ORIGINAL ARTICLE

Cardiovascular Responses and Postexercise Hypotension After Arm Cycling Exercise in Subjects With Spinal Cord Injury

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ABSTRACT. Claydon VE, Hol AT, Eng JJ, Krassioukov AV. Cardiovascular responses and postexercise hypotension after arm cycling exercise in subjects with spinal cord injury. Arch Phys Med Rehabil 2006;87:1106-14.

Objective: To examine postexercise hypotension and contributing factors in subjects with spinal cord injury (SCI).

Design: Prospective clinical research study.

Setting: Rehabilitation center.

Participants: Subjects with chronic cervical-level (n=19) and thoracic-level (n=8) SCI.

Interventions: Not applicable.

Main Outcome Measures: Subjects underwent graded arm-cycling with electrocardiogram and oxygen uptake monitoring to exhaustion. Heart rates and blood pressures were measured before and after exercising. Injury to motor and sensory pathways was determined by American Spinal Injury Association grade, and to autonomic pathways by sympathetic skin responses (SSRs) (n=16).

Results: Resting blood pressures and heart rates were lower in cervical than thoracic SCI (mean arterial pressure [MAP]: cervical, 76.6±2mmHg; thoracic, 93.5±3mmHg; $P<.001$). Following exercise, heart rate responses were greater in thoracic than cervical SCI; MAP increased in thoracic SCI (8.4±5mmHg) and markedly decreased in cervical SCI (−9.3±2mmHg) ($P<.001$). No subject had significant electrocardiographic abnormalities at rest or during exercise. There were correlations between SSR and heart rate and blood pressure responses to exercise; the correlation between the SSR and blood pressure response was due to an interaction between the heart rate and blood pressure responses.

Conclusions: Abnormal cardiovascular responses to exercise and transient postexercise hypotension were common in cervical, but not thoracic SCI. This may be partly related to loss of descending sympathetic nervous control of the heart and vasculature following high SCI.

Key Words: Exercise; Hypotension; Rehabilitation; Spinal cord injuries.

PEOPLE WITH SPINAL CORD injury (SCI) are prone to unstable blood pressure control, including orthostatic hypotension.[1-3] This may cause symptoms of impending loss of consciousness such as dizziness, light-headedness, nausea, or even syncope[2,4] and can severely impact on quality of life for people with SCI. Orthostatic hypotension (OH) classically occurs following the assumption of an upright posture, and can be exacerbated by various factors including heat stress, vasodilator drugs, and after meals.[5] The incidence of OH and syncope is also increased immediately after exercise in susceptible populations.[6,7] Exercise not only provokes transient hypotension and fainting, but may also elicit a sustained reduction in arterial blood pressure that is reported to last from less than 1 hour[8] up to 12 hours.[9] This rather more sustained and variable reduction in arterial pressure following exercise is known as postexercise hypotension. The magnitude and duration of postexercise hypotension is reported to be independent of the exercising muscle mass,[10] exercise duration,[8,11] modality,[12] or intensity.[13,14] However, there seems to be a link between postexercise hypotension and the recovery posture[11] and resting blood pressure prior to undertaking exercise,[9] whereby upright recovery and higher resting blood pressures are associated with a greater postexercise hypotension response.

Although a number of studies have examined cardiovascular control in people with SCI *during* exercise,[15-20] information on the cardiovascular responses *after* exercise is limited. It is the period after cessation of exercise that we were particularly interested in, because it is then that blood pressure is most likely to fall and symptoms of hypotension occur.[6,7]

Therefore, an aim of this study was to examine the incidence and severity of postexercise hypotension by examining the cardiovascular responses to a brief period of exhaustive arm cycling exercise in subjects with cervical- and thoracic-level SCI. There may be a link between the severity of postexercise hypotension and the integrity of sympathetic nervous control of blood pressure,[21] so we also examined whether there would be a correlation between hypotension and the extent of damage to sympathetic circuits after SCI by using sympathetic skin responses (SSRs), a noninvasive assessment of sympathetic integrity.[22]

Finally, people with SCI may also be more prone to cardiac arrhythmias than able-bodied subjects.[23-25] After SCI, the incidence of cardiac arrhythmias during exercise is not known. Thus, a secondary aim of this study was to document any electrocardiographic changes that occurred following exercise in people with SCI, and any relation between cardiac abnormalities and cardiac sympathetic control as inferred using SSR.

The evaluation of the incidence and severity of postexercise hypotension and electrocardiographic abnormalities in people with SCI will help to identify those most at risk for exercise-related complications, in order to facilitate the safe participation in physical activity and rehabilitation in this population.

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Supported by the Rick Hansen Man In Motion Research Foundation (grant no. 05-0747), the Canadian Institutes of Health Research (career scientist award), and Michael Smith Foundation of Health Research.

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit upon the authors or upon any organization with which the authors are associated.

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0003-9993/06/8708-10756$32.00/0
doi:10.1016/j.apmr.2006.05.011

This is of particular importance given that people with SCI are known to be at greater risk of cardiovascular mortality than able-bodied persons.[26,27]

METHODS

Participants

The study was approved by the Vancouver General Hospital and University of British Columbia Research Ethics Committees, and was performed in association with the Declaration of Helsinki (2004) of the World Medical Association. All subjects had chronic traumatic SCI (≥1y duration), were apparently healthy, taking no cardiovascular medication, and none had known cardiovascular disease. All subjects gave written informed consent, and all were able to manually cycle an arm ergometer.

