Varying Physiological Response to Arm-Crank Exercise in Specific Spinal Injuries

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Abstract. The purpose of this arm-crank ergometry (ACE) study was to provide a greater understanding of the influence to which specific cervical and thoracic spinal cord injuries contribute to reduction in optimal cardio-respiratory and metabolic function. Twenty five male volunteers aged 20 to 47 years participated. Twenty disabled wheelchair-confined spinal cord injured (SCI) subjects were equally divided into four 'site-specific' groups based on the lesion being within either high- or low- cervical or thoracic anatomical regions. Five physically non-disabled controls (As) were included. Measured variables tended to decrease progressively from As to high-level quadriplegics. Analysis revealed a high variance in maximum cardio-respiratory performance levels between groups ($P < 0.001$). These findings confirm that limitation to upper body physical capabilities in the SCI during high-intensity ACE is dependent on specific lesion site. Considerable variability in performance levels were measured in those suffering lesions within closely approximating anatomical regions. Results also suggest a greater importance in the location of cervical rather than thoracic injuries in contributing towards higher relative losses in maximal cardio-respiratory and metabolic potential. Alterations in body composition and varying severity of muscle paralysis likely also play a contributing role in reducing optimal metabolic function in SCI individuals. The importance for stringent classification techniques of spinal cord lesion site in predicting upper body physical exercise potential in the SCI has therefore been highlighted in this study.

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Key words: arm-crank ergometry, site-specific spinal cord injury, maximum cardio-respiratory and metabolic potential

Introduction

The physiological and metabolic responses of

physically non-disabled (As) subjects to high-level arm-crank ergometry (ACE) exercise have been well documented (McConnel et al., 1984; Eston and Brodie, 1986; Walker et al., 1986; Sedlock et al., 1990). A reduction of maximal upper body ACE performance in the spinal cord injured (SCI) has been reported (Gass and Camp, 1984; Hooker and Wells, 1992, 1993; Ragnarsson, 1996). However, comparatively little is known of the direct effect spinal cord lesion level contributes to the reduction.

Numerous authors, including Jehl et al. (1991) and Hopman et al. (1992, 1993), have further reported comparisons between high- and low- level spinal paraplegic injury sites to As. Van Loan and colleagues (1987) and Drory et al. (1990) compared paraplegics and As to quadriplegics, but did not adequately distinguish injury level within specific SCI groups. Hjeltnes and Jansen (1990) did segregate each paraplegic and quadriplegic group into more specific cervical or thoracic lesion sub-groups, but only reported on maximum minute oxygen uptake (VO_{2max}) levels during ACE.

A detailed comparative analysis of cardio-respiratory measures during maximal ACE in individuals with varying cervical and thoracic SCI levels to non-injured physically abled subjects has therefore still not to date been adequately addressed.

Reduced cardio-respiratory performance potential of SCI during ACE has been observed to produce specific compensatory mechanisms to improve upper body work ability. Davis and Shephard (1988) and Glaser (1989) have reported on the ability of SCI to extract greater peripheral arterial oxygen loads in upper body musculature during steady state ACE. A paucity exists in the literature, however, reporting on the contribution specific spinal cord injury levels limit an individual's ability to produce and efficiently utilise energy during high intensity upper body exercise.

The primary purpose of this study, therefore, was to detail the contributions that specific cervical and thoracic spinal cord injuries limit cardio-respiratory and metabolic performance during high intensity ACE exercise. It is anticipated that this study may highlight performance

variations within SCI groups traditionally classified as homogenous.

Methods

Subjects

Twenty five male volunteers aged between 20 and 47 years participated in the study. Subjects were divided equally into five (5) distinct groups comprising of high-level quadriplegics (HLQs), low-level quadriplegics (LLQs), high-level paraplegics (HLPs), low-level paraplegics (LLPs) and physically non-disabled (As) controls. Spinal cord lesion level of HLQs was at the fourth to fifth cervical vertebrae (C4-C5), and sixth to eighth (C6-C8) for LLQs. Injury sites for HLPs were between the first and sixth thoracic vertebrae (T1-T6), and seventh to twelfth (T7-T12) for LLPs. Complete and incomplete cord lesions were combined within groups with the exception of HLQs. All injuries in HLQs were incomplete cord transections which enabled sufficient upper body exercise capacity to perform maximum ACE.

