Pulmonary rehabilitation in chronic respiratory insufficiency · 3

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Ventilatory muscle training

Roger S Goldstein

Although in recent years ventilatory muscle function has received considerable attention, the role of ventilatory muscle training within the context of the management of patients with altered ventilatory function remains unclear. It is likely that ventilatory muscles will function less well—that is, will be unable to maintain their required or expected force[1] if their rate of energy consumption exceeds that of their energy supply. The energy demands are influenced by the work of breathing and the integrity of the contractile apparatus,[2–4] whereas the energy supply is determined by the force and timing of muscle contraction and by the delivery of oxygen to the tissues.[5] Training of the ventilatory muscles must follow the basic principles of training for any striated muscle with regard to the intensity and duration of the stimulus, the specificity of training, and the reversibility of training. It is equally important to evaluate the outcome of training with appropriate end points. Although there are many conditions in which the ventilatory muscles are involved, most published reports have been of healthy volunteers or subjects with chronic airflow limitation. This paper will discuss the standard methods used for testing and training of the ventilatory muscles, the rationale for such training, and the results of training programmes. It will end with conclusions for the clinician as to whether it is appropriate to include ventilatory muscle training as part of the management of those in whom the ventilatory system is involved.

Muscle strength

As in any striated muscle, the force of contraction will be determined by (1) the length of the muscle (length-tension relationship), (2) whether the contraction is associated with shortening (force-velocity relationship), (3) the strength and frequency of stimulation (force-frequency relationship), and (4) the integrity of the contractile apparatus.[6] The length of the respiratory muscles varies with lung volume. Inspiratory muscle contractile force is greatest between functional residual capacity (FRC) and residual volume (RV), and expiratory muscle contractile force is greatest at total lung capacity (TLC). Although a bedside evaluation of a short sniff or a strong cough will reflect a subject's respiratory muscle strength, the standardised laboratory method of evaluating strength is that described by Black and Hyatt[7] in which the subject is encouraged to make maximal efforts against a closed airway. More invasive measurements include ballooned catheter assessments of oesophageal (pleural) and gastric (abdominal) pressures and their difference, the transdiaphragmatic pressure (PDI). The PDI recorded during a sniff from FRC provides an accurate and reproducible measure of inspiratory muscle strength[8] and oesophageal pressure recorded during a cough reliably reflects expiratory muscle strength.[9] Recently there has been considerable interest in bilateral simultaneous transcutaneous phrenic nerve stimulation in the assessment of diaphragmatic strength. Not only is this method relatively painless, but it allows for an accurate and reproducible measure of muscle strength without requiring a maximal effort from the subject. The amplitude of the shock is monitored from the muscle mass action potential (M wave) recorded with surface electrodes. A non-invasive measure of diaphragmatic contractility can be achieved by measuring mouth pressure against an occluded airway during phrenic nerve twitch stimulation. With the glottis open and the airway closed at the mouth, mouth pressure is a good estimate of the overall pressure change on the pleural surface.[10 11]

Muscle endurance

Ventilatory muscle endurance will be determined by the composition of muscle fibre types, the adequacy of the blood supply, and the integrity of the contractile apparatus.[6] It will also be influenced by the force and duration of contraction and the velocity of shortening during the contraction. Because the force of contraction is expressed in relation to its maximal value, muscle strength will have an important influence on endurance.

TESTS OF ENDURANCE

Hyperpnoea

The laboratory evaluation of endurance has included measurements of sustained ventilation and of sustained pressure. The simplest is the maximum voluntary ventilation (MVV), a brief period (12 or 15 seconds depending on the study) during which the

University of Toronto, Toronto, Ontario, Canada
R S Goldstein

Reprint requests to:
Dr R S Goldstein, West Park Hospital, 82 Buttonwood Avenue, Toronto, Ontario M6M 2J5, Canada

subject is encouraged to sustain maximum ventilation with no extrinsic ventilatory load. The test reflects voluntary neural drive, airway resistance, and respiratory muscle strength.[12 13] In 1968 Tenney and Reese[14] studied isocapnoeic hyperpnoea and reported that the logarithm of endurance time varied linearly (negative slope) with the percentage of maximal breathing capacity (20 second MVV). They also reported that estimates of power of breathing against endurance time were consistent with a simple model in which energy is derived from a fixed finite store and from a steady supply. The rate of supply was estimated to be adequate to sustain ventilation at 55% of maximal breathing capacity. Leith and Bradley[15] later evaluated the influence of ventilatory muscle endurance using sustained isocapnoeic hyperpnoea and noted that the sustained ventilatory capacity when plotted against endurance time became asymptotic at approximately 80% of MVV. Studies among healthy volunteers, subjects with chronic obstructive pulmonary disease, and those with cystic fibrosis have indicated that their maximum sustained ventilatory capacity for 15 minutes is between 60% and 100% of MVV.[16–18]

