Respiratory Muscle Strength Training: Functional Outcomes versus Plasticity

Christine M. Sapienza, Ph.D., CCC-SLP,[1,2] and Karen Wheeler, Ph.D., CCC-SLP[3]

ABSTRACT

Respiratory muscle strength training is a paradigm that has been used for numerous years with a variety of populations including but not limited to spinal cord injury, chronic obstructive pulmonary disease, multiple sclerosis, Parkinson's disease, voice disordered, sedentary elderly, and healthy young. The respiratory muscle strength program discussed here is an expiratory muscle strength training and uses a pressure threshold device with a regimented treatment protocol. The primary purpose of the expiratory muscle strength training program is to promote strength in the expiratory muscles. The training protocol occurs five times per day, 5 days a week, and consists of ~15–20 minutes per day of training by the user at home. The device threshold is changed weekly by a clinician to maintain a threshold load of 75% of an individual's maximum expiratory pressure. The threshold setting of the device is always based on the individual's recorded maximum expiratory pressure generated into a digital pressure gauge. Results of 4 weeks of expiratory muscle strength training protocols indicate up to a 50% improvement for healthy subjects,[1] those with multiple sclerosis,[2] and those with spinal cord injury.[3] The potential transfer of expiratory muscle strength to functional outcomes is discussed, as well as how strength-training paradigms may influence cortical plasticity.

KEYWORDS: Expiratory, strength, adaptations, functional, plasticity

Learning Outcomes: As a result of this activity, the reader will be able to (1) describe the underlying principles of respiratory strength training, and (2) explain the role of the respiratory strength training on influencing functions related to breathing, cough, speech, and swallow.

[1]Department of Communication Sciences and Disorders, University of Florida; [2]Malcom Randall V.A. Medical Center, Brain Rehabilitation and Research Center, Gainesville, FL; [3]Department of Speech and Hearing Science, Arizona State University, Tempe, Arizona.

Address for correspondence and reprint requests: Christine Sapienza, Ph.D., Department of Communication Sciences and Disorders, 336 Dauer Hall, University of Florida, Gainesville, FL 32611. E-mail: sapienza@csd.ufl.edu.

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There are two main types of respiratory muscle strength training programs including inspiratory and expiratory muscle strength training (EMST). Which method is selected depends on the targeted muscle group of interest and the patient profile of symptoms. For example, patients with spinal cord injury occurring at the cervical level would have difficulty with both inspiratory and expiratory muscle strength and could be a candidate for both types of training, whereas individuals with spinal cord injury at the thoracic level may have more difficulty with expiratory muscle weakness and therefore be a candidate for EMST. Those with chronic obstructive pulmonary disease who have significant dyspnea and exercise intolerance would benefit most from inspiratory muscle strength training.[4] The focus of this article is on EMST and on how improvements in expiratory muscle strength may translate to peripheral functional improvements in breathing, cough, swallow, and speech. As well, the basis of muscle strength training within a framework of neuroplasticity is presented.

The theoretical rationale that supports the use of EMST for those patients with neurological disease and disorder (such as spinal cord injury or multiple sclerosis) and for others (e.g., those with upper airway dysfunction) is based on sound evidence obtained from studying the effects of strength training in the limbs. Increases in the strength of skeletal muscles such as limb muscles occur fairly rapidly when they are exposed to strength-training programs, as a result of neural mechanisms, with eventual hypertrophy of the muscle occurring as training progresses past a period of approximately 4 weeks.[5] The abdominal and internal intercostal muscles are skeletal muscles and have many of the same structural and metabolic properties as limb muscles such as equal distribution of type I and type II fibers. Histochemical examination verifies that abdominal muscles are composed of approximately an equal distribution of type I (slow-oxidative) and type II (fast-twitch) muscles fibers. Likewise, the internal intercostal muscles are similar to limb muscles in the distribution of type I and type II fibers, although they consist of slightly fewer type II fibers.[6] Similarities in muscle fiber type distribution between expiratory muscles and limb muscles indicate that these muscles are capable of using similar metabolic processes for the creation of the energy necessary for muscle contraction.