Neurologic Assessment

We used the American Spinal Injury Association/International Medical Society of Paraplegia (ASIA) Impairment Scale for neurologic classification of the level and severity of SCI through the assessment of motor and sensory impairment.[28] ASIA grades A (motor and sensory complete) and B (motor complete) represent severe SCI. ASIA grades C (motor incomplete, although nonfunctional) and D (motor incomplete, functional) characterize moderate SCI, and ASIA grade E describes normal motor and sensory function.

Arm Cycling Exercise

Subjects performed a graded arm-cycle ergometer task to peak oxygen consumption (Vo_2peak) on an electronically braked arm ergometer.[a] Throughout testing we continuously monitored the 12-lead electrocardiograph (Quark C12)[b] and breath-to-breath Vo_2 and carbon dioxide production using a portable gas analyzer system (Cosmed K4b^2).[b] The "limb leads" of the electrocardiograph were placed on the torso to minimize the effects of arm cycling on the signal-to-noise ratio. Brachial blood pressure was determined by sphygmomanometry after a 10-minute rest period, immediately following the completion of the test, and every minute during a 5-minute recovery period. Blood lactate was determined at rest and peak exercise.

After 10 minutes of seated rest, at the end of which baseline readings were taken, subjects began to arm cycle against no resistance at a comfortable cadence (generally 50−70rpm). After a 1-minute warm-up, work load was increased by 5 or 10W/min for subjects with paraplegia or tetraplegia respectively, until the subject was no longer able to maintain a cycling rate of 30rpm. Peak Vo_2 was considered to have been reached if at least 2 of 3 criteria for maximal effort were fulfilled: (1) a respiratory exchange ratio of 1.0 or higher; (2) a plateau in Vo_2 with increased exercise intensity; or (3) volitional fatigue (ie, decline in cycling rate <30rpm). For safety reasons the test would be terminated early if indicated according to the American College of Cardiology and American Heart Association practice guidelines.[29]

Sympathetic Skin Responses

We recorded SSRs in 16 subjects (10 cervical, 6 thoracic); 11 subjects declined to participate in this test. SSRs were recorded bilaterally and simultaneously from both hands and feet using self-adhesive electrodes. Each stimulus consisted of a single electric pulse of 0.2ms duration and an intensity of 8 to 10mA. In all subjects, a standard stimulation protocol was applied, whereby subjects were stimulated at the left median nerve at the wrist and the left posterior tibial nerve at the ankle. A total of 10 stimuli were applied at each location, with variable time delays in order to eliminate habituation. Data were recorded continuously using an analog-to-digital converter (Powerlab/16SP Model ML795)[c] interfaced with a computer. Data were stored for subsequent offline analysis using specialized software (Powerlab version 5.0.2).[c] Responses were qualified by the number of reproducible SSR elicited.[22] Thus, the maximum response at each site would score 10, whereby all 10 stimuli elicited an SSR.

Statistical Analysis

We performed all statistical analyses using GraphPad software[d] for Windows. Data are expressed as mean ± standard error of the mean (SEM). Values were tested for normality using the Kolmogorov and Smirnov assumptions. Comparisons between the 2 groups were performed using unpaired Student *t* tests. Within-group comparisons were performed using repeated-measures analysis of variance with the Dunnett or Tukey post hoc test. Sex differences between groups were evaluated using the Fisher exact test. Correlations between variables were performed using the Pearson or Spearman correlation coefficient, as appropriate. Statistical significance was assumed at the level of P less than .05.

RESULTS

Participant Characteristics

There were no significant differences in age or sex distribution between the groups. Subjects with cervical injury were taller ($P<.05$) than those with thoracic SCI, although the body mass index (BMI) for the 2 groups was similar (table 1). Subjects with cervical SCI had been injured for longer than those with thoracic SCI ($P<.05$).

All subjects exercised until their Vo_2peak was reached, and in no case was the test discontinued due to adverse cardiovascular symptoms or signs. Compared with subjects with cervical SCI, in subjects with thoracic SCI the Vo_2peak was greater ($25.2\pm2.6 mL\cdot kg^{-1}\cdot min^{-1}$ vs $14.9\pm1.1 mL\cdot kg^{-1}\cdot min^{-1}$; $P<.001$) and the resting blood lactate was greater (2.55 ± 0.40mmol/L vs 1.45 ± 0.16mmol/L; $P<.001$). Both groups showed the expected increase in lactate after exercising ($P<.001$); this increase was greater in the subjects with thoracic than the subjects with cervical SCI (8.71 ± 0.48mmol/L vs 4.94 ± 0.37mmol/L; $P<.001$).

Cardiovascular Responses

Resting seated systolic (SAP) and diastolic (DAP) arterial pressures were significantly greater in subjects with thoracic than cervical SCI (SAP: 115.3 ± 3.9mmHg and 96.2 ± 2.4mmHg,

Table 1: Subject Characteristics of the 2 Groups Studied

SCI Subjects	Cervical	Thoracic
N	19	8
Age (y)	39.2±2.0	32.0±2.0
Male:female	18:1	5:3
Height (m)	1.80±0.02	1.71±0.04*
Weight (kg)	77.9±2.7	69.0±4.9
BMI (kg/m^2)	24.2±0.9	23.5±1.2
Duration of SCI (y)	15.5±2.2	6.0±1.4*

NOTE. Values are mean ± SEM or as otherwise indicated. There were no significant differences in age, sex, weight, or BMI between groups. Subjects with cervical SCI were taller and had been injured longer than those with thoracic SCI.
*$P<.05$.