Resistive loading

Measurements of endurance based on the time for which subjects can overcome alinear inspiratory resistive loads have become popular following a classic study by Roussos and Macklem.[19] In this study healthy volunteers were invited to breath to exhaustion against an alinear inspiratory resistance at a given, predetermined transdiaphragmatic pressure which was measured with two balloons and displayed to the subject on an oscilloscope. At FRC the PDI that could be generated indefinitely was approximately 40% of the PDImax, with contraction and relaxation times being relatively equal. In a subsequent study these authors showed that, if the same inspiratory load applied at FRC was applied at a higher lung volume, the inspiratory muscle endurance time was reduced (fig 1).[20]

Given that the diaphragm contracts during inspiration it should tire more rapidly if, at any given tension, the ratio of inspiratory time to the duration of the breathing cycle (TI/TTOT) is increased. Bellemare and Grassino[21 22] have pointed out that the duty cycle is an important component of inspiratory muscle endurance. For the diaphragm they proposed a tension-time index (TTDI) which incorporated both the pressure (PDI/PDImax) and the duty cycle (TI/TTOT). The TTDI in healthy volunteers becomes critical at about 0·15. This critical index of work is similar to the tensions known to cause limitation of blood flow in other skeletal muscles. In an open chest animal model[23] it was shown that blood flow is limited beyond a TTDI of 0·2. The physiological implication of this finding is that, if the diaphragm is obligated to exceed its critical tension, its ability to perform work will be limited by its $\dot{V}O_2$ during relaxation when blood flow is restored and it may fail. This explanation was also suggested by Tenney and Reese[14] to explain the limitation during sustained hyperventilation at greater than 50% of maximal breathing capacity. In measurements of endurance that use alinear inspiratory resistive loads, the pattern of breathing should be standardised. If this is the case, highly accurate and reproducible measures of inspiratory muscle endurance can be made.

Threshold loading

In 1982 Nickerson and Keens[24] devised a method for measuring ventilatory muscle endurance as the sustainable inspiratory pressure which is the highest pressure a subject can generate in each breath for 10 minutes. A weighted plunger was used as an inspiratory valve which ensured that a minimum pressure was generated with each breath (fig 2). In 15 healthy volunteers aged between 5 and 75 years the mean (SE) sustainable inspiratory pressure was 82 (6) cm H_2O or 68% (3%) of their maximum inspiratory pressure. This method allows for reproducible measure-

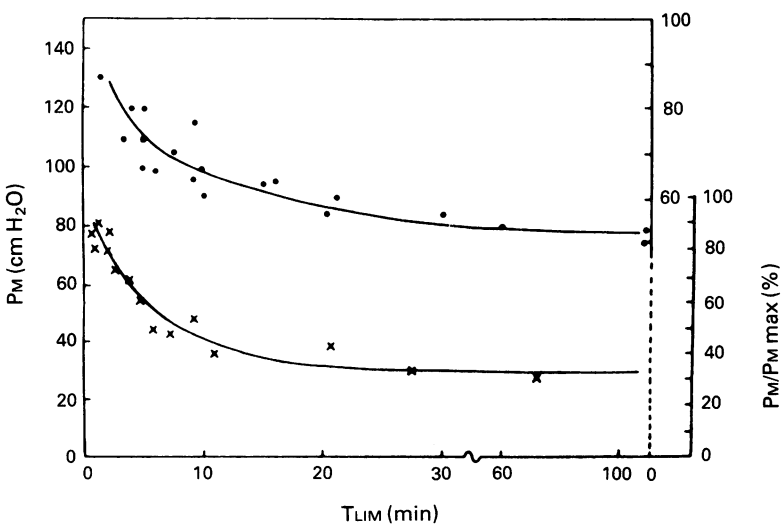

Figure 1 Effect of lung volume on endurance time (TLIM) in minutes. Left ordinate: mouth pressure (PM); right upper ordinate: mouth pressure as percentage of maximum (PM/PMmax) at functional residual capacity (●); right lower ordinate: PM/PMmax at functional residual capacity + 0·5 inspiratory capacity (x). Note difference in asymptotic value of PM/PMmax at the two lung volumes. Reproduced with permission from reference 20.

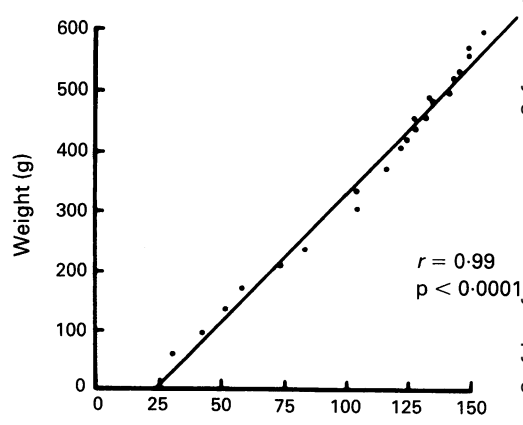

Figure 2 Relationship between weight on plunger and threshold mouth pressure. Reproduced with permission from reference 24.