The criterion for recruitment of subjects for the study was; 1) sufficient upper torso and extremity nerve function and strength to accomplish ACE exercise, 2) no previous history of cardiovascular or respiratory problems, and, 3) for the SCI, wheelchair dependency for at least one year (Ward, 1981; Davis et al., 1984) with no previous history of cardiovascular problems. In accordance with procedures approved by the University of Wollongong Human Experimentation Ethics Committee, all volunteers were medically screened to exclude those with clinically diagnosed cardio-respiratory limitations or any other possible contraindications to strenuous exercise.

Apparatus

Peak minute oxygen uptake ($\text{VO}_{2\text{max}}$) was determined using an electro-magnetic braking cycle ergometer system (Excalibur Sport, V1.47, 1990, Lode B.V. Groningen, The Netherlands). The unit was mounted and secured onto a table. The pedals of the cycle were modified into arm-crank handles. With arms extended (approx. 5 deg. flexion), subjects' shoulders were on a horizontal plane to the handle axis (Ward, 1985; Hooker and Wells, 1992). The hands of all quadriplegics and HLPs were secured to the cranks using tape. Pilot studies revealed LLPs to possess sufficient grip strength to perform ACE without strapping.

All cardio-respiratory exercise responses were calculated and recorded via a 'Q-Plex I Cardiopulmonary Exercise System' (Quinton Instrument Company, Seattle, WA, USA, 1989). Metabolic parameters were calculated by analysis of expiratory gas ratios and volumes using open circuit spirometry. Gas flow rates and volumes (BTPS) were measured using a pneumotachometer. All equipment was calibrated prior to each test. Heart rate was monitored from a standard V5 precordial lead using a 12-lead 'Q 5000 Exercise Test Monitor' (Quinton Instrument Company,

Seattle, USA, 1989). This equipment was non-invasive. Heart rates and ECG patterns were constantly displayed on an S-VGA computer monitor during exercise.

Procedure

Subjects' anthropometric characteristics were determined prior to testing. Disabled individuals were weighed with a pre-calibrated hospital wheelchair scale (Detecto Scales Inc., Brooklyn, N.Y., USA). Percent body fat was then estimated via standard procedures using Harpenden skin calipers (Davis and Shephard, 1988). Total height was calculated using seated height plus lower limb length. Seated trunk height was measured in the disabled group as the distance from the mid-acetabulum to the vertex of the head. Lower limb length was measured from mid-acetabulum with subjects lying supine.

Tests were performed at approximately the same time of day, and in a temperature controlled laboratory (21.0–22.0 deg. C.). The subjects fasted for at least 12 hours prior to testing, and were asked to avoid vigorous exercise or the taking of medications of any sort 24 hours prior to commencement of testing and to refrain from smoking.

Opportunity was given to each individual before commencement of data collection to practice the ACE exercise for familiarisation. Subjects were not permitted to talk during data collection. Five minutes of resting cardio-respiratory data was acquired before commencement of continuous ACE exercise testing. Throughout the entire test subjects were required to maintain an arm pedal cadence of 50 rev.min⁻¹ (Dotson et al., 1982). Cadence (RPM) was monitored via an LED positioned directly in front of each subject at eye level. Initial power outputs (PO) of 15 Watts (W), 30W, and 40W were set for the first 60 seconds of data collection for quadriplegics, paraplegics and As respectively. An additional loading of 5 W was applied every minute thereafter until completion of the test (Lasko-McCarthy and Davis, 1991).

A plateau in the minute oxygen uptake with increasing PO plus a concurrent respiratory quotient (RQ) greater than 1.1 indicated attainment of $\text{VO}_{2\text{max}}$ (Bar-On and Nene, 1990; Davis et al., 1990). Termination of the exercise test occurred if; 1) the subject reported subjective fatigue and hence volitionally aborted the exercise protocol despite verbal encouragement, 2) the subject was unable to maintain the required crank speed (determined by the author) indicating exhaustion (Franklin et al., 1983; Davis and Shephard, 1988), and, 3) predicted maximum heart rate ($220 - \text{age}$) was reached. A 5 minute warm-down period immediately followed the completion of all exercise testing, and, thereafter, data collection continued until the recovery of previous resting cardio-respiratory measures was attained.