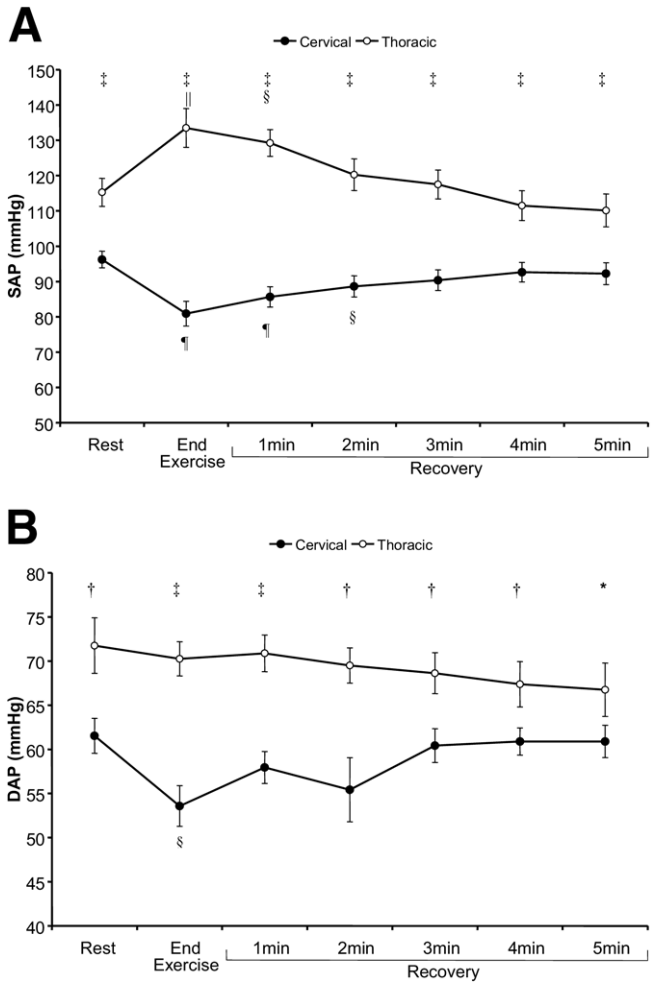

Fig 1. (A) SAP and (B) DAP responses to exercise. Resting seated SAP and DAP were significantly higher in subjects with thoracic than those with cervical SCI. After exercise, SAP was increased in the subjects with thoracic SCI, but was decreased in those with cervical SCI. After exercise, DAP was unchanged in subjects with thoracic-level SCI, but was decreased in those with cervical SCI. During the recovery from exercise, blood pressures began to return to the resting levels in both groups, but were consistently elevated in subjects with thoracic compared with cervical SCI. *$P<.05$; †$P<.01$; ‡$P<.001$ cervical versus thoracic; §$P<.05$; ‖$P<.01$; ¶$P<.001$ within-group comparison against the resting condition. Note the change in scale in the lower panel.

$P<.001$; DAP: 71.8±3.1mmHg and 61.5±2.0mmHg, $P<.01$) (fig 1). After exercise, SAP was increased in thoracic ($P<.01$) and decreased in cervical ($P<.001$) subjects. At the end of the exercise, SAP was 133.5±5.5 and 80.9±3.5mmHg in the subjects with thoracic and cervical SCI, respectively ($P<.001$). After exercise, DAP was decreased significantly ($P<.05$) in cervical SCI, but did not differ significantly from baseline in thoracic SCI (cervical: 53.6±2.3mmHg; thoracic: 70.3±1.9mmHg; $P<.001$).

Mean arterial pressure (MAP) was higher at all stages of testing in subjects with thoracic than cervical SCI ($P<.001$). Resting MAP was 76.6±1.7 and 93.5±3.3mmHg ($P<.001$) in cervical and thoracic SCI subjects, respectively. At the end of the exercise, MAP showed an insignificant increase in thoracic to 101.9±3.4mmHg and a significant decrease ($P<.001$) in cervical SCI subjects to 62.2±2.8mmHg.

Throughout the recovery period, SAP, DAP, and MAP all steadily returned to baseline levels in both groups, but remained significantly higher in thoracic than in subjects with cervical-level SCI throughout.

Five (26%) of the subjects with cervical SCI and 1 (13%) with thoracic SCI reported presyncopal symptoms associated with the initial fall in blood pressure that occurred after exercising. The symptoms improved as the blood pressure returned back toward the resting levels. In no subject were medical interventions required to restore blood pressure.

Resting seated heart rates were significantly greater in thoracic than cervical SCI subjects (81.4±6.5 and 68.3±2.6bpm, $P<.05$) (fig 2). Heart rate increased in both groups after exercise, but this increase was greater in thoracic than in cervical SCI subjects (+80.3±8.1 and +36.9±4.1bpm, $P<.001$), resulting in greater peak heart rates in thoracic than cervical subjects (158.6±4.4 and 105.2±4.4bpm, $P<.001$). Heart rates gradually decreased during recovery in both groups, and returned back to resting levels in both groups after 5 minutes of recovery. Heart rates during recovery were greater at all times in subjects with thoracic SCI ($P<.001$).