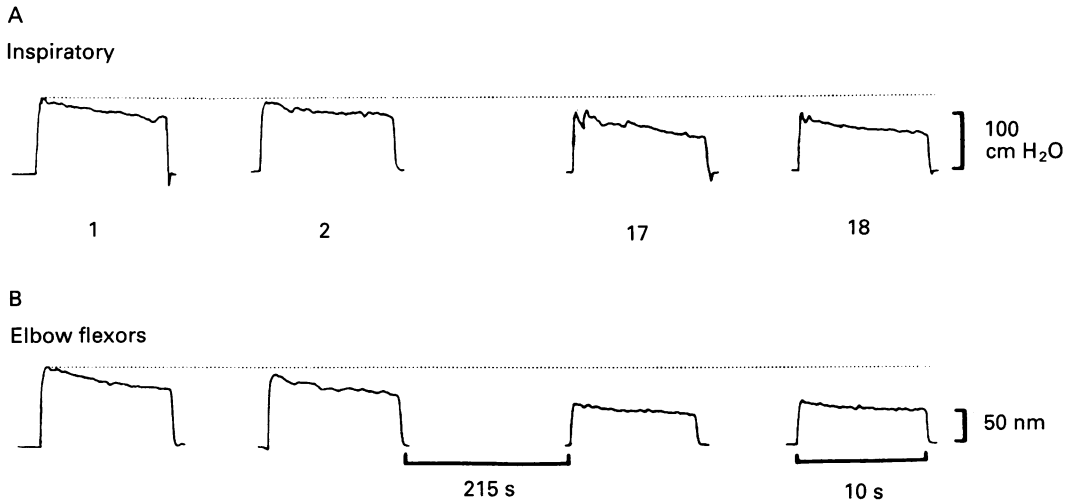

Figure 3 Records from a typical study in a control subject. Repeated maximal static contractions of (A) the inspiratory muscles at functional residual capacity and (B) the elbow flexor. The contractions lasted 10 seconds with rest intervals of five seconds (duty cycle of 67%). Note the decline in peak and average sustained force was less for the inspiratory muscles than for the elbow flexors. Reproduced with permission from reference 29.

ments of ventilatory muscle endurance. Clanton *et al*[25] have shown that both inspiratory flow rate and duty cycle will influence the sustainable inspiratory pressure and therefore these variables should be standardised. Chen and Kuo[26] tested 160 healthy volunteers using this technique and reported that (1) endurance was greater in men who were physically active than those who were sedentary, (2) it was higher in men than in women, and (3) it decreased with age. Martyn *et al*[27] described an incremental test of ventilatory muscle performance in which threshold loads were progressively increased at two minute intervals. Subjects began with a low load and continued to breath until they could no longer inspire. The authors concluded that this two minute incremental loading test was a simple assessment of ventilatory muscle performance. The practical significance of such a test remains to be explored.

Repeated maximal static contractions

A more recent approach to the measurement of respiratory muscle endurance described by Gandevia *et al* involves repeated maximal contractions against a closed airway.[28] With this technique it has been suggested that the inspiratory muscles recover more rapidly from fatigue than do the expiratory muscles or limb muscles (fig 3). This technique has the advantage of allowing respiratory muscles to be compared with limb muscles under similar circumstances and its use has been reported both in healthy volunteers and in subjects with respiratory disease.[28 29]

Histochemical composition

It has become clear that the performance of the respiratory muscles is linked to their histochemical composition (table 1). This property of all skeletal muscle is reflected in its fibre types. Generally type I fibres have a high oxidative capacity and a relatively low concentration of glycolytic enzymes. They are activated by smaller motor neurones recruited early during the orderly recruitment of motor units and, although they produce low levels of force relative to their cross sectional area, they are fatigue resistant. The time from the onset of contraction to peak tension is slow and they are referred to as slow twitch fibres. Type IIA muscle fibres are intermediate in oxidative capacity, enzyme concentration, and fatigue resistance. Type IIB muscle fibres are low in oxidative capacity and have a high concentration of glycolytic enzymes. They are activated by larger motor neurones recruited later during the orderly recruitment of motor units and they fatigue rapidly. The time from the onset of contraction to peak tension is fast and they are referred to as fast twitch fibres. In the diaphragm of normal adults there are approximately 55% type I fibres whereas premature infants, who may be prone to muscle dysfunction, have less than 10%.[30]

Rationale for training

Initial studies of ventilatory muscle training have been on healthy volunteers.[15 24 25] When healthy volunteers are encouraged to breathe to exhaustion[31] at levels of ventilation close to

Table 1 Properties of muscle fibre types. Reproduced with permission from reference 6

	Fibre type		
Characteristic	*I*	*IIA*	*IIB*
Twitch type	Slow	Fast	Fast
Colour	Intermediate	Red	White
Myosin ATPase activity	Low	High	High
Glycolytic capacity	Low	Intermediate	High
Oxidative capacity	High	High	Low
Mitochondrial density	High	High	Low
Endurance capacity	Excellent	Good	Poor