Cardio-respiratory responses were monitored and recorded every 10 seconds during exercise. Variables measured in litres were expired ventilation (V_E BTPS), oxygen consumption (STPD), and carbon dioxide

Table 1 Anthropometric characteristics (mean $\pm$ SE) of subjects

	As	LLPs	HLPs	LLQs	HLQs
Age	33.0	28.2	33.4	29.6	32.4
(years)	(3.6)	(3.3)	(4.7)	(1.3)	(3.2)
Height	175.7	177.7	175.2	179.0	176.5
(cm)	(3.1)	(1.3)	(1.9)	(1.9)	(1.2)
Weight	82.5*	62.6	68.8	61.8	63.1
(kg)	(8.4)	(6.0)	(3.9)	(2.9)	(4.0)
BF	18.3	15.0	19.1	19.5	21.4
(%)	(2.0)	(2.0)	(1.2)	(3.6)	(1.2)

* = $P < 0.05$, SE = standard error.

As = Physically non-disabled controls, LLPs = Low-Level paraplegics, HLPs = High-level paraplegics, LLQs = Low-Level quadriplegics, HLQs = High-level quadriplegics, BF = body fat.

production (STPD). Minute averages were then calculated. Other measures taken included absolute ($\text{l} \cdot \text{min}^{-1}$) and relative ($\text{VO}_2 \cdot \text{kg}^{-1}$) oxygen consumption (VO_2). All these measures were minute values. The RQ and percent expired oxygen and carbon dioxide were also recorded. Additional measurements were minute heart rate (f_c) ($\text{beats} \cdot \text{min}^{-1}$), total (Kcal) and relative ($\text{Kcal} \cdot \text{kg}^{-1}$) energy expenditure, defined as the energy used to achieve $\text{VO}_{2\text{max}}$, and total (W) and relative ($\text{W} \cdot \text{kg}^{-1}$) power output (PO).

Statistical analysis

Prior to analysis, data was tested for the assumptions of normality and homogeneity of variance within groups (Cochrans C Test and Levene Statistic). Shapiro-Wilks Test was applied to residual values to assess assumptions of normal distribution. Violation of normality within groups ($P < 0.05$) resulted in log transformation of the data. The Levene Statistic was incorporated to test for equality of variance between groups. Data found to fall without the 95% level ($P < 0.05$) for homogeneity of variance was also tested using non-parametric Kruskal Wallis methods. Anthropometric, resting and maximal cardio-respiratory and metabolic values were then analysed within and between groups using multivariate analysis of variance statistics techniques (MANOVA). Post-hoc Sheffe's tests were then used to discover the location of variable differences between groups ($P < 0.05$).

Results

Dependent variables were analysed using 'site-specific' groups. This comprised of five groups. The SCI was categorised according to anatomical location of the injury falling within specific spinal cord regions as explained in the methods section.

Anthropometric values for all groups were similar for age, height, and percent (%) body fat. Physically non-disabled subjects weighed heavier ($P < 0.05$) than all other groups. The anthropometric description of the experimental

Table 2 Physiological values (mean $\pm$ SE) of subjects at rest and maximum arm-crank ergometry exercise capacity

	As	LLPs	HLPs	LLQs	HLQs
V_{Erest}	10.1	9.9	9.7	9.2	7.4*
($\text{l} \cdot \text{min}^{-1}$)	(0.5)	(0.3)	(0.2)	(0.8)	(0.4)
$\text{VO}_{2\text{rest}}$	0.33	0.28	0.26	0.25	0.23
($\text{l} \cdot \text{min}^{-1}$)	(0.3)	(0.2)	(0.2)	(0.2)	(0.01)
f_{crest}	74.4	79.4	68.8	64.4*	76.6
($\text{beats} \cdot \text{min}^{-1}$)	(3.0)	(2.8)	(3.1)	(2.3)	(3.2)
V_{Emax}	121.7*	66.7	68.0	41.2**	23.9**
($\text{l} \cdot \text{min}^{-1}$)	(15.8)	(4.3)	(2.9)	(3.9)	(2.3)
$\text{VO}_{2\text{max}}$	2.60	1.66	1.85	1.18**	0.93**
($\text{l} \cdot \text{min}^{-1}$)	(0.2)	(0.1)	(0.1)	(0.1)	(0.1)
$\text{VO}_{2\text{max}}$	32.7	27.6	27.0	18.8**	11.5**
($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$)	(3.9)	(2.1)	(0.8)	(1.5)	(1.1)
f_{cmax}	174.6	174.2	170.0	141.8*	122.2*
($\text{beats} \cdot \text{min}^{-1}$)	(2.1)	(1.1)	(1.0)	(6.1)	(6.6)

* = $P < 0.05$, ** = $P < 0.001$, SE = standard error.