Sympathetic Skin Response

SSRs in each subject following medial and tibial nerve stimulation are shown in table 2. Only one of the subjects tested with cervical SCI (C6 ASIA grade B) had preservation of SSR; we elicited 8 SSRs from the right palmar surface in response to median nerve stimulation. In all other subjects with cervical SCI, the SSRs were absent from both palmar and plantar surfaces in response to both median and tibial nerve stimulation. In all thoracic SCI subjects tested, SSRs were preserved in both left and right palmar surfaces in response to median nerve stimulation. In 2 subjects with thoracic SCI (T4 ASIA grade B, T10 ASIA grade A), we elicited reproducible SSR from both plantar surfaces after median nerve stimulation. Three thoracic SCI subjects had preserved palmar SSR in response to tibial nerve stimulation, and in 2 of these subjects some SSR could be elicited from the plantar surfaces after

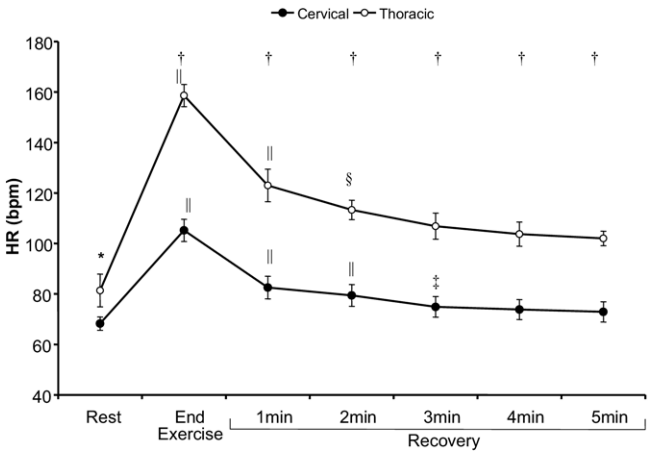

Fig 2. Heart rate (HR) responses to exercise. Resting seated heart rates were significantly lower in cervical than thoracic SCI subjects. The heart rate at the end of exercise was increased in both cervical and thoracic groups, but this increase was greater in those with thoracic SCI. During the recovery from exercise heart rate decreased in both groups toward the resting supine levels, and was significantly greater in those with thoracic SCI at all time points. *$P<.05$; †$P<.001$ cervical versus thoracic; ‡$P<.05$; §$P<.01$; ‖$P<.001$ within-group comparison against the resting condition.

Table 2: The Number of Positive SSRs are Shown for Each Medial and Tibial Nerve Stimulation

	Medial Nerve Stimulation				Tibial Nerve Stimulation			
SCI	LA	RA	LL	RL	LA	RA	LL	RL
C4 ASIA grade A	0	0	0	0	0	0	0	0
C4 ASIA grade A	0	0	0	0	0	0	0	0
C4 ASIA grade A	0	0	0	0	0	0	0	0
C4 ASIA grade A	NT							
C5 ASIA grade A	NT							
C5 ASIA grade A	0	0	0	0	0	0	0	0
C5 ASIA grade A	NT							
C5 ASIA grade B	0	0	0	0	0	0	0	0
C5 ASIA grade B	0	0	0	0	0	0	0	0
C6 ASIA grade A	NT							
C6 ASIA grade A	NT							
C6 ASIA grade B	0	0	0	0	0	0	0	0
C6 ASIA grade B	NT							
C6 ASIA grade B	0	8	0	0	0	0	0	0
C7 ASIA grade A	0	0	0	0	0	0	0	0
C7 ASIA grade A	NT							
C7 ASIA grade A	NT							
C7 ASIA grade B	NT							
C7 ASIA grade C	0	0	0	0	0	0	0	0
T3 ASIA grade A	10	10	0	0	0	0	0	0
T4 ASIA grade B	10	10	10	10	10	10	10	10
T7 ASIA grade A	10	10	0	0	0	0	0	0
T10 ASIA grade A	10	10	9	9	6	4	0	2
T11 ASIA grade A	10	10	0	0	10	10	0	0
T11 ASIA grade A	10	10	0	0	0	0	0	0
T11 ASIA grade A	NT							
T12 ASIA grade A	NT							

NOTE. Ten stimuli were applied to each nerve in random fashion to eliminate habituation. The number of positive SSRs is shown for each subject from the left arm (LA), right arm (RA), left leg (LL), and right leg (RL).
Abbreviation: NT, not tested (in subjects who declined to participate in this part of the study).

tibial nerve stimulation. Only 1 subject with incomplete thoracic SCI (T4 ASIA grade B) had SSR comparable to those seen in able-bodied control volunteers.

Electrocardiographic Analysis

The majority of subjects displayed normal sinus rhythm at rest (table 3). After exercise, all electrocardiograms (ECGs) showed the typical changes associated with tachycardia (decreased RR, QT, and PR interval). There were no clinically significant changes compared with the resting electrocardiographic activity in any subject.