Table 2 Diseases sometimes associated with respiratory muscle weakness. Reproduced with permission from reference 57

Neurological diseases	Quadriplegia
	Myasthenia gravis
	Botulism
	Poliomyelitis
	Guillain-Barré syndrome
Muscle diseases	Myopathy (for example, steroids)
	Specific muscle enzyme deficiencies
Connective tissue diseases	Polymyositis
	Dermatomyositis
	Systemic lupus erythematosus
Endocrine disorders	Thyrotoxicosis
	Cushing's disease
Metabolic disorders	Hypophosphataemia
	Hypocalcaemia, hypomagnesaemia
	Metabolic alkalosis

Table 3 Effect of hyperpnoeic training on ventilatory muscles. Reproduced with permission from reference 51

Reference	No. of subjects	Endurance		Course (weeks)	Response* (%)	Better than control subjects? (Yes or no)
		Duration (min)	Frequency (weeks)			
Leith and Bradley[15]	4 normal	20–30	5	5	19	Yes
Keens *et al*[16]	4 normal	25	5	4	22	—
	4 with cystic fibrosis	25	5	4	55	Yes
Belman and Mittman[18]	10 with COPD	30	5	6	33	—
Levine *et al*[17]	15 with COPD	15	5	6	41	Yes
Reis and Moser[71]	5 with COPD	30	5	6	16	No

*Increase in maximum sustained ventilatory capacity. COPD—chronic obstructive pulmonary disease.

their MVV there are measurable changes in the pressure-frequency relation of the diaphragm, the ratio of high to low frequency power of the diaphragmatic electromyograph, and in the PDImax. These observations suggest that ventilatory endurance at high levels of ventilation may be limited by ventilatory muscle fatigue. Such levels of ventilation are rarely encountered in day-to-day activities and therefore ventilatory muscle training is unlikely to be of value among healthy individuals. When ventilatory muscle training was applied to healthy volunteers in peak athletic condition ($\dot{V}O_2max$ > 60 ml/kg/min) it did not influence maximum oxygen uptake or endurance cycling at 90% of maximal power output.[32]

In contrast, there are several diseases in which the ventilatory muscles may influence gas exchange, the level of physical activity, or the sensation of dyspnoea (table 2). Such limitations unquestionably influence the quality of life and, if muscle training were to result in a useful functional improvement, it would be indicated as part of the management of these conditions.

Methods of ventilatory muscle training

As in testing, the training regimens have focused on repeated maximal inspiratory and expiratory efforts against a closed airway for strength training and isocapnoeic hyperpnoea, resistive and pressure threshold loads for endurance training. Isocapnoeic hyperpnoea has been shown to improve test function among healthy volunteers and subjects with cystic fibrosis and chronic obstructive pulmonary disease (table 3). Leith and Bradley[15] have shown that, after five weeks of ventilatory muscle training, subjects trained for strength increased their strength by 55% whereas those who trained for endurance increased their maximum sustained ventilatory capacity from 81% to 96% of their MVV (table 4). This very well designed study emphasised the importance of the specificity of training and the importance of the stimulus being sufficiently high to effect training. The programme consisted of training for five days a week for five weeks. Strength trainers performed repeated maximal static inspiratory and expiratory manoeuvres against obstructed airways, and endurance trainers performed isocapnoeic hyperpnoea to exhaustion at levels of ventilation equivalent to 50% MVV.

Alinear resistances have also been applied to the inspiratory muscles for training. The response to training has been measured either as the maximum tolerable resistance or as the time during which a given load may be sustained. Pardy *et al*[33] used a simple home programme in which subjects inspired for 30 minutes per day (in two 15 minute sessions) against their critical inspiratory resistance while watching television or reading a book. At the end of two months of training the critical resistance that could be tolerated by the subject had increased. Many training programmes have incorporated this approach. Unfortunately, subjects often change their breathing strategy to one of slow, deep inspirations in order to tolerate more easily the inspiratory resistance. This strategy almost certainly reduces the inspiratory load to a level below that necessary to induce training. It is therefore essential that the breathing pattern is controlled during training. Belman and Shadmehr[34] overcame this issue during resistive ventilatory muscle training by using a single orifice resistance together with a target feedback device. The feedback device regulated breathing frequency, duty cycle, and mean pressure. Two other studies have reported the influence of resistive training during which there was some control over the pattern of breathing and the mean mouth pressure achieved.[35 36]

Clanton *et al*[37] showed the effectiveness of the threshold loading technique in training four healthy volunteers for half an hour each day over 10 weeks. The mean (SD) maximal inspiratory pressure increased by 50 (9) cm H_2O and the endurance time for a load set at 65% of the initial maximal inspiratory pres-

Table 4 Ventilatory muscle response to training. Reproduced with permission from reference 15

	Control group	Strength trainers	Endurance trainers
Strength at FRC			
PEmax	4 (6)	57 (9)*	10 (9)
PImax	−2 (6)	54 (16)*	9 (13)
Lung volumes			
TLC	0·7 (1·3)	4·6 (1·4)*	1·9 (0·8)
VC	−0·3 (2·2)	3·6 (0·8)	3·1 (1·2)
Sustained ventilatory capacity (% MVV)	4·5 (1·5)	3·8 (1·5)	15·5 (5·0)*
MVV at 15 seconds	0·8 (4·0)	2·0 (0·7)	14 (4·7)*

FRC—functional residual capacity; PEmax—maximum expiratory pressure; PImax—maximum inspiratory pressure; TLC—total lung capacity; VC—vital capacity; MVV—maximum voluntary ventilation. Values are Δ% (SE). *Statistically significant change (independent *t* test).

