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Expiratory Muscle Training Increases Pressure Support in High School Band Students

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Summary: An experiment to increase expiratory muscle strength for instrumentalists, using a high-intensity, low-repetition expiratory training method, was conducted with 40 healthy high school band players. Subjects trained five days per week for 2 weeks using four sets of six training breaths for a total of 24 training breaths with a spring-loaded pressure relief valve that provided an adjustable threshold. The training valve pressure was set at 75% of the subject's measured maximum expiratory pressure (up to 80 cm H_2O). Results demonstrated that high-intensity, low-repetition expiratory exercises significantly increased expiratory pressure generating capacity in these subjects and the degree of the training effect was similar regardless of the instrument the band member played. The training effect occurred within 2 weeks of initiating expiratory muscle training. Thus, this simple method of expiratory-specific strength training is effective and efficient for increasing expiratory pressure support in high school band students and has possibilities of a respiratory support device for many high pressure generating purposes. The potential mechanisms of the training effect are discussed. **Key Words:** Pressure—Training—Muscle strength.

INTRODUCTION

The ability to produce sound from a musical instrument that requires the musician to blow air into the instrument is, in part, a function of the pressure the musician can generate. Contraction of the expiratory muscles, the elastic recoil of the respiratory structures, and the contraction of the oral-pharyngeal muscles are the sources used to produce this pressure. The first two pressure sources are responsible for the generation of subglottal pressure, which is referred to as pressure support. Pressure support is the foundation of all other pressure changes. This subglottal pressure provides the driving force for air to move out of the lung, into the upper airways, out of the mouth, and into the instrument. The action of the larynx, pharynx, oral cavity, mouth, and lips shape the subglottal pressure. The frequency of sound (range) is also a function of the driving pressure, that is, the greater the pressure, the higher the note that can be produced. The driving pressure is also essential for the control and duration of a sustained note.

The importance of pressure support has long been recognized and many techniques have been developed to increase support and to control the support pressure. The magnitude of the support is a direct

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function of the musician's ability to generate pressure with active contraction of the expiratory muscles. 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 positive subglottal pressure. Therefore, controlled relaxation of the diaphragm appears to be very important in support control. However, the contraction of the diaphragm counteracts the actions of the expiratory muscles in the generation of the magnitude of support. Thus, inspiratory training techniques that are designed to increase diaphragmatic strength do not increase the subglottal pressure needed for expiratory tasks, although we commonly come across individuals who believe they are contracting their diaphragm to produce positive expiratory pressures.

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, and help maintain a tight abdomen, but do not increase one's ability to generate greater maximum expiratory pressures (MEP) primarily because the muscle training techniques are task specific. This means that in order for a person to increase expiratory pressure generating strength, an exercise that requires them to actively contract the expiratory muscles to blow harder is mandatory.

Several devices have been developed to increase the resistance to expiratory airflow, making it harder to blow air out the mouth. This type of exercise meets the activity specific requirement for muscle training, but provides only modest increases in MEP. The magnitude of the resistance used for training varies but is, generally, a high-repetition, low-intensity exercise that requires the person to generate pressures less than 50% of MEP. The magnitude of the pressure is a function of the airflow rate, which decreases as the expiration progresses. This reduces the expiratory muscle effort with decreasing airflow rate and reduces the expiratory muscle training effect. Forcing the person to maintain a constant airflow rate for the entire expiratory effort in part compensates for this declining expiratory effort effect. With most resistive respiratory muscle training methods, MEP increases ranged from 20% to 30% with training.[1]

Our laboratories developed a high-intensity, low-repetition inspiratory training method that replaces the resistance devices with a pressure relief valve. The pressure relief valve has an adjustable spring that holds a cover over the breathing tube. It keeps it closed until sufficient pressure is produced to overcome the spring strength. The cover is then pushed open and air flows. The method requires the person who is using the device to generate a minimum pressure throughout the entire breath for lung volume to change. This specifically exercises the respiratory muscles in a breathing specific manner. The ability of the respiratory muscles to generate force changes with changing muscle length (related to lung volume change). The pressure relief valve used for inspiratory muscle training increases the respiratory muscle demand as lung volume increases, the diaphragm gets shorter, and the ability of the diaphragm to generate force decreases. This provides a greater muscle training effect over the entire inspiratory effort when compared to resistive methods. The mechanism that is most likely responsible for this change is neural, particularly when the training period is short in duration. The strength of a muscle relies not only on the structural integrity of the muscle but also on the ability of the nervous system to properly activate the muscle. Previous literature on strength training of the limbs suggests that training results in adaptive changes to the nervous system, allowing more specific muscle movements and better coordination of control, resulting in greater force generation.[2,3]