Relationship Between SSR and Cardiovascular Control

Because there were no significant electrocardiographic abnormalities in any subject, it was impossible to examine a relationship between the SSR and electrocardiographic abnormalities after SCI. However, it was noted that sinus bradycardia was more common at rest in subjects with cervical SCI, in whom the SSRs in the upper extremities were usually absent.

One subject with incomplete cervical SCI (C6 ASIA grade B) had some preservation of unilateral palmar SSR, yet still exhibited postexercise hypotension. One subject with thoracic SCI (T12 ASIA grade A) developed postexercise hypotension, but unfortunately declined to participate in SSR testing. All subjects with thoracic SCI in whom SSRs were recorded showed normal palmar SSRs. Interestingly, 2 subjects with cervical SCI (C7 ASIA grade C, C4 ASIA grade A) did not develop postexercise hypotension, despite absent palmar SSR.

There was a significant ($P<.001$) correlation between the number of reproducible palmar SSRs and the maximum heart rate rise during the exercise test (fig 3A). There was also a weak, but statistically significant, correlation between the number of palmar SSRs elicited and the blood pressure immediately after cessation of exercise ($R=.656$, $P<.01$). However, multiple regression analyses revealed that the apparent correlation between the blood pressure after exercise and the SSR was actually due to an interaction between the heart rate and blood pressure responses ($P<.03$; correlation shown in fig 3B) rather than an interaction between blood pressure and SSR per se ($P=.95$).

DISCUSSION

We examined, for the first time, the incidence of postexercise hypotension in subjects with cervical and thoracic SCI. Baseline cardiovascular parameters, and cardiovascular responses following exercise were abnormal, and hypotension immediately after cessation of exercise was common in subjects with cervical, but not thoracic SCI. This appeared to be partly related to the loss of descending sympathetic nervous control of the heart and vasculature following high SCI. However, we did not see a sustained reduction in blood pressure following exercise. We found no evidence that SCI subjects were prone to cardiac arrhythmias or electrocardiographic abnormalities during or after exercise.

The causes of postexercise hypotension, although not fully understood, are probably related to neural and local mecha-

Table 3: Resting 12-Lead ECGs for All Subjects

SCI	Resting ECGs
C4 ASIA grade A	NSR; long QT interval (.471s); 1mm anteroseptal ST elevation
C4 ASIA grade A	Sinus bradycardia (50bpm)
C4 ASIA grade A	Sinus bradycardia (51bpm); 1.5mm anteroseptal ST elevation
C4 ASIA grade A	NSR
C5 ASIA grade A	Sinus bradycardia (59bpm); 2mm anteroseptal ST elevation
C5 ASIA grade A	NSR; 1mm anteroseptal ST elevation
C5 ASIA grade A	NSR; 1.5mm widespread ST elevation
C5 ASIA grade B	NSR with marked RSA; 1.5mm anteroseptal ST elevation
C5 ASIA grade B	Sinus bradycardia (50bpm); 2mm anteroseptal ST elevation
C6 ASIA grade A	NSR; left anterior hemiblock (QRS axis, −60°); 1.5mm widespread ST elevation
C6 ASIA grade A	Sinus bradycardia (49bpm) with marked RSA; left anterior hemiblock (QRS axis, −30°)
C6 ASIA grade B	NSR; left anterior hemiblock (QRS axis, −45°); 1.5mm widespread ST elevation
C6 ASIA grade B	NSR; long QTc (.572s); 1.5mm anterior/lateral ST elevation
C6 ASIA grade B	NSR with marked RSA; 1.5mm widespread ST elevation
C7 ASIA grade A	NSR; 1.5mm widespread ST elevation; long QT (.470s)
C7 ASIA grade A	NSR; left anterior hemiblock (QRS axis, −60°); 2mm widespread ST elevation
C7 ASIA grade A	NSR; borderline 1st degree atrioventricular block (PR interval, .24s); 2mm anteroseptal ST elevation
C7 ASIA grade B	NSR; 1.5mm widespread ST elevation
C7 ASIA grade C	NSR; long QTc (.468s)
T3 ASIA grade A	NSR; 1mm anteroseptal ST elevation
T4 ASIA grade B	NSR; 1mm widespread ST elevation
T7 ASIA grade A	NSR with marked RSA; 1mm anteroseptal ST elevation
T10 ASIA grade A	NSR; 1mm widespread ST elevation
T11 ASIA grade A	NSR
T11 ASIA grade A	Sinus bradycardia (54bpm); 1mm widespread ST elevation
T11 ASIA grade A	NSR; long QTc (.461s)
T12 ASIA grade A	Sinus tachycardia (115bpm); frequent ventricular ectopics

NOTE. Resting electrocardiographic characteristics are shown for each subject. Nonspecific ST-segment changes and sinus bradycardia were common findings. Four subjects had slightly long QTc (>440ms). Three subjects had left axis deviation associated with left anterior hemiblock. Abbreviations: NSR, normal sinus rhythm; RSA, respiratory sinus arrhythmia.

nisms that produce a sustained decrease in regional and systemic vascular resistances.[21] The neural component of this response refers to a marked reduction in sympathetic vasoconstriction, and the vascular component reflects reduced vascular responses to sympathetic stimulation coupled with the presence of local and circulating vasodilators.[21] Persons with SCI often have disruption of descending sympathetic pathways to regions below the lesion level, and thus the degree of sympathetic dysfunction is typically greater in higher-level lesions.[30-33] Although this would be expected to render people with SCI particularly susceptible to postexercise hypotension, people with SCI with sympathetic dysfunction also tend to have lower resting blood pressures, a factor associated with reduced severity of postexercise hypotension.[9]