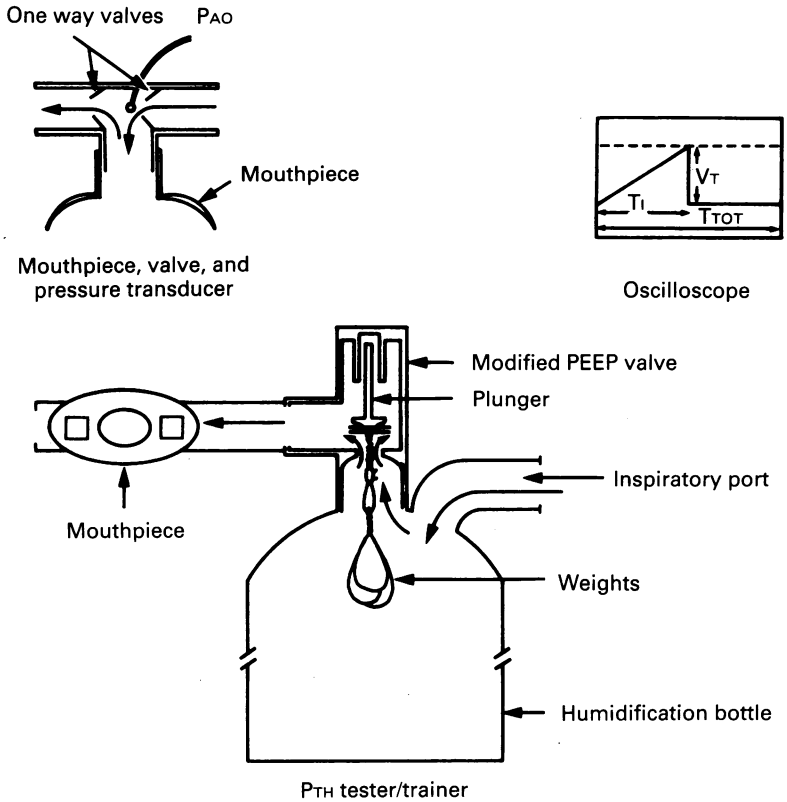

Figure 4 Diaphragm of PTH tester/trainer with details of mouthpiece and information shown to subject during testing. PAO—pressure at the airway opening (expressed as mean mouth pressure); PEEP—positive end expiratory pressure. Reproduced with permission from reference 38.

sure increased by six minutes. Both Clanton *et al*[37] and Goldstein *et al*[38] controlled for duty cycle, frequency, and tidal volume (fig 4).

Response to training

Normal skeletal muscles will adapt to training provided that the appropriate stimulus is applied.[39–43] The basic principles of training are that (1) for muscle fibres to change structure and function they must be stressed (overloaded) above a critical threshold; (2) training is specific for the stimulus—that is, strength training will increase fibre size (muscle hypertrophy) whereas endurance training will increase oxidative enzymes and mitochondrial density, myoglobin content, and capillary density; (3) training is reversible and the effects will be reduced (deconditioning) if the training ceases. Ventilatory muscles will undergo extensive metabolic adaptation to chronically increased respiratory loads, presumably to optimise muscle performance.[40–42 44] It appears, however, that training does not produce any major shifts in the proportion of slow twitch and fast twitch fibres in skeletal muscle although there are no longitudinal studies that have encompassed a large enough sample and included all of the techniques used in the identification of fibre type interconversion.[45] Muscle will also adapt to maximise its length-tension characteristics by altering the number of its sarcomeres.[46–49] This process of adaptability of skeletal muscle may have an important bearing on the indications for training those patients with chronic respiratory disease. Keens *et al*[16] noted that subjects with cystic fibrosis had a 36% higher ventilatory muscle endurance than did healthy volunteers and suggested that a process of adaptation to the chronic stress of breathing against high inspiratory loads had occurred. Similowski *et al*[50] evaluated the contractile properties of the human diaphragm during chronic hyperinflation and concluded that, for the same lung volume, the function of the diaphragm was as good as that measured in healthy volunteers.

