulting in improvements in certain physiological functions in healthy adults and certain clinical populations. However, very few studies have sought to quantify real therapeutic gains directly attributable to the interventions that have employed EMST with regard to breathing, cough, swallow, and speech. Previously, the effects of EMST on cough have quantified in maximum voluntary coughs. Cough is a reflexive event. To examine the effects of EMST on coughs should be measured in reflexive coughs. Most recently, the inhaled irritant of choice for investigation of cough in human has been capsaicin. This irritant is safe and reproducibly elicits cough in virtually all participants (Dicpinigaitis, 2003; Dicpinigaitis & Alva, 2005; Ertekin et al., 1995; Nieto et al., 2003; Prudon et al., 2005). Thus, cough measures in this study were completed using capsaicin-induced cough productions. Additionally, the effects of EMST on swallow have quantified with videofluoroscope or subjective reports from the participants in the previous studies. However, no study has measured the strength changes in the muscles used for swallowing. Surface electromyography (sEMG) has been commonly used to evaluate the strength changes in the swallow muscle group activity. sEMG is a simple, reliable, and noninvasive method to assess temporal and neural activities of complex muscle group. Particularly, the activity of submental muscles which are the primary muscle group of hyolaryngeal elevation during the early stage of swallowing could be repetitively measured using sEMG without invasive procedure in pre- and post-EMST.

Purpose of the Study

The purpose of this study is to investigate the physiological effects of EMST on expiratory muscle strength in otherwise healthy, but sedentary, elderly as measured by the primary dependent variable of maximum expiratory pressure (MEP). Additionally, this study will examine the potential effects of EMST on maximum inspiratory pressure (MIP), breathing, cough, swallow, and speech functions affected by the aging process.

Hypotheses

Central Hypothesis: It is hypothesized that a 4-week EMST program will improve maximum respiratory pressure, breathing, cough, swallow, and speech functions in otherwise healthy, but sedentary, elderly adults due to expected increases in expiratory muscle strength. The specific hypotheses are the following:

Hypothesis 1: Expiratory muscle strength, as indicated by increased MEP, and inspiratory muscle strength, as indicated by increased MIP due to changes in pulmonary mechanics, will improve after 4 weeks of strength training with an expiratory pressure threshold device.

Hypothesis 2: Increased expiratory muscle strength will be translated to improvements in breathing functions. Specifically, improvements in forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and expiratory reserve volume (ERV) will be affected. But the ratio of FEV_1 to FVC will not be changed.

Hypothesis 3: Improved expiratory muscle strength will increase the peak expiratory flow rate (PEFR) and the post-peak plateau duration (PPPD) and the post-peak plateau integral amplitude (PPPIA) as well as decrease inspiratory phase duration (IPD) and compression phase duration (CPD) during the capsaicin-induced reflexive cough production.

Hypothesis 4: Increased cough magnitudes are not the influence of increased sensitivity to capsaicin challenge.

Hypothesis 5: Increased expiratory force and hyolaryngeal displacement will increase the peak amplitude (PA) and integral amplitude (IA) of submental muscle group activity and decrease the duration (DUR) of submental muscle group activity during maximal voluntary dry (saliva) and thin paste bolus (5 cc and 10 cc pudding) swallows.

Hypothesis 6: Increased expiratory muscle strength will increase excess lung pressure (P_{EL}) as well as the maximum phonation durations (MPDs) at comfortable intensity and at loud intensity.

CHAPTER 2
METHODOLOGY

The project design was a prospective, complete repeated measures design. Participants were assigned to use a specific experimental training device for expiratory muscle strength training. The independent variables were training status (Pre-training/Post-training) and gender for all functions, consistency (maximal voluntary dry, 5 cc water, 10 cc water, 5 cc pudding, and 10 cc pudding) for swallow function, and loudness (comfortable/loudest) for the speech function. The dependent variables for pulmonary function were maximum expiratory pressure (MEP) and maximum inspiratory pressure (MIP), force vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), the ratio of FEV_1 to FVC (FEV_1/FVC), and expiratory reserve volume (ERV). Cough dependent variables were inspiratory phase duration (IPD), compression phase duration (CPD), peak expiratory flow rate (PEFR), post-peak plateau duration (PPPD), and post-peak plateau integral amplitude (PPPIA) as well as total number of coughs and total number of expulsive events. Swallow dependent variables were peak amplitude (PA), duration (DUR), and integral amplitude (IA) of submental (SM) rectified surface electromyography (sEMG) during maximal voluntary dry and 5 cc and 10 cc boluses of wet (water) and thin paste (pudding) swallows. Speech dependent variables included excess lung pressure (P_{EL}) as well as maximum phonation durations of sustained vowel production (MPDs) at two levels of intensities, comfortable and maximum loudness.

Sample Size Determination

Sample size calculation was performed using one dependent variable as suggested by Marks (Marks, 2002). The maximum expiratory pressure (MEP) was used as the variable to determine sample size since this measure was considered the primary outcome measure for determining the effect of EMST. The standard deviation of MEP, denoted as σ, was determined from the range of MEP values obtained in a pilot study (Kim, Sapienza, & Davenport, 2005). The range was 75.86 cm H_2O, which yielded a σ of 18.97 cm H_2O. The minimum clinical significant difference, or bound on error (B), between the average MEP values before EMST and average MEP value after EMST was determined to be 20% of the average baseline MEP. Since the average MEP was measured at 73.57 cm H_2O, B value was calculated and yielded a value of 14.71 cm H_2O. Using the obtained values of σ and B, DELTA (δ) was calculated using the formula: $\delta = B/\sigma$ and determined to be as 0.775. The significance level (α), or probability of executing a Type I error, was predetermined at 0.05. The power of the test (1-β), or the ability to reject the null hypothesis if the null is false, was set at 90%. Using the sample size table provided in Marks (2002), the number of participants needed was 18. Therefore, 18 participants were recruited for this study.

Recruitment and Selection

An approval for the study was obtained from the University of Florida Health Science Center Institutional Review Board (IRB# 402-2004) prior to recruiting participants. All participants in this study signed an informed consent document authorized by the IRB. Participants were recruited from local community members (via residential facilities, social and professional organizations, churches, and retirement communities) in the Gainesville area. Printed flyers containing information about the study and contact information were posted at various locations across local communities (Appendix A).

Inclusion Criteria

Participants were included based on the following criteria:

1. Over 65 years.
2. Sedentary: Sedentary was defined as a person with 24-hours (24-h) of maximum exertion time (MET-Time) < 50 in physical activity as described in the physical activity questionnaire (Aadahl & Jorgensen, 2003) (Appendix B).

    - The chosen activities were listed in the physical activity scale in nine levels of physical exertion, ranging from sleep or inactivity to strenuous activities. The physical activity scale was composed of the number of minutes (15, 30, or 45 min) and hours (1 to10-h) spent on each MET activity level on an average 24-h weekday. This allowed for a calculation of the total MET-time, representing 24-h of sleep, work, and leisure time on an average weekday.
    - MET activity level: A = 0.9 MET, B = 1.0 MET, C = 1.5 METs, D = 2.0 METs, E = 3.0 METs, F = 4.0 METs, G = 5.0 METs, H = 6.0 METs, and I ≥ 6 METs).
    - For each activity level (A to I) the MET-Value was multiplied by the time spent on that particular level and MET from each level was added to total 24-h MET-time, representing physical activity level on an average weekday.

3. Able to maintain his/her current level of physical activity during participation in this study.

    - Participants were asked to report to the investigator any significant changes in their level of physical activity during their participation in the study with regards to intensity and frequency of exercise during the entire training (e.g., a sedentary person begins exercising 2 to 4 days per week).

4. Able to complete the informed consent to participate in the study

Exclusion Criteria

Participants were excluded from the study if they reported any of the following:

1. History of the following medical conditions: chronic and acute cardiac disease including untreated hypertension (systolic blood pressure > 140 mmHg, diastolic

blood pressure > 90 mmHg), pulmonary disease, neuromuscular disease, and/or immune system disease, or others as reported on a health questionnaire (Appendix C).

2. Upper respiratory infection at the time of the baseline measurements as reported on the health questionnaire or during the training period. If symptoms persisted for more than 1 week of training, the participant was excluded from the study.

3. Pulmonary function test values below 70% of the predicted normative value (e.g., FEV_1 < 70% or FVC < 70%).

4. History of smoking or tobacco use within the last 5 years.

5. Extreme athletes (e.g., marathon runner, professional weightlifter).

6. Other illness that would prevent patient from completing the protocol.

7. Significant change in activity level.

Participant Demographics

Twenty one participants, 16 women and five men, were recruited in the study. Two women completed only the first pre-training baseline measures and withdrew from the study due to uncomfortable feeling of the capsaicin challenge. One man completed the first and the second pre-training baseline measures and withdrew from the study due to the concern about a low, but potential health risk involved, particularly, with high intensity pressure threshold trainer during the development of high expiratory pressure. Thus, a total of 18 sedentary healthy elderly individuals completed the study. Four of these participants were men and 14 were women. The average age of the participants was 77 years with a range of 68 to 89 years. The age of men ranged from 72 to 89 years (mean age of 78.25 ± 7.80) and the age of women ranged from 68 to 84 years (mean age of 76.64 ± 5.27). Demographic information of participants is summarized in Table 2-1.

Measures

Participants were asked to fill out a physical activity questionnaire (Appendix B) and health questionnaire (Appendix C). Blood pressure was measured by the investigator to determine if the participants qualified to be in the study. This study included a 7-week experimental protocol for each participant. Week 1 and week 2 were two pre-training baseline measurement conditions. Participants were exposed to the training program during weeks 3 to 6. Week 7 was the post-training measurement condition. Measures of pulmonary function (i.e., maximum respiratory pressures and breathing measures), cough, swallow, and speech were obtained for each participant.

Table 2-1. Demographic information for participants in the study.

Participant	Gender	Age (yrs)	Height (cm)	Weight (kg)	Physical Activity (METs)
1	F	77	154.94	63.05	31.70
2	F	73	154.90	49.50	23.45
3	F	68	157.00	93.60	48.30
4	F	83	165.00	58.95	40.80
5	M	73	177.80	112.50	17.55
6	M	72	177.80	77.40	38.90
7	F	74	160.02	74.25	28.82
8	F	81	152.40	70.00	46.90
9	F	81	152.40	53.10	30.45
10	F	75	167.64	70.65	31.92
11	F	84	165.10	61.20	23.45
12	F	77	152.40	65.90	25.45
13	F	69	167.64	74.25	26.10
14	M	79	175.26	N/A	41.70
15	M	89	172.72	74.25	15.70
16	F	83	160.02	74.25	25.70
17	F	71	165.10	72.00	30.45
18	F	77	167.64	72.00	20.95

Note: N/A = Not applicable

Maximum respiratory pressures, MEPs and MIPs, were recorded from all participants in two pre-training conditions and following each week of training for 4 weeks as well as post-EMST. All other measures were recorded from all participants in two pre-training conditions and post-EMST.

Pulmonary Measures

Maximum Respiratory Pressures. Maximum expiratory pressures (MEP) and maximum inspiratory pressure (MIP) were measured using a disposable mouthpiece connected to a Smart 350 series pressure manometer (Meriam Process Technologies, Cleveland, Ohio, USA) by 50 cm of 6 mm inner diameter tubing with a 20-gauge (2 mm) needle air-leak at the mouth to prevent the participant from sustaining pressure with a glottal closure (Berry et al., 1996; Enright et al., 1994; Karvonen et al., 1994; O'Kroy & Coast, 1993). During the completion of the MEP and MIP tasks, each participant stood with his/her nose occluded with a nose clip while he/she used the Smart 350 series pressure manometer. For MEP, after inhaling to total lung capacity, the participant placed his/her lips around a mouthpiece and blew out as forcefully as possible. For MIP, after exhaling to residual volume, the individual placed his/her lips around a mouthpiece and inspired as forcefully and fast as possible through the mouthpiece connected to a pressure gauge with the nose occluded by a nose clip. Repeated measures were taken with a 1 to 2 minute rest between trials for both the MEP and MIP measures, until three measurements were obtained within ± 5% of each other and no further improvement was obtained. The average of these three values was used for analysis. Approximately 5 to 10 trials were necessary per participant to obtain the 3 trials within ± 5% of each other.

Breathing Measures. Pulmonary function tests (PFTs) were completed using a computerized MasterScreen PFT system (Jaeger Toennies, Erich Jaeger Gmbh, Leibnizstrasse 7, D-97204 Hoechberg). During the completion of the PFTs, the participant sat in front of the MasterScreen PFT. To obtain PFTs, the guidelines of the American Thoracic Society were followed. Breathing function parameters measured in this study were forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), the ratio of FEV_1 to FVC (FEV_1/FVC), and expiratory reserve volume (ERV). For FVC and FEV_1, each individual placed their mouth around a disposable mouthpiece. The nose was occluded by a nose clip to prevent air leak. Next, the participant was asked to take a deep breath and inspired to total lung capacity, followed by taking three tidal volume breath cycles. The individual then blew out as forcefully as possible into a mouthpiece, being verbally encouraged to “blast out” all the air in the lungs. FVC was defined as the total volume of air expired during a maximally forced expiration after a full inspiration (Figure 2-1). FEV_1 was defined as a measure of expiratory volume during the first second of expiration during the forced vital capacity maneuver with maximal expiratory effort (Figure 2-1). FEV_1/FVC ratio was calculated by dividing the FVC value by the FEV_1 value taken from the PFTs. This parameter provides a clinically useful index of airflow limitation. All PFT measures were completed a minimum of three times with a 1 to 2 minute rest between trials to ensure similar values were obtained at each attempt. The best three measurements were averaged and recorded for analysis. For ERV, each participant was asked to place their mouth around a disposable mouthpiece. The nose was occluded by a nose clip to prevent air leak and they were asked to rest breathe for three cycles. Next, the participant was asked to carry out, consecutively, a maximal slow inspiration, a maximal slow expiration, and then one more maximal slow inspiration into a mouthpiece connected to the MasterScreen PFT. ERV was defined as the maximum volume of air that can be expired after normal expiration (i.e., tidal volume; Figure 2-2).

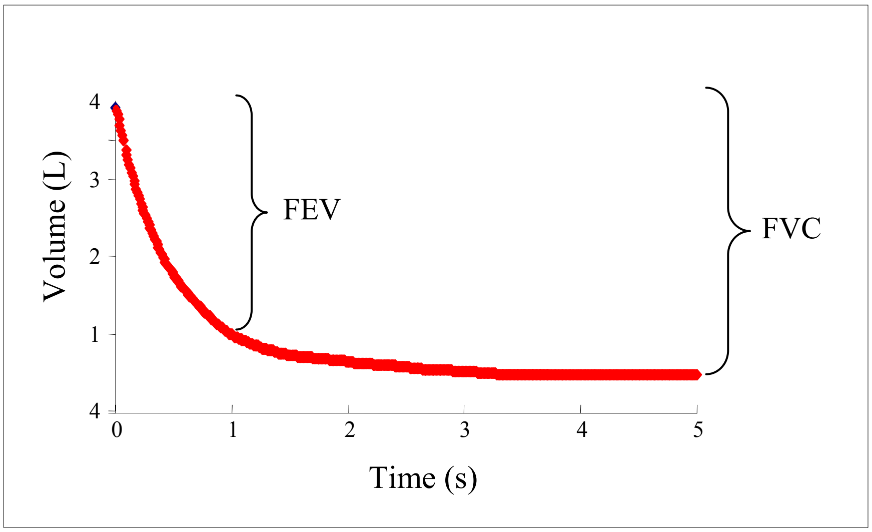


Figure 2-1. Graphical depiction of FVC and FEV_1.

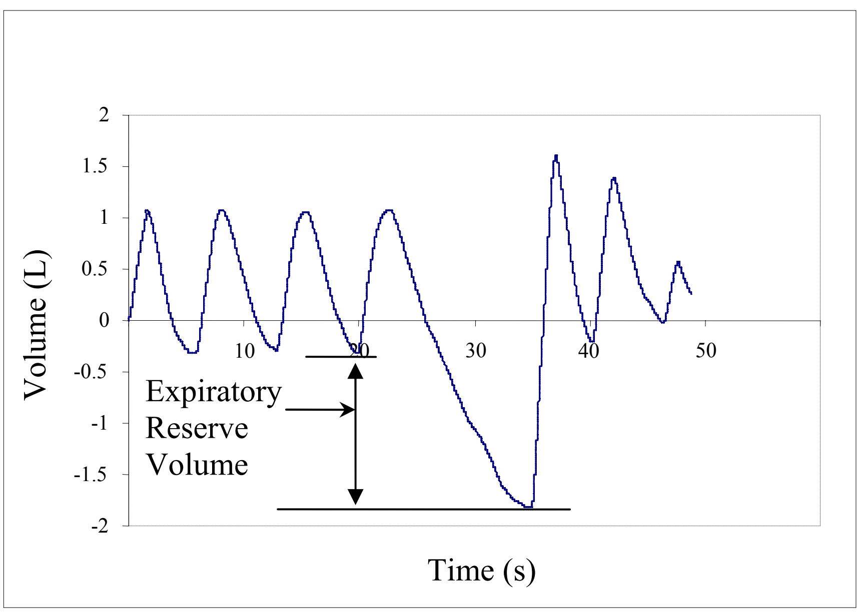


Figure 2-2. Graphical depiction of ERV.

Cough Measures

Cough magnitude was measured from an expiratory flow waveform produced during a capsaicin-induced cough. To obtain an acceptable cough signal, the participants were seated comfortably in a chair. The mask, connected to a pneumotachometer (Hans Rudolph Inc., Kansas City, MO, USA) was placed on the participants face and they were given a verbal cue to take a single vital capacity breath of 100 microMolar (μM) capsaicin in 80% physiological saline, 10% Tween 20, and 10% Ethanol capsaicin solution (Appendix D) via an air-powered nebulizer (KoKo Digidoser; Pulmonary Data Services Instrumentation Inc., Louisville, CO). The nebulizer output was set at 10 μL. A differential pressure transducer (Validyne MP 45-2-871, Validyne Engineering Corp. Northridge, CA, USA) in ranges as low as ± 2 cm H_2O was attached to the facemask pneumotachometer. Next, the participants were asked to take a single deep inspiration of a 10 μL of 0.9% saline solution followed by capsaicin solution to eliminate capsaicin residue from the facemask. Capsaicin and saline was administered five times alternatively in pre-training and post-training conditions. Each test of inspiration was separated by an interval of 1 to 2 minutes. This attachment was fitted directly into the Power Lab/8SP data acquisition system (ADInstruments, ML750, Colorado Springs, CO, USA). Prior to collection of cough measures from each participant in the baseline sessions, the pneumotachometer was calibrated with 1 L/s flow source. A calibration routine within Chart 4.2.3 for Windows software (ADInstruments, Colorado Springs, CO, USA) was used to calculate volume and flow.

All cough signals were recorded using the Chart 4.2.3 software. The signal was low-pass filtered at 300 Hz through the filter in the Powerlab unit, as the filter in the spirometer capable of filtering to 100 Hz, was inadequate to filter the high frequency components of the waveform during cough. Measurements of cough flow were analyzed using Chart 5 for Windows software (ADInstruments, Colorado Springs, CO, USA). The sampling rage was set at 2,000 samples per second for all cough measures.

A cough was defined as having an inspiratory phase, a compression phase, and one or more expulsive events (i.e., cough refractory; CR) on a single inspiration. Expulsive events are composed of one strong expulsive event (SE) followed by reflexive expulsive events (RE; Figure 2-3). The total number of coughs (N of coughs) was counted from the airflow signal taken from each of the five trials of capsaicin inhalation and then averaged. In addition, the total number of expulsive events (N of CR), sum of all SEs and REs, was counted independent of the inspiration from the cough airflow taken from each of the five trials of capsaicin inhalation and then averaged.

