CENTRAL AND PERIPHERAL HAEMODYNAMICS IN INDIVIDUALS WITH PARAPLEGIA DURING LIGHT AND HEAVY EXERCISE

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The purpose of this study was to analyse lower leg skin blood flow (laser Doppler flowmetry) in five individuals with high-level paraplegia (T5–T9), six participants with low-level paraplegia (T10–T12) and six able-bodied controls during 3-minute light and heavy arm-cranking exercises ($\sim$15% and 80% peak power output, respectively). Throughout light exercise, cutaneous vasoconstriction was shown for the control group ($-$20%), but not the low-level ($+$62%) or the high-level paraplegic group ($+$33%). During heavy exercise, vasoconstriction was initially found for controls followed by an increase in skin blood flow during the last 2 minutes, whereas the participants with paraplegia demonstrated skin blood flow increases. Skin blood flow responses were not related to lesion level. Metabolic parameters were not different among the three groups, but heart rates for participants with paraplegia were higher during heavy exercise than in controls. These results suggest impaired sympathetic vasoconstriction in individuals with paraplegia during exercise.

Key words: laser Doppler flowmetry, skin blood flow, vasoconstriction, heart rate, able-bodied, spinal cord injury.

INTRODUCTION

During upper body exercise, individuals with paraplegia with intact sympathetic innervation of the heart demonstrate, at a given submaximal oxygen consumption (VO_2), similar cardiac outputs compared with able-bodied persons (AB). However, cardiac stroke volume and heart rate at these similar submaximal work levels are markedly different, with the spinal cord injured (SCI) individuals having lower stroke volume and higher heart rate values at low (40% of maximum), moderate (60% of maximum) and high (80–90% of maximum) exercise intensities (1–3). These observations have been attributed to an impaired redistribution of blood from the inactive body areas in the lower limbs and the abdomen, leading to a decreased venous return and cardiac filling pressure.

Other studies provide indirect support for the existence of impaired blood redistribution from below the spinal cord lesion during upper body exercise (4–7). However, these studies have not identified the underlying mechanisms of this phenomenon. Figoni et al. (6) reported enhanced cardiac performance in individuals with quadriplegia when exercising in a supine position, compared with an upright seated exercise, suggesting a better venous return. Hopman et al. (7) and Pitetti et al. (8) showed beneficial haemodynamic effects during exercise (lower heart rate and higher stroke volume) in participants with both tetraplegia and paraplegia using an anti-G suit to apply lower body positive pressure. Similar observations were seen in experiments using functional neuromuscular stimulation to induce vigorous pulsatile muscle contractions in the lower limbs of individuals with paraplegia (4, 5). Thus, external compression of the lower body or activation of the peripheral (leg) "muscle pump" by electrical stimulation may support venous return and enhance central haemodynamics in persons with SCI.

Few studies have evaluated blood flow adaptations in the legs of SCI persons. Kinzer & Convertino (9) used impedance plethysmography during an arm-cranking exercise and reported a decrease in fluid accumulation in the legs of AB, whereas an increase was noted in individuals with paraplegia. The authors (9) attributed the increase in fluid accumulation in the legs of their participants with paraplegia to the lack of the venous muscle pump. Hopman et al. (10) reported greater leg volume decreases in AB but not in participants with paraplegia during arm exercise using strain gauge plethysmography. Electromyographic recordings revealed no muscle activity in the legs of the AB subjects in this study (10). Based on these findings, the authors concluded that blood redistribution from the lower limbs was impaired in the SCI participants, owing to a loss of sympathetic vasoconstriction below the lesion.

Given these findings, it is necessary to clarify the role of exercise-induced blood redistribution in the paralysed body regions of individuals with paraplegia. Peripheral vasoconstriction can be observed during the first few minutes of dynamic exercise (11) and is most marked at high workloads (12). Therefore, the aim of the present investigation was to use laser Doppler flowmetry (LDF) to compare the lower leg skin blood flow of individuals with paraplegia having different lesion levels with AB during light and heavy arm-cranking exercises. The aim was to test the hypothesis of impaired vasoconstriction in

the skin of the lower limbs of individuals with paraplegia and the possible effects of the impaired vasoconstriction on cardiac performance.