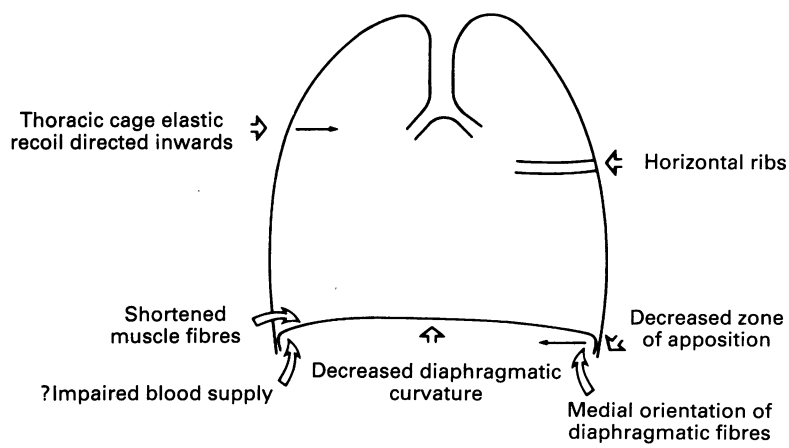

Figure 5 Detrimental effects of hyperinflation on respiratory muscle function. Reproduced with permission from reference 53.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

There are good theoretical reasons to think that subjects with chronic obstructive pulmonary disease may be expected to develop ventilatory muscle dysfunction as their work and energy cost of breathing are substantially increased, whereas their capacity to endure such work may be diminished by mechanical and other influences.[51 52] The main mechanical change is that of hyperinflation which shortens the inspiratory muscles and puts them in a disadvantageous position on their length-tension curve. Hyperinflation results in a significant decrease in the size of the zone of apposition between the costal fibres of the diaphragm and the rib cage (fig 5).[53] The increased airway resistance results in an increased inspiratory load with each breath so the work of breathing is increased at rest and during exercise. Chronic obstructive pulmonary disease also influences the size and mass of the diaphragm as part of the generalised weight loss that patients often experience. This weight loss probably relates to the increased caloric requirements in subjects in whom intake is inadequate. Another influence on ventilatory muscle function in patients with chronic obstructive pulmonary disease may be that of medications such as steroids which could further compromise muscle function. These influences on muscle function may be closely linked to dyspnoea and to limitations in exercise performance.

A superb review of the physiological and clinical aspects on training of the respiratory muscles has recently been published by Grassino.[54] Given that maximum inspiratory

pressure and PDImax is reduced, there may be merit for strength training. Since it has been recognised that the sensation of dyspnoea is related to the percentage of the maximum inspiratory pressure developed with each breath,[55] it is possible that strengthening the inspiratory muscles may lessen dyspnoea. Strengthening of expiratory muscles may assist with clearance of secretions. Endurance training might result in an increased capacity for higher levels of ventilation (such as in physical exercise) or for sudden increases in resistive load (such as in infectious exacerbations). There is some preliminary evidence that inspiratory resistive training may assist those with chronic respiratory failure in being weaned from mechanical ventilation by providing brief periods of fatiguing exertion alternating with periods of complete rest.[56] Subjects with chronic obstructive pulmonary disease (bronchitis, emphysema, asthma, cystic fibrosis, bronchiectasis) could, at least in theory, benefit from training.

Results of training programmes

A very large number of studies have evaluated the influence of ventilatory muscle training and the reader is referred to two recent reviews for details of several published studies.[51 57] In summary, it appears that subjects can be trained to improve their performance as measured by a particular test of endurance which is specific to the training modality. The evidence that such training results in functionally useful changes remains equivocal. Smith *et al*[58] recently reviewed 73 articles from a computerised bibliographical database and identified 17 relevant randomised trials of ventilatory muscle training in chronic airflow limitation. The study quality was assessed and descriptive information concerning the study populations, interventions, and outcome measurements was extracted. The authors then combined effect sizes across studies (the difference between treatment and control groups divided by the pooled standard deviation of the outcome measure). The criteria for methodological quality are shown in table 5. A primary analysis of these results showed that only MVV was associated with a significant p value for the effect size. A secondary analysis was undertaken in which studies were included only if ventilatory muscle training was associated with control of the breathing pattern. These studies were compared with those in which breathing pattern had not been controlled (table 6). The authors concluded that respiratory muscle strength and endurance will improve, but with no associated alterations in functional exercise capacity or laboratory measurements of exercise capacity.

Although most of the above studies have involved subjects with chronic bronchitis and emphysema, ventilatory muscle function has also been improved in subjects with cystic fibrosis.[16 59] Keens *et al*[16] were able to induce changes in ventilatory muscle endurance of equivalent magnitude to that of endurance training in seven subjects who participated in a four week physical activity training programme consisting of 1·5 hours per day of swimming and canoeing. A recent report by Weiner *et al*[60] describes the results of inspiratory muscle training among asthmatic subjects: 15 subjects (group A) received inspiratory muscle training and 15 (group B)

Table 5 Criteria for methodological quality. Reproduced with permission from reference 58*

Sample
- Random or consecutive sample (5)
- Arbitrary sample (or cannot tell) (0)

Similarity of groups
- Number of following variable in which groups were comparable: age, gender, forced expiratory volume, maximum inspiratory pressures, walk test distance (0–5)