Our previous results, with other subject groups, appear to support the limb literature. We have reported an average 50% increase in maximum inspiratory pressure (MIP) in normal young adult subjects.[4] The inspiratory muscles were trained with the valve pressure set at 75% of maximum inspiratory pressure. This increase in maximum inspiratory pressure is greater than previous reports using resistive methods. A similar increase in MIP was obtained in a patient with laryngeal papilloma and documented as having a high laryngeal resistance.[5] We hypothesized that inverting the pressure relief valve to have subjects expire through it would provide an expiratory muscle exercise specific to breathing out and increase muscle strength as measured by the MEP. This hypothesis was tested in high school band students.

METHODS

Subjects

Forty healthy young males and females, naïve to expiratory strength training, volunteered to participate. The nonsmoking, untrained participants were an average of 21 (± 1) years old. Individuals were excluded for a history of chronic respiratory or neuromuscular disease, previous thoracic surgery or trauma, or chest wall compliance limited by obesity.[6] This study was reviewed and approved by the University of Florida Institutional Review Board. The study was explained to the subjects and informed consent obtained from their parent or guardian and assent obtained from the subject prior to enrollment into the study.

Maximum expiratory pressure measurement

Subjects underwent initial pretraining testing of MEP. Subjects refrained from strenuous physical activity prior to the MEP test. Testing occurred at the same time of the day for both pre-expiratory muscle training and postexpiratory muscle training. Expiratory muscle strength, measured as MEP, was recorded at the mouth by a pressure manometer. The measurement apparatus consisted of a mouthpiece connected to the pressure manometer by 50 cm of 2 mm i.d. tubing with a 14-gauge needle airleak. The experimenter read standardized written instructions to each subject. During testing, all subjects were required to stand with the nose occluded with clips. After inhaling to total lung capacity (TLC), subjects placed their lips around the mouthpiece and expired as forcefully as possible. Repeated measurements were taken, with a 1 to 2 minute rest between trials, until three measurements were obtained with 5% variability. Of these three values, the best MEP was recorded. MEP was defined as the greatest positive pressure obtained at the mouth sustained for at least 1 second while performing a maximal expiratory effort from TLC. MEP was retested weekly in the EMT group throughout the 2-week training period. MEP was measured at the beginning and end of the study period at the same time as the EMT and control group. Each EMT subject had his or her own individual training device. Individual training devices were labeled and stored in a locked room.

Expiratory muscle training

The strengthening protocol consisted of high-load EMT designed to improve maximal expiratory force production. Subjects trained with a spring-loaded pressure relief valve that provided an adjustable threshold load to their expiration. A load between 14 and 80 cm H_2O threshold pressure was regulated by adjusting the spring compression. Subjects were instructed to refrain from heavy exertion for 15 to 20 minutes prior to, and upon completion of, performing EMT. The training valve pressure was set at 75% of the MEP for each subject's individual MEP up to a maximum of 80 cm H_2O. The EMT group trained together under the supervision of an investigator 5 days per week, Monday through Friday. All subjects expired at their assigned training load for the daily session. Each EMT daily session consisted of four sets of six training breaths for a total of 24 training breaths. A training breath lasted approximately 3 to 4 seconds, and was separated by a 10 to 15 second rest. The daily training session lasted approximately 10 minutes. At the end of week one, the MEP for the EMT group was measured and the threshold pressure adjusted to 75% of the new MEP. At the end of week 2, MEP was measured in all EMT and control subjects.

Statistical analysis

Baseline differences in anthropometrical data between subjects were compared using an independent Student's *t* test. All *t* tests did not reach statistical difference at $p = 0.05$. A two-way within subjects analysis of variance (ANOVA) analyzed the effects of EMT on MEP. Significant interactions were analyzed using univariate repeated measures and means comparison with Bonferroni correction.