METHODS

Subjects

Eleven male individuals with paraplegia and six male AB controls participated in the study. Written informed consent was obtained from all participants and the study protocol was approved by the Faculty Ethical Committee. The individuals with SCI were divided into two groups based on their neurological injury level. The individuals with "low-level" paraplegia (LP, $n = 6$) had spinal lesions between T10 and T12. The second group was composed of individuals with "high-level" paraplegia (HP, $n = 5$) with lesions between T5 and T9. To avoid the influences of an impaired sympathetic innervation to the heart or the upper limb, subjects with lesions above T5 were not included in the study. All had longstanding and complete spinal cord lesions of traumatic origin (ASIA class A). None of the participants was under medical treatment at the time of the study.

Protocol

All participants visited the laboratory twice. The first visit was to perform a continuous arm-cranking test of incremental 3-minute workloads while being verbally encouraged until volitional fatigue. Initial power output was set at 15 W, followed by increases in work intensity of 15 W. On the second visit, participants performed a continuous 3-minute exercise bout at a constant load of approximately 80% of their individual maximal power output reached in the first test. Both tests were performed at a pedalling frequency of approximately 60 rpm. The initial workload of the maximal arm-cranking test (3 minutes at 15 W) was the "light" exercise protocol and the second exercise test (3 minutes at 80%) was the "heavy" exercise protocol.

All participants had refrained from caffeine, alcohol and nicotine for at least 8 hours prior to testing. After they were equipped for the test, they rested for 30 minutes in a wheelchair in the exercising position, in order for all variables under study to reach a stable basal value. All tests were administered in the morning, with the laboratory room temperature at $22.9 \pm 1.6°C$ and a relative humidity of $53.9 \pm 2.2\%$.

Materials and measurements

The arm-cranking exercise was performed on a modified electromagnetic Rodby cycle ergometer. The axis of the ergometer was placed at the shoulder height of the seated participant and a fore/aft distance which allowed for a slight bend in the elbow at maximal reach. Both feet were suspended in special footholders and the knees were bent approximately $30°$ with respect to the horizontal line. The aim was to avoid muscle contractions in the lower limbs of the AB and to minimize the influence of gravitational forces on leg blood flow.

During both tests, metabolic measurements (minute ventilation, oxygen and carbon dioxide concentrations of expired air) were continuously monitored, using open-circuit spirometry. A computerized Siemens ELEMA spirometer (Erlangen, Germany) provided values integrated over time intervals of 30 seconds. Expired ventilation was measured with a pneumotachograph and the O_2 and CO_2 concentrations were assessed with an Oxymat-M paramagnetic O_2 analyser and an Ultramat-M infrared CO_2 analyser, respectively. Both analysers were calibrated before each test with a reference gas of known concentrations (15.9% O_2, 5% CO_2). The parameters studied were expired minute ventilation (VE, l/minute, STPD), oxygen consumption (VO$_2$, l/minute, STPD) and carbon dioxide production (VCO$_2$, l/minute, STPD).

Central haemodynamics were evaluated by impedance cardiography, using a Minnesota Impedance Cardiograph (model 304b, Instrumentation for Medicine, Greenwich, U.S.A.) and the classical tetrapolar aluminium band electrode configuration was applied (Cardiograph Electrode Tape, IFM). The first time derivative of transthoracic impedance changes (dZ/dt) and the electrocardiogram (ECG) were registered on a personal computer (PC) via a 16-bit analogue-to-digital data acquisition card (AT-MIO-16XE-50, National Instruments, Austin, U.S.A.), using a sample frequency of 300 Hz. Custom-made software

was used to perform off-line filtering and ensemble averaging of the dZ/dt curve and to calculate cardiac stroke volume and heart rate (13). Cardiac output was taken as the product of stroke volume and heart rate. All metabolic and central haemodynamic parameters were determined over the last 30 seconds of each exercise stage of the maximal arm-cranking test, as well as at the end of the second (80%) test.