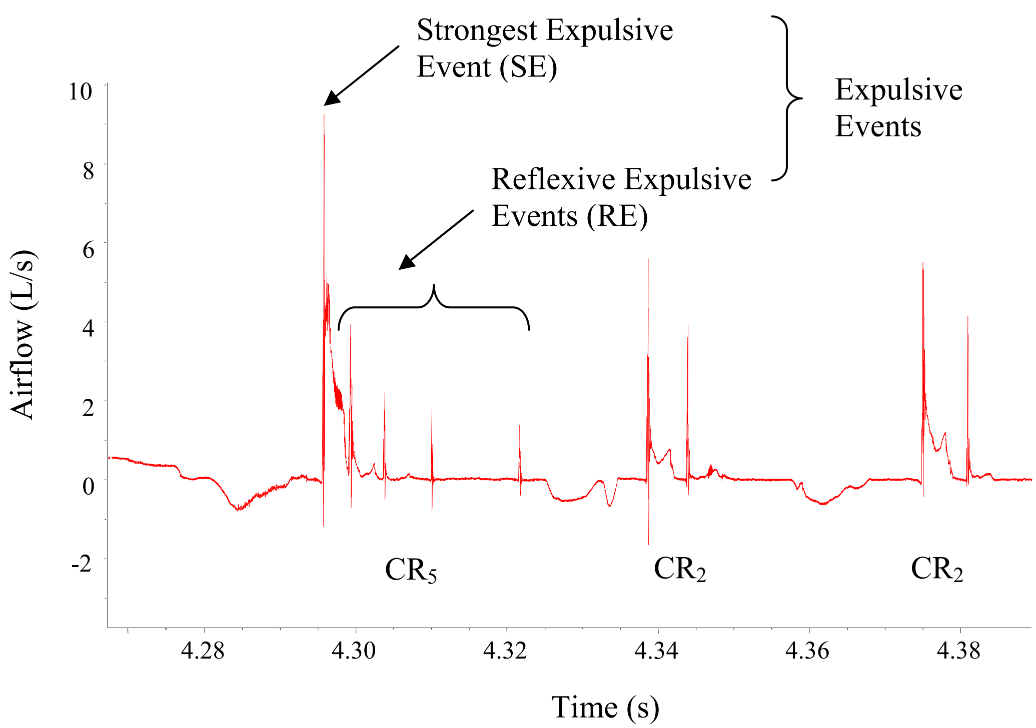


Figure 2-3. Airflow during reflexive cough production. CR_5 represents five expulsive events. The total number of coughs is three and the total number of expulsive events is nine in this Figure.

Cough magnitudes measured in this study were inspiratory phase duration (IPD), compression phase time (CPD), peak expiratory flow rate (PEFR), post-peak plateau duration (PPPD), and post-peak plateau integral amplitude (PPPIA; Figure 2-4). The magnitudes of the coughs having the highest PEFR, were taken from each of the five trials of capsaicin inhalation, were collected and then averaged.

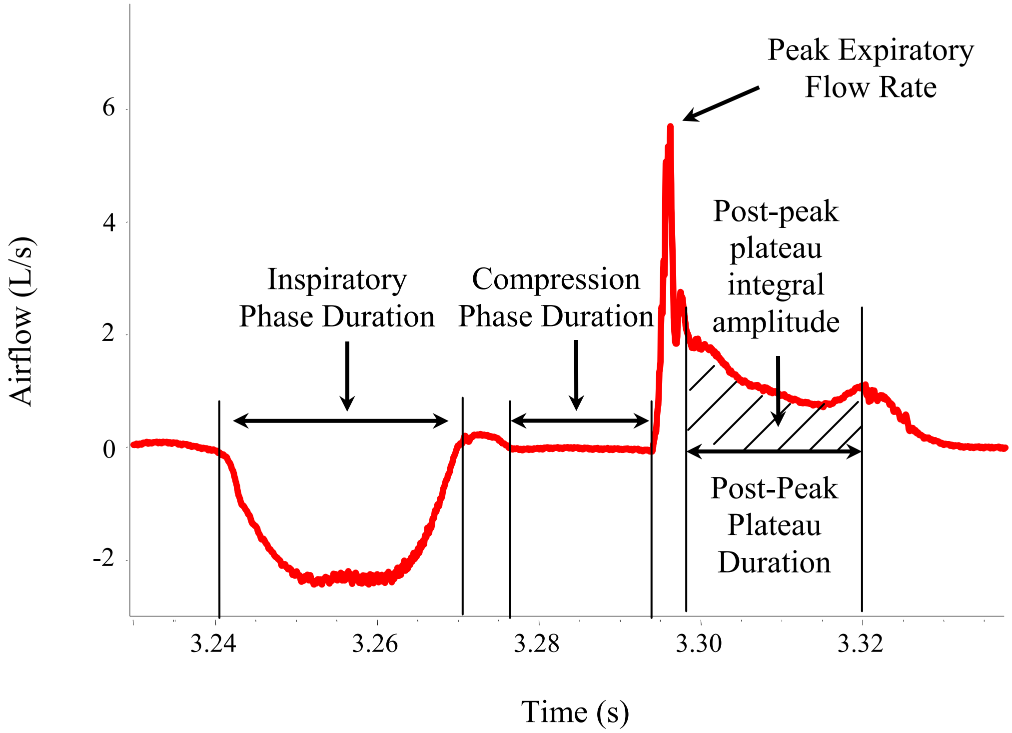


Figure 2-4. Cough magnitudes in one cough.

IPD (in seconds) was defined as the time from the beginning to the end of the inspiratory phase marked by departing and returning of the airflow to 0 L/s. PEFR (L/s) was defined as the highest peak of expiratory flow rate following the inspiratory phase during the capsaicin-induced cough. CPD (in seconds) was defined as the duration between the end of the inspiratory phase and the beginning of the expiratory phase. PPPD (in seconds) was defined as the time of sustained airflow that occurred after the peak expiratory flow. This was visually determined by observing the expiratory flow waveform. The initiation point for PPPD was marked as the time immediately after the transient overshoot of the peak airflow signal when the flow became stable. PPPD termination was marked as the final time-point where the stable flow ended prior to another rapid descent in the expiratory flow rate. PPPIA (L/s × s) was defined as the area under cough airflow curve between PPPD initiation and PPPD termination.

Swallow Measures

Submental surface electromyographic signals (SM-sEMG) were obtained using an 8-channel Bagnoli EMG System (Delsys Inc., Boston, MA, USA) to record submental muscle group activity. Two surface electrodes (DE-2.1 single differential electrodes, Delsys Inc., Boston, MA, USA) were attached and taped to the skin beneath the chin, bilaterally, to record the strength of submental (SM: suprahyoid) muscles over the mylohyoid, geniohyoid, and anterior digastric muscle complex during three trials of each consistency. A paper ruler, width of 1 cm, measured the length from the tip of the nose to the point where two electrodes were attached on the chin in the first pre-training baseline measurement session and recorded to help keep consistent place for electrodes attachment in baseline measurements and post-training measurement. The output of the Bagnoli systems was connected directly to the Power Lab/8SP data acquisition system. The software program, Chart 4.2.3 for Windows from ADInstruments, was used to record the swallow measures online and Chart 5 for Windows was used to analyze the data off-line. The signal from two sEMG electrodes was amplified with a gain of 1000 V/V, low-pass filtered at 1,000 Hz, and high pass-filtered at 100 Hz in order to remove any DC offset and high-frequency noise. The original EMG signal was rectified by the root mean square (RMS) method. This method used the square root of the average of the squared values of the preceding data points over the time set (20 ms). Chart 4.2.3 was set up to display 4 different channels. Channel 1, channel 2, channel 3, and channel 4 displayed the right SM-sEMG raw data, the left SM-sEMG raw data, the right SM-sEMG RMS data, and the left SM-sEMG RMS data, respectively. SM-sEMGs were collected during five different consistencies which included dry voluntary maximal effortful swallow, wet (5 cc and 10 cc water) swallow, and thin paste (5 cc and 10 cc pudding) swallow. A total of 15 trials were randomly assigned to the participants.

Swallow dependent variables were peak amplitude (PA), duration (DUR), and integral amplitude (IA) of SM-sEMG activity during swallowing (Figure 2-5). The maximum strength of SM muscles was defined as the PA (mV) of the RMS of SM-sEMG signal activity during swallowing. The DUR (s) of SM-sEMG during swallowing was determined by measuring the interval between the onset and the offset (offset time minus onset time) of SM-sEMG activity (Ding et al., 2002; Ertekin et al., 1995). The IA (mV/s × s) of SM muscles was also calculated as the area of RMS under the SM-sEMG activity curve between the onset and offset of the swallow. This measured the output of total SM muscles activity during swallowing.

The onset and offset in SM-sEMG activity were determined by Cycle Variables function in Chart 5 (Figure 2-6). Cycle Variables function is defined as channel calculation that identifies cycles in the SM-sEMG waveform. It calculates and displays cycles extracted from the waveform and explicitly takes into account the waveform's cyclic aspects. Chart 5 software provides two different types of cyclic variables including temporal quantities, such as period, frequency, or rate and amplitude-related characteristics, such as cyclic maximum, cyclic minimum, cyclic mean, or cyclic height. In this analysis, cyclic mean was used as cyclic variables in channel 1.

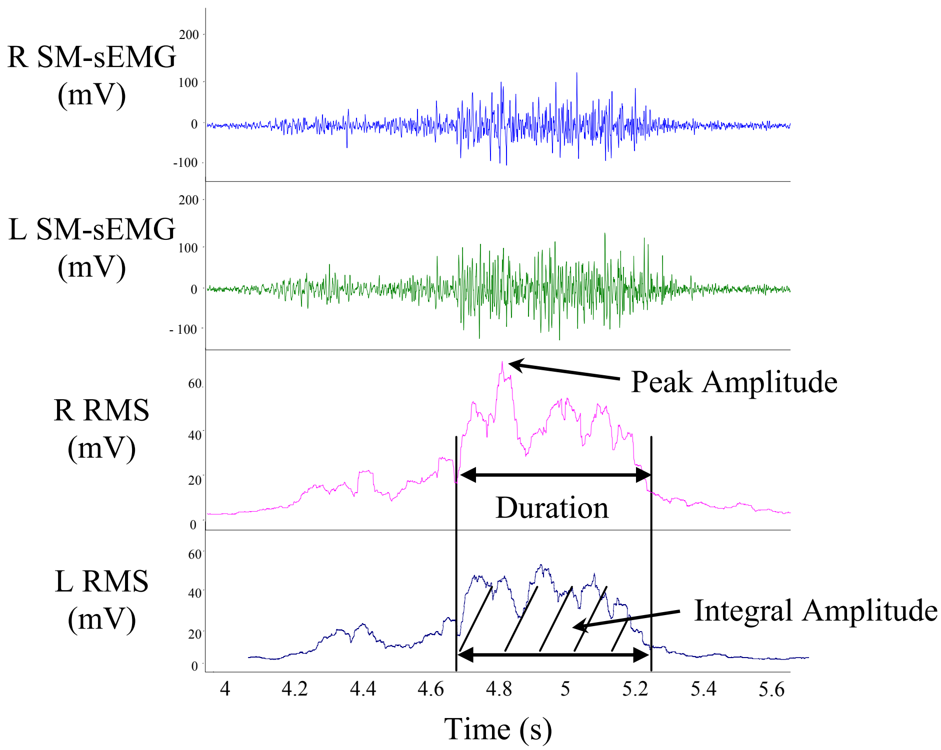


Figure 2-5. SM-sEMG Activity. R SM, L SM, and RMS denote right, submental muscle, left submental muscle, and root mean square, respectively.

Cyclic mean is the mean value of the data points contained in one cycle of a waveform to display the cycle-by-cycle mean value of SM-sEMG recording. SM-sEMG baseline before each swallow task was measured in an individual and was used for setting up the sensitivity of cycle detection algorithm by adjusting 2% noise threshold of baseline, a percentage of the selected data range. Cycle Variables ignore fluctuations in the waveform less than the set-up noise threshold value. The onset and offset of SM activity during swallowing were determined the zero points which were the closest time before the peak of SM-sEMG and the closest time after the peak of SM- sEMG, respectively.

Before measuring swallow function, as a standardized test of SM-sEMG activity, participants were asked to blow into the expiratory pressure threshold trainer which was set up at 50 cm H_2O. These were taken during two pre-training baseline measurements and compared in PA, DUR, and IA of SM-sEMG activity.

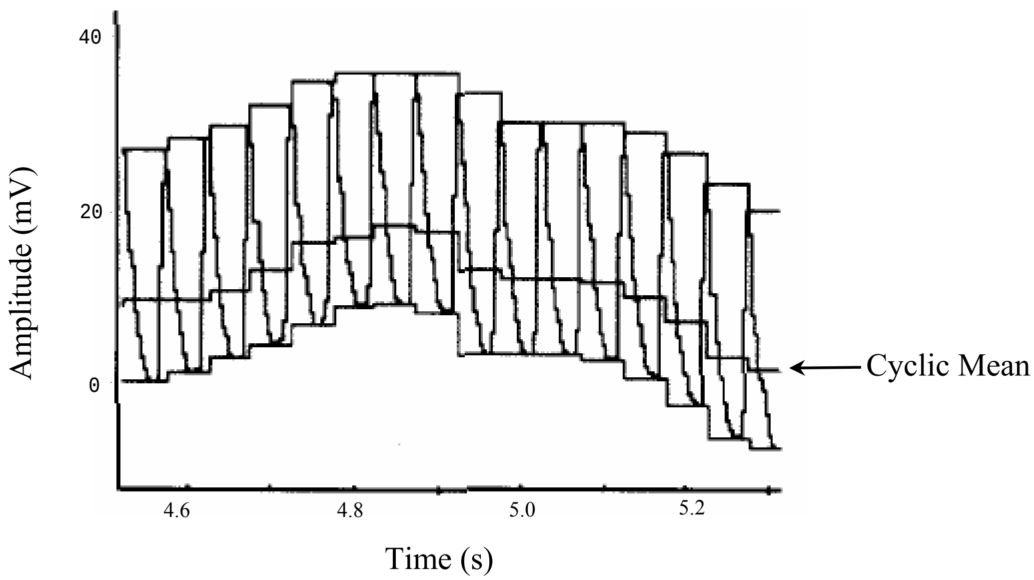


Figure 2-6. Cycle variables function. From Chart 5 for Windows software (ADInstruments, Colorado Springs, CO, USA).

Speech Measures

Dependent variables related to speech production were examined using aerodynamic and acoustic analysis. Aerodynamic measures included air pressure measures (explained below). Participants were asked to repeat syllables while a small disposable plastic pitot tube (2 mm diameter) was placed into the oral cavity between the lips and behind the front teeth. The tube was connected to a pressure transducer (PTL-1, Glottal Enterprises, Syracuse, NY, USA) low pass-filtered at 30 Hz which recorded the air pressure signal. The pressure transducer was calibrated with 5 cm H_2O prior to data collection (MCU-4, Glottal Enterprises, Syracuse, NY, USA). All pressure measures were recorded using Power Lab/8SP data acquisition system with Chart 4.2.3 for Windows software. The sampling rate was set at 10,000 samples per second for all pressure measures. Air pressure measures were obtained by three tasks. The first task was the repetition of a syllable train /pɑ/ seven times in one breath at the softest vocal intensity level. The second task was the gradual increase in the vocal intensity to the maximum effort level. The third task was the repetition of a syllable train /pɑ/ seven times in one breath at the maximum effort level. The participants were instructed to take a breath just before starting the next task. The first and third were completed randomly. If individuals initiated from the third task, they were asked to decrease the vocal intensity gradually to the softest possible intensity level. During these tasks, participants were instructed to produce each consonant in the syllable with approximately equal stress and to maintain a syllable rate of about 1.5 syllables per second. In repeating a syllable train at the softest possible intensity level, participants were instructed to initiate voice at the lowest possible intensity level without whispering. The initiating effort level of syllable repetition was randomly assigned. Five trials of this task in each individual were recorded on Chart 4.2.3 for Windows and were analyzed on Chart 5. The recordings for air pressure were completed in a quiet room.

Air pressure measures included excess lung pressure (P_{EL}) calculated from phonation threshold pressure (P_{th}) and lung pressure (P_L). P_{th} and P_L values were estimated from intraoral pressure (P_o) measurements (Hodge et al., 2001; Rothenberg, 1982; Smitheran & Hixon, 1981). P_o was defined as the pressure within the oral cavity during the production of the voiceless stop segment /p/ produced in the syllable train /pɑ/ at both the softest and the loudest possible intensity levels. P_{th} was defined as the minimum pressure required for initiating vocal fold vibration (Titze, 1994). Measurement of P_o at the softest possible level was utilized for estimating P_{th}. P_L used in this study was defined as the P_o at the loudest possible level. P_{EL} was defined as the difference between the P_L and P_{th} (Hodge et al., 2001). The pressure peaks of P_o values from the middle five of seven repeated /pɑ/ at both the softest and loudest possible intensity levels were measured and averaged to estimate P_L and P_{th} values from P_o. The relative change in pressure was calculated in cm H_2O.

Acoustic measures included maximum phonation durations (MPDs) of sustained vowel phonation at two levels of intensities, comfortable and loud. MPD was defined as the greatest length of time over which sustained vowel could be prolonged (Baken & Orlikoff, 1998). Participants sat on a chair comfortably and wore a cardioid headset microphone (ATM73a, Audio-Technica, Japan) placed 2 cm from the right corner of the mouth. Participants' phonations were recorded on a portable digital audio tape (DAT) recorder (TAS CAM DA-P1, TEAC Corporation, Japan). Participants were instructed to take a deep breath and sustain the vowel /ɑ/ as long as they could at the comfortable and the loudest effort levels. Each task was performed three times and the order of the tasks was randomly assigned. Three trials of MPD in each intensity level was analyzed and averaged using the software program, TF32 for 32-bit Windows (Milenkovic, Wisconsin, USA).

Training Protocol

After completion of the two pre-training baseline sessions discussed above, each participant was provided with the expiratory pressure threshold trainer. The expiratory pressure threshold trainer used to complete the EMST program was a cylindrical plexiglass tube that consisted of a mouthpiece and an adjustable one-way spring-loaded valve (Figure 2-7). This device allowed the pressure threshold to be set up to 150 cm H_2O. The spring contained in this device was adjustable to allow for the required pressure threshold to be increased. The valve blocked expiratory airflow until a sufficient threshold pressure was reached to overcome the spring force. Participants had to overcome a threshold load by generating an expiratory pressure sufficient to open the expiratory spring-loaded valve.

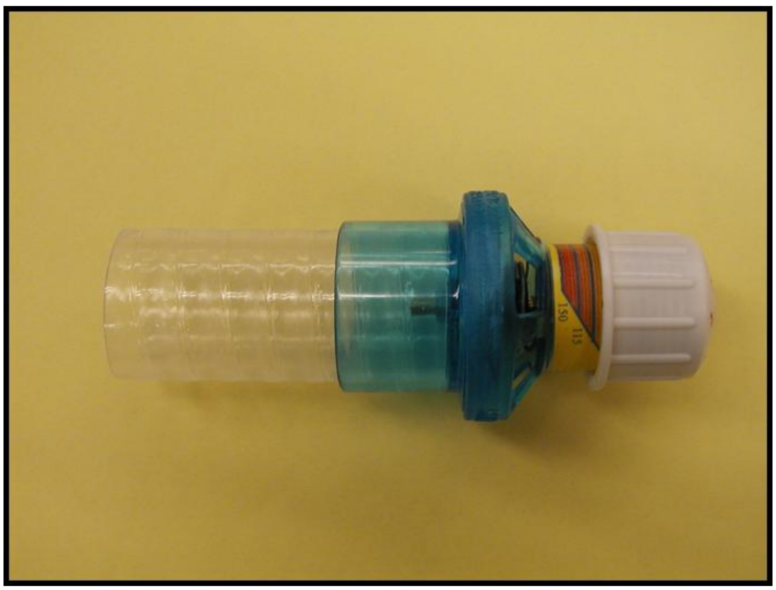


Figure 2-7. Expiratory pressure threshold training device.

