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A. J. Thomas, G. M. Davis,
J. R. Sutton

Cardiovascular and Metabolic Responses to Electrical Stimulation-Induced Leg Exercise in Spinal Cord Injury

Rehabilitation Research Centre,
The University of Sydney, Sydney,
Australia

Abstract: Electrical stimulation-induced leg muscle contractions provide a useful model for examining the role of leg muscle neural afferents during low-intensity exercise in persons with spinal cord-injury and their able-bodied cohorts. Eight persons with paraplegia (SCI) and 8 non-disabled subjects (CONTROL) performed passive knee flexion/extension (PAS), electrical stimulation-induced knee flexion/extension (ES) and voluntary knee flexion/extension (VOL) on an isokinetic dynamometer. In CONTROLS, exercise heart rate was significantly increased during ES (94 ± 6 bpm) and VOL (85 ± 4 bpm) over PAS (69 ± 4 bpm), but no changes were observed in SCI individuals. Stroke volume was significantly augmented in SCI during ES (59 ± 5 ml) compared to PAS (46 ± 4 ml). The results of this study suggest that, in able-bodied humans, Group III and IV leg muscle afferents contribute to increased cardiac output during exercise primarily via augmented heart rate. In contrast, SCI achieve raised cardiac output during ES leg exercise via increased venous return in the absence of any change in heart rate.

Keywords: Exercise, Spinal Cord Injury, Cardiovascular Responses

1. Introduction

In humans, the clear separation of central and peripheral neurogenic drive during exercise is often difficult to elicit. Unlike studies which utilise animal models and invasive techniques, human experimentation in this area has been limited to examination of exercise responses under different conditions of muscular contraction, limb movement and pharmacological manipulation. For example, passive limb movements have been utilised to model the condition in which voluntary effort ("central command") is eliminated, and afferent neural information arising from muscle contraction is minimal. Electrically stimulated muscular contractions have been used to model the situation whereby "central command" is eliminated, but afferent receptor stimulation is enhanced. Manipulations of these protocols with spinal cord-injured subjects provides another model for determining the cardiorespiratory responses during exercise when the pathways for

both ascending (peripheral afferent feedback) and descending feed-forward neural drive ("central command") are attenuated or abolished (depending upon the site and extent of spinal cord injury).

The purpose of the present study was to examine the potential contribution of central and peripheral neurogenic signals on the cardiovascular and metabolic responses to dynamic, low-intensity leg exercise in individuals with spinal cord injury and able-bodied subjects.

2. Methods

Eight spinal cord-injured subjects (SCI) and 8 able-bodied individuals (CONTROL) volunteered to participate in the present investigation. All subjects gave written informed consent and underwent medical examination according to guidelines established by the Human Ethics Committee of the University of Sydney. SCI were paraplegics with sensori-motor complete spinal le-

sions between T₅ and T₁₂, but were otherwise healthy. SCI and CONTROL were matched for age, stature and body mass.

All subjects performed knee flexion and extension exercise on a motor-driven isokinetic dynamometer at an angular velocity of 0.8 rad/sec during three randomly assigned conditions. The first condition, passive knee flexion/extension (PAS), was intended to differentiate the reflex effects of Group I and II afferent nerves, from neural signals transmitted by Group III and IV afferent fibres. The second condition, electrically-stimulated knee flexion/extension (ES) was included to promote responses associated primarily with Group III and IV afferent reflexes from the stimulated leg muscles. Neuromuscular stimulation was elicited by a 10-channel laboratory stimulator, which provided monophasic pulses of 0.30 ms duration at 50 Hz. The stimulator output was limited to 150 mA delivered percutaneously to the gluteal, hamstrings, quadriceps, tibialis anterior and

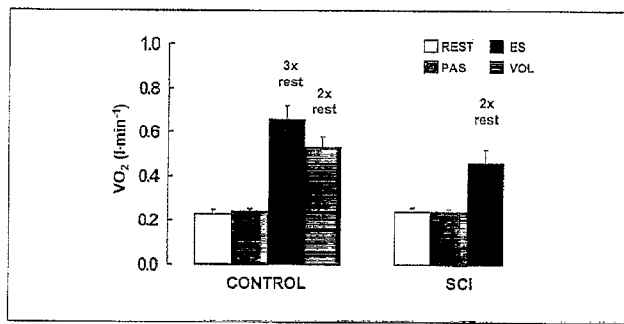


Fig. 1 Oxygen uptake in CONTROL versus SCI subjects. "2x rest" or "3x rest" indicate $p < 0.05$ compared to REST. Data are mean \pm SE.