Leg skin blood flow was assessed using LDF, a technique which provides a reliable measurement of the relative variations in skin blood flow (14). The measurements are not influenced by underlying muscle blood flow (15), since the measurement depth is approximately 1 mm. The Periflux 4001 Master (Perimed AB, Stockholm, Sweden) was used in this study, with a 1 mW diode laser (780 nm) and a bandwidth that allowed for Doppler shift measurements between 20 Hz and 25 kHz. The signal recorded was represented by an arbitrary value (perfusion unit, PU), resulting from the number of blood cells moving within the measured volume multiplied by the mean velocity of the cells. The instrument was calibrated on a daily basis to the two measuring points 0 PU and 250 PU using the PF1001 Motility Standard. Two integrating probes were used simultaneously to transmit/receive light both to and from seven different scattering volumes. This results in a seven-fold increase in the measuring volume with respect to standard LDF probes, over an area of approximately 1 cm^2, plus a significant decrease in the average intrasubject coefficient of variation (16). Each probe was inserted into a heating probe holder (PF 450 Thermostat Probe Holder) applied to the skin by means of a double-sided adhesive ring. The heater unit (PF 4005 PeriTemp Heater, Perimed AB, Stockholm, Sweden) connected to the probe holders maintained local skin temperature standardized at 39°C, a typically used temperature facilitating investigations of skin blood flow changes without abolishing reflex cutaneous vasoconstriction (17). Measurements were performed on the left lower leg, the middle of the tibialis anterior muscle and the middle of the triceps surae muscle. These skin sites were shaven and cleaned with alcohol prior to testing. A PC continuously recorded the LDF signal using a frequency of 1 Hz. Biological zero, i.e. the continuous low LDF signal during vascular occlusion, was registered after 20 minutes of pre-exercise rest. A pressure cuff applied proximally to the LDF probes, just below the left knee, was rapidly inflated to over 200 mm Hg for 2 minutes. The stable value read during the last 30 seconds was taken as biological zero and subtracted from all other LDF readings (16). The values thus obtained from the two probes were then averaged and expressed with respect to the mean value over 2 minutes at the end of the 30-minute rest period, since LDF allows for semiquantitative measurements only. The recordings during both exercise bouts (light and heavy protocol) were analysed over 30-second intervals to account for minor temporal variations in skin blood flow. The left foot of each participant was immobilized laterally between two vertical bars, and the fibre-optic cables of the LDF probes were secured to the nearest solid object to avoid movement artifacts in the LDF signal during each arm-cranking exercise test.

Statistics

Group differences were assessed by one-way analyses of variance (ANOVA) and the Student–Newman–Keuls post-hoc procedure, when appropriate. Two-factor ANOVA procedures for repeated measures were applied to compare the variation in skin blood flow of the three groups. Spearman rank order correlations were calculated between the maximal skin blood flow values recorded during the two exercise tests and the lesion level of the participants with paraplegia. Significant effects were considered at the 5% level. Data are presented as means $\pm$ S.D., unless otherwise indicated.

RESULTS

The three groups studied had similar physical characteristics, except for body mass, which was lower in the LP subjects (Table I). The power output reached during the first maximal arm-cranking test was similar for all three groups. During the light exercise protocol (3 minutes at 15 W) participants performed at $15 \pm 3\%$ of their individual maximal power

Table I. *Major physical characteristics of able-bodied persons (AB), and individuals with low-level (LP) and high-level (HP) paraplegia*

	AB ($n = 6$)	LP ($n = 6$)	HP ($n = 5$)
Age (years)	28.3 ± 5.6	31.3 ± 8.3	32.2 ± 7.0
Body mass (kg)	78.7 ± 10.1	$60.2 \pm 11.2*$	82.0 ± 10.7
Height (m)	1.80 ± 0.08	1.74 ± 0.10	1.81 ± 0.07
Body mass index (kg/m^2)	24.2 ± 2.0	$19.9 \pm 2.9*$	24.7 ± 2.6
Time since injury (years)	–	8.4 ± 6.5	11.9 ± 6.8
PO$_{max}$ (W)	110.0 ± 7.8	110.0 ± 22.6	99.0 ± 22.8

Data are means $\pm$ S.D. PO$_{max}$ = maximal power output. *Significantly different from the other two groups ($p < 0.05$).

output, whereas during the heavy exercise protocol, the second test, relative intensity was $80 \pm 1\%$.

