Ventilatory Muscle Endurance Training in Quadriplegia: Effects on Breathing Pattern

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Summary

We examined the effects of ventilatory muscle endurance training on resting breathing pattern in 12 C6-C7 traumatic quadriplegics at least 1 year post-injury. All subjects had complete motor loss below the lesion level. Subjects were randomly assigned to a training (N = 6), or a control group (N = 6). Baseline tests included measurement of resting ventilation and breathing pattern using mercury in rubber strain gauges for 20 minutes in a seated position; maximum inspiratory mouth pressure (MIP) at FRC, and sustainable inspiratory mouth pressure for 10 minutes (SIP); lung volumes, and arterial blood gases (ABG's). The training protocol consisted of breathing through an inspiratory resistor equivalent to 85% SIP for 15 minutes twice daily, 5 days a week for 8 weeks. Both trainers and controls attended the lab every 2 weeks for reassessment of MIP and SIP and the inspiratory resistance was increased in the training group as SIP increased. At the end of 8 weeks, baseline tests were repeated. All subjects had normal ABG's. There was a significant increase in mean MIP and SIP in both the control group ($30\% \pm 19\%$ and $31\% \pm 18\%$ respectively), and in the training group ($42\% \pm 24\%$ and $78\% \pm 49\%$ respectively). Although the absolute values for both MIP and SIP were greater in the training group than in the control group, the differences were not significant. The alterations in resting breathing pattern were also the same in both groups. Mean frequency decreased significantly in the control group (20.2/minute to 16.9/minute) and, while insignificant, the change in frequency in the training group was the same, 19.4/minute to 16.4/minute. Mean tidal volume (V_t) increased 18.2% of baseline V_t in the control group and 17.0% baseline in the trainers, resulting in no change in minute ventilation. As MIP and SIP increased similarly in both groups, the data from the control and trainers was pooled and timing changes re-evaluated pre- and post-study. A significant decrease in mean T_i/T_{tot} was observed, while no change in V_t/T_i was found. We concluded that the testing procedure

itself provided the stimulus resulting in a significant increase in MIP and SIP. The addition of training did not increase MIP and SIP further. The increased MIP and SIP resulted in a slower and deeper breathing pattern and a significantly shorter T_i/T_{tot} in both trainers and control subjects.

Key words: *Paraplegia; Quadriplegia; Respiratory muscles; Exercise training; Breathing control*

The breathing pattern of quadriplegics, as in other patients with respiratory muscle weakness, appears to be more rapid and shallow than in normals (Bergofsky, 1964). Maximum inspiratory mouth pressure (MIP), an index of ventilatory muscle strength, is often also markedly reduced as compared to normals (Gross *et al.*, 1980; Leith and Bradley, 1976). It has been demonstrated that the strength and endurance of the ventilatory muscles can be improved through a programme of inspiratory resistive training in normal subjects (Leith and Bradley, 1976; Clanton *et al.*, 1985a), chronic obstructive pulmonary disease (COPD) patients (Pardy *et al.*, 1981; Belman and Mittman, 1980), as well as quadriplegics (Gross *et al.*, 1980). Such training regimes have reported symptom relief and subjective improvements in breathing in quadriplegics (Gross *et al.*, 1980), and increased exercise performance in COPD (Grassino *et al.*, 1979). While such training regimes are thought to be important in protecting the ventilatory muscles against fatigue (Gross *et al.*, 1980; Pardy *et al.*, 1981; Grassino *et al.*, 1979), little information is available concerning the effects such training may have on resting breathing pattern or breathing control. We hypothesised that increasing the strength and endurance of the ventilatory muscles would alter resting breathing pattern by increasing tidal volume (V_t) and decreasing frequency (F). Such a slower, deeper breathing pattern is more efficient and might lessen the adverse effects of chronic hypoventilation which may occur in this group.

