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# Effect of aerobic training on ventilatory muscle endurance of spinal cord injured men

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The functional consequences of ventilatory muscle impairment of spinal cord injured (SCI) subjects has been evaluated through spirometric and maximal respiratory pressure tests. Nevertheless, underlying functional abnormalities may be evident only under dynamic conditions, such as with a ventilatory muscle endurance test (VME). In order to evaluate the VME of thoracic SCI men and the effect of physical training on it we evaluated 12 SCI subjects (Group I) and 12 able-bodied controls (Group II). The subjects were submitted to clinical evaluation, spirometry, maximum voluntary ventilation in 12 s (MVV-12sec) and a test of VME – the highest time of sustained ventilation at 70% of the maximum voluntary ventilation in isocapnic conditions (MVV-70% time). Gr. II was evaluated before and after an arm cranking aerobic training program (30 min/session, three times/week, 6 weeks) with training target heart rate corresponding to ventilatory anaerobic threshold. On the initial evaluation, Gr. I subjects presented a significantly reduced forced vital capacity (FVC), forced expiratory volume in 1 s ($FEV_1$) and MVV-12 sec when compared to controls ($P<0.05$). Also, the VME was severely reduced in Group I (median, ranges; 1.15, 0.61 – 12.22) when compared to Group II (14.60, 1.20 – 15.00) – $P<0.001$. When Gr. I subjects were separated by the level of lesion, the VME was lower in high injured (T1 – T7) than intermediate (T8 – T10) and low injured patients (T11 – T12) – $P<0.05$. After aerobic training, Group I subjects incremented significantly the FVC ($P<0.05$) and the VME ($P<0.001$), so that MVV-70% time values post-training were not different from the initial values of the Gr. II. In conclusion, (i) the VME of thoracic SCI men was severely reduced when compared to able-bodied controls; (ii) a 6-weeks arm cranking aerobic training program was efficient to normalize the VME of SCI subjects.

**Keywords:** spinal cord injury; pulmonary function testing; paraplegia; rehabilitation; aerobic training

## Introduction

Spinal cord injured (SCI) subjects frequently show a reduction in pulmonary volumes (a restrictive pattern on spirometry) and in maximal static respiratory pressures, which are correlated with the functional loss of ventilatory muscles.[1–3] Therefore, the assessment of ventilatory impairment in SCI subjects has been made with easily obtainable functional indices, such as the forced vital capacity (FVC) and the maximal inspiratory pressure (MIP).[4]

Few studies have approached the dynamic ventilatory performance of SCI individuals.[5,6] Vital capacity and its subdivisions are relatively insensitive indicators of respiratory muscle weakness. Indeed, respiratory muscle strength has to fall below half normal before the vital capacity is significantly impacted.[6] Additionally, SCI subjects may show mechanical alterations during prolonged endurance activities that predispose to muscular fatigue, and such changes could not be detectable by functional resting evaluation.[6]

Ventilatory muscle endurance (VME) can be assessed by different tests, which vary in complexity and the necessity for technical expertise.[6,7] A simple and reliable test of VME is the longest time that a subject is able to voluntarily sustain 70% of the maximum breathing capacity or maximum voluntary ventilation (70%-MVV time).[8] This ventilatory intensity is close to the level of ventilation available for prolonged ventilation at maximal exercise in young healthy subjects.[9,10] To the best of our knowledge, no previous study has analyzed 70%-MVV time in SCI subjects with different levels of injury.

Thus, the objectives of the present study were (a) to compare the VME (as indicated by the 70%-MVV

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time test) of complete spinal cord injured and able-bodied physically active men and (b) evaluate the effect of an arm cranking aerobic training program on the VME of SCI subjects.

## Methods

*Subjects*
Twelve wheelchair-dependent SCI men (ASIA Class A,[11] with complete sensory and motor block below the level of the injury - Group I) and twelve able-bodied controls (Group II) matched for age, body mass and training status participated in this study. All participants were non-smokers. Healthy medical students personally volunteered for the control group.