Strength training as a form of exercise produces adaptations in muscle that improve motor performance.[7,8] There are two main guiding principles that underlie the concept of strength training. The first is that the exercise stimulus must be sufficient to elicit a change in muscle function. This is referred to as stimulus intensity and is usually defined in terms of the amount of load, as well as the duration, of the exercise task and of how long it is performed (minutes per day × days per week × total weeks). The stimulus must be of a magnitude great enough to make the neuromuscular system respond beyond its typical level of activity, thereby forcing the muscular system to adapt to the increased demand. The degree of improvement is dependent on the amount of load placed on the muscle during the conditioning and the load specificity, related to the projected muscle task for which the conditioning is desired, discussed in more detail later.[9] Peak improvements in muscle strength are attained through the use of loads of between 70% and 90% of the maximum load for periods of 30–60 seconds, repeated three to four times, 3–4 days a week. The next concept is specificity, which refers to how closely the exercise task aligns with the targeted outcome. Specificity of an exercise means that it matches the biomechanics, energy system use, and physiological and psychological control factors of an intended performance. Simply targeting "endurance" or "strengthening" of a specific system or muscle group in a general sense may be enough to force some of the adaptations necessary to improve performance on a specific task. However, the greatest functional benefits are elicited when the training task resembles the end goal as much as possible for specific gains in the activity of interest to occur. Therefore, if becoming a better runner is the goal, then running is the training task; if improved swallowing is the goal, then swallowing is the optimal training task. In some instances, however, this may not be possible. For example, although using a task such as effortful swallowing as a therapy

tool may be more task specific, it may not be advantageous if the patient is aspirating. Therefore, use of another exercise that works to strengthen swallow muscles or enhance swallow muscle coordination may need to precede an actual swallow therapy paradigm. The final concept is that of transference, which is the rationale for using a nonspecific strength training, such as EMST, to improve performance in related, but more specifically defined, functional tasks.

The goal of EMST is to increase expiratory muscle strength. Its paradigm is distinct from traditional breathing resistance training, as it does not allow airflow rate to be modified during the training stimulus.[11,12] The resistance devices marketed for clinical use (PEEP, incentive spirometry, etc.) allow the rate of airflow produced into the trainer to be varied, thus minimizing the training effect that could be potentially gained by a user. A unique program of EMST occurs with a pressure-threshold device that provides a consistent pressure load to expiration. Participants must overcome the threshold load by generating an expiratory pressure sufficient to open the expiratory spring-loaded valve within the device. The individual must sustain this pressure level throughout the expiration. If the participant does not generate the threshold pressure, the valve remains closed. See Figure 1 for a depiction of the device.

The potential functional outcomes that may translate from the use of EMST include influences on breathing, cough, speech, and swallow. These hypotheses are modeled on the general process of strength and endurance training, which demonstrates that following training, respiratory muscles show an increase in the number of mitochondria, capillary density, and type I muscle fibers, leading to an overall decrease in the metabolic demands on those muscles for completing a task (e.g., speaking, coughing, etc.).[5] If an expiratory muscle can generate more expiratory force with more efficiency, expiratory driving pressure can be increased, which is the primary variable necessary for improving expiratory volumes, cough flow magnitude, speech sound quality, sound energy, and overall speech naturalness, and it can also potentially influence swallow function. Specific to swallow, a secondary training effect may also be occurring with EMST by virtue of increases in the activation of the submental muscle group. This is discussed later under the swallowing section.

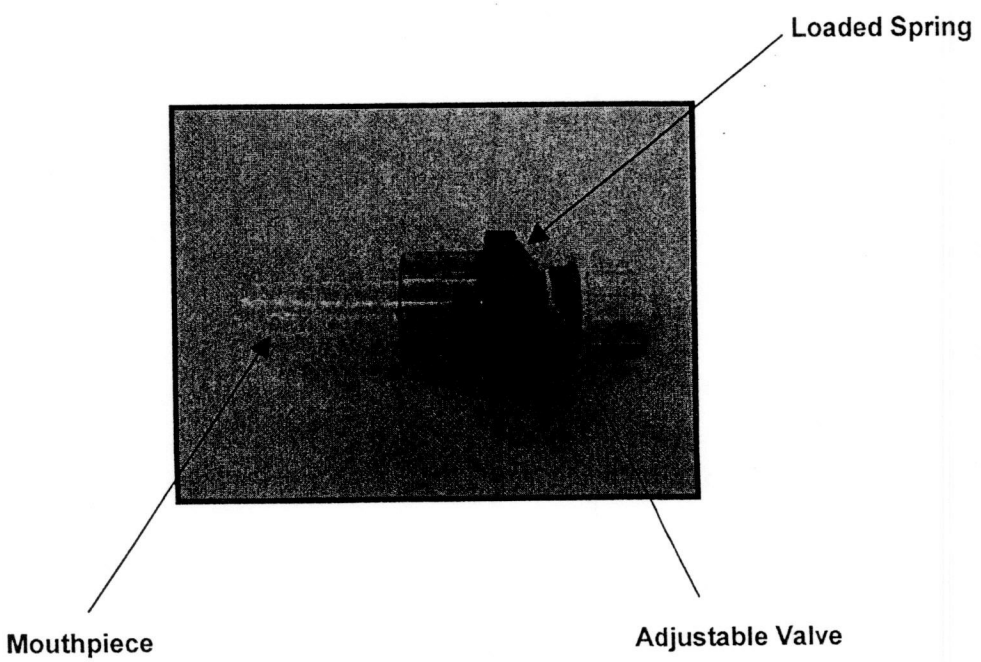

Figure 1 The expiratory muscle strength trainer.