Methods

Twelve quadriplegics with complete motor loss below the level of the sixth and seventh cervical vertebrae were recruited from the outpatient department of the Rehabilitation Hospital's Spinal Cord Unit. All subjects were at least 1 year post-injury and were medically stable at the time of entry into the study. Informed consent was obtained from all individuals. Subjects were randomly assigned to a control ($N = 6$) and training group ($N = 6$). The baseline characteristics of the subjects are listed in Table I. A pre-test questionnaire was completed with each subject to document drugs, activity level, symptoms with activity, and electric versus standard wheelchair. Baseline pulmonary function tests, lung volumes, and arterial blood gases (ABG's) were measured. Functional residual capacity (FRC) was determined using a closed circuit nitrogen washout technique. Total lung capacity (TLC) was calculated by adding inspiratory capacity (IC) to the FRC measurement, and residual volume (RV) was calculated by subtracting FVC from TLC.

Maximum inspiratory pressure (MIP) was assessed using a comparable technique to that described by Black and Hyatt (1971), using a ± 100 cm H_2O Vali-

Table I Characteristics of quadriplegic subjects

	Level of lesion	Age	Height (cm)	FVC (% Pred)	FEV _{1.0} /FVC (%)
Trainers					
1	C6	27	175	47	100
2	C6	35	188	71	95
3	C7	28	180	85	86
4	C6 ACS	28	173	46	92
5	C6 ACS	31	164	85	69
6	C6 ACS	37	178	41	89
$\bar{X}$		31	176	63	
S.D.		4.1	7.9	20	
Controls					
1	C6	49	175	60	100
2	C7	22	180	58	91
3	C7	28	175	60	86
4	C6	26	183	85	72
5	C7	49	188	114	83
6	C6	36	195	55	100
$\bar{X}$		35	183	72	
S.D.		12	7.8	23	

ACS: Anterior Cord Syndrome.

dyne pressure transducer. Measurements were recorded at FRC. Subjects wore noseclips, and were instructed to breathe normally through a mouthpiece to establish FRC. Then at the end of a normal expiration subjects were instructed to breathe in against an occluded mouthpiece. Measurements were repeated a minimum of 10 times until 3 measurements within 5% of each other were recorded. The average of the 3 best measurements was calculated as MIP.

Sustainable inspiratory pressure (SIP) was used as an index of ventilatory muscle endurance. A modification of the technique described by Knickerson and Keens (1982) was employed which required that the quadriplegics only had to complete 2 endurance runs during each testing session. We built a similar threshold load device to that of Knickerson and Keens (1982) (Fig. 1). The relation between the weight applied to the threshold load device and the pressure that must be generated to produce airflow was linear, with an opening pressure of -8 cm H₂O. Once opened, flow was independent of pressure. The initial SIP test was conducted with the subject generating a pressure equivalent to 75% MIP. The subject was instructed to continue breathing against this load as long as possible. A single lead ECG and end-tidal CO₂ (Perkin-Elmer Medical Gas Analyzer, model 1100) were monitored throughout the run. The patient was also instructed to come off the device if subjective symptoms of dizziness, lightheadedness, or undue fatigue were experienced. If the subject was able to breathe against the load for more than 10 minutes, the second test was performed with a load equivalent to 85% MIP. If the first run was less than 10 minutes, the load was decreased to 65% MIP for the second run. A rest period equal to at least 10 times the length of the preceeding test was provided between each run (Nickerson and Keens, 1982). A pressure/time plot was constructed and the pressure that the subject could breathe against for 10 minutes was extrapolated from these two points. This extrapolated pressure at 10 minutes was called SIP.

ENDURANCE TESTING RIG

Figure 1 Ventilatory muscle endurance testing rig.