All subjects in Group I were paraplegic, with the level of injury between T1 and T12. Etiology of injury was gunshot wound in eight subjects, road and traffic accident in three and one patient had a fall. All lesions had existed for at least 3 years. They were separated into 3 groups according to an anatomical and functional criteria of ventilatory muscle impairment: high thoracic injury, with paralysis of intercostal muscles in different degrees and total paralysis of abdominal and pelvic muscles (T1–T7, $n=4$); intermediate thoracic injury, with paralysis of abdominal muscles in different degrees and total paralysis of pelvic muscles (T8–T10, $n=5$); low thoracic injury, with paralysis of low abdominal and pelvic muscles (T11–T12, $n=3$).[12]

*Procedures*
Each test session began with the completion of an identification and subjective information sheet. The following questions were asked and recorded by the investigator: (1) level of habitual physical activity (Saltin and Grimsby questionnaire, modified);[13] (2) etiology and time since injury; (3) actual or past illnesses or interventions; (4) smoking history.

Subjects of Group I were weighed in the sitting position on a hospital scale. All subjects underwent a comprehensive medical examination and none of the subjects had any cardiovascular disease or used medicines likely to affect the results. All participants gave informed consent for participation in the study.

The following protocol was carried out in all subjects, after approbation of Ethical Committee of UNIFESP-EPM.

*Spirometry* Spirometric tests were performed using a calibrated 10-liters Collins Survey Spirometer (Collins, Braintree MA, USA). The subjects completed at least three acceptable maximal expiratory maneuvers. For Group I the tests were conducted with subjects sitting in their wheelchairs. According to standard protocols of the American Thoracic Society, 1991[14] for technique, acceptability and reproducibility the following spirometric variables were recorded and expressed in BTPS conditions: forced vital capacity (FVC, L); forced expiratory volume in 1 s ($FEV_1$, L), $FEV_1$/FVC ratio (%), forced expiratory flow between 25 and 75% of the FVC ($FEF_{25-75\%}$, $L.sec^{-1}$) and peak expiratory flow rate (PEFR, $L.sec^{-1}$). Predicted normal values for all spirometric variables were those of Knudson *et al*[15].

*Maximal voluntary ventilation (MVV-12sec)* The maximum voluntary ventilation (MVV) is the largest volume that can be breathed into and out of the lungs during 10–15 s interval with voluntary effort. In this study, the MVV was measured by having the subject sitting in his wheelchair and breathing deeply (with a volume greater than the tidal volume but lower than the vital capacity) and rapidly for a 12 s interval in the 10-liters Collins Survey Spirometer (Collins, Braintree MA, USA). The values were recorded in $L.min^{-1}$ (BTPS), by extrapolating the 12 s accumulated volume to 1 min. Predicted normal values were those of Cherniack and Raber.[16]

*Ventilatory muscle endurance (70%-MVV time)* Ten minutes after the MVV-12sec test, the ventilatory muscle endurance was measured as the highest time that the subject was able to voluntarily sustain a target ventilation corresponding to 70% of his MVV-12sec value (70%-MVV time).[8]

Ventilation was measured breath-by-breath by electronic integration and summation of the signals by a flow-volume module of a computerized metabolic cart (Vista TurboFit, Vacumed, Ventura CA, USA). The actual ventilation was continuously displayed on-line in a video monitor of a host computer, while the target ventilation for each individual (70% of his MVV-12sec) was being identified as a row to be followed.

Hypocapnia was avoided during forced ventilatory maneuvers by the use of partial rebreathing of expired air, using a hose connected to a mixing chamber (volume of 5 L). Expired air was continuously monitored (Gas Analyzer Set, Vacumed, Ventura CA, USA) from a sample line connected to the mouthpiece. The expired fraction of carbon dioxide ($F_ECO_2$) was maintained in a narrow range ($5\pm1\%$).