Cointervention
- Number of following criteria met: comparable frequency of visits, comparable medication changes, comparable number of intercurrent illnesses (5 for 3/3; 3 for 1/3; 0 for 0/3)

Sham or masking
- Sham or masking undertaken (5)
- No sham or masking (0)

Compliance
- Training was hospital supervised (5)
- Home programme with reporting diary and patients periodically (4)
- Home programme with either diary or periodic review (3)
- Compliance not measured or cannot tell (0)

Observer masking
- Observers masked as to treatment groups (5)
- Not masked or cannot tell (0)

Standardised testing
- Encouragement standardised (5)
- Not standardised or cannot tell (0)

Follow up
- 90–100% follow up (5)
- 80–89% follow up (3)
- <80% subjects accounted for (1)
- Cannot tell (0)

*Numbers in parentheses indicate number of points in summary score.

Table 6 Sensitivity analysis of resistance studies with flow rates controlled versus those with flow rates not controlled. Reproduced with permission from reference 58

Variable/condition	No. of studies	Effect size (standard deviation units)	Effect size (natural units)	p	Homogeneity p	p value on difference in effect size between controlled and uncontrolled studies
Respiratory muscle strength (Pimax), controlled flow rate	5	0·51	8·2 cm	0·01	0·92	0·02
Respiratory muscle strength (Pimax), uncontrolled flow rate	6	−0·09	−1·8 cm	0·57	0·23	
Respiratory muscle endurance, controlled flow rate	4	0·41	10·3 l/min	0·06	0·93	0·09
Respiratory muscle endurance, uncontrolled flow rate	3	−0·08	−14·6 l/min	0·67	0·23	
Laboratory exercise capacity, controlled flow rate	2	−0·002	−0·02 ml/kg/min	0·99	0·10	0·30
Laboratory exercise capacity, uncontrolled flow rate	5	−0·17	−1·57 ml/kg/min	0·10	0·09	
Functional exercise capacity, controlled flow rate	3	0·30	88·9 m*	0·22	0·75	0·45
Functional exercise capacity, uncontrolled flow rate	4	0·07	18·4 m	0·71	0·05	
Functional status, controlled flow rate	2	0·65	1·87†	0·02	0·004	0·02
Functional status, uncontrolled flow rate	3	−0·13	−0·70	0·49	0·92	

Pimax—maximal inspiratory pressure; *12 minute walk test distance; †dyspnoea rate of chronic respiratory questionnaire in which 0·5 is minimal important difference.

were assigned to sham training in a double blind trial. Unfortunately the pattern of breathing during testing and training was not controlled. Subjects reported improvements in several indices of asthma in association with improvements in strength and endurance (fig 6) including a reduction in the amount of inhaled β_2 agonists.

NON-OBSTRUCTIVE VENTILATORY DISEASES

Although various other conditions have been associated with ventilatory muscle weakness (table 2), there have been few studies evaluating the role of ventilatory muscle training for individuals with such conditions. An early study reported by Gross *et al*[61] concerned six chronic quadriplegic subjects in whom muscle function was measured during breathing against several inspiratory resistances. This was also the first report of the use of power spectral changes in the electromyogram of respiratory muscles in the assessment of muscle fatigue. The maximum static inspiratory mouth pressure was measured at functional residual capacity and the critical inspiratory mouth pressure was established below which electromyographic changes of diaphragmatic fatigue did not develop. Following inspiratory muscle training for 30 minutes daily, six days a week, with loads sufficient to induce the electromyographic changes of fatigue, a significant increase in maximum inspiratory pressure and in critical inspiratory mouth pressure was noted. Whether such training protects against the clinical sequelae of an acute respiratory tract infection is unknown and has not been explored further. Ventilatory muscle training in tetraplegics has resulted in only small changes in their peak expiratory flow.[62] In patients with neuromuscular disease strength and endurance can be improved by training, but the influence of such training on exercise tolerance, coughing, and talking remains to be explored.[63]

Attempts at training in subjects with Duchenne's muscular dystrophy have not been found to be of clinical benefit.[64 65]

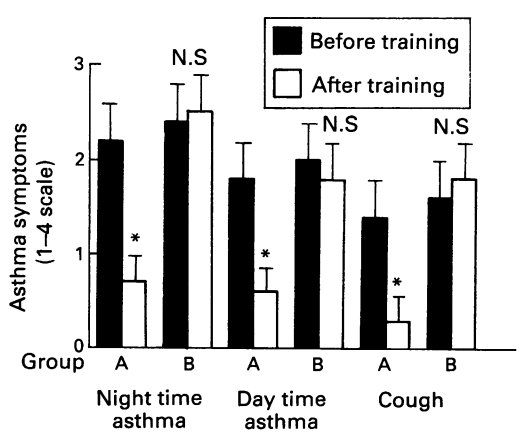

*Figure 6 Mean (SE) diary card data for asthma symptoms judged by the patients on a scale of 0 = no symptoms to 4 = very severe symptoms before and during the last two weeks of training. Group A received inspiratory muscle training and group B received sham training. *p < 0·05; N.S—not significant. Reproduced with permission from reference 60.*

Outstanding issues

The above studies of ventilatory muscle training raise a number of interesting issues. Firstly, the use of whole body exercise to evaluate ventilatory muscle function may not be appropriate as it involves many other muscle groups unrelated to breathing. The evidence that physical exercise is associated with ventilatory muscle dysfunction rests, in fact, on a few uncontrolled studies in a small number of subjects.[57 66] In these subjects exercise was associated with measured changes in the ratio of the high to low frequencies in the power spectrum of the diaphragmatic electromyogram.