The trainers were provided with an inspiratory resistor that would require the generation of a pressure equivalent to 85% SIP at normal resting flow rates. The patient was instructed to breathe on this resistor while wearing nose clips, twice daily for 15 minutes, 5 days a week, for 8 weeks. Patients were allowed to set their own breathing rate and pattern during training and the training strategy employed was not monitored. Each patient was required to keep a daily diary to monitor performance. Both trainers and controls attended the lab every 2 weeks and repeated the tests for MIP and SIP. The inspiratory resistor was increased for the trainers as SIP increased.

Ventilation and breathing pattern were assessed using mercury in rubber strain gauges. Subjects were seated in their wheelchairs for a period of 20 minutes, and a single lead electrocardiogram was also recorded during this period. Subjects watched a videotaped movie to distract them from the monitoring devices. They were instructed to sit still and to avoid arm or trunk movements, both of which introduce artifacts in the strain gauge signals.

The ribcage strain gauge (RC) was positioned approximately 1 inch below the nipple level on the chest wall, and the abdominal strain gauge (ABD) was placed half way between the umbilicus and the xiphoid process, around the upper abdomen. These positions provided the maximum signal for both strain gauges. A 3 inch wide and half inch thick foam pad was placed over the spinous processes posteriorly under the strain gauges to protect the patient from undue pressure in this region during the test. The output signals from both strain gauges were converted into a DC voltage and were sampled at 20 Hz by a Hewlett Packard microcomputer (Model 9816) and simultaneously recorded on a polygraph (Model 2600S, Gould Recorder).

The calibration technique has been previously described in detail using respiratory inductance plethysmography (RIP) (Loveridge *et al.*, 1983), and strain gauges have been demonstrated to be equally as accurate as the RIP in normal

Table II Pulmonary function and lung volume data pre- and post-training

% Pred	Trainers (N = 6)		Controls (N = 6)	
	Pre	Post	Pre	Post
TLC	77.3 ± 9.5	82 ± 6.9	91.6 ± 17	91.6 ± 17
RV	107 ± 15	122 ± 24	125.5 ± 41	126 ± 38
FRC	78 ± 11	83 ± 15	82 ± 23	79 ± 18
IC	80 ± 12	95 ± 15	9 ± 25	104 ± 27
FVC	63 ± 20	64 ± 10	72 ± 23	75 ± 23

subjects using this calibration technique (Loveridge *et al.*, 1984a). Briefly, the strain gauges were calibrated in the seated position and its stability was checked at the end of the data acquisition period. During calibration, the subject wore noseclips and breathed through a pneumotachygraph for a period of 1 minute. The pneumotachygraph signal was integrated digitally. The analog signals from the RC, ABD, and the pneumotachygraph were sampled simultaneously at 20 Hz by a microcomputer. Only inspiratory data were analysed. Multiple linear regression analysis was used to determine RC and ABD calibration factors. The strain gauges were considered calibrated on the basis of the tightness of fit between the pneumotachygraph derived volume and the predicted volume from the summed RC and ABD signals. A summed RC and ABD volume less than 10% deviation from the pneumotachygraph measured volume was considered to be an acceptable calibration. At the end of the study, subjects again breathed through a pneumotachygraph for a 1 minute period and the accuracy of the RC and ABD calibration factors derived during the initial calibration were verified.

The best fit multiple linear regression equation from calibration was used to reconstruct a spirogram from the summed RC and ABD signals. Segments in which there were movement artifacts were identified from the chart recording and deleted from the computer data file prior to analysis. Subjects were indirectly observed through the test and any movement was noted on the chart recording. The peak (end of inspiration), and the trough (beginning of the next inspiration) were identified manually with the assistance of an *x-y* plotter on the computer. These values were then stored by the computer and used to determine tidal volume (V_t), inspiratory time (T_i) and expiratory time (T_e). The remaining timing components of the spirogram were derived from these values. The mean, standard deviation and coefficient of variation of each breath component was calculated. A computer histogram program was used with the tidal volume data to identify sighs (breaths greater than $3 \times$ mean V_t).