As stated previously, the participants' MEP was measured at the initiation of the study and following each week of training as well as post-training. The training protocol for each participant lasted 4 weeks and consisted of five sets of five breaths, 5 days per week with the pressure threshold set at 75% of the participant' MEP at the time of measurement (Baker, 2003; Chiara, 2003; Saleem, 2005; Wingate et al., in press). This percentage was based on skeletal muscle training research that demonstrates that the most effective muscle strengthening occurs when a near maximal load is placed on the muscle (Powers & Howley, 2001). Each training breath lasted 3 to 4 seconds. In the initial training session, an individual was informed of the time frame of training, proper device handling procedures, appropriate mouth closure around the device's mouthpiece, and air leak prevention techniques. To prevent possible air leak, the individual was instructed to place his/her lips tightly around the device's mouthpiece and one of his/her hands held his/her cheeks around the lips firmly. The individual was then instructed to blow as forcefully as possible into the device's mouthpiece from total lung capacity (TLC) to open the valve. The individual was also trained how to correctly discriminate the sounds between success and failure of opening valve by successive opening valve trials. As air passes through the device following the opening the valve, the individual could listen to a distinct audible sound such as whistle sound or air popping sound.

A weekly readjustment meeting was maintained with each participant and the investigator. At that meeting, the participant's MEP and MIP was measured in the same manner stated earlier, and the average value of each was used in the dataset. The device was readjusted by the investigator according to the newly measured average MEP value. To ensure that the new training load was appropriate, the participant was needed to complete the one set of five breaths in the clinician's attendance. Any participant concern regarding the training program was addressed at that time.

Compliance

To insure participant's compliance with the training protocol, participants were provided with written (Appendix E) and verbal instructions for the use of their devices and the EMST protocol. During home training period, participants recorded their completion of training sets daily at home on a log sheet (Appendix F) during the 4 weeks of EMST. Participants were also instructed to call the investigator at any time if they had questions or if problems arose in their practice procedure.

Statistical Analysis

The mean, standard deviation, and percent change were calculated from the database to describe the trends in the dependent variable from pre- to post-training. If the first pre-training measures were not statistically different from the second pre-training measures, the two datasets were averaged and used as the average pre-training measures to be compared with the post-training measures. Differences between the pre-training condition 1 and the pre-training condition 2 were examined by paired-samples *t*-test for all dependent variables. There were no significant difference between the pre-training condition 1 and pre-training condition 2 for any of the dependent variables of pulmonary, cough, swallow, and speech functions, therefore those were averaged and then used as the average pre-training measures that were compared with the post-training measures (Table 2-2, 2-3, 2-4). Three doubly multivariate repeated measures analyses of variance (MANOVA) were used to examine the effects of EMST on pulmonary and cough functions. Repeated measures univariate analysis of variances (ANOVAs) were conducted to evaluate the effects of EMST on swallow and speech functions. Doubly multivariate repeated measures design has multiple dependent variables measured in different levels of one or more within-subjects factors or in different levels of between-within (mixed) design with multiple repeated dependent variables (Tabachnick & Fidell, 1996). In these repeated measures designs, sphericity assumptions (i.e., homogeneity of variance assumption) were checked using Mauchly's test of sphericity. Sphericity assumption is a mathematical assumption that assumes all variances of the differences for each pair of categories of the within-subjects factor are equal in the populations sampled.

In advance, it is expected that the observed samples variances of the differences are similar if the sphericity assumption is met. In this study, if this assumption was violated, Greenhouse-Geisser adjustment test for the within-subjects effect was conducted. This test adjusts the degrees of freedom (df) downward for the usual F test statistic to overcome the reduced p-value for the within-subjects effect (Agresti & Finlay, 1999).

However, Pillai's Trace was used in this study since unequal sample sizes occurred for the men and women involved in this study. Usually, Pillai's Trace provides good power and is most unlikely to violate statistical assumptions as well as it is more appropriate when sample sizes are small or cell sizes are unequal (Olsen, 1976; Tabachnick & Fidell, 1996; Walker, 1998).

Table 2-2. Paired-samples *t*-test between the two pre-training conditions for the pulmonary and cough function dependent variables.

DV	1^{st} Pre-training		2^{nd} Pre-training		*t*	df	*p*
	M	*SD*	*M*	*SD*			
MEP (cm H_2O)	75.956	20.513	78.317	20.558	-1.351	17	0.194
MIP (cm H_2O)	38.497	13.304	39.661	15.081	-0.462	17	0.650
FEV_1 (L)	1.907	0.527	1.981	0.464	-1.810	17	0.088
FVC (L)	2.525	0.707	2.568	0.574	-0.947	17	0.357
FEV_1/FVC	76.232	8.546	77.109	6.253	-0.611	17	0.549
ERV (L)	0.995	0.656	0.947	0.592	0.823	17	0.422
IPD (s)	1.094	0.277	1.196	0.401	-0.956	17	0.352
CPD (s)	0.384	0.215	0.311	0.205	1.815	17	0.087
PEFR (L/s)	4.885	3.252	5.076	2.091	-0.246	17	0.809
PPPD (s)	0.232	0.087	0.240	0.086	-0.357	17	0.725
PPPIA (L/s×s)	3.369	2.750	3.607	2.940	-0.350	17	0.730
N of Coughs	40.778	15.125	37.611	12.391	1.826	17	0.085
N of CR	13.944	4.734	14.444	3.974	-0.486	17	0.633

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 2-3. Paired-samples *t*-test between the two pre-training conditions for the swallow function dependent variables.

DV	Consistency	1st Pre-training		2nd Pre-training		*t*	df	*p*
		M	*SD*	*M*	*SD*			
PA (mV)	DRY	56.042	20.569	65.251	29.250	-1.600	17	0.128
	5W	53.503	31.790	51.812	39.797	0.352	17	0.729
	10W	53.830	30.736	51.009	37.893	0.502	17	0.622
	5P	52.866	23.594	54.752	37.564	-0.282	17	0.781
	10P	52.120	25.592	58.301	41.531	-0.987	17	0.338
DUR (s)	DRY	0.995	0.171	0.926	0.149	1.905	17	0.074
	5W	0.912	0.154	0.939	0.147	-0.915	17	0.373
	10W	0.958	0.147	0.928	0.170	1.018	17	0.323
	5P	1.012	0.132	0.974	0.134	1.465	17	0.161
	10P	1.014	0.142	1.012	0.160	0.071	17	0.944
IA (mV)	DRY	25.168	8.626	28.123	11.441	-1.100	17	0.286
	5W	22.347	12.912	21.524	13.622	0.385	17	0.705
	10W	23.197	12.369	21.333	13.113	0.741	17	0.469
	5P	25.124	10.583	25.171	14.679	-0.017	17	0.987
	10P	22.677	8.502	26.071	15.607	-1.106	17	0.284

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 2-4. Paired-samples *t*-test between the two pre-training conditions for the speech function dependent variables.

DV	1st Pre-training		2nd Pre-training		*t*	df	*p*
	M	*SD*	*M*	*SD*			
P_{EL} (cm H_2O)	12.879	5.122	14.392	5.480	-1.819	17	0.087
MPD – COMF (s)	17.529	8.766	18.025	7.312	-0.626	17	0.540
MPD – LOUD (s)	19.449	11.149	19.394	11.229	0.052	17	0.959

* indicates that the mean difference is significant at $\alpha = 0.05$.

If the effect of gender was not significant at $\alpha = 0.05$, it was eliminated from the MANOVA or ANOVA, then another MANOVA or ANOVA was carried out only using the within-subjects factors. If any MANOVA or ANOVA indicated a significant interaction among factors at $\alpha = 0.05$, simple main effects tests using paired-samples *t*-tests were conducted with the α level set at 0.01. If any MANOVA or ANOVA indicated a significant main effect at $\alpha = 0.05$, univariate comparisons of the specific outcome variables were explored. In univariate comparisons (i.e., multiple pairwise comparisons), the inflated α level was adjusted by using the Bonferroni adjustment to reduce Type I error rate when multiple tests are performed on the same data (Tabachnick & Fidell, 1996). Type I error occurs when one rejects the null hypothesis when it is true. All analyses were carried out using SPSS software version 11.5.

In addition, the relationship between the change in MEP and other pulmonary, cough, swallow, and speech functions were investigated using Pearson *r* correlation.

Inter- and intra-judge reliability were also completed on 10% of the data that were measured by hand. To test the inter-judge reliability of the dependent variables, a different examiner, a student trained by the investigator in analyzing and scoring the various measures, re-analyzed the data. The student was blinded to the purpose of the study. Pearson *r* correlations were used to determine if there was any significant difference between the values obtained by different examiners. To test intra-judge reliability, the investigator repeated the analyses of 10% of the data sets and compared the first set of measures to the second set using Pearson *r* correlations again.

CHAPTER 3
RESULTS

This study determined the effects of a 4-week expiratory muscle strength training (EMST) program on pulmonary, cough, swallow, and speech functions in otherwise healthy, but sedentary, elderly adults.

Reliability

Pearson *r* correlations were calculated to evaluate the intra-judge measurement reliability for cough, swallow, and speech functions (Table 3-1). Strong correlations were found, indicating high intra-judge reliability. For intra-judge reliability, the Pearson *r* correlation between the first and second sets of measurement ranged from 0.905 to 1.000. Likewise, Pearson *r* correlations were calculated to test the inter-judge measurement reliability for cough, swallow, and speech functions. Results also showed moderate to strong correlations between ratings made by the two different judges. For inter-judge reliability, the Pearson *r* correlation between two measurers for cough, swallow, and speech functions ranged from 0.743 to 1.000. Given these data, the reliability of the all measures in cough, swallow, and speech functions was considered adequate for the purpose of the present experiment.

Correlation Between MEP and Other Dependent Variables

A Pearson *r* correlation was performed between MEP and the other dependent variables included in the study. The results of the correlation are presented in Table 3-2 and show that MEP was both moderately positively and negatively correlated with variables such as MIP ($r = 0.532$, $p = 0.001$), CPD ($r = -0.367$, $p = 0.028$), PEFR ($r =$

0.526, $p = 0.001$), PPPIA ($r = 0.472$, $p = 0.004$), and P_{EL} ($r = 0.363$, $p = 0.029$). However, MEP was not significantly correlated with other pulmonary and any swallow dependent variables.

Pulmonary Function

Table 3-3 depicts the descriptive statistics for all of the pulmonary function measures pre- and post-training as a function of gender.

A 2 × 2 doubly multivariate repeated measures analysis of variance (MANOVA) was conducted to analyze the results of MEP, MIP, FEV_1, FVC, FEV_1/FVC, and ERV as affected by training and gender. Sphericity assumptions were met. The results of the MANOVA indicated a non-significant two-way interaction between training and gender at $\alpha = 0.05$ (Table 3-4). The main effect of training significantly affected the combination of MEP, MIP, FEV_1, FVC, FEV_1/FVC, and ERV (Pillai's Trace = 0.827, F (6, 11) = 8.766, $p = 0.001$, $\eta^2 = 0.827$). Gender did not significantly affect the combination of pulmonary function dependent variables. Hence, a one-way repeated measures MANOVA without the gender effect was conducted. Again, sphericity assumptions were met. The results of MANOVA indicated that the main effect of training was significant on the combination of pulmonary dependent variables (Wilks' $\Lambda = 0.189$, F (6, 12) = 8.576, $p < 0.001$, $\eta^2 = 0.811$). Therefore, one-way repeated measures univariate ANOVAs to determine the specifics of the training effect were conducted (Table 3-5). The ANOVA results indicated that MEP was significantly greater in post-training (F (1, 17) = 40.978, $p < 0.001$, $\eta^2 = 0.707$). MIP also significantly increased with training (F (1, 17) = 18.513, $p < 0.001$, $\eta^2 = 0.521$). However, no significant effects of training were found on FEV_1, FVC, FEV_1/FVC, and ERV. MEP and MIP increased from pre- to post-training by 44% and 49%, respectively (Figure 3-1).

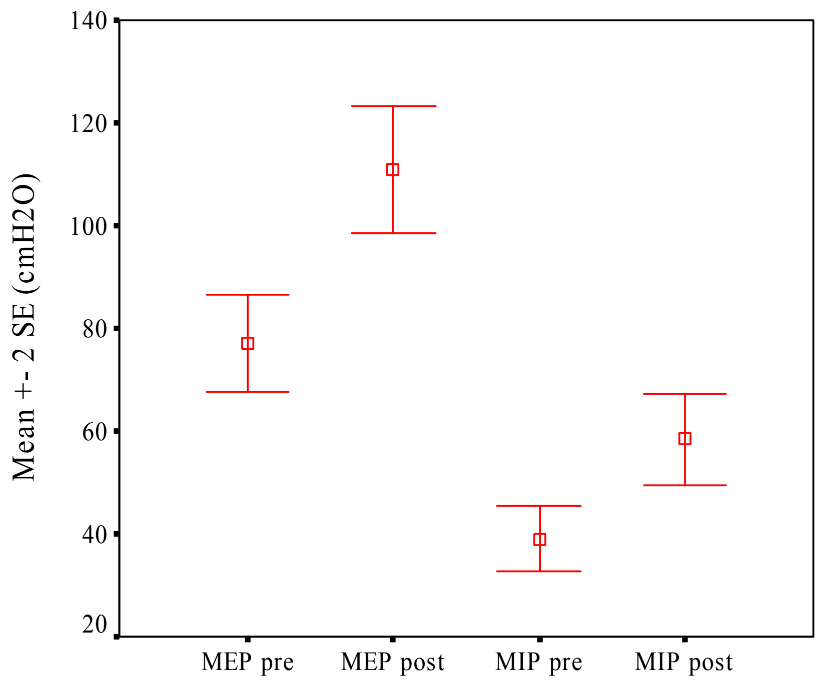


Figure 3-1. Effects of training on MEP and MIP.

Cough Function

Table 3-6 shows the descriptive statistics for the dependent variables associated with cough function pre- and post-training as a function of gender.

A 2 × 2 doubly multivariate repeated measures analysis of variance (MANOVA) was used to analyze the results of IPD, CPD, PEFR, PPPD, and PPPIA as affected by training and gender. Sphericity assumptions were met. Table 3-7 shows the MANOVA results and indicates that the two-way interaction between training and gender was not significant. The main effects of training and gender were then examined and indicated that training was not significant on the combination of dependent variables of IPD, CPD, PEFR, PPPD, and PPPIA, but close to significance of $\alpha = 0.05$ (Pillai's Trace = 0.520, F (5, 12) = 2.598, $p = 0.081$, $\eta^2 = 0.520$). Gender was not significant. Hence, a one-way repeated measures MANOVA without the gender effect was conducted. The result of MANOVA without the gender effect indicated a significant effect of training on the combination of cough dependent variables (Wilks' $\Lambda = 0.351$, F (5, 13) = 4.803, p = 0.010, $\eta^2 = 0.649$). One-way repeated measures univariate ANOVAs of the training effect were then completed (Table 3-8). The ANOVA results revealed that CPD was significantly decreased with training (F (1, 17) = 13.590, $p = 0.002$, $\eta^2 = 0.444$). PEFR was also significantly increased with training (F (1, 17) = 29.620, $p < 0.001$, $\eta^2 = 0.635$) and was PPPIA (F (1, 17) = 16.826, $p = 0.001$, $\eta^2 = 0.497$). However, no significant effect of training was found for IPD as well as PPPD. The results for CPD, PEFR, and PPPIA are illustrated in Figure 3-2, Figure 3-3, and Figure 3-4, respectively. CPD decreased from pre- to post-training by 53% and PEFR and PPPIP increased from pre- to post-training by 61% and 96%, respectively.

To evaluate the effects of training and gender on the total number of coughs (N of coughs) and the total number of expulsive events (i.e., N of cough refractories; N of CR), a 2 × 2 doubly multivariate repeated measures analysis of variance (MANOVA) was conducted. Sphericity assumptions were met. There was no significant interaction between training and gender (Table 3-9). The main effects of training and gender were not significant. Therefore, a univariate MANOVA was completed to test for the training effect after excluding the gender effect. The results of the MANOVA indicated no significant effect of training on the N of coughs and the N of CR (Pillai's Trace = 0.210, F (2, 16) = 2.130, $p = 0.151$, $\eta^2 = 0.210$).

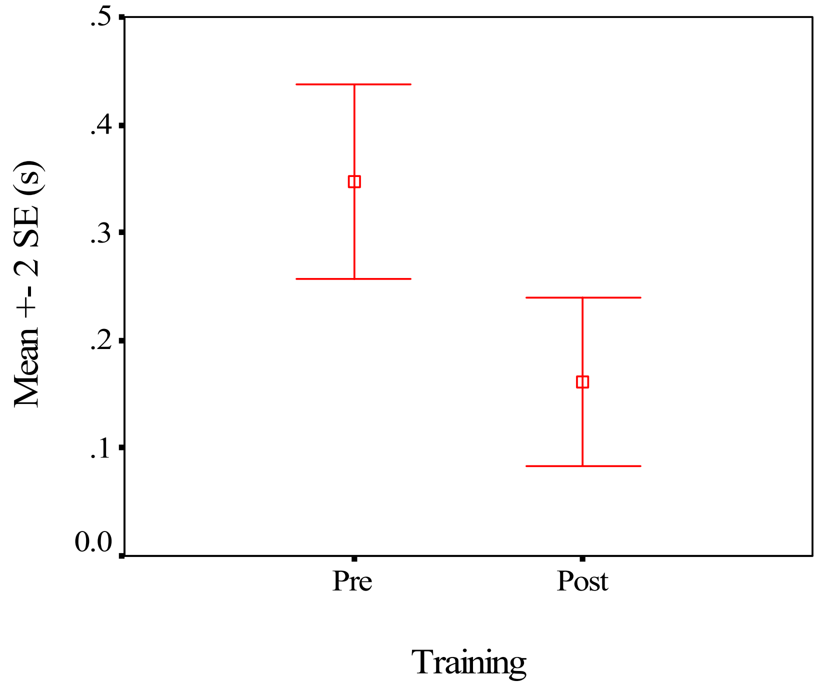


Figure 3-2. Effects of training on CPD.

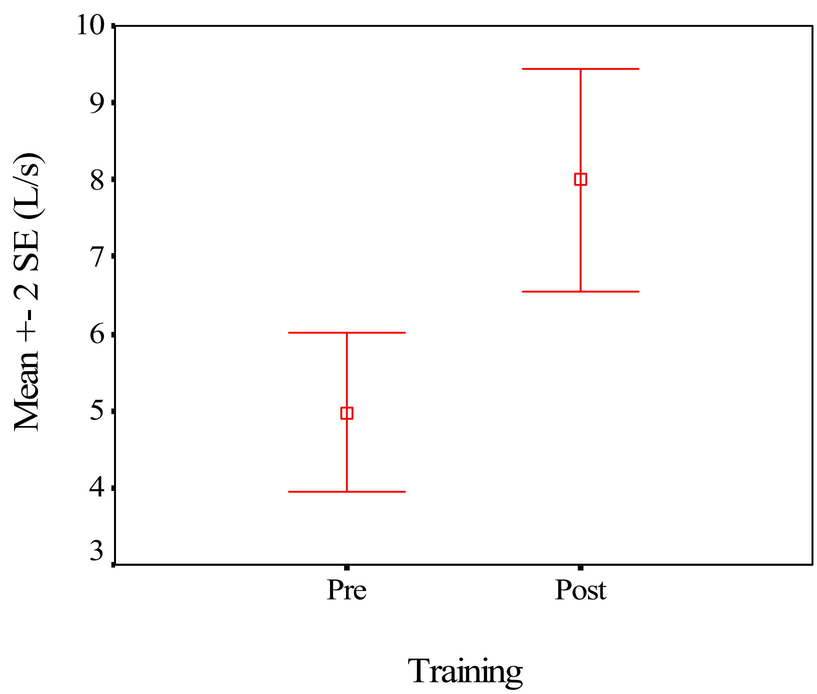


Figure 3-3. Effects of training on PEFR.