BREATHING

Previously, inspiratory and expiratory muscle strength in healthy individuals has not been strongly correlated with a person's ability to generate a forced vital capacity.[13] More recently, there is evidence that maximum inspiratory and expiratory pressures are strongly correlated with forced vital capacity production, and some work even indicates that a decrease in respiratory muscle strength may contribute to a decrease in observed forced vital capacity, particularly in extreme environmental conditions.[14] Furthermore, we also know that a loss of respiratory muscle strength with disease or aging can deteriorate ventilatory functions.[6] During expiration at rest, the passive elastic recoil of the lungs is typically sufficient in generating the expiratory force/pressure needed for basic ventilatory function. However, normal ventilatory function depends on equilibrium between ventilatory requirements and ventilatory capacity. The respiratory muscles play an important part in accomplishing this balance. For patients experiencing ventilatory dysfunction, such as the elderly, the frail, and some patients with neurodegenerative diseases, therapies that improve respiratory muscle function serve to increase ventilatory capacity, thus improving ventilatory function. Koessler's 2001 work on 27 patients with neuromuscular disease who were followed for 2 years showed that maximum inspiratory pressure along with vital capacity increased with strength training in 18 patients with Duchenne's muscular dystrophy and in nine patients with spinal muscular atrophy.[15] Most recently, Kim's dissertation work showed that ventilatory capacity was increased following EMST training with the sedentary elderly.[16]

COUGH

The degree of forced expiratory volumes that can be generated by an individual directly relates to the degree of expiratory driving pressure. Likewise, when expiratory driving pressures are increased, the ability to clear an airway improves by increasing the necessary cough airflow rate to expel the substance. It is assumed that peak expiratory airflow rate during cough will increase if an expiratory strength training protocol increases the expiratory driving pressures.[17] This increase, in turn, will diminish the need to use increased vocal fold compression to generate increased driving force during cough. As a result, cough compression time will decrease.

The speed of vocal fold closure during cough will increase as a result of increasing the degree of lateral vocal fold movement during the cough expulsion phase, thus returning the vocal folds medially at a faster rate as a result of the increased recoil forces of the vocal fold tissue. Evidence of these effects has been shown most recently by Kim in her work with the sedentary elderly during capsaicin-induced cough.[16]

SPEECH

With regard to speech, improvements in sound quality, speech naturalness, speech duration, and speech loudness are a function, to some extent, of the degree of expiratory pressure that is developed.[18–20] Pressure support is the foundation of all other pressure changes. The subglottal pressure for speech provides the driving force for air to move out of the lung, into the upper airways, and out the mouth. The action of the larynx, pharynx, oral cavity, mouth, and lips shapes the subglottal pressure. The frequency of sound (range) also is a function of the driving pressure; that is, the greater the pressure, the higher the fundamental frequency of the voice. The driving pressure also is essential for the control and duration of a sustained note. The importance of pressure support has been long recognized, and many techniques have been developed to increase and control the support pressure. The diaphragm is the primary inspiratory muscle and must relax for the expiratory muscles, particularly the abdominal muscles, to produce lung compression and thereby generate the necessary positive subglottal pressure. Training methods for increasing expiratory muscle strength to assist in developing higher-pressure support have been used with mixed success. Abdominal exercises, such as sit-ups, increase abdominal muscle strength for moving the body trunk, helping to maintain a tight abdomen, do not increase one's ability

to generate greater maximum expiratory pressure. When inspiratory volumes are limited and the subglottal pressure demand for particular speech tasks cannot be met (e.g., during long durations of speech or loud speech), active expiratory muscles must be recruited to generate the positive airway pressure for these tasks. In Hixon's classic 1987 text on respiratory function in speech and song, Putnam and Hixon clearly indicated the positive relationship between weak expiratory muscles of the ribcage and the abdomen to inadequate loudness levels, stress contrasts, and short breath groups during speech. By increasing expiratory muscle strength, certain pathological conditions can be compensated for with more ease, and expiratory muscle force can be developed for generating the necessary positive pressures, resulting in increased sound durations, greater sound pressure level, and improved sound quality and speech naturalness.[21]