Results

Arterial blood gases were normal, and routine pulmonary function tests and lung volume measurements remained unchanged in both groups post-study (Table II). In 3 trainers with lower initial IC's (67–72% predicted), there was substantial improvement in IC post-training. However, the other 3 trainers

Table III Strength (MIP) and endurance (SIP) of the ventilatory muscles

	Strength (-CM H ₂ O Press)			Endurance (-CM H ₂ O Press)		
	Pre	Post	% Increase	Pre	Post	% Increase
Controls	81.7 ± 21.9	105.3* ± 16.3	30.2 ± 18.9	56.2 ± 15.4	71.8* ± 10.5	31.3 ± 18.0
Trainers	72.3 ± 17.3	100.7* ± 19.3	44.2 ± 32.7	50.2 ± 12.1	76.5* ± 15.2	58.7 ± 49.2

*P < 0.05.

with mean baseline IC's of 89% predicted showed little or no improvement with ventilatory muscle training. Although the increase in IC was insignificant for the group as a whole, it appears that those quadriplegics with low IC's can improve their IC considerably following inspiratory muscle training.

Only 1 patient experienced an elevation in end-tidal CO₂ consistently during the SIP runs with pre-test values ranging from 35–40 mm Hg and end SIP test values ranging from 50–58 mm Hg. This subject complained of no symptoms with this elevation in CO₂. The mean CO₂ for all other subjects were within normal limits. No ECG abnormalities were observed, and the mean elevation in heart rate at the end of the SIP runs for both groups was 16.2 beats/min. over the baseline heart rate that day.

Both the trained group and the controls significantly increased MIP and SIP post study (Table III). Although the changes appeared greater in the trainers, the standard deviations were large. The mean pattern of increase in SIP and MIP for the trainers appeared to be progressive over the 8 week study period, while the increases in the control group appear to level off after their fourth testing session (Fig. 2). There were no significant differences in MIP or SIP at any time period between the 2 groups.

Breathing pattern changes observed were the same for both the trainers and the controls (Table IV). Mean frequency was significantly reduced in the control group ($p < 0.05$), and while the difference was insignificant in the trainers ($p < 0.50$), quantitatively the decrease in frequency was the same. Mean tidal volume increased slightly in both groups allowing for minute ventilation to remain unchanged. No significant differences in timing were observed between the 2 groups following training, and there were no changes in the variability of any breath component between the 2 groups as assessed by comparing the coefficients of variation.

Only 2 controls and 1 trainer demonstrated any sighs during the pre-training evaluation, and there were no changes in sighing frequency observed post-training.

As both trainers and controls significantly increased MIP and SIP, the data was pooled, and the effects of increased MIP and SIP on breathing pattern was analysed for the group as a whole. This analysis confirmed the significant decrease in frequency previously observed in the control group only, and found that this was due to a significant increase ($p < 0.05$) in Te ($1.91 \pm .43$ to $2.48 \pm .75$). Mean Ti/Ttot for the 12 subjects was $0.405 \pm .049$ pre-test and decreased significantly ($p < 0.05$) to $0.374 \pm .046$ with the increased MIP and SIP.

Figure 2 Mean sustainable inspiratory pressures (SIP) and mean maximum inspiratory pressures (MIP) with standard deviation bars for both the trainers (solid line) and the controls (dashed line) at 2 weekly intervals.

Discussion

The 12 quadriplegics studied showed the same restrictive lung function pattern observed by previous investigators (Stone and Keltz, 1963; McKinley *et al.*, 1969). In spite of this and a rapid shallow breathing pattern, these subjects were able to maintain normal arterial blood gases at rest. This is comparable to the observations in previous studies of chronic quadriplegia (McKinley *et al.*, 1969).