As = Physically non-disabled controls, LLPs = Low-level paraplegics, HLPs = High-level paraplegics, LLQs = Low-level quadriplegics, HLQs = High-level quadriplegics, V_{Erest} = resting minute ventilation, $\text{VO}_{2\text{rest}}$ = resting minute oxygen uptake, f_{crest} = resting minute heart rate, V_{Emax} = peak minute ventilation, $\text{VO}_{2\text{max}}$ = peak minute oxygen uptake, f_{cmax} = peak minute heart rate.

Table 3 Power production and energy expenditure (mean $\pm$ SE) of subjects

	As	LLPs	HLPs	LLQs	HLQs
PO	1276.3**	651.3	662.3	335.0**	128.5**
(W)	(230.3)	(96.5)	(62.2)	(37.0)	(8.6)
PO	15.5**	10.4	9.7	5.4	2.0**
($\text{W} \cdot \text{kg}^{-1}$)	(2.2)	(0.8)	(0.5)	(0.5)	(0.1)
Energy	127.1*	65.0	72.6	35.9**	13.0**
(Kcal)	(20.5)	(8.0)	(5.7)	(4.7)	(0.9)
Energy	1.6	1.2	1.1	0.6**	0.2**
($\text{Kcal} \cdot \text{kg}^{-1}$)	(0.2)	(0.2)	(0.05)	(0.06)	(0.01)

** = $P < 0.001$, SE = standard error.

As = Physically non-disabled controls, LLPs = Low-level paraplegics, HLPs = High-level paraplegics, LLQs = Low-level quadriplegics, HLQs = High-level quadriplegics, PO = total power output, W = Watts.

and control As subjects is shown in Table 1.

Table 2 provides a summary of both resting and maximal cardio-respiratory values for each group prior to, and during maximal ACE exercise.

Individual groups at rest

All groups generally showed similarity in all parameters. The exception was HLQs which showed significantly lower minute ventilation (V_{Erest}). LLQs also produced lower minute resting heart rates (f_{crest}).

Individual groups during maximum ACE performance

Group values are summarised in Tables 2 and 3.

Fig. 1 Peak minute oxygen uptake values (mean + SE) for site-specific groups. Asterisk (*) denote that LLPs, HLPs were significantly different to LLQs and HLQs but (=) denotes that LLPs and HLPs were similar to each other.

Reference to Table 2 indicates that performance levels progressively decreased from highest values in As, to lowest in HLQs. Differences were highly significant ($P < 0.001$) unless otherwise specifically identified. Cervical rather than thoracic spinal cord lesions contributed to the higher relative losses in cardio-respiratory and metabolic potential. All LLQs performance levels were superior to HLQs.

Table 2 also indicates significant differences ($P < 0.001$) in maximum minute ventilation (V_{Emax}) between most groups. The exception was LLPs and HLPs which showed no difference.

VO_{2max} levels (l.min⁻¹) in Table 2 were similar between As, HLPs, and LLPs. Both LLQs and HLQs individually showed significant difference ($P < 0.001$) to all other groups. Figure 1 shows that VO_{2max} levels (ml.kg⁻¹.min⁻¹) for both HLQs and LLQs remained significantly lower to all other paraplegic and As groups, even after weight discrepancy between groups was accounted for. Differences between HLQs and LLQs disappeared however. LLPs, HLPs and As also produced similar levels.

Analysis of maximum heart rates (f_{cmax}) in Fig. 2 revealed similarities between LLQs and HLQs, but significant differences ($P < 0.05$) to all other groups. These other groups measured similar levels to each other.

Fig. 2 Peak heart rate values (mean + SE) for site-specific groups. (=) denotes similarities between LLQs and HLQs. *Significantly different from all other groups (controls, LLPs and HLPs).

Results of total power output (W) and energy expenditure (Kcal) are presented in Table 3. Significantly greater ($P < 0.001$) PO levels were generated in As compared to all others. Both HLPs and LLPs groups displayed similar levels. HLQs and LLQs recorded significantly different ($P < 0.001$) levels. The significant limitation cervical cord lesion places on total PO (W) is seen in Fig. 3. Both LLQs and HLQs had appreciably greater deficit in total attainable PO than LLPs, HLPs, and As. Figure 4 reveals that correction of weight against PO (W.kg⁻¹) eliminated differences between HLQs and LLQs.