The subjects wore noseclips and a standard posture was adopted: sitting in their wheelchair (Group I) with the trunk supported by the backrest, avoiding any trunk stabilization or anchorage of accessory ventilatory muscles. The participants were instructed to breathe with any pattern or rhythm that they preferred and were given an active standard encouragement to maintain the target ventilation as long as possible.

SVV-70% time was measured from the moment that target ventilation was reached; whenever the subject was unable to reach it within the first minute of the test, it was interrupted and another attempt was performed after 10 min. The test was ended when the subject was not able to sustain the target ventilation, with a reduction higher than 10%.

Subsequently they were asked about the nature of the limiting symptoms.

*Exercise testing and training program* Prior to sessions of training, all SCI subjects underwent a cardiopulmonary exercise testing on a computerized metabolic gas exchange system (Vista Turbo Fit, Vacumed, Ventura CA, USA) using a calibrated electromagnetically-braked arm crank ergometer (Cybex MET-300, Cybex, USA).

The data were calculated automatically using standard formulae and displayed in descriptive numerical and graphical forms (average of 20 s). The following data were obtained: oxygen uptake, $mL.min^{-1}$ STPD); carbon dioxide production ($VCO_2$, $mL.min^{-1}$ STPD); respiratory exchange ratio (RER); minute ventilation (VE, $L.min^{-1}$ BTPS), respiratory rate (f, bpm); ventilatory equivalent for $O_2$ and $CO_2$ ($VE/VO_2$ and $VE/VCO_2$); expired fraction of $O_2$ and $CO_2$ ($FEO_2$ and $FECO_2$, mmHg); heart rate (HR, bpm) and oxygen pulse ($VO_2$/HR, mL/beat). $VO_2$ at the anaerobic threshold ($VO_2AT$) was estimated by the ventilatory method, when $VE/VO_2$ and $FEO_2$ increased while $VE/VCO_2$ and $FECO_2$ remained stable. In this study the $VO_2AT$ was identified in all of the patients being studied.

The supervised arm cranking aerobic training program consisted of 30 min sessions, three times a week for 6 weeks with the training target heart rate corresponding to ventilatory anaerobic threshold.

*Statistical analysis*
The values are reported as median and ranges. The following statistical tests were used: (a) Mann–Whitney two sample test to compare the unpaired data of the two groups in the initial evaluation; (b) Wilcoxon's test to evaluate the differences between pre- and post-training paired values of the Group I; (c) One Way Analysis of Variance (ANOVA) with Bonferroni's $P$ correction in the comparison of the 70%-MVV time values among groups. Level of statistical significance was always set at $P<0.05$.

## Results

Group I patients had median (range) values of age of 31 years (22–54 years) and body mass of 75.3 kg (65.8–82.5); Group II able-bodied had median value of age of 30 years (22–52 years) and body mass of 73.8 kg (67.2–80.0) - $P>0.05$. All participants were classified as moderately active to active, with physical activity score higher than three (i.e. habitual exercise at least 3 h/week).[13]

Spirometric, MVV-12sec and 70%-MVV time values in the initial evaluation for both groups are reported on Table 1. We found a significant reduction in Group I for all studied variables, with exception of $FEV_1$/FVC ratio. The spirometric abnormalities followed a restrictive pattern in five individuals (four with high thoracic injury) being 3 graded as mild and 2 as moderate.[14] Although median values were reduced when compared to Group II, the spirometric and MVV-12sec values of Group I were higher than 80% of predicted[15,16] for the FVC in 7/12, for the $FEV_1$ in 9/12 and for the MVV-12sec in all patients (Figure 1).