Our inspiratory resistive ventilatory muscle training protocol produced similar increases in muscle strength (MIP) as those observed in training regimes with other quadriplegic patients (Gross *et al.*, 1980); and although endurance was assessed differently in this study as compared to ours, both studies found improved ventilatory muscle endurance with training. Similar improvements in ventilatory muscle strength and endurance following a variety of training protocols has been confirmed in patients with cystic fibrosis (Keens *et al.*, 1977), chronic obstructive pulmonary disease (COPD) (Pardy *et al.*, 1981; Belman and Mittman, 1980), and in normals (Leith and Bradley, 1976). However, our control group also showed significant increases in MIP and SIP. These subjects

Table IV Timing components of ventilation pre- and post-training

	Trainers		Controls	
	Pre	Post	Pre	Post
# Breaths	358 ± 93	320 ± 91	380 ± 47	313 ± 66
TI	1.42 ± .48	1.50 ± .40	1.21 ± .30	1.36 ± .23
TE	2.00 ± .56	2.47 ± .72	1.82 ± .27	2.49 ± .84
VT	.299 ± .07	.339 ± .08	.325 ± .09	.396 ± .14
F	19.5 ± 5.3	17.4 3.9	20.2 ± 3.4	16.9* ± 3.1
VE	5.5 ± .77	5.3 ± .76	6.4 ± 1.5	6.4 ± 2.0
VT/TI	.223 ± .03	.236 ± .03	.274 ± .07	.302 ± .11
TI/TTOT	.417 ± .04	.383 ± .03	.393 ± .06	.367 ± .06

*p < 0.05.

did not participate in any resistive training sessions, except for the stimulus of the testing procedures which was repeated every 2 weeks. Enhanced endurance has been observed in repeated testing in COPD subjects (Richardson *et al.*, 1988). These authors found that experienced COPD subjects who had previously performed an endurance test similar to that described by Knickerson and Keens (1982) on 6 occasions could breathe against a significantly higher maximum baseline load during a 2 minute incremental endurance test than could naive COPD patients. Repeated endurance testing at weekly intervals in these experienced COPD patients, however, did not show a progressive improvement in endurance beyond the baseline values. The COPD study also found no difference in strength (MIP) measurements between naive and experienced COPD subjects, and no improvement in MIP measurements on 3 separate tests conducted 1 week apart. Knickerson and Keens (1982) noted a slight improvement in both MIP and SIP on the second testing of normal subjects but found no additional increases after that time period. Clanton *et al.* (1985a) found no significant differences in MIP or endurance in normal control subjects who exercised at a subthreshold stimulus (10% MIP), in contrast to a normal training group who exercised at a higher stimulus which produced training effects.

While learning could certainly account for the improvement in MIP and SIP between the first and second testing sessions in our study, previous authors experience (Clanton *et al.*, 1985a; Nickerson and Keens, 1982; Richardson *et al.*, 1988) cannot explain the progressive improvement observed beyond this point in both the control and trainer groups (Fig. 2). The testing procedure appears to have induced a similar response in both groups.

The improvement in MIP and SIP observed in previous studies following ventilatory muscle training has been attributed to either alterations in the ventilatory muscles themselves as a result of training, or to an altered strategy of breathing. As breathing pattern in the controls improved similarly to the trainers, this suggests that the testing stimuli delivered at 2 weekly intervals was sufficient to induce a 'training effect'. It is unlikely that these changes reflect a

training response in the ventilatory muscles themselves as it has been suggested that the ventilatory muscles respond to training in a similar manner to other skeletal muscles (Belman and Sieck, 1982). While, the minimal intensity, frequency and duration of training required to induce training effects in the ventilatory muscles, particularly in quadriplegics is unknown, a training regime consisting of one session every 2 weeks has never been shown to induce training effects in other skeletal muscles. It is more likely that the rigorous testing procedures using a threshold load device resulted in the controls altering their strategy to breathe in a similar manner as the trainers. This altered strategy appears to be able to be maintained, and improved with infrequent reinforcement, and appears to induce changes in resting breathing pattern. Belman *et al.* (1986) has previously reported that the strategy of breathing in COPD patients during inspiratory resistive breathing significantly alters the patient's ability to sustain a higher load. Previous authors have also observed that ventilatory muscle endurance in normal subjects increased when inspiratory flow was decreased, despite a nearly constant pressure load and duty cycle (Clanton *et al.*, 1985b). Such an altered breathing strategy could account for the enhanced performance during ventilatory muscle endurance testing.