SWALLOW

An individual's respiratory status plays a significant role in the critical integration between breathing and swallowing activity and in sustaining the obligatory apneic pause that must occur during oropharyngeal swallowing as the bolus passes into the distal esophagus.[22] Thus, the effects that EMST may have on swallow function are related to two different mechanisms: first, the ability to generate adequate expiratory pressure; and second, the ability to protect the airway. Subglottal pressure has been shown to play a role in healthy swallowing, as is demonstrated by persons with tracheostomy, whereby simply occluding the tracheostomy tube improves the timing of the swallow and subsequently improves the overall swallow safety.[23] Although the exact mechanisms that dictate the relationship between expiratory pressure, subsequent positive subglottal pressure, and swallowing remain unclear, it has been hypothesized that laryngeal mechanoreceptors sensitive to change in subglottal pressure during the obligatory apneic period surrounding the oropharyngeal swallow may play a role in online modification of the swallow motor program.[24–28] It has been demonstrated that with manipulation of lung volumes to very high or low percentages of vital capacity, the mechanics of a swallow change in healthy individuals.[29] Thus, the ability to achieve adequate lung volume on inspiration, followed by the passive recoil forces associated with the lung–thorax unit during expiration, coupled with expiratory muscle activation to enhance expiratory airflow, leads to increased subglottal pressure against the closed upper airway, which may play a role in modifying or generating the swallow motor program. Research is currently underway to examine the possibility of target lung volumes for swallow initiation within the motor plan generated for a swallow, and how that volume may change with bolus size or viscosity.

A second mechanism by which we believe the technique of EMST may be beneficial to swallowing is via strengthening of the suprahyoid muscles and thus aiding hyolaryngeal elevation and laryngeal vestibule closure.[30] Improvements in hyolaryngeal elevation should aid in maintaining the integrity of laryngeal vestibule closure during the apneic period. Electromyographic investigation has confirmed increased activation of the anterior suprahyoid muscles during use of the EMST device compared with a normal discrete swallow, indicating that those muscles are being subjected to a degree of overloading with EMST.[31] Further, we anticipate increased velopharyngeal muscle activity with training. This is based on our pilot observations in the videofluorographic suite, which revealed velopharyngeal closure during the training as well as evidence that nonspeech blowing tasks of greater than 12 cm H_2O result in increased single–motor unit firing of the levator veli palatini.[32] In addition, genioglossus and pharyngeal constrictor muscles also may be EMST targets, as is suggested by preliminary data.[33] In all, the relative distance and temporal duration measures we plan to apply in our methods are indirect indicators of oropharyngeal muscle strength and coordination. If the durations and relative distances change as we hypothesize, it seems reasonable to conclude that the muscles known to be responsible for these airway protective and bolus propulsive actions have increased in strength and coordinative abilities.

EXERCISE AS A MECHANISM FOR INDUCING NEUROPLASTIC CHANGES

Using exercise to induce neuroplastic changes has been demonstrated in animal studies as well as in humans. Chen et al recently revealed the potential neuroprotective effect of intensive exercise in those who incorporated a great amount of physical exercise.[34] Those who exercised more were at less risk for Parkinson's disease (PD). Specifically, Chen's group found that vigorous exercise in early adult life was inversely related to PD. If men vigorously exercised for greater than or equal to 10 months, they had a 60% lower risk of getting PD. Likewise, women who exercised vigorously in their early adulthood had a lower risk of getting PD later in life.

Neuroprotection refers to strategies to protect neurons from injury or degeneration. Exercise is one rehabilitation tool that is free and that appears to offer a neuroprotective effect as well as improving function—as outlined above for EMST—thus relieving symptoms and improving muscle strength. By understanding the effects of exercise on neuroplasticity, novel nonpharmacological therapeutic modalities may be designed to delay or reverse disease progression of different types of neurological disorders.

Neuroplasticity refers to the ability of the brain to physically change in response to stimulus and activity.[35,36] Cortical neuroplastic changes may include changes in functional organization of the cortex or changes in the threshold of stimulation needed to elicit a motor or sensory response.[37,38] It is here that the distinction between skilled and unskilled exercise training, which may translate to task-specific versus task nonspecific exercise, must be made. Functional reorganization of the cortex as well as threshold changes have been shown to occur in response to skilled but not unskilled training.[39–41] For example, Kleim and colleagues found that rats trained in a skilled reaching task experienced synaptogenesis associated with physiologically distinct areas of motor cortex, whereas rats trained in a motor activity control group did not.[42] These immediate, short-term changes following the training of skilled exercises are thought to be a result of increased neuronal excitability, with the subsequent addition or loss of synaptic material leading to enduring changes within 7–10 days of skill exercise learning.[39,42–44] Underlying mechanisms that may explain these changes include an increase in the number of synapses per neuron, increased arborization of dendrites on pyramidal cells, or enhanced synaptic responses. In addition, increased glucose uptake by muscles during any type of exercise may lead to prolonged and increased neurotransmitter release in neocortex, which contributes to the above-mentioned corticoplastic mechanisms.[39,42–44]