Energy expenditure patterns (Kcal) in Fig. 5 revealed similar trends between groups to PO. Significantly greater ($P < 0.001$) metabolic activity was recorded in As to all others. LLPs and HLPs attained similar measurements. HLQs and LLQs had significantly lower ($P < 0.001$) readings to not all other groups. HLQs also recorded significantly different levels ($P < 0.001$) to LLQs. Calculations against body weight (Kcal.kg⁻¹) depicted in Fig. 6 revealed similarities in energy utilisation between HLQs and LLQs. These measures remained significantly different ($P < 0.001$) to all other groups. Similar values were measured between As and both HLPs and LLPs. LLQs produced similar total energy consumption values (Kcal.kg⁻¹) to HLPs. HLQs were unable to produce energy outputs similar to either LLPs, HLPs, or As.

Fig. 3 Total (W) power output values (mean + SE) for site-specific groups. Asterisk (*) denote that LLQs and HLQs were significantly different in total attainable PO than LLPs and HLPs.

Fig. 4 Relative (W.kg⁻¹) power output values (mean + SE) for site-specific groups. Asterisk (*) denotes that correction of weight eliminated differences between LLQs and LHQs.

Fig. 5 Total (Kcal) energy expenditure values (mean + SE) for site-specific groups. Asterisk (*) denote that LLQs were significantly different to HLQs and both were significantly different to all other groups.

Fig. 6 Relative (Kcal.kg⁻¹) energy expenditure values (mean + SE) for site-specific groups. The (=) denotes that energy utilization was similar between LLQs and HLQs and was also significantly different to all groups. (E) denotes that HLPs showed similar energy consumption to LLQs.

Discussion

The present study provided significant insight into the extent to which spinal cord lesions within specific anatomical regions limit cardio-respiratory and metabolic function during high intensity upper body physical exercise. These results provide evidence of the diversity in maximal upper body physical exercise potential of individuals within traditionally categorised similar SCI populations. The findings suggest a greater importance in the location of cervical spinal cord lesions to thoracic for contributing towards overall relative greater loss in maximal physiological and metabolic function.

Group measures at rest

The lower minute ventilation results of HLQs to all others recorded at rest may possibly be the result of increased primary and accessory respiratory muscle compromise. Interruption to neurological innervation of the diaphragm by the phrenic nerve (C3-C5) in the HLQs would produce reduced diaphragmatic activity at rest. A concurrent reduced ability for adequate chest expansion through both interrupted intercostal nerve activity, and the inability to maintain an optimal thorax posture whilst seated, would both also further contribute to limit efficient resting ventilation. These limitations in ventilation performance were obviously compounded further during higher intensity ACE exercise, when even greater ventilation demands are needed to provide increased oxygen concentrations to the hard-working upper body musculature.

Group measures at maximal exercise capacity

Comparison of maximum cardio-respiratory levels between site-specific groups in the present study highlighted a large distribution of performance levels of individuals suffering spinal cord lesions within close approximating anatomical regions.

The present findings support little change in the relative loss of maximal cardio-respiratory and metabolic function in spinal lesions between the first and twelfth thoracic vertebrae (T1-T12). The close proximity of values between LLPs, HLPs, and As in the current study was unexpected. Differences have been reported in past literature (Jehl et al., 1991; Hooker and Wells, 1992; Hopman et al., 1992, 1993; Lin et al., 1993). An understanding of the spinal anatomical origins of the sympathetic nervous system to the heart and upper limbs would also suggest limitations in the ability of SCI to adequately respond to the higher workloads placed on their upper body musculature. Cardiac sympathetic control originates between T1-T5, and an appreciable extent of nervous supply for upper limb blood vessel regulation, including vasodilation, which occurs during increased

exercise, is derived from the high-thoracic spinal cord levels (T4-T6). This is especially relevant in persons with lesion sites at or between T1-T6, those found in the current HLPs group (Cowell et al., 1986; Glaser, 1989; Lehmann et al., 1989; Bhambhani et al., 1991; Jehl et al., 1991; Hopman et al., 1994).

It is possible that the paraplegic subjects in this study were able to withstand greater relative upper body musculature demands than As because of delayed onset of physical fatigue. It is also postulated, that despite limitations to central physiological mechanisms such as sympathetic cardiac drive, paraplegics were able to match As high intensity ACE performance levels through superior peripheral upper body muscle adaptations such as increased muscle strength and aerobic endurance (Burkett et al., 1990). Paraplegic subjects are more likely to have developed improved strength and endurance in their upper body musculature from the everyday physical demands of wheelchair ambulation.