The fact that there was such variability in the improvement in MIP and SIP in the training group could be explained at least in part from the training protocol chosen. The flow rate could be altered with the resistive training device employed, and the pressure generated is dependent on flow. The trainers were allowed to set their own flow rates during training and it is possible that some of them decreased their flow rates sufficiently during the training sessions that they were no longer generating a pressure sufficient to elicit an additional training effect. However, the training pressure was selected assuming a flow rate of approximately 0.3 litres/sec. $\pm$ 0.05 litres and all trainers were exercising on orifices of 2.5 mm or less during the last 2 weeks of their training. It has been previously suggested that COPD patients may alter their strategy to breathe against inspiratory resistances and improve performance in ventilatory muscle testing without inducing training effects on the ventilatory muscles themselves (Belman *et al.*, 1986). This could explain the large variation in both the improvements in MIP and SIP, and in breathing pattern alterations observed in our training group. The addition of threshold load training, rather than a flow-dependent resistive device may have more conclusively addressed the question of whether there would be an additive effect of training to that observed by testing alone. A larger sample size would also have assisted, but large groups of quadriplegics with similar lesion levels who would meet the criteria for inclusion in such a study are difficult to find without conducting a multicentre trial. Finally, it is possible that the additional training is unnecessary, as a less frequent stimulus is all that is required to induce an altered strategy to breathe in quadriplegics.

While altering one's strategy to breathe may account for an improvement in test values, it does not explain the changes in resting breathing that were observed. Similar changes in resting breathing pattern were observed in both controls and trainers. Frequency decreased significantly in the control group and similarity, but insignificantly in the trainers. Mean tidal volume increased 18% of baseline V_t in the controls and 17% baseline V_t in the trainers. This slight

increase in V_t coupled with a decreased frequency resulted in an unchanged minute ventilation. The tendency towards a slower, deeper breathing pattern following increases in MIP and SIP is quite apparent. Such changes could account for the subjective improvements noted by previous authors in quadriplegics following a ventilatory muscle training regime (Gross *et al.*, 1980). Breathing patterns, and the timing components of the spirogram have not been previously examined following ventilatory muscle training. The pre-study mean Ti/T_{tot} for both trainers and controls was 0.405 ± 0.49 . It has been previously suggested that such long Ti/T_{tot} 's predispose individuals to inspiratory muscle fatigue (Bellemare and Grassino, 1983). Following the stimulus of the threshold load testing sessions mean Ti/T_{tot} for the combined groups was reduced to 0.374 ± 0.046 . This post study Ti/T_{tot} is comparable to values that we observed in normal individuals in a previous study (Loveridge *et al.*, 1984b). A shorter Ti/T_{tot} is thought to allow for recovery of the diaphragm during expiration and therefore minimise fatigue (Bellemare and Grassino, 1983). Increasing MIP and SIP appears to have resulted in a decrease in Ti/T_{tot} towards normal values.

Conclusions

The repeated presentation of an inspiratory threshold load on the respiratory muscles of quadriplegics appears to induce positive changes in ventilation. These changes likely represent an altered strategy to breathe, rather than any training effects in the ventilatory muscles themselves. This altered strategy to breathe against a resistive load appears to induce an effect on the inspiratory timing mechanism resulting in a shorter Ti/T_{tot} , and a slower, deeper respiration during resting ventilation. Whether or not these alterations in resting breathing pattern can be sustained, or whether further threshold loaded inspiratory muscle training can produce greater improvements in breathing remains unclear.

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