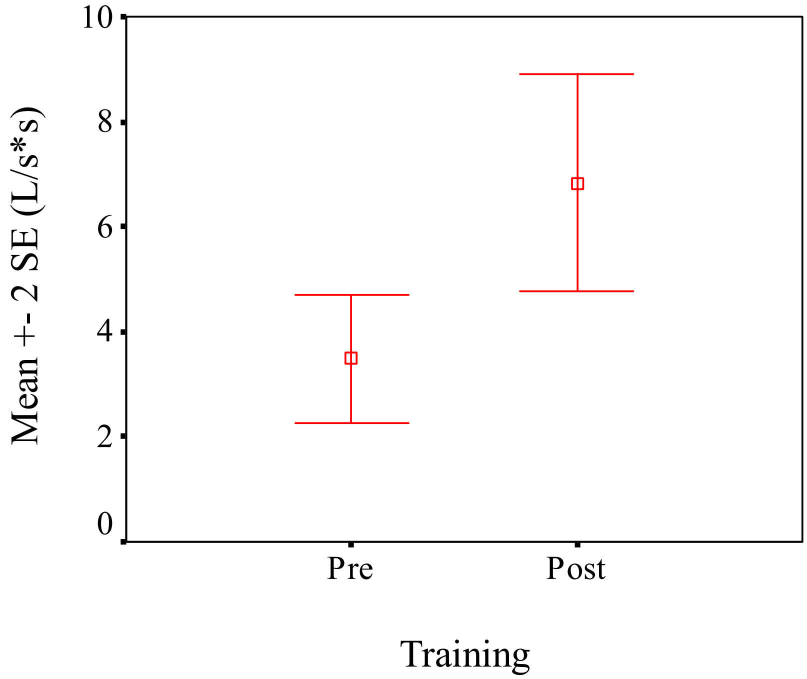


Figure 3-4. Effects of training on PPPIA.

Swallow Function

Table 3-10 shows the descriptive statistics for pre- and post-training values of all of the swallow function measures as a function of gender.

A 5 × 2 × 2 MANOVA could not be conducted since the small sample size ($N = 18$) and unequal sample sizes in gender were used, resulting in a reduction of power. Instead, 5 × 2 × 2 repeated measures univariate ANOVAs were examined to determine the effects of training, consistency, and gender on PA, DUR, and IA of submental muscle group activity. Sphericity assumptions, presented in Table 3-11, were significantly violated by the interaction between training and consistency on IA (Mauchly's W (9) = 0.066, $p <$ 0.001), by consistency on PA (Mauchly's W (9) = 0.025, $p < 0.001$), and by consistency on IA (Mauchly's W (9) = 0.121, $p < 0.001$). Therefore, Greenhouse-Geisser correction for the violation of sphericity assumption was applied. Sphericity assumptions, however, were met by training on all dependent variables, consistency on DUR, interaction between training and consistency on PA and DUR. The results of the ANOVA indicated no significant three-way interaction among the three factors on PA, DUR, and IA (Table 3-15). No significant two-way interactions between training and gender on PA, DUR, and IA, between consistency and gender on PA, on DUR, and on IA, and between training and consistency on any of swallow dependent variables were found. Accordingly, the main effects of training, consistency, and gender on PA, DUR, and IA were assessed for further evaluation (Table 3-12). Training significantly increased IA (F (1, 16) = 6.744, p = 0.019, η^2 = 0.297), but did not change PA and DUR. Consistency also significantly increased IA (F (2.368, 37.884) = 4.143, p = 0.018, η^2 = 0.206), but did not change both PA and DUR. No significant effects of gender were found on any of the swallow dependent variables. Therefore, 2 × 5 repeated measures univariate ANOVAs were conducted after excluding the gender effect to verify the effects of training and consistency on PA, DUR, and IA. Again, sphericity assumptions, presented in Table 3-13, were checked and were also violated by the interaction between training and consistency on IA (Mauchly's W (9) = 0.069, p < 0.001), by consistency on PA (Mauchly's W (9) = 0.029, p < 0.001), and by consistency on IA (Mauchly's W (9) = 0.121, p < 0.001). Greenhouse-Geisser corrections were applied for violations of these ANOVA assumptions of sphericity. Sphericity assumptions, however, were met by training on all swallow dependent variables, consistency on DUR, the interaction between training and consistency on PA and DUR.

Table 3-14 shows the univariate ANOVA results without the gender effect. For PA, a significant two-way interaction between training and consistency was observed (F (4, 68) = 3.122, p = 0.020, η^2 = 0.155). Thus, simple main effect tests using paired-samples *t*-tests were submitted to further explore the effects of training and consistency on PA at $\alpha = 0.01$ (Table 3-15). The results of these tests indicated that the PAs between any of two different consistency pairs in pre-training were not significantly different from each other. However, two different consistency pairs in post-training had significantly different PAs. Specifically, the PA measured for 10 cc pudding swallow in post-training was significantly higher than the 5 cc water swallow in post-training ($t = -3.388$, $p = 0.003$) and also significantly higher than the 10 cc water swallow in post-training ($t = -3.319$, $p = 0.004$). Additionally, one pair having different consistencies in different training levels had significantly different PAs. The PA for 10 cc pudding swallow in post-training was significantly greater than the 10 cc water swallow in pre-training ($t = -3.041$, $p = 0.007$). Furthermore, the profile plots were created by consistency as the horizontal axis, each pre- and post-training as the separate bar in PA (Figure 3-5). The plots illustrated the significantly different amount for the increases in PA from pre- to post-training in five different consistencies. Specifically, the PAs for maximal voluntary dry swallow (17% change), the 5 cc pudding swallow (16% change), and the 10 cc pudding swallow (18% change) had larger increases than the 5 cc water swallow (5% change) and the 10 cc water swallow (3% change) from pre- to post-training.

The results of univariate ANOVA for DUR indicated no significant two-way interaction between training and consistency (Table 3-14). The main effect of training on DUR was significant (F (1, 17) = 4.966, $p = 0.040$, $\eta^2 = 0.226$) and the main effect of consistency on DUR was also significant (F (4, 68) = 3.869, $p = 0.007$, $\eta^2 = 0.185$). Thus, multiple pairwise comparisons using the Bonferroni adjustment were completed to examine the effects of training and consistency on DUR (Table 3-16).

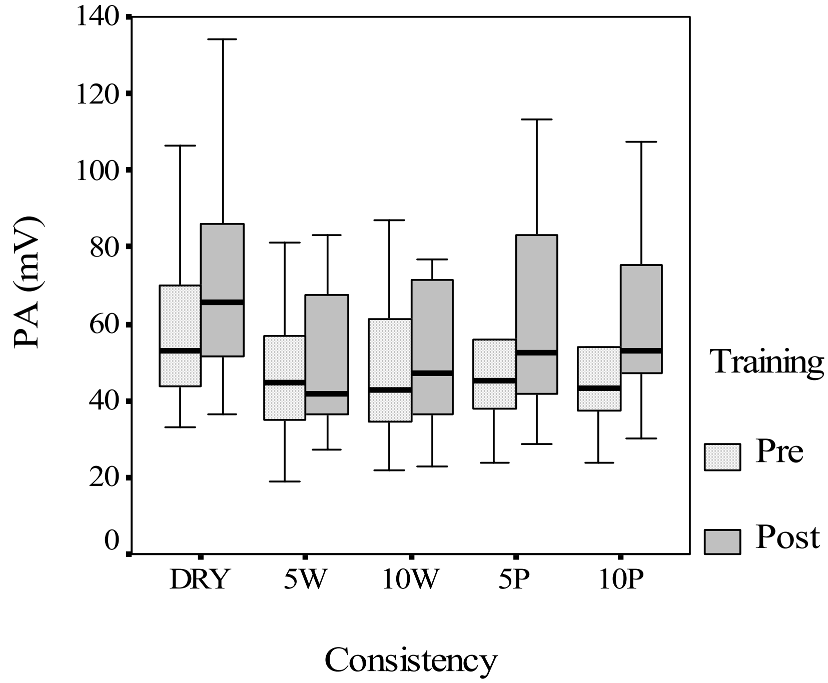


Figure 3-5. Effects of training and consistency on PA.

The training effect on DUR is illustrated in Figure 3-6 and the consistency effect on DUR is in Figure 3-7. Those indicated that post-training (M = 1.016, SE = 0.032) had significantly longer DUR than pre-training (M = 0.967, SE = 0.026; p = 0.040). However, there was no significant training effect on DUR in each consistency. Ten cc pudding swallow (M = 1.039, SE = 0.036) had significantly longer DUR than 5 cc water swallow (M = 0.948, SE = 0.029; p = 0.016) and 10 cc water swallow (M = 0.958, SE = 0.030; p = 0.012).

Examining the univariate ANOVA results without the gender effect on IA, also presented in Table 3-14, indicated a significant two-way interaction between training and consistency (F (1.987, 33.778) = 3.396, p = 0.046, η^2 = 0.167). Thus, simple main effect tests using paired-samples t-tests were evaluated to examine the effects of training and consistency on IA at α = 0.01.

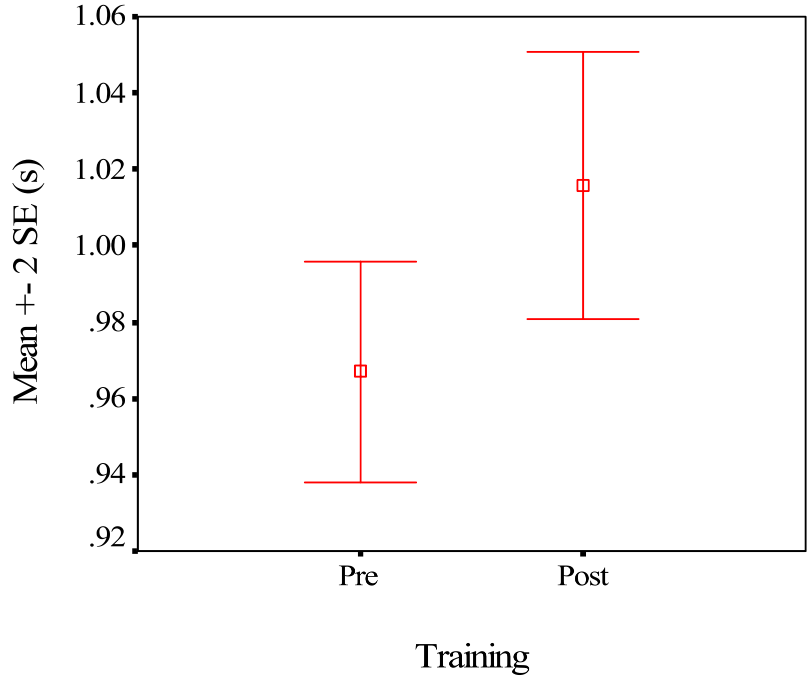


Figure 3-6. Effects of training on DUR.

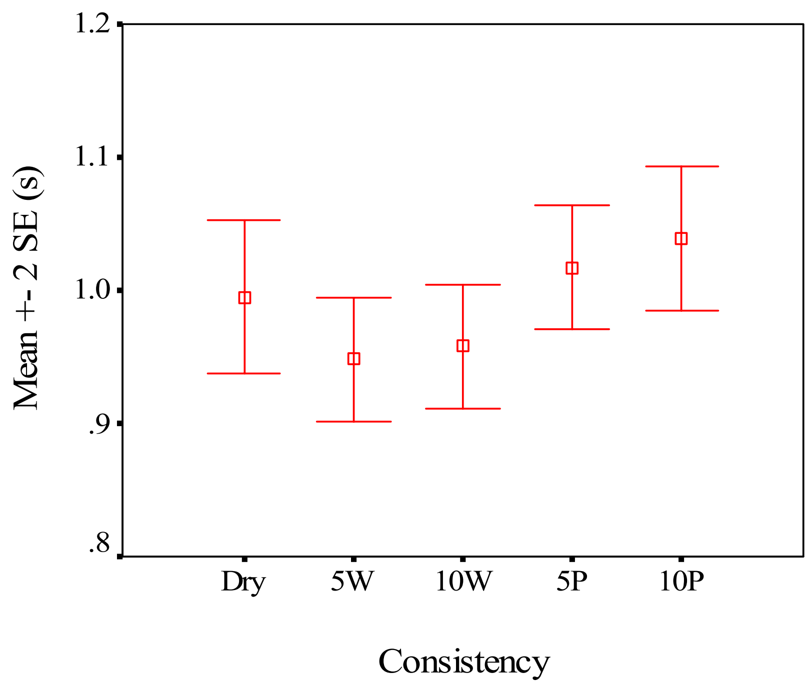


Figure 3-7. Effects of consistency on DUR.

Table 3-17 demonstrates the results of these tests and indicates that only one different consistency pair in pre-training had significantly different IAs. The IA for the 5 cc pudding was significantly higher than the 5 cc water (t = -2.811, p = 0.012). Five different consistency pairs in different training levels had significantly different IAs. The IA for 10 cc pudding swallow in post-training was significantly higher than the 5 cc water swallow in pre-training (t = -3.570, p = 0.002), than the 10 cc water swallow in pre-training (t = -3.610, p = 0.002), and than the 10 cc pudding swallow in pre-training (t = -2.867, p = 0.011). The IA for maximum voluntary dry swallow in post-training was also significantly higher than the 5 cc water swallow in pre-training (t = -2.800, p = 0.012) and the 10 cc water swallow in pre-training (t = -2.725, p = 0.014). Additionally, four different consistency pairs in post-training had significantly different IAs. Specifically, the IA for 5 cc water swallow in post-training was significantly lower than the dry swallow in post-training (t = 3.291, p = 0.004), than the 5 cc pudding swallow in post-training (t = -2.974, p = 0.009), and than the 10 cc pudding swallow in post-training (t = -3.485, p = 0.003). The IAs for 10 cc water swallow in post-training was also significantly lower than the 10 cc pudding swallow (t = -3.041, p = 0.007).

The profile plots were created with consistency as the horizontal axis, each pre- and post-training as the separate bars in IA (Figure 3-8). The plots depicted the significantly different amounts of IA increases from pre-training to post-training in different consistencies. The IAs for maximal voluntary dry swallow (16% change) and the 10 cc pudding swallow (33% change) dramatically increased from pre- to post-training. However, the IAs for 5 cc water swallow (6% change), the 10 cc water swallow (12% change), and the 5 cc pudding swallow (9% change) increased with small amounts from pre- to post-training.

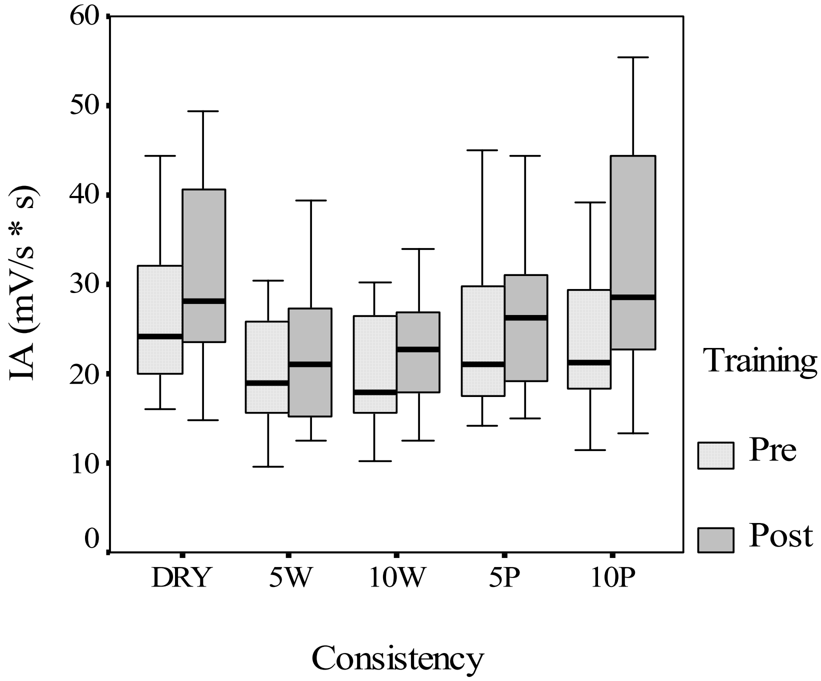


Figure 3-8. Effects of training and consistency on IA.

Speech Function

Table 3-18 shows the descriptive statistics for the speech function measures pre- and post-training as a function of gender.

The ANOVA result on P_{EL}, presented Table 3-19, indicated a significant two-way interaction between training and gender (F (1, 16) = 5.866, p = 0.028, η^2 = 0.268). However, no main effect of gender was found. Therefore, a one-way repeated measures ANOVA without gender effect on P_{EL} was further examined. Again, sphericity assumptions were met on P_{EL}. The ANOVA result indicated P_{EL} was significantly increased by training (F (1, 17) = 24.031, p < 0.001, η^2 = 0.586). The result for P_{EL} is illustrated in Figure 3-9. P_{EL} increased from pre- to post-training by 45%.

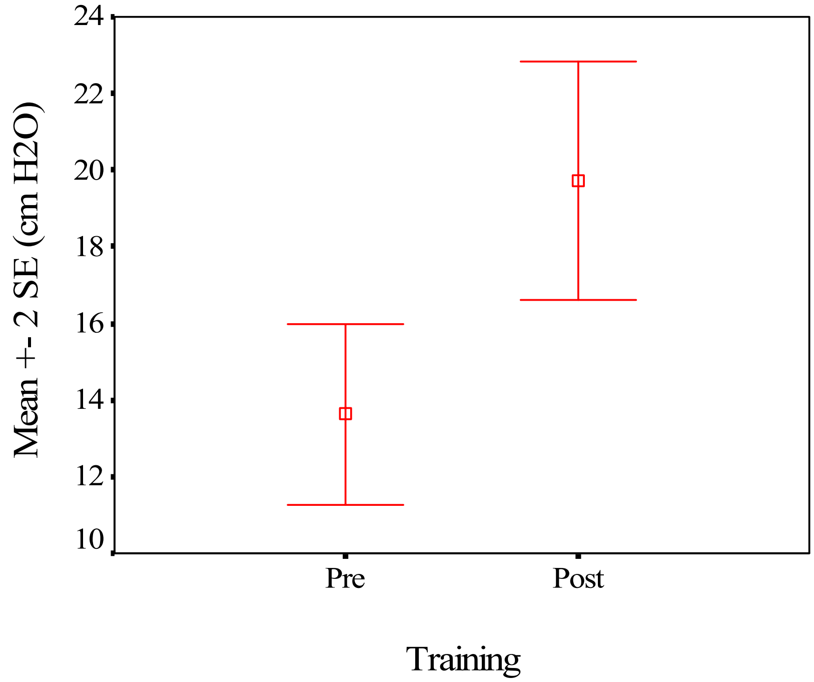


Figure 3-9. Effect of training on P_{EL}.

For MPD, a 2 × 2 × 2 repeated measures univariate ANOVA was examined to analyze the effects of training, loudness, and gender. For P_{EL}, a 2 × 2 repeated measures univariate ANOVA was conducted to determine the effects of training and gender. Sphericity assumptions were met by all factors in those ANOVAs. The ANOVA results on MPD indicated no significant three-way interaction among training, loudness, and gender (Table 3-20). Non-significance of two-way interactions between training and gender, between loudness and gender, or between training and loudness were found. Further, all main effects of training, loudness, and gender were not significant. Thus, a 2 × 2 repeated measures univariate ANOVA without the gender effect on MPD were evaluated. Sphericity assumptions of MPD were not violated. Table 3-21 shows the ANOVA results on MPD without gender which indicates a significant two-way interaction between training and loudness (F (1, 17) = 10.431, p = 0.005, η^2 = 0.380).

Simple main effect tests using paired-samples *t*-tests were then followed (Table 3-22). The MPDs at the comfortable intensity level between pre- and post-trainings were significantly different at the $\alpha = 0.01$ ($t = -3.070$, $p = 0.007$). The profile plots created by pre- and post-training as the horizontal axis, two intensity levels as the separate bars in MPD show a large increase of MPD for the comfortable intensity level from pre- to post-training by 26% compared to the small change for the loudest intensity level from pre- to post-training by 3% (Figure 3-10).