The decrease in the median 70%-MVV time was severe in Group I (1.15 min; 0.61–12.22) compared to Group II (14.60 min; 1.20–15.00) - Table 1. When the patients of Group I were separated by the level of injury (see Methods) the VME was lower in high injured patients (T1–T7: 0.65 min; 0.61–0.71) compared to intermediate (T8–T10: 1.96; 0.72–3.90) and

**Table 1** Results of the functional evaluations of the Group I thoracic SCI men ($n$ = 12) and of the Group II able-bodied controls ($n$ = 12)*

| Variables | Group I Initial | Group I Final | Group II Initial |
|---|---|---|---|
| FVC | | | |
| (L) | 4.05† | 4.40‡ | 5.63 |
| (% pred) | 81† | 90‡ | 103 |
| FEV | | | |
| (L) | 3.62† | 3.70 | 4.46 |
| (% pred) | 84† | 88 | 107 |
| $FEV_1$/FVC | | | |
| (%) | 85 | 80 | 81 |
| MVV-12sec | | | |
| ($L.min^{-1}$) | 175† | 178 | 212 |
| (% pred) | 124† | 126 | 141 |
| 70%-MVV time | | | |
| (min) | 1.15‡ | 9.10‡ | 14.60 |

*Values are displayed as median. See text for abbreviations. †Significantly lower than Group II in the initial evaluation ($P<0.05$–Mann-Whitney test). ‡Significant differences between initial and final evaluations ($P<0.01$–Wilcoxon test). Obs: post-training 70%-MVV time values of Group I were not significantly different from the values of Group II in the initial evaluation ($P>0.05$; Mann-Whitney test)

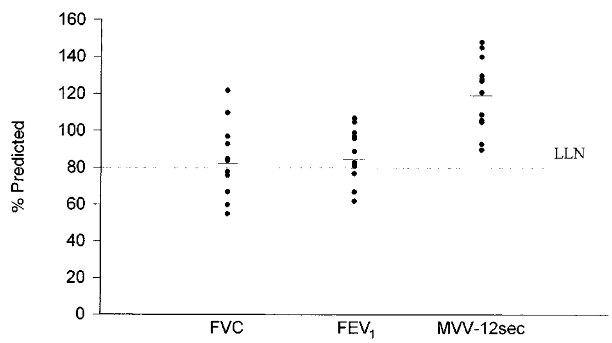


**Figure 1** Individual and median spirometric* and MVV-12sec** values in % of predicted (% pred) of the Group I thoracic SCI men ($n$=12). *Predicted from Knudson *et al.*[15] **Predicted from Cherniack and Raber.[16] LLN: Lower limit of normality

low (T11–T12: 4.50 min; 1.60 12.22) ones (Figure 2). However, when compared to Group II, the reduction on VME was significant in both high and intermediate injured patients (Figure 2). Individual analysis showed that only one individual of Group I sustained 70% of his MVV for more than 10 min and none sustained for 15 min; these findings contrast with those from Group II, where seven subjects sustained the target ventilation for more than 10 min and five individuals for 15 min (Figure 2).

Analyzing the limiting symptoms at the end of 70%-MVV test, nine subjects of Group I said that dyspnea was the main symptom, 2 said muscle pain or discomfort and one said general fatigue; this contrasted with Group II where only two subjects complained of dyspnea at the end of the test, and nine subjects muscle pain or discomfort. No participant from both groups related any symptom compatible with hypocapnia.

After arm cranking aerobic training, Group I presented a moderate improvement on FVC ($P<0.01$) and a pronounced increment on 70%-MVV time ($P<0.001$), so that the post-training values of 70%-MVV time were not significantly different from that of Group II at initial evaluation (Table 1). Furthermore, we found that all subjects increased their 70%-MVV time and 5 of them were able to sustain the target ventilation for 15 min (Figure 3).

## Discussion

To our knowledge, this is the first investigation to examine the effects of supervised aerobic training on ventilatory muscle endurance (VME) of spinal cord injured (SCI) subjects. Our data support the hypothesis that these individuals have a severe reduction of VME when compared to able-bodied controls. Arm cranking aerobic training was adequate to increase the VME so that the post-training VME values were similar to able-bodied ones.

These results are similar to those of Estrup *et al.*[8] who studied 12 patients with neuromuscular disease and six normal subjects in an 8-weeks strength and endurance respiratory muscle training program. They were able to show a significant rise in vital capacity and VME (70%-MVV time) with the highest relative improvement found in patients who were most severely disabled. Nevertheless, their investigation was focused on subjects with progressive muscular dystrophy, and their training program was quite different from the present study where a systemic aerobic exercise program was used.