RESULTS

The dependent variable MEP, was measured in all subjects prior to training with the pressure threshold device, and measured again following the subject's training with the device. The mean and standard deviation data for the females and males within each of the study groups (control versus trained) is presented in Table 1. As a function of sex, males produced higher MEP's than females regardless of study group. As a function of training, it is clear that the control group experienced no increase in MEP; in fact the female control MEP decreased by approximately 6 cm H_2O and the male control MEP slightly increased by less than 2 cm H_2O. On the other hand, the trained females' MEP increased 43 cm H_2O and the trained

TABLE 1. *Descriptive Statistics for Maximum Expiratory Pressure (MEP) as a Function of Sex (Female versus Male) and Study Group (Control versus Trained). Pre-MEP means are pressures developed prior to the training program. Post-MEP means are pressures developed after the training program.*

	Sex	Study Group	Mean	SD	N
Pre-MEP	Female	Control	95.07	20.10	7
		Trained	90.93	19.25	15
	Male	Control	106.68	14.29	7
		Trained	111.76	28.19	11
	Total	Control	100.87	17.81	14
		Trained	99.74	25.21	26
Post-MEP	Female	Control	89.26	16.03	7
		Trained	133.94	25.32	15
	Male	Control	108.86	8.73	7
		Trained	164.86	32.40	11
	Total	Control	99.06	16.03	14
		Trained	147.02	31.96	26

males' MEP increased 53 cm H_2O. These increases represent a 47% and 48% increase, respectively.

Initial analysis showed that the effect of instrument (wood versus brass) was not statistically significant [F (1,34) = 0.126, p = 0.725], nor was the effect of class (underclassmen versus upperclassmen), [*F* (1,34) = 0.534, p = 0.462]. These effects were subsequently removed from the model to enhance the power of the final analysis.

Statistically significant effects were found for the between subject main effects of sex and study group (Table 2). Test of within subject effects indicated a significant statistical interaction for MEP by study group (Table 3). These statistical results verify the direction and magnitude of the mean data described above, indicating that males produced higher overall MEPs than females and that MEP significantly increased for both sexes as a function of training.

DISCUSSION

The results of this study are the first demonstration that high-intensity, low repetition expiratory specific exercises significantly increase expiratory pressure generating capacity in adolescents. Further, the results also show that, regardless of the instrument played by the band member, the degree of the training effect is similar. This training effect occurred in both males and females with significant sex effects but no age differences. The training effect occurred in all subjects following 2 weeks of expiratory muscle training. Thus, this simple method of expiratory specific strength training is not only effective but also efficient for increasing expiratory pressure support in high school band students.

The mechanism of the training effect is quite simple to understand and should be understood in order to know how the outcome of reduced respiratory muscle effort is achieved. The process of moving air requires a driving force. The driving force for the respiratory system is the pressure gradient between the alveoli and the atmosphere. To inflate the lung, the inward driving force must be an alveolar pressure less than atmospheric pressure creating a pressure gradient into the lung. For expiration to occur, the driving force must be an alveolar pressure greater than atmospheric pressure, generating a pressure gradient out of the lung. In order for sound to be produced, air must be moved from the alveolar spaces through the lower conducting airways to the larynx, through the glottis, pharynx, and mouth. The driving force for this air movement is a pressure in the alveolar space greater than atmospheric pressure. The alveolar pressure is changed by two forces: (1) the passive, elastic properties of the respiratory system and, (2) active contraction of the expiratory muscles.

TABLE 2. *Results of Analysis of Variance for Between Subject Effects*

Source	Type III Sum of Squares	*df*	Mean Square	*F*	*p*
Intercept	916851.747	1	916851.747	1029.082	0.000*
Sex	9723.575	1	9723.575	10.914	0.002*
Study Group	11427.119	1	11427.119	12.826	0.001*
Error	32964.847	37	890.942		

*Significant at $p \leq 0.025$

TABLE 3. *Results of Analysis of Variance Tests for Within Subject Effects*

Source	Type III Sum of Squares	*df*	Mean Square	*F*	*p*
MEP	9651.895	1	9651.895	61.820	0.000*
MEP by Sex	429.331	1	429.331	2.750	0.106
MEP by Study Group	11228.155	1	11228.155	71.916	0.000*
Error (MEP)	5776.774	37	156.129		