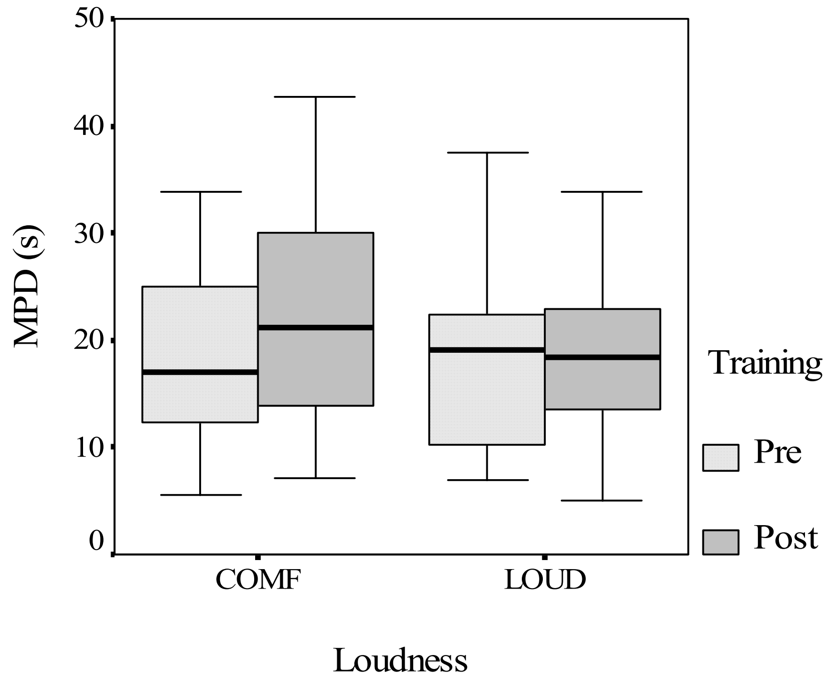


Figure 3-10. Effects of training and loudness on MPD.

Table 3-1. Results of intra- and inter-judge reliability of cough, swallow, and speech function variables.

Measures	Intra-Judge		Inter-Judge	
	r	*p*	*r*	*p*
Cough				
IPD	0.962	< 0.001*	0.939	0.005*
CPD	0.992	< 0.001*	0.957	0.003*
PEFR	1.000	< 0.001*	0.999	< 0.001*
PPPD	0.939	< 0.001*	0.882	0.020
PPPIA	0.974	< 0.001*	0.996	< 0.001*
Swallow				
PA	0.992	< 0.001*	0.983	< 0.001*
DUR	0.905	< 0.001*	0.743	< 0.001*
IA	0.910	< 0.001*	0.987	< 0.001*
Speech				
MPD	0.998	< 0.001*	0.974	< 0.001*
P_{EL}	1.000	< 0.001*	1.000	< 0.001*

* Correlation is significant at the 0.05 level.

Table 3-2. Correlation matrix of dependent variables.

	Pulmonary					Cough					Speech		
	MIP	FEV_1	FVC	FEV_1/FVC	ERV	IPD	CPD	PEFR	PPPD	PPPIA	COMF	LOUD	P_{EL}
Pulmonary													
MEP	0.532*	0.232	0.215	0.082	0.194	-0.115	-0.367*	0.526*	-0.305	0.472*	0.312	0.139	0.363*
MIP		0.257	0.161	0.275	0.087	0.090	-0.194	0.350*	-0.229	0.105	0.233	0.043	0.183
FEV_1			0.952	0.151	0.554*	-0.088	-0.245	0.465*	0.065	0.391*	0.577*	0.543*	0.018
FVC				-0.140	0.635*	-0.225	-0.303	0.420*	0.097	0.409*	0.464*	0.443*	0.054
FEV_1/FVC					-0.198	0.456*	0.179	0.194	-0.123	0.056	0.384	0.326	0.056
ERV						0.045	-0.117	0.245	0.067	0.378*	0.403*	0.294	0.004
Cough													
IPD							0.125	0.034	0.059	-0.011	0.361*	0.159	-0.195
CPD								-0.625*	0.023	-0.498*	-0.005	0.023	-0.301
PEFR									-0.319	0.847*	0.492*	0.384*	0.402*
PPPD										-0.136	-0.128	-0.250	0.038
PPPIA											0.462*	0.264	0.464*
Swallow - PA													
DRY											-0.089	0.198	-0.119
5W											0.256	0.651*	-0.314
10W											0.200	0.587*	-0.280
5P											0.028	0.346*	-0.282
10P											0.142	0.489*	-0.301
Swallow - DUR													
DRY											0.169	-0.024	0.554*
5W											-0.039	-0.071	0.254
10W											0.125	0.056	0.259
5P											0.177	0.112	0.154
10P											0.262	0.128	0.307
Swallow - IA													
DRY											0.120	0.110	0.256
5W											0.010	0.073	0.177
10W											0.026	0.039	0.154
5P											0.032	-0.014	0.107
10P											-0.061	-0.059	0.339*
Speech													
COMF												0.703*	-0.006
LOUD													-0.194

Table 3-2. Continued.

	Swallow - PA					Swallow - DUR					Swallow - IA				
	DRY	5W	10W	5P	10P	DRY	5W	10W	5P	10P	DRY	5W	10W	5P	10P
Pulmonary															
MEP	-0.034	0.075	0.096	-0.018	-0.024	0.139	0.072	0.053	0.016	0.084	0.257	0.026	0.082	0.020	0.084
MIP	-0.136	0.022	0.006	-0.089	-0.024	-0.015	0.103	0.185	0.196	0.093	0.268	0.012	0.153	0.097	0.219
FEV_1	-0.179	0.244	0.192	-0.036	0.121	0.306	0.036	0.269	0.154	0.188	-0.158	-0.393*	-0.402*	-0.407*	-0.310
FVC	-0.235	0.197	0.172	-0.056	0.082	0.342*	0.082	0.248	0.110	0.213	-0.271	-0.465*	-0.493*	-0.496*	-0.413*
FEV_1/FVC	0.139	0.108	0.028	0.019	0.080	0.053	-0.007	0.200	0.236	0.078	0.401	0.315	0.360	0.361	0.379
ERV	-0.272	0.010	0.022	-0.143	-0.022	0.274	0.116	0.184	0.032	0.235	-0.345*	-0.285	-0.279	-0.335*	-0.247
Cough															
IPD	0.123	0.107	0.073	0.096	0.153	-0.065	-0.054	0.048	0.048	0.009	0.265*	0.375*	0.435*	0.390*	0.386*
CPD	-0.216	-0.085	-0.082	-0.129	-0.090	-0.196	0.062	0.125	0.066	0.246	0.085	0.256	0.261	0.403*	0.129
PEFR	0.132	0.216	0.198	0.074	0.150	0.420*	-0.001	0.023	0.069	0.039	0.286	-0.084	-0.077	-0.167	0.069
PPPD	-0.089	-0.351*	-0.353*	-0.288	-0.218	-0.022	0.173	0.191	0.154	0.233	-0.310	-0.320	-0.288	-0.279	-0.248
PPPIA	0.171	0.159	0.153	0.091	0.128	0.541*	0.148	0.117	0.124	0.223	0.167	-0.043	-0.067	-0.116	0.039
Swallow - PA															
DRY		0.643*	0.620*	0.844*	0.803*	-0.172	-0.010	-0.089	-0.133	-0.196	0.360*	0.386*	0.346*	0.300	0.314
5W			0.970*	0.862*	0.915*	-0.213	-0.030	-0.036	-0.077	-0.095	0.197	0.178	0.125	0.104	0.032
10W				0.850*	0.909*	-0.263	0.017	-0.026	-0.163	-0.106	0.150	0.161	0.122	0.055	0.034
5P					0.940*	-0.249	-0.018	-0.169	-0.192	-0.199	0.150	0.241	0.184	0.177	0.107
10P						-0.242	0.048	-0.005	-0.122	-0.102	0.206	0.225	0.188	0.163	0.126
Swallow - DUR															
DRY							0.428*	0.441*	0.531*	0.530*	0.180	0.058	-0.037	0.052	0.027
5W								0.749*	0.594*	0.692*	-0.041	0.111	0.104	0.127	0.024
10W									0.632*	0.754*	0.200	0.149	0.152	0.245	0.177
5P										0.744*	0.163	0.031	-0.002	0.226	-0.061
10P											0.170	0.049	0.024	0.243	0.022
Swallow - IA															
DRY												0.609*	0.625*	0.733*	0.656*
5W													0.971*	0.884*	0.765*
10W														0.871*	0.808*
5P															0.766*

Note: All abbreviations are listed in Appendix G.
* Correlation is significant at the 0.05 level.

Table 3-3. Descriptive statistics for pre- and post-training on pulmonary function variables.

DV	Gender	Pre		Post		Change (%)
		M	*SD*	*M*	*SD*	
MEP (cm H_2O)	Men	76.503	23.623	132.285	21.491	72.915
	Women	77.316	20.115	104.695	24.548	35.412
	Average	77.136	20.199	110.826	26.108	43.676
MIP (cm H_2O)	Men	42.130	10.915	62.790	17.361	49.039
	Women	38.206	13.998	57.176	19.515	49.652
	Average	39.078	13.179	58.423	18.713	49.504
FEV_1 (L)	Men	2.079	0.611	2.213	0.687	6.445
	Women	1.906	0.468	1.974	0.477	3.568
	Average	1.945	0.488	2.027	0.517	4.216
FVC (L)	Men	2.863	0.672	2.928	0.858	2.270
	Women	2.467	0.624	2.557	0.644	3.648
	Average	2.555	0.638	2.639	0.687	3.288
FEV_1/FVC	Men	71.742	6.578	76.947	6.312	7.255
	Women	78.078	6.449	78.076	5.721	-0.002
	Average	76.670	6.840	77.826	5.683	1.508
ERV (L)	Men	1.089	0.249	1.190	0.424	9.275
	Women	0.938	0.687	1.171	0.670	24.840
	Average	0.972	0.613	1.176	0.613	20.988

Table 3-4. MANOVA result for the effects of training and gender on pulmonary function variables.

Factor	Statistic	Value	*F*	Hypothesis df	Error df	*p*	η^2
Intercept	Pillai's Trace	1.000	4726.314	6	11	0.000	1.000
Gender	Pillai's Trace	0.259	0.640	6	11	0.698	0.259
Training	Pillai's Trace	0.827	8.766	6	11	0.001*	0.827
Training × Gender	Pillai's Trace	0.372	1.086	6	11	0.427	0.372

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-5. Univariate ANOVA results for training effect on pulmonary function variables.

Factor	DV	SS	df	MS	F	p	η^2
Training	MEP	10215.482	1	10215.482	40.978	0.000*	0.707
	MIP	3368.061	1	3368.061	18.513	0.000*	0.521
	FEV_1	0.062	1	0.062	3.556	0.077	0.173
	FVC	0.064	1	0.064	3.806	0.068	0.183
	FEV_1/FVC	12.020	1	12.020	0.873	0.363	0.049
	ERV	0.374	1	0.374	1.853	0.191	0.098
Error	MEP	4238.002	17	249.294			
	MIP	3092.882	17	181.934			
	FEV_1	0.294	17	0.017			
	FVC	0.285	17	0.017			
	FEV_1/FVC	234.145	17	13.773			
	ERV	3.430	17	0.202			

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-6. Descriptive statistics for pre- and post-training on cough function variables.

DV	Gender	Pre *M*	Pre *SD*	Post *M*	Post *SD*	Change (%)
IPD (s)	Men	0.932	0.224	0.758	0.350	-18.669
	Women	1.206	0.243	1.357	0.495	12.521
	Average	1.145	0.262	1.224	0.524	6.899
CPD (s)	Men	0.229	0.097	0.122	0.068	-46.689
	Women	0.382	0.201	0.173	0.185	-54.563
	Average	0.348	0.192	0.162	0.166	-53.412
PEFR (L/s)	Men	4.985	2.810	6.483	2.969	30.155
	Women	4.979	2.097	8.431	3.056	69.127
	Average	4.981	2.181	7.998	3.064	60.635
PPPD (s)	Men	0.206	0.028	0.213	0.041	3.538
	Women	0.244	0.078	0.227	0.084	-7.066
	Average	0.236	0.071	0.224	0.075	-5.008
PPPIA (L/s×s)	Men	3.308	4.210	4.043	3.746	22.210
	Women	3.539	1.948	7.624	4.033	115.411
	Average	3.488	2.457	6.829	4.155	95.767
N of coughs	Men	2.700	0.983	2.650	1.025	-1.852
	Women	2.879	0.720	3.400	1.109	18.114
	Average	2.839	0.757	3.233	1.109	13.894
N of CR	Men	6.850	2.965	6.700	3.118	-2.190
	Women	8.121	2.622	9.729	4.229	19.789
	Average	7.839	2.665	9.056	4.132	15.521

Table 3-7. MANOVA result for the effects of training and gender on cough function variables.

Factor	Statistic	Value	*F*	Hypothesis df	Error df	*p*	η^2
Intercept	Pillai's Trace	0.982	129.655	5	12	0.000	0.982
Gender	Pillai's Trace	0.451	1.968	5	12	0.156	0.451
Training	Pillai's Trace	0.520	2.598	5	12	0.081	0.520
Training × Gender	Pillai's Trace	0.373	1.427	5	12	0.283	0.373

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-8. Univariate ANOVA results for training effects on cough function variables.

Factor	DV	SS	df	MS	*F*	*p*	η^2
Training	IPD	0.056	1	0.056	0.664	0.426	0.038
	CPD	0.310	1	0.310	13.590	0.002*	0.444
	PEFR	4609.588	1	4609.588	29.620	0.000*	0.635
	PPPD	0.001	1	0.001	1.259	0.277	0.069
	PPPIA	100.427	1	100.427	16.826	0.001*	0.497
Error	IPD	1.429	17	0.084			
	CPD	0.388	17	0.023			
	PEFR	2645.640	17	155.626			
	PPPD	0.017	17	0.001			
	PPPIA	101.465	17	5.969			

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-9. MANOVA result for the effects of training and gender on total number of coughs and total number of expulsive events.

Factor	Statistic	Value	*F*	Hypothesis df	Error df	*p*	η^2
Intercept	Pillai's Trace	0.915	80.710	2.000	15.000	0.000	0.915
Gender	Pillai's Trace	0.081	0.661	2.000	15.000	0.531	0.081
Training	Pillai's Trace	0.068	0.548	2.000	15.000	0.589	0.068
Training × Gender	Pillai's Trace	0.096	0.794	2.000	15.000	0.470	0.096

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-10. Descriptive statistics for pre- and post-training on swallow function variables.

DV		Gender	Pre *M*	Pre *SD*	Post *M*	Post *SD*	Change (%)
PA (mV)	DRY	Men	43.566	2.603	52.510	10.196	20.528
		Women	65.527	22.897	76.102	25.812	16.139
		Average	60.647	22.144	70.860	25.094	16.840
	5W	Men	34.993	5.715	33.841	5.293	-3.293
		Women	57.704	37.814	61.352	31.135	6.322
		Average	52.658	34.548	55.239	29.745	4.902
	10W	Men	36.030	6.266	32.799	4.509	-8.967
		Women	57.103	35.430	60.256	30.509	5.522
		Average	52.420	32.374	54.154	29.212	3.309
	5P	Men	39.736	6.731	41.968	10.016	5.616
		Women	57.829	30.586	68.533	29.898	18.509
		Average	53.809	27.987	62.630	28.817	16.393
	10P	Men	36.339	3.976	37.767	7.109	3.930
		Women	60.888	34.138	73.304	30.197	20.392
		Average	55.432	31.691	65.407	30.616	17.994
DUR (s)	DRY	Men	0.946	0.116	1.080	0.122	14.127
		Women	0.964	0.151	1.016	0.217	5.310
		Average	0.960	0.141	1.030	0.199	7.240
	5W	Men	0.892	0.208	0.947	0.209	6.192
		Women	0.935	0.120	0.977	0.125	4.494
		Average	0.926	0.138	0.971	0.141	4.858
	10W	Men	0.935	0.146	0.988	0.178	5.627
		Women	0.945	0.151	0.968	0.129	2.424
		Average	0.943	0.145	0.972	0.136	3.130
	5P	Men	0.955	0.161	1.019	0.146	6.707
		Women	1.004	0.112	1.047	0.164	4.284
		Average	0.993	0.121	1.041	0.156	4.802
	10P	Men	0.976	0.093	1.010	0.135	3.522
		Women	1.024	0.149	1.081	0.196	5.590
		Average	1.013	0.137	1.065	0.183	5.147
IA	DRY	Men	18.724	1.787	30.241	11.982	61.509
		Women	28.905	8.145	31.125	10.223	7.680
		Average	26.643	8.382	30.929	10.267	16.086
	5W	Men	16.388	0.641	19.299	3.925	17.765
		Women	23.521	13.824	24.345	11.962	3.503
		Average	21.936	12.471	23.223	10.807	5.871
	10W	Men	16.698	0.772	20.457	4.925	22.513
		Women	23.855	12.758	26.143	11.850	9.592
		Average	22.265	11.574	24.880	10.844	11.746
	5P	Men	19.825	2.872	23.975	2.748	20.930
		Women	26.668	12.490	28.555	12.808	7.075
		Average	25.147	11.372	27.537	11.428	9.502
	10P	Men	19.294	1.816	26.837	6.874	39.093
		Women	25.826	11.840	34.008	17.802	31.682
		Average	24.374	10.751	32.415	16.127	32.986

Table 3-11. Mauchly's test of sphericity for training, consistency, and gender effects on swallow function variables.

Factor	DV	Mauchly's *W*	χ^2	df	*p*
Training	PA	1.000	0.000	0	1.000
	DUR	1.000	0.000	0	1.000
	IA	1.000	0.000	0	1.000
Consistency	PA	0.025	53.437	9	0.000*
	DUR	0.389	13.602	9	0.140
	IA	0.121	30.486	9	0.000*
Training × Consistency	PA	0.616	6.990	9	0.640
	DUR	0.721	4.724	9	0.859
	IA	0.066	39.234	9	0.000*

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-12. Univariate ANOVA (mixed design) results for the combined effects of training, consistency, and gender on swallow function variables.

Factor	DV	SS	df	MS	*F*	*p*	η^2
Within-Subjects							
Training	PA	738.360	1	738.360	1.265	0.277	0.073
	DUR	0.096	1	0.096	4.260	0.056	0.210
	IA	637.877	1	637.877	6.744	0.019*	0.297
Training × Gender	PA	324.115	1	324.115	0.555	0.467	0.034
	DUR	0.005	1	0.005	0.210	0.653	0.013
	IA	65.220	1	65.220	0.690	0.419	0.041
Error (Training)	PA	9336.621	16	583.53			
	DUR	0.362	16	0.023			
	IA	1513.351	16	94.584			
Consistency	PA	2698.133	1.408	1915.649	2.792	0.097	0.149
	DUR	0.126	4	0.031	2.205	0.078	0.121
	IA	787.303	2.368	332.515	4.143	0.018*	0.206
Consistency × Gender	PA	236.542	4	59.136	0.245	0.912	0.015
	DUR	0.028	4	0.007	0.499	0.737	0.030
	IA	7.088	4	1.772	0.037	0.997	0.002
Error (Consistency)	PA	15460.024	22.536	686.030			
	DUR	0.912	64	0.014			
	IA	3040.167	37.884	80.250			
Training × Consistency	PA	422.414	4	105.603	2.041	0.099	0.113
	DUR	0.011	4	0.003	0.536	0.710	0.032
	IA	175.570	1.871	93.853	2.404	0.111	0.131
Training × Consistency × Gender	PA	78.743	4	19.686	0.380	0.822	0.023
	DUR	0.009	4	0.002	0.424	0.791	0.026
	IA	87.985	4	21.996	1.205	0.317	0.070
Error (Training × Consistency)	PA	3311.880	64	51.748			
	DUR	0.339	64	0.005			
	IA	1168.390	29.931	39.036			
Between-Subject							
Intercept	PA	328872.375	1	328872.375	53.101	0.000	
	DUR	120.846	1	120.846	869.716	0.000	
	IA	73087.519	1	73087.519	74.724	0.000	
Gender	PA	19296.632	1	19296.632	3.116	0.097	
	DUR	0.014	1	0.014	0.102	0.754	
	IA	1165.650	1	1165.650	1.192	0.291	
Error	PA	99094.255	16	6193.391			
	DUR	2.223	16	0.139			
	IA	15649.637	16	978.102			

Note: η^2 = effect size.