Cardiovascular Responses to Exercise in Subjects With SCI

The cardiovascular responses in subjects with SCI during exercise have been described in detail previously,[16,17,20] and are beyond the scope of the present study. Complete cervical SCI disrupts descending sympathoexcitatory input to spinal autonomic neurons located in the T1-L2 segments,[34] resulting in complete loss of descending sympathetic control of both the heart and vasculature. In complete mid-thoracic injury below the level of the cardiac sympathetic neurons (T1-5), the heart is under coordinated sympathetic and parasympathetic (vagal) control, although the major vascular capacitance region (the splanchnic bed), lacks descending sympathetic control. Thus, resting seated blood pressures and heart rates were lower in those with cervical than thoracic SCI, as observed previously.[35]

Blood pressure responses in subjects with thoracic SCI were similar to those within the able-bodied population.[8] Blood pressure was elevated immediately after exercise, and gradually returned to baseline within 5 minutes of recovery. This physiologic increase in blood pressure is due to a combination of reduced vagal activity to the heart, increased sympathetic activity to the heart and vasculature, and pronounced vasoconstriction.[7,8,21] Coupled with increased cardiac output this produces a marked increase in systolic blood pressure, with only a modest rise or little change in diastolic blood pressure; vasodilatation in the exercising muscle acts to buffer the increase in diastolic pressure.[8] We did not see a sudden blood pressure fall after exercise in thoracic SCI, nor did we see prolonged hypotension indicative of postexercise hypotension. This is probably because blood pressure regulation is only moderately impaired in thoracic SCI and descending sympathetic control of the main capacitance region of the body, the splanchnic bed, is likely preserved (particularly in low thoracic SCI) as demonstrated by the presence of SSR in the upper body in most thoracic SCI subjects. Furthermore, muscle atrophy and decreased leg blood flow after SCI[36] will minimize venous pooling in the lower limbs during seated recovery after exercise and may compensate for impaired leg vasoconstriction in subjects with thoracic SCI. In addition local veno-arterial reflexes (independent of descending cardiovascular control) could contribute to blood pressure maintenance after exercise in thoracic SCI.[37] The absence of postexercise hypotension in thoracic SCI is not surprising given that the presence of postexercise hypotension in able-bodied subjects is greatly dependent on the resting seated blood pressure of the person, with higher resting

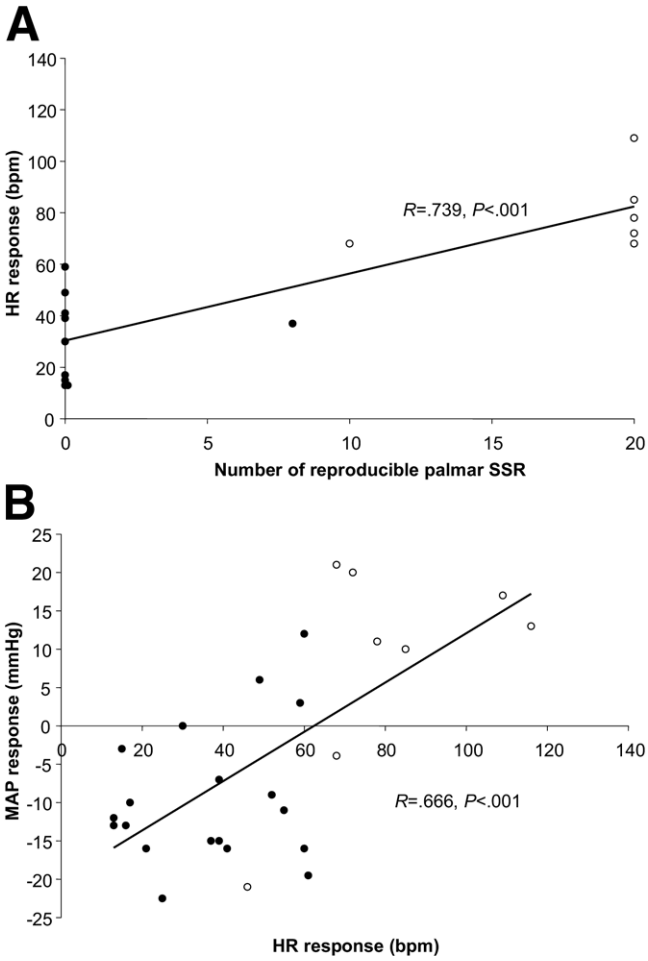

Fig 3. Correlation between the maximum heart rate (HR) during exercise and (A) the number of reproducible SSR and (B) the MAP response immediately after stopping exercise. Data from thoracic SCI subjects are shown in the open circles and cervical SCI subjects in the closed circles. There was a significant positive correlation between the maximum heart rate achieved during the exercise test and the number of reproducible palmar SSR elicited. There was also a significant correlation between the maximum heart rate response during the exercise test and the change in blood pressure immediately after cessation of the exercise test.

blood pressures leading to larger and more prolonged postexercise hypotension.[8,9,21] In able-bodied subjects with resting seated blood pressures below 130/85mmHg, postexercise hypotension is rare,[9] and the mean blood pressure of the subjects with thoracic SCI of approximately 115/72mmHg is well below this threshold value.