*Significant at $p \leq 0.025$

Sound production usually begins at volumes above the functional residual capacity (FRC). The volume is increased by the action of the inspiratory muscles. At the end of the inflation, the inspiratory muscles relax and the respiratory system's elastic forces produce an alveolar pressure greater than atmospheric pressure that is predicted from the pressure-volume curve. Vocalization intensity decreases in proportion to the decrease in collapsing pressure as the lungs deflate to FRC. Thus, the passive pressure-volume nature of the respiratory system provides one component of the driving force for expiratory airflow. This passive elastic pressure component is regulated by the magnitude of the active inspiration. That is, the greater the inspiratory volume the greater the magnitude of passive elastic pressure. The passive elastic pressure is the basic driving force that is modified by the action of the expiratory muscles.

Active expiratory pressure is generated by contraction of the muscles that decrease the diameter of the abdomen and push the diaphragm up into the thorax. A decrease in thoracic diameter is achieved by pulling the ribs downward. Muscles, which act to pull the ribs down, assist in producing an active expiratory driving force. The active expiratory driving force squeezes the lung from the outside and the force of the squeeze generates a pressure that adds to the passive elastic recoil pressure. The muscles that produce this squeeze pressure are usually identified as the internal intercostal muscles and abdominal muscles. Contraction of the internal intercostal muscles decreases the intercostal space width and pulls the ribs down. The sheets of abdominal muscle surrounding the abdominal cavity (external oblique, internal oblique, and transversus abdominis) act to decrease the diameter of the entire abdomen. Because the contents of the abdomen are primarily noncompressible fluids, the pressure in the abdomen increases. The relaxing diaphragm provides an outlet for the pressurized abdominal contents. Thus, the decrease in the diameter of the abdomen pushes the relaxing diaphragm up into the thorax, compressing the lung and the air in the lung. To this action is added the decrease in diameter of the lower portion of the rib cage. The lower ribs are connected to muscles that have the spine or pelvic girdle as their opposite attachment. These muscles (external oblique, internal oblique, longissimus dorsi, ileocostalis dorsi, ileocostalis lumborum, serratus posterior inferior, and portions of the quadratus lumborum) form a circumferential band around the base of the chest wall. Contraction of these muscles pulls the lower ribs downward, which decreases the diameter of the lower thorax and prevents expansion of this part of the rib cage by the upward force of the pressurized abdominal contents. The lungs are being squeezed from the bottom. This means that the decrease in the thoracic base and upward push from the pressurized abdominal contents will compress the lungs, generating the active expiratory air pressure.

In order to produce sound, an instrumentalist must employ expiratory muscles to generate active expiratory air pressure. The motion of the lips provides the source of sound for brass instruments. Just as vocal fold motion is produced with sustained expiratory pressure at a particular threshold, sustained expiratory pressure is essential to vibrate the tissue of the lips. This threshold pressure requirement is as complex as the pressure threshold requirement for initiating vocal fold vibration. Lip tension, the acoustic impedance of the instrument, and the player's mouth cavity are all considered in determining the pressure threshold needed to initiate lip movement. To instrumentalists, this pressure minimum is commonly referred to as blowing pressure threshold.

In order to effectively produce a blowing pressure one must have an intact and strong respiratory pump to power the instrument as well as an intact oral cavity structure. That is why it is imperative for instrumentalists to have strong expiratory muscles. Without adequate expiratory muscle strength, contractile force would be decreased, lung volume could not be actively decreased, and pressure would not be increased. Thus, the active mechanism discussed above for increasing expiratory pressure could not be used. By employing the expiratory pressure threshold training program we were able to actively recruit the expiratory muscles and target their use at a maximal level. The respiratory muscles, like other skeletal muscles, responds to a conditioning program designed to *selectively* improve muscle strength.[7] When this technique occurs with the repetition of stimuli in the presence of a muscle load, a significant increase in strength occurs. Recall that in this study the load was 75% of the individual instrumentalists maximum pressure-generating ability and the experimental protocol required that the subjects trained with four sets of six training breaths. These parts of the protocol are key in achieving an increased effect of muscle strengthening.