Although EMST may not be specific to a task such as swallowing, it is a skill task that requires the integration and coordination of multiple muscle systems and thereby may lead to neuroplastic changes in targeted muscles. For example, during swallowing, the anterior suprahyoid muscles are activated for about 800 ms, submaximally.[45–48] Similarly, short bouts of activity are seen in these muscles during speech tasks, as well as during mastication.[49,50] However, their pattern of activation is different as seen on surface electromyography with EMST, and the motor tasks associated with their activation are distinctively different than those associated with swallowing, Therefore, it is reasonable to hypothesize that there may be enhancement of the sensory and motor representation and excitability of areas with which those muscles are associated with EMST, which is a seemingly novel skill task. Subsequently, this may lead to carryover both of increased cortical drive and of coordination of those muscles associated with EMST to tasks that require precise timing and strength targets, such as the act of swallowing.

DETRAINING

One question that remains with regard to the EMST training protocol is what happens when the training is stopped. That is, does the strength-training effect remain established once the training stimulus is removed (e.g., detraining)? A true detraining effect can only be examined if the stimulus is completely removed. Detraining in skeletal muscles is defined as a decrease in the adaptations that occur to a muscle as a result of training. The

reduction of these adaptations results in eventual loss of maximum force output, and therefore in a loss in the functions that improved with the increased force output. As indicated previously, respiratory and limb muscles are structurally and functionally similar, and because of this, it might be assumed that respiratory muscles follow a similar pattern of strength decline during a detraining period as do limb muscles.[51] However, this hypothesis has not been tested. Knowledge of the time course of the reversibility of training effects gained from these programs will guide the successful development of a strength-training program. Clinically, detraining effects have to be taken into account, maintenance programs have to be developed, and patients need to be informed so that their motivation can be maintained. The effects of detraining, if they occur rapidly, can influence an individual's motivation to train and deter generalization of treatment to functional activities

Some human limb muscle research indicates that strengthened limb muscles are able to maintain near-maximal force-generating ability for up to 2–4 weeks after the cessation of a training program.[52–55] Longer periods of detraining demonstrate more significant decreases in limb muscle strength. To the authors' knowledge, only a few respiratory muscle-training studies have addressed the concept of detraining following a strength-training program. In a recent study, Romer and McConnell examined detraining effects following an inspiratory muscle pressure threshold training program in healthy participants.[56] The participants completed a 9-week training program and were followed for 18 weeks after the completion of the training segment. The largest decrease in strength occurred during the first 9 weeks of the detraining period (~7%). Trends were not followed between the first and ninth week of the detraining period. As stated previously, the limb muscle literature indicates that training effects last up to 4 weeks. Additional data points during the first 9 weeks of the detraining period might have provided more specific information regarding the time course of strength loss in the respiratory muscles. In 2000, Gosselink and colleagues examined detraining effects following an EMST program completed with individuals with multiple sclerosis.[57] These participants completed a 12-week training program in which they increased their expiratory muscle strength by 35% from baseline. The participants were followed for 6 months following the program and were able to maintain 30% of their strength above baseline at 3 months after training; at 6 months following training, the participants maintained 9% of their strength above baseline. Information regarding the week-to-week time course of strength loss is unknown for these participants.

Baker, Davenport, and Sapienza studied detraining effects for EMST in 32 healthy participants. Sixteen participants trained for 4 weeks (Group 1) and 16 participants trained for 8 weeks (Group 2).[1] All 32 participants were detrained for 8 weeks. The gains in maximum expiratory pressure for both groups were on the order of 40%–50%, and the detraining effects after 8 weeks of no training indicated approximately a 12% decrease in the maximum expiratory pressure gains regardless of the initial training duration.

SUMMARY

Respiratory muscle strength-training paradigms act, mechanistically, similar to limb muscle strength-training paradigms. The functional outcomes can be broad, affecting multiple functions related to breathing, cough, speech, and swallow. More work on patient groups is necessary to define the individual patient outcomes, define the boundary between strengthening and fatigue, and determine the potential central plastic changes that may occur with this exercise protocol.

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