The often greater reported performances of As to paraplegics in upper body high intensity ACE exercise may also be directly related to the usual heavier body weights of the physically non-disabled. This is supported by data produced by Hooker et al. (1993) and Lin et al. (1993). These authors found no disparity in $\text{VO}_{2\text{max}}$ ($\text{ml.kg}^{-1}.\text{min}^{-1}$) results between these two populations, despite As displaying significantly higher absolute $\text{VO}_{2\text{max}}$ ($\text{ml.kg}^{-1}.\text{min}^{-1}$) levels. Jehl and colleagues (1991) have also recorded similar maximum cardiac outputs and heart rates, but dissimilar $\text{VO}_{2\text{max}}$ (l.min^{-1}) between paraplegics (T1-T6) and As. The authors suggested major alterations in vasomotor regulation below the injury site in the paraplegics to explain these differences. Indeed differences in $\text{VO}_{2\text{max}}$ ($\text{ml.kg}^{-1}.\text{min}^{-1}$) between As and both LLPs and HLPs groups in the present study were further diminished when weight variations ($\text{ml.kg}^{-1}.\text{min}^{-1}$) were considered in the calculations.

Maximum metabolic performances in groups

Despite As subjects producing impressively greater total PO (W) values to all other groups, comparison of the energy expenditure required to produce each W of work (W.Kcal^{-1}) revealed similarities between groups. Measures of 10.02, 9.12, 9.3 and 9.9 were recorded for LLPs, HLPs, LLQs and HLQs respectively. This would suggest that much of the greater physical upper body work capacity As subjects possessed in the current study was contributed not by any apparent differences in metabolic potential, but possibly more towards As subjects' significantly heavier body weights. This would result in a greater proportion of absolute workable muscle mass and obvious greater potential to perform higher levels of absolute work (PO).

Interpretation of PO results indicated greater relative power outputs per unit body weight (W.kg^{-1}) for As to all other SCI. When physically non-disabled subjects' weight

discrepancy for this variable was controlled, and differences in metabolic efficiencies between groups already disregarded, it would appear that As were able to perform more efficiently during their upper body exercise because of additional reasons. SCI cardio-respiratory limitations alone are not sufficient to explain these differences, since both absolute ($\text{l}\cdot\text{min}^{-1}$) and relative ($\text{l}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) $\text{VO}_{2\text{max}}$ levels were similar between As and all paraplegic subjects.

It is postulated that perhaps As were able to adopt better seating postures in which to perform ACE. More successful recruitment of trunk stabilising musculature through better neuromusculature control and/or greater amounts of available muscle mass would permit improved upper body positioning. This would likely provide enhanced biomechanics for more efficient upper body muscle force application to the handles of the arm crank ergometer. This is supported in the results since LLQs had similar power ($\text{W}\cdot\text{kg}^{-1}$) and energy expenditure ($\text{Kcal}\cdot\text{kg}^{-1}$) outputs to both paraplegic groups. HLQs have significantly greater relative losses in both function and strength of trunk stabilising and upper limb musculature due to involvement of higher (C3-C5) cervical nerve roots. This likely results in less efficient upper body muscle ACE work. Muscles affected include pectoralis major, latissimus dorsi, rhomboids, rotator cuff muscles, triceps and biceps brachii.

Anthropometric variability between groups may also have played a part in explaining measured differences in work and metabolic performances. Although As were significantly heavier to all others, HLPs were on average 9% heavier than LLPs. This may provide reason for their unexpected greater, albeit non-significant, absolute levels for $\text{VO}_{2\text{max}}$ ($\text{l}\cdot\text{min}^{-1}$), total PO (W) and energy expenditure (Kcal).

Since HLQs were on average only 2% heavier than their LLQs counterparts, such differences in $\text{VO}_{2\text{max}}$ could be partially rationalised by variance in the body composition of members from each group. HLQs subjects probably contained a less proportion of active muscle mass due to greater relative muscle paralysis and a more sedentary lifestyle. Despite estimated body fats being similar, an increase in intramuscular fat, fibrotic and other connective tissue deposition is likely.

The results of this study therefore suggest that spinal cord injury, regardless of site, does not appear to effect metabolic function. However, other consequences of different spinal cord lesion location, including variations in the severity of muscle paralysis and alterations in body composition such as proportion of lean body mass, appear to reduce the ability to optimise metabolic function during high intensity upper body ACE exercise.

This study highlights the diversity in maximal cardio-respiratory and metabolic potential within SCI populations traditionally categorised as homogenous. The present research reveals the potential inaccuracies that may be

encountered if interpretation of such data is not further categorised into more distinct SCI lesion sites. The importance for well defined spinal cord lesion site classification techniques within thoracic and cervical regions for differentiating upper body physical exercise potential between SCI individuals has been justified by the findings of this study.

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