In subjects with cervical SCI, however, the blood pressure response after exercise was markedly abnormal. Immediately after exercise blood pressure decreased in cervical SCI subjects to levels sufficient to cause symptoms of presyncope in 5 subjects. The occurrence of presyncope or even frank syncope following exertion is relatively common, particularly in individuals with low orthostatic tolerance.[6] The fact that only 5 of the 19 cervical subjects reported symptoms of presyncope despite marked hypotension in 16 subjects is not surprising, because people with cervical SCI are remarkably tolerant to hypotension,[2] as are other populations with autonomic dysfunction,[38,39] probably due to some alteration in cerebral blood flow autoregulation.[40] The mechanism underlying this marked hypotension immediately after exercise in people with cervical SCI is likely related to a persistent drop in systemic vascular resistance that is not adequately buffered by increases in cardiac output.[7,21] After exercise, cardiac output declines from the peak exercising level more rapidly than systemic vascular resistance recovers,[7,21] which leads to a decrease in blood pressure. In subjects with cervical SCI and impaired descending sympathetic control of the vasculature as demonstrated by the SSR, this decrease is likely to be even more pronounced because there is an inability to constrict capacitance vessels and maintain blood pressure. Furthermore, the smaller cardiac responses after exercise, as described earlier, will contribute to the decreases in cardiac output and blood pressure following exercise. It is interesting to note, however, that the initial hypotension after exercise was rapidly restored to baseline levels. The mechanism for the rapid restoration in blood pressure is unknown, particularly given the disrupted sympathetic nervous control in these individuals. It may be that local reflexes independent of descending control, and muscle atrophy and decreased leg blood flow, will help to limit venous pooling in the dependent limbs as mentioned above.[36,37] In addition, it has been reported that subjects with cervical SCI have increased vasopressin release to hypotensive challenges[41] and possible peripheral α-adrenoceptor hyperresponsiveness,[42] both of which could contribute to their relatively rapid blood pressure recovery after exercise. Also, although all subjects with cervical SCI exercised until exhaustion, the level of exercise performed was less than in subjects with thoracic SCI, as demonstrated by the lower Vo_2peak and lactate levels. This could attenuate the metabolic vasodilatation that occurred during the exercise and facilitate a rapid blood pressure recovery after the initial hypotensive response. Finally, the fact that there was no sustained postexercise hypotension in subjects with cervical SCI may be related to their low resting arterial pressures as mentioned above.[8,9,21]

It is interesting that in 1 subject with cervical SCI in whom there was some, albeit minimal, preservation of unilateral palmar SSR, there was still an abnormal hypotensive response immediately after exercise. Similarly, one of the subjects with thoracic SCI exhibited marked, symptomatic hypotension after exercise. This subject had low thoracic SCI (T12 ASIA grade A) and thus, presumably, sympathetic innervation above this level would have been intact (she declined to participate in SSR testing). Furthermore, 2 of the subjects with cervical injury and absent SSR did not develop hypotension. This confirms previous reports in other populations with disordered blood pressure control, in which, although the control of sympathetic vasoconstriction is important in defending against hypotension,[43-45] other factors such as plasma or blood volumes,[46,47] salt intake,[48] and control of cerebral autoregulation[49] are known to make a significant contribution to blood pressure regulation. These additional contributing factors were not examined in the present study.

Heart rate responses after exercise were directionally similar in both groups, but more marked in subjects with thoracic SCI who had faster heart rates at rest, greater increases in heart rate following exercise, and faster heart rates throughout recovery compared with subjects with cervical-level SCI. The slower heart rates throughout testing in subjects with cervical-level SCI probably reflects a lack of descending sympathetic control of the heart, as shown by the absence of palmar SSR, and the significant correlation between the number of palmar SSR and the heart rate response to the exercise test. In these subjects, the maximum heart rate rise in the absence of descending sympathetic control of the heart is likely mediated by vagal with-

drawal. In support of this, the maximum heart rate in the subjects with cervical SCI at peak exercise was similar to that obtained from complete vagal blockade with atropine in able-bodied subjects.[50] The faster heart rates and larger heart rate responses following exercise in the thoracic subjects are probably due to preserved autonomic control of the heart with mid-lower thoracic SCI, as inferred from the presence of palmar SSR. These data support the notion that palmar SSR can be used as an index of preserved descending sympathetic control to the upper thoracic spinal cord and consequently to the heart.

Electrocardiographic Changes in Response to Exercise in Subjects With SCI

We did not see any significant electrocardiographic abnormalities in any subject at rest, during, or after the exercise protocol. We did see a high incidence of nonspecific ST-segment changes indicative of early repolarization, and mild axis deviations in our cohort of SCI subjects. We do not suggest these electrocardiographic findings are signs of pathology. They have been observed in the SCI population previously[51,52] and probably occur because the ECGs were recorded in the seated position and with the limb leads placed on the torso to minimize background noise during arm cycling. Both of these factors are known to affect the ST-segment morphology and QRS axis.[53-55] It is common to perform seated 12-lead electrocardiographic screens in wheelchair users, rather than have them transfer to a bed. Thus, positional changes and lead modifications should be accounted for in the interpretation of ECGs in the SCI population.