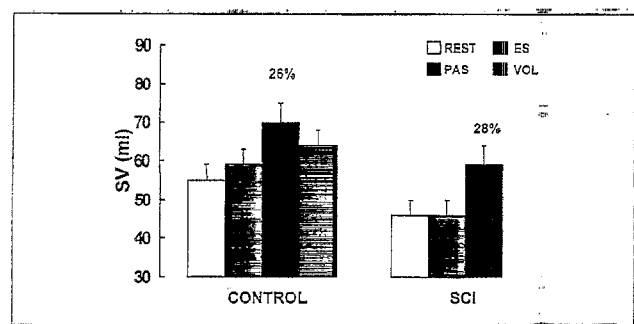


Fig. 3 Stroke volume in CONTROL versus SCI subjects. Percentages indicate $p < 0.05$ compared to REST. Data are mean \pm SE.

gastrocnemius muscle groups of both legs in a fashion to elicit bilateral knee flexion and extension. The last condition, voluntary exercise (VOL), which could be performed only by CONTROLS, was intended to differentiate the role of "central command" on the responses observed.

Cardiovascular, respiratory and metabolic responses were assessed via open circuit spirometry and impedance cardiography, while haemodynamic responses were assessed non-invasively from beat-to-beat blood pressure.

3. Results and Discussion

In SCI and CONTROL, oxygen uptake (Fig. 1) was significantly higher during ES compared to REST or PAS, but VOL was no greater than ES in the CONTROL subjects.

The observation of an increased oxygen uptake (VO_2) during electrically stimulated leg exercise in able-bodied

subjects has been a finding of previous investigations [1, 2]. The magnitude of VO_2 increase in this study was similar to that of Adams et al. [1] who used a similar exercise paradigm and reported a doubling of resting metabolism during ES. Interestingly, the magnitude of VO_2 increase during VOL was lower than during ES. The finding was in contrast to Adams et al. [1], who noted slightly greater metabolism during voluntary exercise than electrically stimulated muscle contractions. We speculate that the observed differences in oxygen cost between VOL and ES may reflect differences of muscle fibre recruitment between the two exercise modes or a more precise control of motor unit recruitment during voluntary exercise.

SCI also demonstrated significant elevations of VO_2 in response to ES, however these increments were uniformly lower compared to CONTROLS. Adams et al. [2] observed smaller increments of VO_2 in response to electrically stimulated leg muscle exercise in their

SCI volunteers (60% increase over rest) compared to able-bodied cohorts (93% increase from rest). The lower VO_2 rise observed in SCI is probably a reflection of their decreased leg muscle mass which is a sequelae of chronic spinal cord injury. Scelsi et al. [3] have reported significant and progressive decreases in muscle fibre diameter as well as a myriad of histopathological and ultrastructural differences between normal and chronically paralysed muscle, including a reduction in the density and size of mitochondria. These chronic changes probably reduce the ability of paralyzed muscle to utilise oxygen when electrically stimulated.

Expired ventilation was significantly elevated over resting conditions (7.2 ± 0.6 l/min) in CONTROL during both VOL (16.1 ± 0.9 l/min) and ES (22.4 ± 2.3 l/min). In the SCI group, ventilation was also increased over REST (8.1 ± 0.7 l/min) during ES (17.3 ± 1.6 l/min). The pattern of expired ventilation (VE) for both SCI and CONTROL groups in this

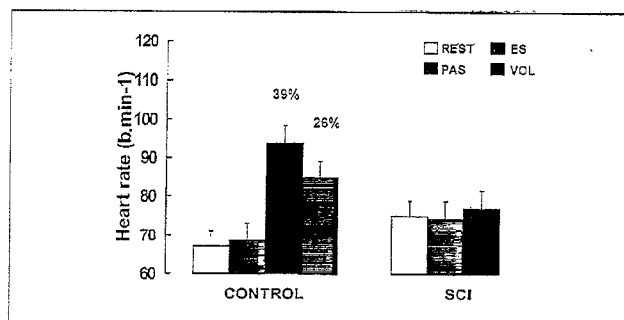


Fig. 2 Heart rate in CONTROL versus SCI subjects. Percentages indicate $p < 0.05$ compared to REST. Data are mean \pm SE.