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-13. Mauchly's test of sphericity for training and consistency on swallow function variables.

Factor	DV	Mauchly's *W*	χ^2	df	*p*
Training	PA	1.000	0.000	0	1.000
	DUR	1.000	0.000	0	1.000
	IA	1.000	0.000	0	1.000
Consistency	PA	0.029	54.371	9	0.000*
	DUR	0.402	14.055	9	0.122
	IA	0.121	32.622	9	0.000*
Training × Consistency	PA	0.628	7.183	9	0.620
	DUR	0.744	4.552	9	0.872
	IA	0.069	41.306	9	0.000*

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-14. Univariate ANOVA results without gender effect for the combined effects of training and consistency on swallow function variables.

Factor	DV	SS	df	MS	*F*	*p*	η^2
Training	PA	1998.888	1	1998.888	3.517	0.078	0.171
	DUR	0.107	1	0.107	4.966	0.040*	0.226
	IA	623.956	1	623.956	6.720	0.019*	0.283
Error (Training)	PA	9660.735	17	568.279			
	DUR	0.366	17	0.022			
	IA	1578.571	17	92.857			
Consistency	PA	3748.024	1.441	2600.175	4.059	0.042*	0.193
	DUR	0.214	4	0.053	3.869	0.007*	0.185
	IA	1122.603	2.367	474.187	6.263	0.003*	0.269
Error (Consistency)	PA	15696.566	24.505	640.554			
	DUR	0.940	68	0.014			
	IA	3047.256	40.246	75.715			
Training × Consistency	PA	622.583	4	155.646	3.122	0.020*	0.155
	DUR	0.007	4	0.002	0.365	0.833	0.021
	IA	251.011	1.987	126.329	3.396	0.046*	0.167
Error (Training × Consistency)	PA	3390.623	68	49.862			
	DUR	0.348	68	0.005			
	IA	1256.374	33.778	37.195			

Note: η^2 = effect size.

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-15. Simple main effect tests of training and consistency on PA.

Factor (I)	Factor (J)	M (I)	M (J)	(I-J)	SE	*t*	*p*
Pre, DRY	Pre, 5W	60.647	52.658	7.989	5.829	1.371	0.188
	Pre, 10W		52.420	8.227	5.203	1.581	0.132
	Pre, 5P		53.809	6.838	3.544	1.929	0.071
	Pre, 10P		55.432	5.215	4.181	1.247	0.229
	Post, DRY	60.647	70.860	-10.213	4.537	-2.251	0.038
	Post, 5W		55.239	5.410	5.246	1.031	0.317
	Post, 10W		54.154	6.492	5.002	1.298	0.212
	Post, 5P		62.630	-1.983	4.608	-0.430	0.672
	Post, 10P		65.407	-4.760	4.775	-0.997	0.333
Pre, 5W	Pre, 10W	52.658	52.420	0.238	1.350	0.176	0.862
	Pre, 5P		53.809	-1.151	3.271	-0.352	0.729
	Pre, 10P		55.432	-2.776	2.959	-0.938	0.362
	Post, DRY	52.658	70.860	-18.202	8.012	-2.272	0.036
	Post, 5W		55.239	-2.581	3.400	-0.759	0.458
	Post, 10W		54.154	-1.497	2.707	-0.553	0.587
	Post, 5P		62.630	-9.972	6.365	-1.567	0.136
	Post, 10P		65.407	-12.749	4.934	-2.584	0.019
Pre, 10W	Pre, 5P	52.420	53.809	-1.389	2.842	-0.489	0.631
	Pre, 10P		55.432	-3.012	2.487	-1.211	0.242
	Post, DRY	52.420	70.860	-18.440	7.410	-2.488	0.023
	Post, 5W		55.239	-2.819	3.055	-0.923	0.369
	Post, 10W		54.154	-1.735	2.487	-0.697	0.495
	Post, 5P		62.630	-10.210	5.594	-1.825	0.086
	Post, 10P		65.407	-12.987	4.271	-3.041	0.007*
Pre, 5P	Pre, 10P	53.809	55.432	-1.623	1.691	-0.960	0.350
	Post, DRY	53.809	70.860	-17.051	6.306	-2.704	0.015
	Post, 5W		55.239	-1.430	3.830	-0.373	0.714
	Post, 10W		54.154	-0.345	3.491	-0.099	0.922
	Post, 5P		62.630	-8.821	5.079	-1.737	0.100
	Post, 10P		65.407	-11.598	4.469	-2.595	0.019
Pre, 10P	Post, DRY	55.432	70.860	-15.427	7.075	-2.181	0.044
	Post, 5W		55.239	0.193	3.900	0.050	0.961
	Post, 10W		54.154	1.278	3.458	0.370	0.716
	Post, 5P		62.630	-7.198	5.635	-1.277	0.219
	Post, 10P		65.407	-9.974	4.602	-2.167	0.045
Post, DRY	Post, 5W	70.860	55.239	15.621	5.792	2.697	0.015
	Post, 10W		54.154	16.705	6.233	2.680	0.016
	Post, 5P		62.630	8.230	3.727	2.208	0.041
	Post, 10P		65.407	5.453	4.699	1.161	0.262
Post, 5W	Post, 10W	55.239	54.154	1.084	2.251	0.482	0.636
	Post, 5P		62.630	-7.391	4.236	-1.745	0.099
	Post, 10P		65.407	-10.168	3.001	-3.388	0.003*
Post, 10W	Post, 5P	54.154	62.630	-8.475	4.523	-1.874	0.078
	Post, 10P		65.407	-11.252	3.390	-3.319	0.004*
Post, 5P	Post, 10P	62.630	65.407	-2.777	3.171	-0.876	0.393

* indicates that the mean difference is significant at $\alpha = 0.01$.

Table 3-16. Multiple pairwise comparisons for DUR by training and by consistency.

Factor		*M*		(I-J)	SE	*p*
(I)	(J)	(I)	(J)			
Pre, DRY	Post, DRY	0.960	1.030	-0.070	-1.870	0.079
Pre, 5W	Post, 5W	0.926	0.971	-0.045	-1.422	0.173
Pre, 10W	Post, 10W	0.943	0.972	-0.030	-1.118	0.279
Pre, 5P	Post, 5P	0.993	1.041	-0.048	-1.634	0.121
Pre, 10P	Post, 10P	1.013	1.065	-0.052	-1.923	0.071
DRY	5W	0.995	0.948	0.047	0.037	1.000
	10W		0.958	0.037	0.035	1.000
	5P		1.017	-0.022	0.031	1.000
	10P		1.039	-0.044	0.035	1.000
5W	10W	0.948	0.958	-0.010	0.020	1.000
	5P		1.017	-0.069	0.025	0.135
	10P		1.039	-0.091	0.024	0.016*
10W	5P	0.958	1.017	-0.059	0.022	0.139
	10P		1.039	-0.081	0.021	0.012*
5P	10P	1.017	1.039	-0.022	0.019	1.000

* indicates that the mean difference is significant at $\alpha = 0.05$ using Bonferroni adjustment for multiple comparisons.

Table 3-17. Simple main effect tests for the effects of training and consistency on IA.

Factor		*M*		(I-J)	SE	*t*	*p*
(I)	(J)	(I)	(J)				
Pre, Dry	Pre, 5W	26.643	21.936	4.707	2.121	2.219	0.040
	Pre, 10W		22.265	4.378	2.043	2.143	0.047
	Pre, 5P		25.147	1.496	1.618	0.924	0.368
	Pre, 10P		24.374	2.268	1.679	1.351	0.194
	Post, Dry	26.643	30.929	-4.286	2.111	-2.030	0.058
	Post, 5W		23.223	3.419	1.799	1.901	0.074
	Post, 10W		24.880	1.763	1.550	1.138	0.271
	Post, 5P		27.537	-0.894	2.000	-0.447	0.661
	Post, 10P		32.415	-5.772	3.005	-1.921	0.072
Pre, 5W	Pre, 10W	21.936	22.265	-0.329	0.651	-0.506	0.620
	Pre, 5P		25.147	-3.212	1.143	-2.811	0.012*
	Pre, 10P		24.374	-2.439	1.099	-2.220	0.040
	Post, Dry	21.936	30.929	-8.993	3.212	-2.800	0.012*
	Post, 5W		23.223	-1.288	1.169	-1.102	0.286
	Post, 10W		24.880	-2.944	1.302	-2.262	0.037
	Post, 5P		27.537	-5.601	2.382	-2.352	0.031
	Post, 10P		32.415	-10.479	2.935	-3.570	0.002*
Pre, 10W	Pre, 5P	22.265	25.147	-2.883	1.210	-2.381	0.029
	Pre, 10P		24.374	-2.110	0.943	-2.236	0.039
	Post, Dry	22.265	30.929	-8.664	3.180	-2.725	0.014*
	Post, 5W		23.223	-0.959	1.344	-0.713	0.486
	Post, 10W		24.880	-2.615	1.316	-1.987	0.063
	Post, 5P		27.537	-5.272	2.405	-2.193	0.043
	Post, 10P		32.415	-10.150	2.811	-3.610	0.002*
Pre, 5P	Pre, 10P	25.147	24.374	0.773	0.695	1.111	0.282
	Post, Dry	25.147	30.929	-5.781	2.780	-2.080	0.053
	Post, 5W		23.223	1.924	1.019	1.889	0.076
	Post, 10W		24.880	0.267	1.006	0.266	0.793
	Post, 5P		27.537	-2.390	1.763	-1.356	0.193
	Post, 10P		32.415	-7.267	3.063	-2.373	0.030
Pre, 10P	Post, Dry	24.374	30.929	-6.554	2.805	-2.337	0.032
	Post, 5W		23.223	1.151	1.043	1.103	0.285
	Post, 10W		24.880	-0.505	0.909	-0.556	0.586
	Post, 5P		27.537	-3.162	1.966	-1.609	0.126
	Post, 10P		32.415	-8.040	2.805	-2.867	0.011*
Post, Dry	Post, 5W	30.929	23.223	7.705	2.341	3.291	0.004*
	Post, 10W		24.880	6.049	2.263	2.673	0.016
	Post, 5P		27.537	3.392	2.053	1.652	0.117
	Post, 10P		32.415	-1.486	3.122	-0.476	0.640
Post, 5W	Post, 10W	23.223	24.880	-1.656	0.637	-2.601	0.019
	Post, 5P		27.537	-4.313	1.451	-2.974	0.009*
	Post, 10P		32.415	-9.191	2.638	-3.485	0.003*
Post, 10W	Post, 5P	24.880	27.537	-2.657	1.499	-1.772	0.094
	Post, 10P		32.415	-7.535	2.478	-3.041	0.007*
Post, 5P	Post, 10P	27.537	32.415	-4.878	2.831	-1.723	0.103

* indicates that the mean difference is significant at $\alpha = 0.01$.

Table 3-18. Descriptive statistics for pre- and post-training on speech function variables.

DV	Loudness	Gender	Pre		Post		Change (%)
			M	*SD*	*M*	*SD*	
P_{EL}		Men	11.902	3.303	22.983	5.774	93.102
		Women	14.131	5.423	18.814	6.730	33.140
		Average	13.636	5.002	19.741	6.610	44.771
MPD	COMF	Men	16.816	12.141	16.476	9.744	-2.023
		Women	18.052	6.866	24.048	9.813	33.216
		Average	17.777	7.895	22.366	10.044	25.809
	LOUD	Men	13.248	5.572	13.251	6.303	0.022
		Women	21.186	11.616	21.870	13.099	3.232
		Average	19.422	10.964	19.955	12.321	2.745

Table 3-19. Univariate ANOVA result for the combined effects of training and gender on P_{EL}.

Factor	SS	df	MS	*F*	*p*	η^2
Within-Subjects						
Training	386.541	1	386.541	35.619	0.000*	0.690
Training × Gender	63.654	1	63.654	5.866	0.028*	0.268
Error (Training)	173.634	16	10.852			
Between-Subject						
Intercept	7157.024	1	7157.024	123.795	0.000	0.886
Gender	5.851	1	5.851	0.101	0.755	0.006
Error (Gender)	925.013	16	57.813			

Note: η^2 = effect size.

* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-20. Univariate ANOVA result for the combined effects of training, loudness, and gender on MPD.

Factor	SS	df	MS	F	p	η^2
Within-Subjects						
Training	31.299	1	31.299	1.487	0.240	0.005
Training × Gender	38.308	1	38.308	1.820	0.196	0.102
Error (Training)	336.794	16	21.050			
Loudness	26.506	1	26.506	0.446	0.514	0.027
Loudness × Gender	46.711	1	46.711	0.785	0.389	0.047
Error (Loudness)	951.781	16	59.486			
Training × Loudness	19.200	1	19.200	3.209	0.092	0.167
Training × Loudness × Gender	24.870	1	24.870	4.157	0.058	0.206
Error (Training × Loudness)	95.724	16	5.983			
Between-Subject						
Intercept	16340.793	1	16340.793	48.378	0.000	0.751
Gender	500.476	1	500.476	1.482	0.241	0.085
Error (Gender)	5404.406	16	337.775			

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-21. Univariate ANOVA result for the combined effects of training and loudness on MPD.

Factor	SS	df	MS	F	p	η^2
Within-Subjects						
Training	118.024	1	118.024	5.349	0.034*	0.239
Error (Training)	375.102	17	22.065			
Loudness	2.642	1	2.642	0.045	0.835	0.003
Error (Loudness)	998.492	17	58.735			
Training × Loudness	73.994	1	73.994	10.431	0.005*	0.380
Error (Loudness)	120.593	17	7.094			

Note: η^2 = effect size.
* indicates that the mean difference is significant at $\alpha = 0.05$.

Table 3-22. Simple main effect tests of training and loudness on MPD.

Factor (I)	Factor (J)	*M* (I)	*M* (J)	(I-J)	SE	*t*	*p*
COMF, Pre	COMF, Post	17.777	22.366	-4.588	1.495	-3.070	0.007*
	LOUD, Pre		19.422	-1.644	1.991	-0.826	0.420
	LOUD, Post		19.955	-2.178	2.532	-0.860	0.402
COMF, Post	LOUD, Pre	22.366	19.422	2.944	1.601	1.838	0.084
	LOUD, Post		19.955	2.411	1.830	1.317	0.205
LOUD, Pre	LOUD, Post	19.422	19.955	-0.533	1.003	-0.532	0.602

* indicates that the mean difference is significant at $\alpha = 0.01$.

CHAPTER 4
DISCUSSION

This study investigated the physiological effects of expiratory muscle strength training (EMST) with the sedentary healthy elderly using a pressure-threshold training device over a 4-week time frame in order to assess the effects on pulmonary, cough, swallow, and speech functions.

Pulmonary Function

Maximum Respiratory Pressure. It was hypothesized that EMST would increase both maximum expiratory pressure (MEP) and maximum inspiratory pressure (MIP). The results indicated significant improvements in both MEP and MIP following the 4-week EMST program. MEPs significantly increased by an average of 44% (range of 8% to 158%) from pre- to post-training. Increases in MEP represent improved expiratory muscle strength. The MEP gains in the current study are comparable to previous studies completed in healthy young adults as well as clinical populations that used the same pressure-threshold training device (Baker, Davenport, & Sapienza, 2005; Chiara, 2003; Hoffman-Ruddy, 2001; Saleem, 2005; Sapienza et al., 2002; Wingate et al., in press). Table 1-2 shows that the change in MEP following a 4-week EMST program in healthy young adults ranged from 25% to 47%. Suzuki et al. (1995) reported an increase in MEP from 165 ± 71 cm H_2O pre-training to 202 ± 77 cm H_2O post-training, a 25% increase for six healthy men. Suzuki's group used a threshold pressure breathing device (Threshold Inspiratory Muscel Trainer, Healthscan Products, Cedar Grove, New Jersey, USA). Sapienza et al. (2002) reported that MEP increased from 99.7 ± 25.2 cm H_2O pre-training to 147.0 ± 31.9 cm H_2O post-training, which is a 47% increase, in 22 healthy men and women. Baker et al. (2005) reported MEPs pre- to post-training from 99.1 ± 34.7 cm H_2O to 127.5 ± 41.1 cm H_2O, respectively, with an increase by 29% in 32 healthy participants. Together, these results suggest that an EMST program is applicable to both young and old healthy individuals to enhance the strength of the expiratory muscles.

Age-related muscle atrophy in the respiratory musculature is observed primarily in the expiratory intercostal muscles, however not in the inspiratory intercostal muscles (Mizuno, 1991). The atrophy of these muscles during the normal aging process is similar to what can be expected for those living a sedentary lifestyle. However, combining a sedentary lifestyle with normal aging can aggravate sarcopenia, or loss of muscle mass with age, in the respiratory musculature. The current study demonstrates that expiratory muscles in the sedentary elderly can be strengthened. The strength may translate to gains in muscle mass/hypertrophy, in response to increased load delivered via EMST over a long enough time frame. The respiratory muscle strength increases achieved with the sedentary elderly are likely related to rapid increases in the neural adaptations at the level of the motor unit. As described earlier, strength gains in skeletal muscles result from a combination of both neural adaptation and muscle mass adaptation. Neural adaptations commonly occur in the early stage (4 to 6 weeks) of training in both young and elderly individuals. After 4 to 6 weeks of training, strength gains in young people are predominately related to muscle hypertrophy, while in elderly people strength gains are mainly due to neural adaptation, even after 6 weeks of training (Moritani & deVries, 1980). These findings suggest that a continual EMST program in the healthy sedentary elderly could be helpful in preventing the alterations in muscle architecture in the expiratory muscles. The time frame in which muscle structure changes is not known but would be a reasonable study to design particularly in an animal model where exercise in a sedentary animal could be compared to a control group pre- and post-exercise. In a human model, some limitations exist for documenting changes in muscle architecture particularly since the respiratory muscles are less accessible, less amenable to biopsy, particularly if recruiting a large sample, and more difficult to image. However, if the animal model proved a positive outcome with respiratory strength training then translation of theory could occur to human application.

Maximum inspiratory pressure (MIP) also increased by an average of 49%, with a range of 10% to 287%, following 4 weeks of training. Several studies have demonstrated increases in MIPs using inspiratory muscle strength training (IMST: Gozal & Thiriet, 1999; Hsiao et al., 2003; Larson et al., 1988; Lotters, van Tol, Kwakkel, & Gosselink, 2002; Martin et al., 2002; McCool & Tzelepis, 1995; Olgiati et al., 1989; Ramirez-Sarmiento et al., 2002; Sturdy et al., 2003; Trueblood et al., 2004; Weiner et al., 2004), a combined program of IMST and EMST (Watsford et al., 2004; Weiner et al., 2003a), or EMST alone (Chiara, 2003; Gosselink et al., 2000).

Since no previous study has used an EMST program independently with a healthy elderly population, the investigator sought to determine the effects of EMST on MIP in the sedentary elderly population. As reported previously, with age gradual reduction in inspiratory muscle strength is accompanied by a decrease in the elastic recoil properties of the lungs. The results of the present study indicated that inspiratory muscle strength increased following the EMST program. Enhancing expiratory muscle strength with EMST increases expiratory reserve volume (ERV) and decreases residual volume (RV), which can increase elastic recoil pressure, thus increasing MIP. Furthermore, during the EMST program, participants are told to inhale to total lung capacity before exhaling to overcome the threshold load set on the training device. This repeated maneuver increases the use of the inspiratory muscles, thus stimulating a potential training effect. These assumptions have been put forth by other investigators (Chiara, 2003; Gosselink et al., 2000). Gosselink et al. (2000) demonstrated EMST effects on MIP in nine patients with multiple sclerosis (MS), with a mean change by 39 ± 41% of initial after 3 months of training. They suggested two possibilities for the significant change in inspiratory muscle strength with EMST. First, EMST would decrease RV, resulting in an increased inspiratory lung volume. This mechanism would allow inspiratory muscles to move easily with increased elastic recoil pressure. The other assumption was that EMST would reduce expiratory lung volume indirectly, thus improving the length-tension of the inspiratory muscle mobility. Likewise, Watson and Hixon (2001) support that abdominal wall trussing places the diaphragm length in an optimum length-tension position thereby resulting in an increase of the potential transdiaphragmatic pressure. EMST may be platforming the diaphragm similarly, thus resulting in greater activity of the diaphragm during the production of MIPs. EMST moves the diaphragm in a headward position, which lengthens the muscle fibers of the diaphragm. Repeated practice of the EMST task may lead to modulation of the sarcomere of the diaphragm, which increase the neural drive to the diaphragm toward inspiration (Watson & Hixon, 2001).