Previous work from our laboratory[56] and others[4,25,57-61] suggests that people with SCI are prone to cardiac arrhythmias during certain stresses, which, at first, appears to be contradictory to the present study. However, the previous reports of arrhythmic events in people with SCI have almost always occurred during autonomic dysreflexia.[56,57,59-61] Autonomic dysreflexia refers to a sudden and inappropriate rise in blood pressure following sensory stimulation below the level of SCI. This represents a unique stimulus to the heart whereby sympathetic drive is extremely high, and is coupled with high vagal tone through the baroreflex.[62] Generally in physiological control of the heart, high sympathetic activity would be coupled with vagal withdrawal. It seems, therefore, that autonomic dysreflexia represents a particularly potent arrhythmic stimulus. Exercise, on the other hand, is associated with vagal withdrawal and increased sympathetic stimulation of the heart in those with low level SCI or incomplete SCI where the descending sympathetic cardiac control is intact, and thus does not appear to be arrhythmogenic. In those with disrupted descending sympathetic control of the heart, there is vagal withdrawal in isolation, which again does not appear to be arrhythmogenic.

Study Limitations

There are some potential confounding factors that should be considered when interpreting the present study. Subjects with thoracic SCI were able to exercise at higher workloads than those with cervical SCI, as shown by their higher V_{O_2}peak and lactate levels following exercise. Although this could potentially affect the results, we do not believe this to be a significant problem because although the responses during exercise would have been related to the exercise intensity, the cardiovascular responses immediately afterward and the magnitude of postexercise hypotension are reported to be independent of exercise intensity and duration.[8,11,13] Furthermore, we aimed to examine the incidence of postexercise hypotension and arrhythmia in the SCI population during typical exercise to volitional exhaustion. Thus, our data are likely to be relevant to the cervical and thoracic SCI population as a whole, in whom exercise work rates are likely to be different in daily life.

Ideally, it would have been preferable to measure beat-to-beat blood pressure responses noninvasively throughout testing using finger plethysmography. Using recently developed software, this technique also allows an estimation of changes in total peripheral resistance, stroke volume, and cardiac output. Unfortunately, finger plethysmography is technically unsatisfactory with large arm movements that occur with arm cycling. An alternative would be to measure beat-to-beat intra-arterial pressures. However, this would be unsuitable for the present study because intravascular techniques are known to increase the incidence of hypotension and syncope,[63] our main outcome measure. A further option would be to examine responses to functional electric stimulation of the legs, while recording beat-to-beat blood pressure with finger plethysmography. However, responses to leg exercise are likely to be very different to arm exercise due to activation of the skeletal muscle pumps in the dependent limbs. We wanted to examine cardiovascular control in a situation as close as possible to that encountered during normal activities, so that we could draw direct inferences about the risks of postexercise hypotension and arrhythmia during daily activities in people with SCI, and so this lower-extremity approach was also undesirable.

Finally, although there were no statistically significant differences in age or sex between groups, there was a trend for the thoracic group to be slightly younger and to include more females than the cervical group. This may have influenced our results. However, we believe our sample was representative of the SCI population in western Canada, in that there were more subjects with cervical than thoracic injuries, more men than women, and a mean age of approximately 35 years.[64] Unfortunately, some of our volunteers declined to participate in SSR testing. We do not know why they declined this procedure; for ethical reasons subjects were entitled to withdraw from the study at any time, and without giving a reason. However, we do not believe that this nonparticipation would have introduced a sample bias, and power calculations reveal the sample size of this part of the study to be adequate.

CONCLUSIONS

We have described the blood pressure response during recovery from exercise, and the associated incidence of postexercise hypotension and cardiac arrhythmia in the SCI population. We found that most subjects with thoracic SCI did not show blood pressure dysregulation following exercise, although hypotension immediately after exercise can occur in thoracic SCI subjects, even those with low thoracic SCI. Subjects with cervical SCI are likely to experience marked hypotension following exercise, which may be symptomatic. This could render them at risk of syncopal events, particularly if the exercise stress is combined with other vasodilator stresses such as a warm environment. Subjects with SCI do not appear to be at increased risk of cardiac arrhythmias during or after exercise. However, there is evidence in the literature that SCI subjects are prone to arrhythmia during episodes of autonomic dysreflexia[56,57,59-61] and are at increased risk of cardiovascular mortality than the able-bodied population.[27] If autonomic dysreflexia were to accompany exercise, for example, through "boosting," there may an associated increase in cardiovascular risk. We advocate that subjects with SCI be encouraged to exercise, and reassured that it is safe for them to do so. This is particularly important given the high incidence of cardiovascular mortality and morbidity in SCI individuals,[26,27] and the known beneficial effects of exercise on cardiovascular risk.[65] It

is, however, important to counsel people with SCI, particularly those with cervical SCI, concerning the risks of hypotension immediately after exercise. Appropriate advice should be given on techniques to minimize the extent of the hypotension, for example, maintenance of an adequate fluid intake, avoidance of overheating, and adopting physical countermaneuvers or postures that minimize venous pooling. In our study, subjects with SCI did not exhibit prolonged postexercise hypotension as is reported in some able-bodied populations, and this is likely related to their low resting blood pressures.

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Suppliers

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