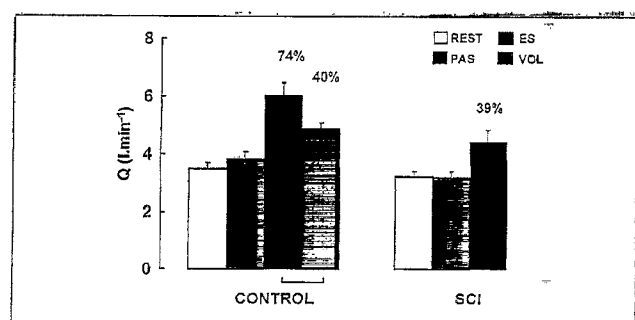


Fig. 4 Cardiac output in CONTROL versus SCI subjects. Percentages indicate $p < 0.05$ compared to REST. \square indicates $p < 0.05$ between VOL and ES. Data are mean \pm SE.

investigation was consistent with an augmented metabolic carbon dioxide production during exercise compared to REST. Previous investigators have confirmed that a significant linear relationship exists between VE and CO₂ production during electrically stimulated leg exercise in both able-bodied individuals [1, 4] and spinal cord-injured subjects [2, 5]. Since exercise induced by ES attenuated or eliminated the effect of "central command" on the effector response (i. e., such exercise was involuntary), it is difficult to perceive how this mechanism could be responsible for the accurate matching of respiratory motor output and metabolic rate. Similarly, the findings of an increased expired ventilation in SCI during ES-exercise clearly demonstrated that the normal relationship between ventilation and CO₂ production was maintained in the absence of neural afferent feedback (i. e., spinal cord lesions abolish or reduce ascending neural drive) from peripheral muscle metabolism. It would appear that neither "central command" nor peripheral afferent reflexes are critical for the normal respiratory response to leg muscle exercise.

The heart rate (HR) responses among PAS, VOL and ES exercise in SCI individuals and their able-bodied cohorts highlighted some significant differences (Fig. 2). In both groups, PAS elicited no increases of HR over REST. These findings are comparable to previous studies which have investigated passive exercise in SCI [5] and able-bodied [6] subjects. Passive lower-limb movements were used in this study to differentiate the HR responses which might be mediated by proprioceptors (carried by Group I and II neural afferents) versus those initiated by muscle contractions (Group III and IV afferent fibres). The lack of any change in HR (or VO₂) during slow velocity passive knee flexion and extension, supports the findings of Waldrop et al. [11] that the activation of Group I or Group II muscle afferents have no central cardiorespiratory effects. In CONTROL subjects, HR was increased during both ES (by 39%, $p < 0.05$) and VOL (by 26%, $p < 0.05$) exercise compared to REST or PAS. However, the differences between exercise conditions was not statistically significant. In marked contrast, the HR

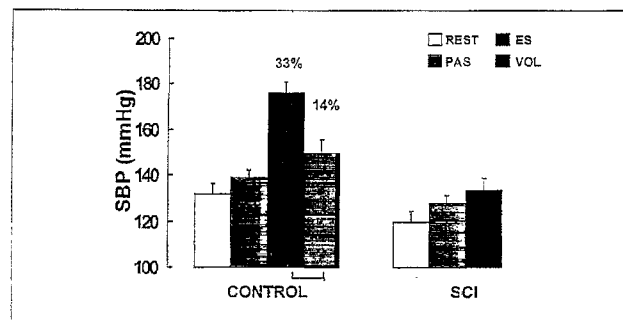


Fig. 5 Systolic blood pressure in CONTROL versus SCI subjects. Percentages indicate $p < 0.05$ compared to REST. \square indicates $p < 0.05$ between VOL and ES. Data are mean \pm SE.

responses among REST, PAS and ES exercise in SCI subjects were not different. Adams et al. [2] and Brown et al. [7] have previously reported similar findings in paraplegic subjects, but Figoni and colleagues [8] have observed increased HR during electrically stimulated leg exercise.

The mechanism underlying the augmented HR in able-bodied subjects during ES leg muscle contractions is probably the afferent neural reflex. Since electrical stimulation provokes muscular exercise in the absence of "central command", an increase of HR in CONTROLS must be due to reflexes arising in the legs as a result of involuntary muscular contractions. We propose that electrically invoked muscular contractions either increase intramuscular pressures and/or raise the concentrations of chemical substances capable of stimulating receptor sites serving small diameter afferent fibres. When stimulated, these mechanoreceptor and metaboreceptor sites initiate centrally directed neural traffic along Group III/IV fibres which act on cardiovascular neurons controlling sympathetic outflow to the heart.

Neural afferent traffic capable of modulating medullary cardiovascular neurons in CONTROL individuals are conveyed in a cephalad direction via the intact spinal cord. In the present study, the absence of an increased HR during ES exercise in SCI subjects suggested that neural afferent feedback was unable to ascend the spinal cord. Consequently, these afferent reflexes were unable to provoke a response from the medullary cardiovascular centers to initiate cardioacceleration.