The ability to sustain a high ventilatory rate depends on anatomical and functional integrity of the inspiratory and expiratory muscles.[17] In quiet breathing, the most important inspiratory muscle is the diaphragm, as for its direct inspiratory action on the rib cage, and for the appositional component activated by the rise in intra-abdominal pressure.[18] The external intercostals, especially the parasternal portion and the scalenes seem to have a secondary significance.[12] When inspiratory requirement increases or in case of functional reduction of the primary inspiratory muscles, the accessory muscles (*sternocleidomastoids, pectoralis major, pectoralis minor, serratii*) can operate to actively elevate the ribs.[17]

On the other hand, expiration with quiet breathing is passive, secondary to the dissipation of the elastic energy accumulated at the lung-ribcage system at the end of inspiration. When the expiratory requirement is increased, the abdominal muscles, supplied by branches of the lower six thoracic nerves and the first lumbar, are progressively recruited. As they contract, these muscles pull the lower ribs down and inwards (an expiratory action) and also draw the abdominal wall inwards producing an increase in intra-abdominal pressure. This latter effect may have an expiratory action, moving the diaphragm cranially and leading to a reduction in lung volume, or an inspiratory action transmitting the pressure to the diaphragm's zone of apposition and elevating the lower rib cage.[18,19]

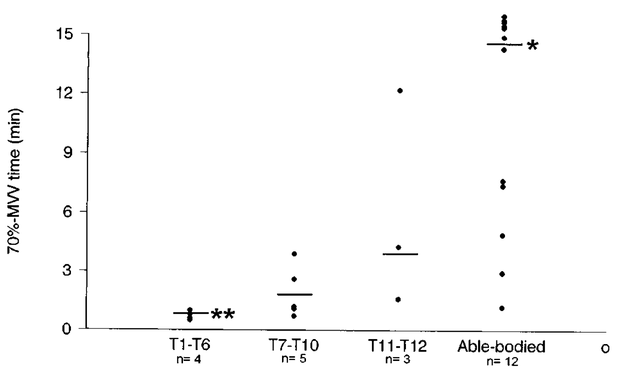


**Figure 2** Individual and median 70%-MVV time values of the Group I thoracic SCI men separated by the level of injury# and of the Group II able-bodied controls. *Classification criteria are reported on Methods. *Higher than T1–T7 and T8–10; **Lower than T8–T10 and T11–T12 ($P<0.05$ - ANOVA test with Bonferroni's $p$ correction)

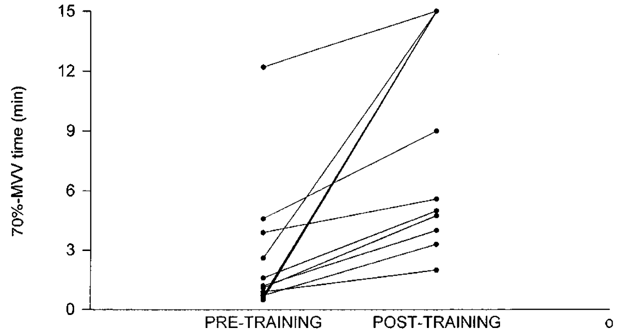


**Figure 3** 70%-MVV time values pre and post arm cranking training in Group I thoracic SCI men

As expected, the functional reduction of the ventilatory muscles in individuals with neuromuscular disease due to weakness or paralysis has a major importance on respiratory mechanical performance, notably during moderate to intense activities for a long period of time.[6] Our results suggested that reduction in VME was related to functional muscle loss, with a more severe impairment in those who are paraplegic above T7. The loss of function of the abdominal muscles in these individuals probably leads to an increase in compliance of abdominal wall and therefore lowers the normal augmentation of internal pressure impairing the adequate thorax-abdomen coupling.[18–20] Additionally, during intense voluntary ventilation a vigorous abdominal muscle contraction is necessary to overcome the high airway resistance of rapid expiratory flow rates and to decrease the expiratory time in order to achieve high breathing frequencies.[20]