Chiara (2003) also reported MIP gains for both MS and healthy controls ranging from 72.3 ± 4.6 cm H_2O to 79.0 ± 4.8 cm H_2O following 8 weeks of EMST. In that study, the MS group had an 8% MIP gain pre- to post-training. Chiara (2003) proposed that EMST requires the inspiratory muscles to be activated repeatedly in order to reach a near-total lung capacity necessary to achieve the pressure requirements imposed by the pressure-threshold device. However, it is doubtful that a substantial training effect on the inspiratory muscles would occur in the absence of any moderate to maximum load on the inspiratory muscles. If that were the case then tasks such as incentive spirometry would result in a strength gain increase and the concept of imposing a physiological load on muscles to increase strength would not be as strongly advocated as they are (Powers & Howley, 2001). Therefore, the presumption of Chiara may not be supported. Another study examining abdominal muscle recruitment in patients with chronic obstructive pulmonary disease (COPD) suggested that increasing the strength of the expiratory muscles in patients with severe COPD allows increases in the diaphragm muscle length and force generating capabilities at the onset of inspiratory muscle contraction by increasing the load on the inspiratory muscles (Gorini et al., 1997). This in turn would result in a greater MIP. Whether this translates to the biomechanics of healthy subjects is unknown.

Hence, it is reasonable to conclude that EMST is an effective program for increasing overall respiratory muscle strength in the sedentary healthy elderly. Older respiratory muscles preserve a high degree of adaptability in response to strength training similar to other limb muscles (Fiatarone et al., 1994; Gauchard, Tessier, Jeandel, & Perrin, 2003; Narici, Reeves, Morse, & Maganaris, 2004). Thus, strength training specifically targeting respiratory muscles may compensate for the age-related changes in function and morphology of the aging human respiratory muscle. This implies that if elderly individuals are involved in a formal exercise program targeting respiratory muscle strength training, the deleterious repercussions of sarcopenia may be reduced. If so, this may result in improvement of breathing, cough, swallow, and speech functions, and further the quality of life in the elderly. The effects of EMST on these functions are discussed next.

Breathing. It was hypothesized that breathing function would be enhanced following EMST. However, the results of the training program did not support this hypothesis. There was no significant improvement in FEV_1, FVC, and ERV pre- to post-training. Exploration of the relationship between MEP and these pulmonary values indicated no significant correlation as well. The current results are congruent with the results of Simpson (1983).

There has been one previous study using EMST with healthy elderly women to observe the effects on breathing function. This respiratory muscle strength training program, using both inspiratory and expiratory muscle strength training, revealed no changes in breathing function (Watsford et al., 2004). Thirteen elderly women (mean age of 64.4 ± 2.7 years) were trained with a pressure-threshold device (Powerlung™ , PowerLung Inc., Houston, Texas, USA) focusing on three modalities (hypertrophy, endurance, and strength based) of training. No significant changes in FEV_1 or FVC pre- to post-training occurred. The study with healthy young people examining the effect of EMST on breathing function with eight healthy adults (mean age of 36.8 ± 8.8 years) using a combination of IMST and EMST with a commercially available resistive device (Powerlung™, PowerLung Inc., Houston, Texas, USA) indicated non-significant changes in FEV_1 and FVC during 4 weeks of training (Amonette & Dupler, 2001).

Strength training with a method other than resistive loading has also been designed to increase abdominal muscle strength. Following 12 sessions of isokinetic trunk curls-up, breathing function in 16 healthy adults (10 women, 6 men; mean age of 22 years) showed no significant gains from pre- to post-training in FEV_1 and FVC (Simpson, 1983). MEP was not measured in this study. One study examined the breathing-control training in singers and wind-instrument players to determine the effect on breathing function. No significant differences were found in FEV_1 and FVC compared to a control group (Schorr-Lesnick, Teirstein, Brown, & Miller, 1985). It would seem that breathing function in healthy young or elderly individuals is not affected by training to increase expiratory muscle strength.

Like healthy populations, clinical populations have also exhibited no improvements in breathing function following EMST. In a study including patients with MS, Chiara (2003) used EMST with the same device used in the current study. No changes in FEV_1 nor FVC with any of the 17 MS patients (14 women, 3 men; mean age of 48.7 years) or 14 healthy controls (12 women, 2 men; mean age of 43.4 years) occurred. The changes of slow vital capacity in patients with MS were also not significant following 3 months and 6 months of EMST (Gosselink et al., 2000). Furthermore, FEV_1 and FVC in 8 patients (1 women, 7 men; mean age of 63.1 ± 3.1 years) with COPD trained with EMST and in 8 patients (2 women, 6 men; mean age of 62.7 ± 3.0 years) with COPD trained with the combination of EMST and IMST also were unchanged (Weiner et al., 2003a). Patients with COPD and sedentary healthy elderly adults have compromised expiratory flow including a low FEV_1 and FVC due to changes in the lung tissue with low elastic recoil of the lungs. Expiratory flow may depend on the status of the lung tissue (Chiara, 2003; Turato et al., 2003), thus increasing respiratory muscle strength might not alter the lung tissue in patients with COPD and sedentary healthy elderly adults, having no significant changes in breathing function.

There was one study which reports improvements in breathing function following EMST in a clinical population. Saleem (2005) reported significant improvement in FEV_1 by 9% and in FVC by 8% from initial pre-training baseline in patients with PD following 4 weeks of EMST using the same device as used in the current study. Saleem's positive results in comparison to the other studies that showed no change in FEV_1 and FVC might be explained by the differences in the participant population being given the training. FEV_1 and FVC in healthy young and elderly adults are already within normal limits before the training. No further benefit from EMST in healthy populations would be expected. In addition, the study of Saleem (2005) used multiple observations in each training condition, which might systematically increase the overall variance associated with the training effect. This could cause the variance portion related to the training main effect to be misinterpreted as the variance associated with the between-subject difference, resulting in an increase in the significance level.

In the current study, there was no significant effect found for EMST on ERV, but descriptive analysis demonstrated that ERV increased by 21% from pre- to post-training from 0.97 ± 0.61 L and 1.18 ± 0.61 L, respectively. Gosselink (2000) speculated that an increase in MIP followed by improved expiatory muscle strength might be related to reduced RV. Decrease in RV would increase ERV, which allows the lungs to expand more easily during breathing. However, this was not well documented by the current study. Since available outcomes examining the effects of EMST on ERV or RV are limited, further studies are needed to explain the effect of EMST on ERV and RV.

In general, expiratory muscle strengthening with an EMST program does not appear to improve FEV_1 or FVC in sedentary healthy elderly. However, the effect of increasing the strength of expiratory muscles on ERV or RV in either healthy or clinical populations may be promising.

Cough Function

It was hypothesized that EMST would be effective in enhancing cough production as a result of increased expiratory pressure. The results in the current study substantiated this hypothesis in some cough parameters.

It is known that declines in expiratory muscle strength lead to decreases in peak expiratory flow rate (PEFR) and peak-post plateau integral amplitude (PPPIA) during coughing in the elderly (Beardsmore et al., 1987; Irwin et al., 1998). In the current study, the average value of PEFR obtained before training was 4.98 ± 2.18 L/s. This value is comparable to previous reported values (Ebihara et al., 2003; Smith Hammond et al., 2001). A study comparing aspiration risk in 18 healthy men controls (mean age of 65 ± 3 years) and men with stroke demonstrated the PEFR values produced by the healthy elderly adults was 3.62 ± 0.34 L/s (Smith Hammond et al., 2001). Sixteen healthy elderly women controls (mean age of 69.8 ± 10.3 years) in a study examining cough efficacy in the patients with PD had a PEFR value of 5.27 ± 1.17 L/s (Ebihara et al., 2003).

As predicted, significant improvements in PEFR and PPPIA during the capsaicin-induced cough were found in the current study following EMST. After the EMST program the average PEFR values increased by 61%, from 4.98 ± 2.18 L/s to 8.00 ± 3.06 L/s; the PPPIA values increased by 96%, from 3.49 ± 2.46 L to 6.83 ± 4.16 L.

Once the glottis opens following the laryngeal compression phase, the high intrathoracic pressure which has built up promotes a burst of expiratory flow, with a rate as high as 12 L/s and extends through the expiratory phase with a gradual decrease to lower flow rates between 3 and 4 L/s (Irwin et al., 1998; McCool & Leith, 1987). During the sustained expiratory flow (i.e., post-peak plateau in the current study), lung volume declines and intrathoracic pressure decreases. This is because intrathoracic pressure decreases leading to decreased cough expiratory flow. It is therefore expected that increasing intrathoracic pressure by providing an efficient force-length relationship in the expiratory muscles may enhance cough expiratory flow. However, enhancing expiratory muscle force with increasing intrathoracic pressure does not affect the PEFR values during coughs by healthy individuals because flow is independent of expiratory effort. As expiratory force is increased, increased resistance within the airway to expiratory flow compresses the airways dynamically. This was observed in the EMST study with young healthy adults which did not show changes in the PEFR from pre- to post-training, regardless of MEP changes (Baker, 2003). However, individuals with expiratory muscle weakness, who begin with low intrathoracic pressures during cough might increase cough expiratory flow because they are able to develop greater intrathoracic pressures (Baker, 2003; Irwin et al., 1998). This has been demonstrated in previous studies with other groups. The PEFR in individuals with PD increased following 4-weeks of EMST (Saleem, 2005). Additionally, PEFR increased in individuals with amyotrophic lateral sclerosis following insufflation and/or exsufflation or manual assistance (Mustfa et al., 2003).

There is a close relationship between PEFR and lung volume and between PPPIA and lung volume. Both PEFR and PPPIA increase with increases in expiratory force at high lung volumes. However, at mid or low volumes they do not increase with additional intrathoracic pressure associated with increasing expiratory force since elastic recoil is reduced at those lung volumes (West, 1995). In individuals with respiratory muscle weakness, breathing up to higher lung volumes prior to the expiratory phase of cough may increase and sustain the intrathoracic pressures, which would subsequently result in increased PEFR and PPPIA. In fact, increased inspiratory muscle strength, and possibly reduced RV, resulting from EMST would lead to increased lung volumes in the inspiratory phase, which would then result in greater intrathoracic pressures, thereby improving PEFR and PPPIA. Unfortunately, inspiratory volumes were not measured as part of this study.

Further, increasing expiratory force with EMST would enhance the dynamic compression of the airways during the expiratory phase of cough due to the higher intrathoracic pressures, which increases expiratory flow velocities (Irwin et al., 1998). Higher velocities of expiratory airflow may promote airway clearance that reduces the aspiration rate in individuals with respiratory muscle weakness such as sedentary elderly adults. In summary, increased expiratory force resulting from EMST may increase cough efficiency by increasing intrathoracic pressure and by increasing lung volume. This leads to increased expiratory flow rate and dynamic airway narrowing, ultimately resulting in higher velocity of expiratory airflow.

The compression phase duration (CPD) was significantly reduced from 0.35 ± 0.19 seconds to 0.16 ± 0.17 seconds, a 53% decrease following 4 weeks of EMST. The CPD of young healthy people commonly lasts an average of 0.2 seconds (Chung et al., 2003; Irwin et al., 1998; McCool, 2006; Smith Hammond et al., 2001). The duration of the compression phase measured post-training was similar to the average CPD value reported previously. The effect of EMST on CPD in the current study agrees with others who have examined effects of EMST. CPD significantly decreased from 0.62 ± 0.51 seconds to 0.54 ± 0.52 seconds by a 13% change after 4 weeks of EMST in young healthy individuals (Baker, 2003). The CPD in patients with PD, particularly the female participants, also significantly decreased from a mean of 0.35 seconds pre-training to 0.22 seconds post-training by a change of 37% (Saleem, 2005). Shortening duration of vocal fold closure before the expiratory phase starts during cough production may be explained by changes in the speed of the neural mechanism of cough. The airway receptors in the subglottal area detect increases in intrathoracic pressure following EMST. Increased intrathoracic pressure builds up the speed of airflow, resulting in high velocity of airflow. Increased velocity of airflow at the vocal folds decreases the pressure rapidly, thus closing the vocal folds quickly (Bernoulli effect), leading to decreases in CPD. Reduced vocal fold closure time also aids in reducing expiratory muscle shortening velocity (Irwin et al., 1998; McCool, 2006).

In addition, it is expected that the activities of muscles involving laryngeal adduction during coughing may be affected by EMST. It has been reported that laryngeal valving capacity is reduced in the elderly (Hoit & Hixon, 1987; Honjo & Isshiki, 1980; Ptacek & Sander, 1966; Titze, 1994). This may increase the risk of aspiration. A previous study reported that increases in lung volume and expiratory force influence the activation of the lateral cricoarytenoid muscle (Koizumi, Kogo, & Matsuya, 1996). The lateral cricoarytenoid muscle is a laryngeal adductor muscle that causes the vocal folds to close tightly (Kogo, Kurimoto, Koizumi, Nishio, & Matsuya, 1992). Kuna and Vanoye (1994) observed that the activity of the laryngeal adductor muscles are increased by elevated expiratory force. Therefore, increasing lung volume and force during EMST should improve laryngeal adduction during coughing, leading to a reduced risk of aspiration. These hypotheses were not tested in the current study, and there are no known studies which have investigated the effect of EMST on laryngeal closure.

Also, given the significant increase in PEFR, cough effectiveness is enhanced. CPD can be minimized with increasing PEFR. Shortening of CPD with co-occurring increases in PEFR makes sense since the primary cough clearance mechanism is increased expiratory flow. As PEFR increased post-training, the need to increase laryngeal closure to generate intrathoracic pressures was minimized.

The inspiratory phase duration (IPD) and post-peak plateau duration (PPPD) following EMST were not changed in the current study. A longer duration of the inspiratory phase may be needed to increase FVC to acquire a sufficient inspiratory lung volume before initiating expulsive events of cough (Saleem, 2005). The lack of findings with regard to PD accompanied by increased MIP with EMST may indicate that higher lung volumes can be achieved without increasing the duration of inhalation. An absence of significant effects of EMST on the measure of PPPD in the face of a significant effect on PPPIA indicates that EMST likely increases the strength of expiratory muscles without changing the capability of expiratory muscles to sustain their expiratory driving force.

On the other hand, the total number of coughs and the total number of expulsive events were counted and compared to investigate whether changes in cough parameters are solely attributed to EMST, or possibly better explained by other factors such as the sensitivity of cough receptors. Since the current study utilized capsaicin challenge to induce reflexive coughing from the participants, it is possible that increased cough sensitivity may be attributable to repeated capsaicin challenges. Counting the number of coughs has been commonly employed to examine cough sensitivity (Hara et al., 2005; Nieto et al., 2003; Plevkova, Brozmanova, Pecova, & Tatar, 2006). Theoretically, as cough sensitivity to capsaicin increases, the number of coughs should increase. However, neither the number of coughs nor expulsive events significantly changed from pre- to post-training. This finding indicates that enhanced cough efficiency in sedentary healthy elderly individuals was affected mainly by EMST not by changes in cough sensitivity to capsaicin challenge.

This study was the first attempt to examine the effects of EMST on reflexive coughs induced by capsaicin challenge. Previous studies have examined maximal voluntary coughs to determine effects of an EMST program (Baker, 2003; Chiara, 2003; Saleem, 2005). The results demonstrated in those studies were inconsistent. This may be due to differences in the instructions given to elicit voluntary coughs from the participants, differences in the maximal effort level of individual participants, or in the overall populations recruited in each study. However, cough is a reflexive event and as such the coughs should be reflexive in nature in order to clearly measure cough parameters and accurately determine the effects of EMST on cough function. Therefore, the results found for the capsaicin induced cough, in the investigator's opinion, clearly reflect the potential EMST has an enhancing cough function.

In conclusion, EMST is an effective program to increase the expiratory muscle strength and enhance the laryngeal valving mechanism in sedentary healthy elderly individuals. These changes contribute to an enhanced cough production mechanism, which may provide protection from aspiration and effectively remove substances and pathogens trapped in the airways of the elderly population.

Swallow Function

The effects of EMST on peak amplitude (PA), duration (DUR), and integral amplitude (IA) of submental (SM) muscle activity obtained from surface electromyography (sEMG) with various bolus consistencies were also examined. The bolus consistencies were divided into three categories: maximal voluntary dry (saliva), wet (water), and thin paste (pudding), with two different volumes (5 cc and 10 cc). It was hypothesized that swallow function, especially with maximal voluntary dry and 10 cc pudding swallows, would improve as a result of the EMST program. This hypothesis was supported by noted increases in the peak amplitude (PA) and integral amplitude (IA) of SM muscle group activity from pre- to post-training. There were noticeable increases on the PAs in maximal voluntary dry and thin paste (i.e., 5cc pudding and 10cc pudding) swallows even though those increases were not statistically significant. The PAs increased by 17% in maximal voluntary dry swallow, by 16% in 5 cc pudding swallow, and by 18% in 10 cc pudding swallows following EMST. However, the PAs did not significantly change in wet swallows for both 5 cc and 10 cc volumes. The IA of SM muscle group activity significantly increased in 10 cc pudding swallow and was not significant but evident increase in maximal voluntary dry swallow. The percent change from pre- to post-training was 33% in 10 cc pudding and 16% in maximal voluntary dry swallows. The IAs of the other consistencies and volumes also increased, but those increases were not significant. Small amount of volumes and wet swallows are submaximal tasks which likely did not need to recruit maximal motor units, so these tasks may not have responded to EMST or a more sensitive test, such as needle EMG for examining the motor unit changes in submental muscle group may be necessary. Since no previous study examined the effects of EMST on SM muscle activity utilizing sEMG, the changes of SM muscle activity in the current study was not compared with others.

The PAs and IAs of SM muscle group activity were also significantly different among each consistency. Examining overall PAs and IAs of the SM muscle group revealed that the maximum and integral activity were significantly higher in maximal voluntary dry swallow than the two other consistencies. Further, the PA and IA of SM-sEMG activity was significantly higher in thin paste swallow than in wet swallow, which was consistent with the previous findings (Ding, Logemann, Larson, & Rademaker, 2003; Reimers-Neils, Logemann, & Larson, 1994). However, there were no volume dependent differences in the PAs and IAs of SM-sEMG activity, which is also concurrent with the findings from the previous studies (Dantas & Dodds, 1990; Ding et al., 2003).

As described earlier, elderly individuals experience a reduction in swallow function. Vaiman, Eviatar, and Segal (2004b) tested voluntary dry swallows as well as water swallows in groups aged 18 to 30, and over 70 years and found that the elderly group presented with significantly lower activity of SM-sEMG than the younger group. In addition, an age group between those two groups demonstrated a significant decline in the range of SM muscle group activity. Decreased SM muscle group activity with aging is generally accompanied by lower motor unit discharge rates, which can impact the swallow mechanism (Kamen, 2005). However, the EMST program may alter this age-related swallow dysfunction. The EMST device augmented employment of the SM muscle group, thus increasing the PAs and IAs of SM-sEMG activity recorded during swallowing. Increased amplitude of sEMG activity can be interpreted as increased neural drive from the central nervous system to peripheral muscle fibers, resulting in enhancement of muscle strength (Gabriel, Kamen, & Frost, 2006; Kamen, 2005; Powers & Howley, 2001). The neural drive is related to motor unit discharge rate, motor unit recruitment, rate coding, double firing (doublet; 2 closely spaced neural firing), and motor unit synchronization (Gabriel et al., 2006; Kamen, 2005). This mechanism is quite resilient to strength training in the elderly. With only a brief period of retraining, elderly people regain muscle strength and with short-term detraining, they lose the muscle strength (Sforzo, McManis, Black, Luniewski, & Scriber, 1995; Taaffe, 1997). In fact, the results from the current study indicate that an EMST program could increase neural drive to SM muscle group, resulting in increased strength of the SM muscle group during swallowing in healthy elderly adults. Further, long term application of this program may facilitate sustained strength of muscles associated with swallow function.