The stroke volume (SV) responses during PAS, ES and VOL exercise for

SCI versus CONTROL subjects are shown in Figure 3.

SV was significantly elevated over REST in both groups by the addition of ES leg muscle contractions. The relative magnitude of increase in SV was 26% for CONTROLS and 28% for SCI. Glaser et al. [9] reported substantially similar increments of SV (12%-20%) in their SCI subjects undergoing leg ES exercise, but Figoni and coworkers [8] observed up to 61% greater SV in their subjects undergoing ES-induced leg cycling. Since SV in the current study was elevated in the absence of any appreciable increase of HR for the SCI subjects, it was unlikely that a strong neurohumoral sympathetically mediated increase of cardiac contractility contributed to this increased SV. A more likely explanation of augmented SV was that the addition of involuntary muscle contractions produced an increase of preload by augmenting venous return from the exercising legs (i. e., reactivation of the leg "muscle pump").

Cardiac output (Q) was significantly elevated over REST in CONTROL during VOL and ES exercise, and in SCI during ES alone (Fig. 4). These findings are consistent with the data of Glaser et al. [9], who noted significant elevations of resting Q in response to electrically stimulated contractions of the thigh and calf muscles (13%-15%) or pulsed static electrical stimulation of the same muscles (18%-20%).

For CONTROL, the exercise conditions which significantly increased Q over rest or passive limb movements (i. e., ES and VOL), were those conditions which also elevated HR. Despite the fact that SV was augmented during ES, it appeared that the increased Q in

CONTROL subjects was primarily due to neurally mediated cardioacceleration, rather than increases of SV elicited by enhanced cardiac preload.

In contrast, the condition which significantly elevated Q over REST or PAS in the SCI group (i.e., ES), was that which significantly increased SV. Since resting HR did not change in these subjects, increased Q in response to ES exercise in SCI subjects was driven more by the volume load imposed by augmented venous return from the leg muscles, than by neural regulation of HR.

In the current study, both ES-induced muscular contractions and VOL exercise elicited significant increases of systolic blood pressure in CONTROL subjects (by 33% and 14% respectively; Fig. 5). An exercise induced rise of systolic blood pressure (SBP) is a consistent finding of able-bodied individuals undertaking dynamic or static exercise, whether such exercise is produced by voluntary muscular effort or involuntary muscular contraction [10]. During ES, the increase in SBP is presumably facilitated by an increase in the activity of chemosensitive afferents within working muscles. Neural afferent signals elicit increased efferent sympathetic activity mediating vasoconstriction of the systemic resistance vessels (and also cardioacceleration). In the current study, the 39-44 mmHg increase in SBP during electrically stimulated lower limb contractions, may have been exclusively mediated by this peripheral afferent reflex, since ES presumably elicits muscle contraction without an increase in central motor command.

In contrast, the SBP of SCI subjects failed to significantly increase above resting levels during dynamic ES exercise. Previous studies have confirmed this finding in other SCI groups [2, 8, 9].

Since voluntary exercise of the lower limbs could obviously not be achieved in SCI subjects, it is difficult to surmise how centrally generated efferent outflow (i.e., "central command") might mediate the control of blood pressure in these individuals. Furthermore, afferent signals arising in the electrically stimulated muscles have diminished ability to ascend towards the vasomotor centers due to the spinal cord lesion. We suggest that the absence of any increase of SBP during electrically stimulated exercise in SCI subjects is likely due to the inability of afferent signal generation to reach the vasomotor center and elicit the reflexive change in the blood pressure.

4. Conclusion

The cardiovascular and metabolic data in this study demonstrated significant differences between the responses of able-bodied and SCI subjects during electrically stimulated lower limb exercise, confirming the role of the skeletal muscle afferent reflexes in the regulation of adaptations to low-intensity dynamic leg exercise. In contrast, the metabolic and respiratory responses to electrically stimulated exercise in AB and SCI groups were similar. Spinal cord injury does not appear to affect the ability of the central nervous system to elicit metabolic or ventilatory responses to electrically induced muscular contractions.

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Address of the authors:

A. J. Thomas,
G. M. Davis, Ph. D. FACSM,
Rehabilitation Research Centre,
Faculty of Health Sciences,
The University of Sydney,
P. O. Box 170,
Lidcombe, NSW 2141 Australia
E-mail: G. Davis@acchs.usyd.edu.au