In order to understand the importance of strengthening the expiratory muscles in instrumentalists, one must appreciate the degree of blowing pressure that is required by them when playing tones of particular intensities and frequencies. Fletcher and Tarnopolsky[8] measured the blowing pressure required for playing wind instruments and described the relationship between the degree of power emitted from the instrument and the blowing pressure requirement. Their findings showed that the amount of pressure that an instrumentalist might have to generate for a C5 and F5 tone on the order of 90 to 100 dB sound pressure (SPL) would fall within a range of 7 to 11 kPa, which converts to 72 to 113 cm H_2O or about 10 to 15 times more pressure than one would need to speak. This is a substantial level of pressure to generate and sustain if the tone must be held for a specific time, which is often the case. Also Fletcher and Tarnopolsky indicated a strong positive linear relationship between increasing frequency and increasing threshold-blowing pressure. Therefore, if an instrumentalist is required to sustain a tone of a high loudness level, that is, of 90 to 100 dB SPL at a moderate to high frequency of 500 to 1200 Hz, the task would require anywhere from 4 to 8 kPA of pressure generation.

The ability to generate a blowing pressure is dependent on many physical factors: the physique of the instrumentalist, the behavior of the lip-valve generator, and the size and shape of the instrument. But, the fact remains: if the expiratory muscles are too weak to initiate and sustain a tone, the behavior of the lip-valve generator and the size and the shape of the instrument are not meaningful. The idea of strengthening the expiratory muscles first in a training protocol for young band members is a bottom-up approach that is based on models of fluid dynamics. Simply put, you cannot bypass a resistance unless you have an adequate pressure source, and you cannot have an adequate pressure source if you do not have the ability to generate high contractile forces of the expiratory muscles, particularly when the demands, that is, frequency, intensity, and duration of the tone are critical for the performance of a musical piece.

High-intensity, low-repetition inspiratory muscle training regimens have been shown to increase inspiratory muscle force generating capacity.[5] During the present study, expiratory muscle strength training (EMST) duration was probably insufficient to elicit significant changes in muscle fiber type or cross-sectional area. As a result of strength training, changes in the motor program, excitability of the neuromuscular system, or both may occur. While the present study did not measure, and therefore cannot confirm which, if any, of these neural adaptations occurred in response to EMST, it is evident that neural adaptations can occur within the initial phases of strength training. An increase in strength is measurable far in advance of any significant increase in muscle mass. While improved strength is elicited within several days of training, significant increases in limb muscle cross-sectional area occur only after about eight weeks of training.[9] Additional evidence of neural adaptation is observed with crossover training. Crossover training occurs when unilateral strengthening of a limb muscle results in a small but significant improvement in strength of the untrained, contralateral limb.[10] Finally, significant strength gains are highly specific to the training mode, speed, and task.[3] Neural adaptations that may have potentially occurred post-EMST include improved ability to attain a maximal volitional contraction,[11] decreased co-activation of antagonist muscle groups, enhanced synchrony of motor unit firing,[12] increased reflex potentiation,[13] and more efficient motor programming.[13] In the future, such dependent measures should be included in order to describe these neural adaptations to training.

Finally, any metabolic changes to the muscle, at this point, can only be hypothesized. From the exercise literature, we do know that there is a certain metabolic responses to exercise and these responses are influenced by the duration and intensity of the exercise. The exact mechanism responsible for the production of ATP is dominated by different metabolic systems depending on the intensity of the task. For the type of task that was described in this paper, the energy to perform the exercise came primarily from an aerobic mechanism. This means that ATP is produced inside the mitochondria and involves a complex interaction between the Krebs cycle and the electron transport chain.[2] How the metabolism of the muscle may be enhanced or changed by this type of training as it varies in duration and/or intensity requires continued study that would focus on the biochemical pathways that result in the production of ATP.

CONCLUSION

After 2 weeks of high-intensity EMST at approximately 75% of MEP, expiratory pressure significantly increased. The rapid strength gains suggest neural and muscle mechanical adaptations to EMST. The exact mechanism of these adaptations remains unknown. Neural adaptations to training include increased neuromuscular excitability, enhanced coordination, improved ability to obtain a maximum vital capacity and more efficient motor programming. Future research is required to elucidate the specific mechanisms of expiratory pump adaptations to EMST.

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