Secondly, only a few studies have explored measurements of dyspnoea and of quality of life before and after ventilatory muscle training. Patessio *et al*[67] reported that there was a reduction in dyspnoea at each inspiratory load in trained subjects but not in a placebo group and concluded that training against an inspiratory resistance decreased the sensation of breathlessness in association with an increase in inspiratory muscle strength and endurance. Dekhuijzen *et al*[35] found no differences in psychometric measures of anxiety, depression, and physical complaints between those who completed a respiratory rehabilitation programme with inspiratory muscle training and control subjects who completed only a respiratory rehabilitation programme. Harver *et al*[36] reported that study subjects showed a decrease in dyspnoea after eight weeks of training which was reflected in an improvement in scores of the transition dyspnoea index (fig 7); control subjects did not. There is therefore a need for better quantification of the sensation of dyspnoea before and after ventilatory muscle training.

Thirdly, the role of expiratory muscle training has been relatively unexplored. During isocapnoeic hyperpnoea expiratory muscle training almost certainly occurs, but during inspiratory resistive loading the role of the expiratory muscles is less clear. Recent studies among healthy volunteers have confirmed that during exercise and during inspiratory resistive loading the expiratory muscles are increasingly activated and there may be changes to the power spectrum of their electromyogram.[68–70] Whether there is a specific role for expiratory muscle training remains to be clarified.

Finally, although harder to evaluate than strength or endurance training, improvements in the coordination of respiratory muscle function may be of benefit to some patients. Following whole body exercise, improvements in performance in the absence of cardiovascular or skeletal muscle training, may relate to improvements in neuromuscular coordination and the efficiency of breathing. Pursed lip breathing, which is learned naturally by some patients, may minimise lung hyperinflation. Emphasis on diaphragmatic and abdominal breathing may reduce dyspnoea. The role of training coordination needs to be defined as it might be complementary to whole body exercise and to specific training of the ventilatory muscles for strength or endurance.

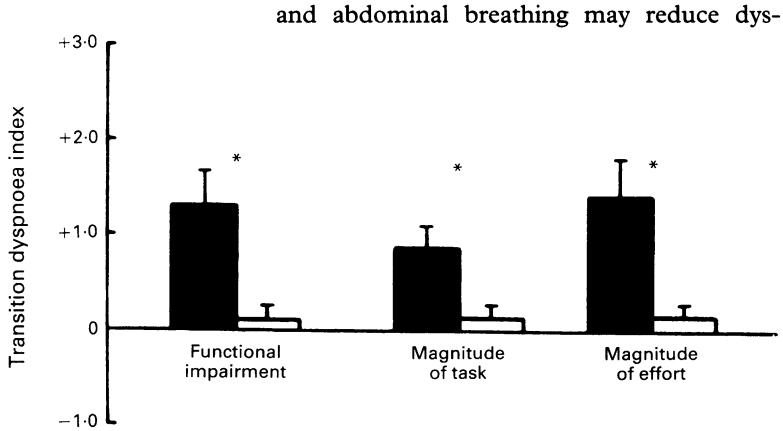

Figure 7 Mean scores for each category of the transition dyspnoea index for both experimental (solid bars) and control (open bars) subjects. Bar represents one standard error. Asterisk indicates significant difference between experimental and control conditions. Reproduced with permission from reference 36.

Conclusion

The subject of ventilatory muscle training is a fascinating study of the application of the principles of training any striated muscle to the muscles of the ventilatory system. Training for strength and endurance has been achieved in healthy subjects and in those with chronic airflow limitation but only a few studies are available that describe the influence of training in other conditions. When training has occurred the evidence that it results in changes that are functionally useful is, at best, equivocal. Training against resistive loads or pressure threshold loads has generally focused on the muscles of inspiration and there is little information on the effect of training the expiratory muscles. Natural adaptations of the muscles of respiration might mitigate against the need for training by maximising their function against chronically increased loads. Whole body exercise is probably an inappropriate outcome measure but studies of gas exchange, quality of life, and dyspnoea are still needed. The design of training programmes must take into consideration an adequate training stimulus, both in terms of intensity and duration, and must control for the pattern of breathing. Ventilatory muscle training is not recommended at present by this author as part of the routine management of those with chronic ventilatory conditions. However, there is considerable potential for research that might identify the appropriate population for training, the best modality of training, and the role of training within the context of other modalities of rehabilitation.

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