Enhanced SM muscle group activity may also be achieved via increased expiratory lung volume and expiratory force resulting from EMST. Subsequently, resulting increases in expiratory airflow may facilitate the swallow sensory detection mechanism. Conceivably, increased afferent feedback to the brainstem could stimulate efferents to deliver information to motor units of the SM muscle group and other muscles involved in the swallow process. The increased activity and recruitment of motor units would theoretically increase the activity of swallow musculature, including the SM muscle group. However, this assumption regarding timing parameters, specifically the duration of SM muscle group activity, was not supported by the findings. The duration of SM activity during swallowing was not significantly different from pre- to post-training for any consistency tested, thus not supporting the hypothesis.

It is possible that an increase in SM muscle group activity as a result of the EMST program would increase hyolaryngeal displacement. During oropharyngeal swallowing, the hyoid bone moves superiorly, thereby elevating the larynx, via contraction of SM muscles (Iwarsson & Sundberg, 1998). As hyolaryngeal displacement increases during swallowing, laryngeal glottal closure should be enhanced, thus allowing movement of the bolus into the esophagus more easily (Logemann et al., 2000; Yokoyama et al., 2000). Increased hyoid elevation during 5 cc and 10 cc thin paste (barium) swallows as a result of EMST has been observed using videofluoroscopy in 10 patients with PD and one healthy young adult (Saleem, 2005; Wheeler & Sapienza, 2005).

There were significant effects of consistency on the duration of SM muscle group activity. SM-sEMGs for maximal voluntary dry and thin paste swallows were longer in duration than for wet swallow. In other words, with increasing bolus viscosity the duration of SM muscle group activity significantly increased. Ding et al. (2003) revealed that differing bolus viscosities altered the duration of SM activity and infrahyoid muscle activity in both young and old participant groups. Reimers-Neils et al. (1994) also found a significant effect of bolus viscosity on total swallow duration and on average sEMG activity of the SM muscle group. However, the current study did not find a significant effect of bolus volume on the duration of SM muscle group activity, which was in agreement with in a study by Dantas and Dodds (1990). These investigators concluded that bolus volume did not affect the duration of the oropharyngeal phase. The duration of SM muscle group activity is affected only by bolus viscosity but not by bolus volume during swallowing.

Based on the results of this study, it is concluded that EMST may have positive effects on the swallow function of sedentary healthy elderly individuals. The long-term continuation of an EMST program may prevent the sedentary healthy elderly from experiencing a decline in normal swallow function. This may be important for reducing the risk of aspiration. Additionally, the current study found little variation in the range of electrical activity from the sEMG measures within each consistency or training task, further supporting the use of sEMG as a reliable technique to measure the muscle activity during swallowing. This method is a simple, noninvasive, reproducible way to measure the changes of muscle activity (Ding et al., 2002; Vaiman et al., 2004a, 2004b).

Speech Function

Aerodynamic and acoustic measures were employed to assess speech function. Excess lung pressure (P_{EL}) was used to measure the aerodynamic component, and the acoustic component was measured based on maximum phonation durations (MPDs) of sustained vowel phonation at two levels of intensities, comfortable and maximum loudness.

For the aerodynamic measure, it was hypothesized that P_{EL} would increase significantly after a 4-week EMST program. P_{EL} was defined as the difference between phonation threshold pressure (P_{th}) and lung pressure at the loudest possible intensity (P_L). As was predicted, P_{EL} was significantly affected by the EMST program. The mean P_{EL}

was 13.63 ± 5.00 cm H_2O in pre-training and 19.74 ± 6.61 cm H_2O post-training. P_{th} was not changed by EMST. The P_{th} was 3.11 ± 1.17 cm H_2O in pre-training and 2.94 ± 0.77 cm H_2O in post-training. These values are congruent with previous findings which reported average P_{th} values ranging from 3 to 4 cm H_2O (Baken & Orlikoff, 1998; Grini-Grandval, Bingenheimer, Maunsell, Ouaknine, & Giovanni, 2002; Hodge et al., 2001; Titze, 1994). In contrast to P_{th}, P_L significantly increased from pre-training with a mean of 16.36 ± 6.05 cm H_2O to post-training with a mean of 22.85 ± 6.77 cm H_2O. Therefore, it is concluded that the increased P_{EL} resulted from an increase in P_L and not by a change in P_{th}. This indicates that by increasing expiratory muscle strength following EMST, chest wall rigidity in the sedentary healthy elderly may be compensated for by development of the more adequate positive pressures, leading to increases in sound pressure level (Hixon, 1973; Isshiki, 1964). As explained earlier, EMST also increased inspiratory muscle strength and possibly reduced RV, resulting in increasing inspiratory lung volume. Increased inspiratory lung volume can add more positive subglottal pressure necessary to produce loud speech.

Furthermore, an increase in lung pressure following EMST may play a role in compensating for age-related decreases in the ability of the laryngeal system to control vocal loudness (Baker et al., 2001). It is known that both the respiratory system and laryngeal mechanisms play an important role in controlling vocal loudness (Baker, Ramig, Luschei, & Smith, 1998; Hodge et al., 2001; Holmberg, Hillman, & Perkell, 1988; Stathopoulos & Sapienza, 1993a, 1993b)}. The laryngeal system in the elderly is not an efficient mechanism to increase vocal loudness secondary to insufficient vocal fold closure resulting from senescent changes in the laryngeal musculature, joints, and nervous innervation (Honjo & Isshiki, 1980; Linville, 1992; Paulsen & Tillmann, 1998; Tanaka et al., 1994). Baker et al. (2001) suggested that higher expiratory efforts may be needed to compensate for the age-related stiffness of the vocal folds that cause a reduction in laryngeal adductory mechanism. Accordingly, EMST would theoretically be conducive in overcoming the inefficient laryngeal adductory mechanism in elderly individuals, thereby improving the control of vocal loudness by augmenting the expiratory driving force.

The MPD of a sustained vowel at the comfortable effort intensity level significantly increased after EMST as predicted in the hypothesis. However, MPD at the loudest possible intensity did not change. MPD at the comfortable intensity level ranged from 5.51 to 33.91 seconds (mean of 17.78 ± 7.90) pre-training and from 7.05 to 42.73 seconds (mean of 22.37 ± 10.04) post-training. This increase in MPD during the comfortable loudness level may be explained by increased expiratory driving force as supported by increased MEP or increased inspiratory lung volume.

It is important to note that these elderly participants were hesitant to produce loud phonation and that may have influenced the outcome of MPD during the loud task. Most participants reported that phonating loudly was not commonly used in their day to day routine and that they felt uncomfortable phonating at their loudest possible level. This may not have resulted in the best elicitation of loud phonation, even with prompting from the investigator. To minimize this limitation in maximum phonation task, the use of white noise would be recommended to increase the intensity level of speaker's voice. Speakers increase their vocal loudness in the presence of increasing white noise increased (Howell, 1990).

Summary

With age, physical functions decline that can influence respiratory performance. Reductions in respiratory muscle strength, elastic recoil of the lungs, and chest wall compliance change the lung pressures necessary for ventilatory and non-ventilatory functions in the elderly. This study was designed to investigate the physiological effects of a 4-week EMST program on pulmonary, cough, swallow, and speech functions in the sedentary healthy elderly. The program was easy for the sedentary healthy elderly to learn and to utilize. The EMST program employed a user-friendly small device that was adjustable to each individual's capabilities and which provided significant improvements in various aspects of physiological function over a short period of time. Furthermore, most participants in the present study reported the changes in their attention to breathing and reduced number of choking events.

Admittedly, there were some limitations to this study. First, eighteen elderly people participated, four men and 14 women. One of the aims of the current study was to document the gender differences in the effect of EMST. However, the unbalanced sample size for gender did not allow for comparison of differential effects of EMST based on gender. There are more elderly women than men in the general population. According to a recent report, 60% of people 65 years or older and 72% of those 85 years or older are women (U.S. Census Bureau, 2003). Additionally, elderly women tend to have higher social engagement than elderly men (Smith & Baltes, 1998; Strawbridge, Cohen, & Shema, 2000). Many elderly men were reluctant to participate in this study when the investigator was recruiting participants from retirement communities. For these reasons, the number of healthy elderly men participating in the study was much smaller number than healthy elderly women.

Second, EMST involves production of high expiratory efforts which are associated with health risks, particularly for individuals with high blood pressure or hernia. One study participant withdrew because he was concerned about increases in blood pressure, even though he had no history of it. EMST is not applicable for the elderly who have heart or vascular problems or those with untreated hypertension since EMST needs high pressure to overcome the pressure threshold set in the training device. Thus clinicians or researchers should use caution when examining the health status of the elderly before implementing the EMST program.

Third, the present study explored the effect of only 4 weeks of EMST. A recent study in a patient with early idiopathic Parkinson's disease reported that a longer duration of the EMST program leads to more improvement in expiratory muscle strength (Saleem et al., 2005). This result is a very important finding, especially for the elderly population. Since aging is a continuous process, continuing the EMST program in the elderly may help to prevent or compensate for age-related deteriorations in pulmonary, cough, swallow, and speech mechanisms. However, the present study does not have direct evidence to support this explanation.

In conclusion, the results from this study supported that EMST may be an effective way to change or compensate for age-related neuromuscular deterioration in pulmonary, cough, swallow, and speech functions. These effects have the potential to decrease the risk of aspiration in the sedentary elderly by increasing expiratory flow rate during cough, by augmenting submental muscle activation during swallowing, and by enhancing the laryngeal adductory mechanism. Results of this study also suggest that sarcopenia might be reversible, and that continued EMST may prevent sarcopenia and the subsequent impact on pulmonary, cough, swallow, and speech functions. This may lead to improvement in the quality of life of elderly individuals. EMST seems to be a viable treatment tool for overcoming functional decline resulting from sarcopenia in the healthy sedentary elderly.

APPENDIX A
INFORMATION FLYER

Are You Interested in Exercising Your Breathing Muscles?

The Communication Sciences and Disorders Department is looking for subjects to participate in a research study to measure the effect of breathing exercise training in healthy elderly.

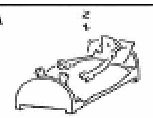
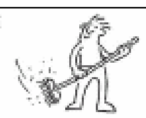
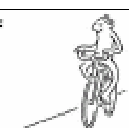
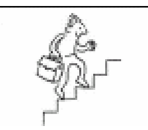
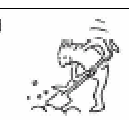
To be eligible you must:

- be over 65 years of age and sedentary
- have no history of cardiac, lung, neuromuscular, immune system disease, or untreated hypertension
- have no history of smoking or tobacco use in the last five years

For more information, please contact Jaeock Kim at jokim@csd.ufl.edu. Phone: (352) 392-2046, ext. 221. To gain more information about her dissertation project supervised by Dr. C. Sapienza at sapienza@csd.ufl.edu.

APPENDIX B
SCREENING PHYSICAL ACTIVITY QUESTIONNAIRE

Describes total amount of physical activity on an average weekday.

Examples		Minutes	Hours	Time:
A 	Sleep, rest	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
B 	Sitting quietly, watching television, listening to music or reading	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
C 	Working at a computer or desk, sitting in a meeting, eating	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
D 	Standing, washing dishes or cooking, driving a car or truck	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
E 	Light cleaning, sweeping floors, food shopping with grocery cart, slow dancing or walking downstairs	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
F 	Bicycling to work or for pleasure, brisk walking, painting or plastering	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
G 	Gardening, carrying, loading or stacking wood, carrying light object upstairs	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
H 	Aerobics, health club exercise, chopping wood or shoveling snow	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	
I 	More effort than level H: Running, racing on bicycle, playing soccer, handball or tennis	☐ ☐ ☐ 15 30 45	☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 1 2 3 4 5 6 7 8 9 10	

Source: Aadahl, M. & Jorgnensen, T. (2003). Validation of a new self-report instrument for measuring physical activity. *Medicine and Science in Sports and Exercise, 35*(7), 1196-1202.

APPENDIX C
SCREENING HEALTH QUESTIONNAIRE

I. Demographics

Name____ Sex___________
Address____
City____________________ State________ Zip Code_________
Birth of date________________
Tel: (H) ________________ (W)________________

II. Physical Characteristics

1. Height______________ Weight____________

2. Rate your health on this scale <u>compared to others your age</u>.

1	2	3	4	5
1= very good	2= good	3= fair	4= poor	5= very poor

3. List the major surgeries you have had within the last 5 years.

4. Are you being treated at the present time for any medical conditions? If yes, please specify.

5. Please list your medications.

6. Blood pressure (It will be measured by the investigators.)
   Systolic / Diastolic (mmHg) ______________ / ______________

III. Physical Activity

Rate your daily physical activity on this scale.

1 2 3 4 5

1= not active 5= extremely active

2. Do you plan to increase or decrease your physical activity over the next several months? Yes/No If yes, how do you plan to change? _____________

IV. Medical History

1. Do you currently have or have a history of the following: Check as many as apply.

_____ Smoking in the last 5 years
_____ Respiratory/breathing problems/asthma
_____ Diagnosis of lung cancer in the last 1 year
_____ Upper of lower respiratory tract infection in the last 2 weeks
_____ Musculoskeletal disorders
_____ Hypertension (treated or untreated)
_____ Immune system diseases
_____ Aneurysm
_____ Stroke
_____ Hernias
_____ Gastroesophageal reflux disease
_____ Cardiovascular problems
_____ Neuromuscular problems

2. __Y __N Do you have asthma or other conditions that affect your breathing?

3. __Y __N Have you had a recent cold or flu?

4. __Y __N Do you have allergies?

8. __Y __N Have you ever smoked tobacco products?
If you used to smoke, for how many years and when did you stop?

Developed by Laryngeal Function Laboratory, Department of Communication Sciences and Disorders, University of Florida. July, 2004.

APPENDIX D
CAPSAICIN SOLUTION PREPARATION

→0.06108g of Capsaicin dissolved in 0.40ml of EtOH (500mM)

Add 0.200ml of Tween 80 and 1.400ml of Saline

Final = 100mM Capsaicin in 70% Saline, 20% EtOH, 10% Tween 80

→0.300ml of 100mM Capsaicin Stock + 149.700ml of Saline

Final Concentration = 200µM Capsaicin in Saline (0.02% Tween, 0.04% EtOH)

* Make 500mls of Saline with final concentration 0.02% Tween, 0.04% EtOH to be used in all remaining dilutions

→150µM Capsaicin in Vehicle = 52.5ml of 200µM + 17.5ml of Vehicle

→100µM Capsaicin in Vehicle = 35ml of 200µM + 35ml of Vehicle

→50µM Capsaicin in Vehicle = 20ml of 150µM + 40ml of Vehicle

→25µM Capsaicin in Vehicle = 15ml of 100µM + 45ml of Vehicle

→10µM Capsaicin in Vehicle = 10ml of 50µM + 40ml of Vehicle

→5µM Capsaicin in Vehicle = 10ml of 25µM + 40ml of Vehicle

Provided by Department of Physiological Sciences, College of Veterinary Medicine, University of Florida. July, 2004.

APPENDIX E
RESPIRATORY MUSCLE TRAINING PROGRAM

INSTRUCTIONS

- This training is to increase the strength of your respiratory muscles just like a general body exercise program.
- You will complete this training program 5 days per week. You will complete 5 sets of the exercises with 5 repetitions each time you complete your training.
- You have been given a respiratory trainer to complete your training at home. You will use this same trainer for the next 4 weeks that you are participating in this study.

EACH WEEK OF TRAINING

1. Place the nose clip on your nose.
2. Breathe in as much air as you can, and place the mouth piece in your mouth.
3. As soon as the mouth piece is your mouth, breathe out as much air as you can.
    - Keep a tight seal with your mouth around the mouth piece.
    - When the expiratory pressure is strong enough to open the valve, you will hear a rush of air move through the device.
4. Repeat this expiratory exercise **5** times (steps 1-4), resting for 30 seconds to 1 minute in between each inspiration.
5. When you have finished all **5** expirations, rest for 2 minutes (you have completed 1 set)
6. After you have rested for 2 minutes, repeat steps 1-5 (for the **5** repetitions)
7. You will continue with this pattern of **5** expirations and 2 minute breaks until you have completed the **5** expirations procedure **5** times (now you have completed 5 sets)
8. On your training log, record the date and the time you completed these exercises
9. You will need to complete steps 1-8, **5** times during the week.
10. At the end of this training week, you will have an appointment during which your maximum expiratory pressure will be taken and your respiratory trainer will be reset.

APPENDIX F
PRESSURE THRESHOLD TRAINING LOG

TRAINING LOG

Week 1

Start Date	MEP 1	MEP2	MEP3	Avg. MEP	Trainer Setting

Date	Time	SET 1 (5 breaths)	SET 2 (5 breaths)	SET 3 (5 breaths)	SET 4 (5 breaths)	SET 5 (5 breaths)

Week 2

Start Date	MEP 1	MEP2	MEP3	Avg. MEP	Trainer Setting

Date	Time	SET 1 (5 breaths)	SET 2 (5 breaths)	SET 3 (5 breaths)	SET 4 (5 breaths)	SET 5 (5 breaths)

Week 3

Start Date	MEP1	MEP2	MEP3	Avg. MEP	Trainer Setting

Date	Time	SET 1 (5 breaths)	SET 2 (5 breaths)	SET 3 (5 breaths)	SET 4 (5 breaths)	SET 5 (5 breaths)

Week 4

Start Date	MEP1	MEP2	MEP3	Avg. MEP	Trainer Setting

Date	Time	SET 1 (5 breaths)	SET 2 (5 breaths)	SET 3 (5 breaths)	SET 4 (5 breaths)	SET 5 (5 breaths)

If you have any question or comment, contact Jaeock Kim at (352) 392-2046 x 221 or after hours at (352) 871-3361; E-mail at jokim@csd.ufl.edu

APPENDIX G
ABBREVIATION TABLE

Abbreviation	Titles
5W	5 cc water swallow
5P	5 cc pudding swallow
10W	10 cc water swallow
10P	10 cc pudding swallow
COMF	Comfortable intensity level
CPD	Compression phase duration
DRY	Maximal voluntary dry swallow
DUR	Duration of submental muscle group activity
DV	Dependent variable
EMST	Expiratory muscle strength training
ERV	Expiratory reserve volume
FEV_1	Forced expiratory volume in 1 second
FEV_1/FVC	The ratio of FEV_1 to FVC
FVC	Forced vital capacity
IA	Integral amplitude of submental muscle group activity
IMST	Inspiratory muscle strength training
IPD	Inspiratory phase duration
LOUD	Loudest possible intensity level
MEP	Maximum expiratory pressure
MIP	Maximum inspiratory pressure
MPD	Maximum phonation duration
PA	Peak amplitude of submental muscle group activity
PEFR	Peak expiratory flow rate
P_{EL}	Excess lung pressure
P_L	Lung pressure
P_{th}	Phonation threshold pressure
P_O	Intra-oral pressure
PPPD	Post-peak plateau duration
PPPIA	Post-peak plateau integral amplitude
SM	Submental muscle group
sEMG	Surface electromyography

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BIOGRAPHICAL SKETCH

Jaeock Kim received a Bachelor of Nursing Sciences degree from the Catholic University, Seoul, South Korea, in February of 1995. After graduation, she worked as a registered nurse in the medical intensive care unit of St. Mary's Hospital, Seoul, South Korea, for 2 years. In 1997, she started her graduate studies at the school and obtained her master's degree in Nursing Sciences in February of 1999. After graduation, she worked as a clinical instructor in several university setting hospitals. She enrolled in the doctoral program in the Department of Communication Sciences and Disorders at the University of Florida under the direction of Alice Dyson, Ph.D., in August of 2000. Following Dr. Dyson's move to another university, Jaeock began working under the mentorship of Christine Sapienza, Ph.D. Jaeock's subsequent research during her doctoral program has focused on the mechanics of the human respiratory system and its relationship to voice production, cough mechanism, and swallow function. She will graduate from the University of Florida with a Doctor of Philosophy in May of 2006.