Other factors could be involved in the reduction of VME in these patients, such as the loss of tonic abdominal expiratory activity resulting in higher residual volume, which induces a raise in the elastic work.[19] On the other hand, the loss of the motion of the intercostal muscles after SCI probably reduces inspiratory performance, and also elevates the grade of distortion of chest wall, with consequent increment of elastic inspiratory load.[17]

From a physiological standpoint measurement of VME has a more practical significance than do spirometric tests, since it tries to simulate the actual action of ventilatory muscles during exercise. Nevertheless the simulation is not perfect, as exercise hyperpnea occurs with higher volumes, has a different pattern of muscle recruitment and is influenced by the bronchodilatation effect of circulating catecholamines.[21] Furthermore, the humidity and temperature are clearly distinct from experimental conditions.

An interesting finding of this study was the highest frequency of dyspnea as the main symptom in Group I. This finding is in agreement with the mechanical inappropriateness theory, which suggests that perception of dyspnea happens as the consciousness of the imbalance between intensity of neural drive and actual thorax displacement.[22] Thus, SCI subjects might have interpreted the mechanical impairment to sustain target ventilation as dyspnea sensation.

The marked increase of VME time of SCI subjects after training is consistent with the findings of Powers *et al*[23] who showed that high intensity aerobic training in rats resulted in significant improvements in the oxidative and anti-oxidant capacities of the costal diaphragm, rectus abdominalis and external oblique muscles. Other authors pointed out that the mammalian respiratory muscle (ins and expiratories) respond to whole-body endurance exercise training with small (10–30%), but significant, increases in mitochondrial oxidative capacity and modifications in muscle fiber size.[24,25] Moreover, in our study the arm cranking exercise training might have induced direct benefits on cervical and upper extremity accessory inspiratory muscles that contributed to the observed improvement. Thus, in our opinion the dramatic improvement on VME of SCI men after training was probably linked to an effective training-induced alteration on primary and accessory functionally active respiratory muscles.

The present study has some technical limitations. The use of a more sensitive indicator of ventilatory fatigue than 70%-MVV time test could have detected other alterations.[6] Some individuals could have a high time of endurance but with a pronounced reduction in post-test maximal static respiratory pressures. Another factor to be considered is the substantial flow resistance on the breathing circuit and the non-conditioning of the inspired air that could affect the time of endurance. However, these factors should influence both groups equally, since no previous study showed that paraplegic individuals would have a higher sensitivity to these conditions. Finally, care must be taken in the analysis of the relationship between reductions in VME and the level of lesion considering the small number of individuals studied.

This work raised some interesting practical questions about the ventilatory performance of SCI subjects. Firstly, the results of the traditional pulmonary function tests for prediction of VME must be considered with caution. The usual procedure for indication ventilatory limitation in exercise (VEmax/MVV-12sec ratio $>0.7$)[26] can be misleading in SCI subjects, since it differs from normal, they cannot sustain 70% of the MVV-12sec for a long time. Secondly, the impact of the improvement on VME in the quality of life and in the athletic performance of these patients is unknown.[27] Thirdly, we did not evaluate whether or not this improvement can be achieved or enhanced with isolated respiratory muscle training. Fourthly, it was very surprisingly the high degree of VME impairment of these particularly active SCI subjects; it suggests that their regular physical activity pattern was not sufficient to improve their ventilatory performance. Considering the progressive increase in the SCI subjects' daily exertion, both in active and sedentary individuals, the dynamic ventilatory impairment study deserves more specific attention.

## Conclusion

In this study we found that (i) the VME (as evaluated by the 70%-MVV time test) of thoracic SCI men was severely reduced when compared to able-bodied controls; (ii) a 6-weeks arm cranking aerobic training program was effective to normalize the VME of SCI subjects.

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