Table II shows an overview of relevant metabolic parameters assessed during the two tests. During both the light exercise test and the heavy exercise test, VO_2, stroke volume and heart rate were not significantly different among the groups. During the light arm-cranking exercise, heart rate was slightly (but not significantly) higher for HP and higher for LP compared with AB. During the heavy arm-cranking exercise, both SCI groups had higher heart rate values than those of the AB participants.

The skin blood flow values recorded during the two exercise bouts are shown in Fig. 1. During the light arm-cranking exercise, a significant time effect ($F = 7.231$, $p < 0.001$) and group by time interaction ($F = 6.576$, $p < 0.001$) was noted for leg skin blood flow (Fig. 1, upper graph). In the AB group, skin blood flow decreased slightly, whereas marked increases were noted in both SCI groups. During the heavy arm-cranking exercise (Fig. 1, lower graph), skin blood flow adaptations did not follow the same pattern (time effect: $F = 5.549$, $p < 0.001$; group by time interaction: $F = 0.297$, $p = 0.988$). Significant differences were found between the HP group and the AB group at the start (0.5 min) of the exercise bout.

The correlations determined between the maximal value of skin blood flow recorded during exercise and the spinal lesion level ($n = 11$) were not significant at either 15% ($r_s = 0.32$, n.s.) or 80% ($r_s = -0.03$, n.s.) of maximal power output.

DISCUSSION

The most important findings of the present study are that lower leg skin blood flow adaptations during the dynamic arm-cranking exercise were different in individuals with paraplegia compared with AB. There was no exercise-induced vasoconstriction in either the HP or LP group, probably owing to a lack of sympathetic innervation in the lower limbs. Heart rate was lower in the AB group (Table II), in accordance with previous studies (2, 9). This might suggest that persons with SCI, compared with AB, have a less efficient redistribution of blood from the inactive lower body territories to the heart (1–3).

LDF has been used in previous studies involving individuals with SCI (18, 19), but not to assess exercise-induced vasoconstriction in persons with paraplegia, as was the purpose of this study. LDF has become increasingly popular in recent years and has been applied when studying AB participants performing lower body exercise (11, 12). An exercise-induced decrease in skin blood flow has been ascribed to enhanced active vasoconstrictor tone, acting via sympathetic efferent pathways and using norepinephrine as a transmitter (11). Taylor et al. (12) found that this phenomenon was best observed during dynamic exercise with a "significant rate of external work", which led to the present study using a heavy exercise protocol (80% of maximal power output), in addition to the light exercise protocol. However, the 80% workload revealed a less marked cutaneous vasoconstriction in the legs of the AB participants (Fig. 1, lower graph). The discrepancy with Taylor et al. (12) might be explained by the fact that they measured forearm skin blood flow at the level of the heart with participants seated while exercising, whereas the measuring site used in the present study was below the level of the heart. The increased cardiac output during the heavy exercise test in combination with the hydrostatic pressure

Table II. *Metabolic data recorded for the three groups [able-bodied persons (AB), and individuals with low-level (LP) and high-level (HP) paraplegia] during the last 30 seconds of the 3-minute light (~15% of maximal exercise capacity) and heavy (~80% of maximal exercise capacity) exercise tests*

Protocol	Group	VO_2 (l/minute)	HR (bpm)	SV (ml/beat)	Q (l/minute)
Light	AB	0.655 ± 0.058	84.1 ± 9.3	106 ± 17	8.838 ± 1.396
	LP	0.806 ± 0.132	$114.2 \pm 17.3*$	94 ± 29	10.570 ± 2.982
	HP	0.831 ± 0.160	97.1 ± 20.1	106 ± 20	10.139 ± 1.962
Heavy	AB	1.570 ± 0.217	126.6 ± 15.2	104 ± 15	12.942 ± 0.610
	LP	1.531 ± 0.430	$157.9 \pm 12.2*$	79 ± 17	12.476 ± 2.868
	HP	1.620 ± 0.240	$146.9 \pm 18.7*$	100 ± 39	14.172 ± 1.962

Data are means $\pm$ S.D. VO_2 = oxygen consumption; HR = heart rate; SV = stroke volume; Q = cardiac output. *Significantly different from AB ($p < 0.05$).

Light arm cranking exercise test

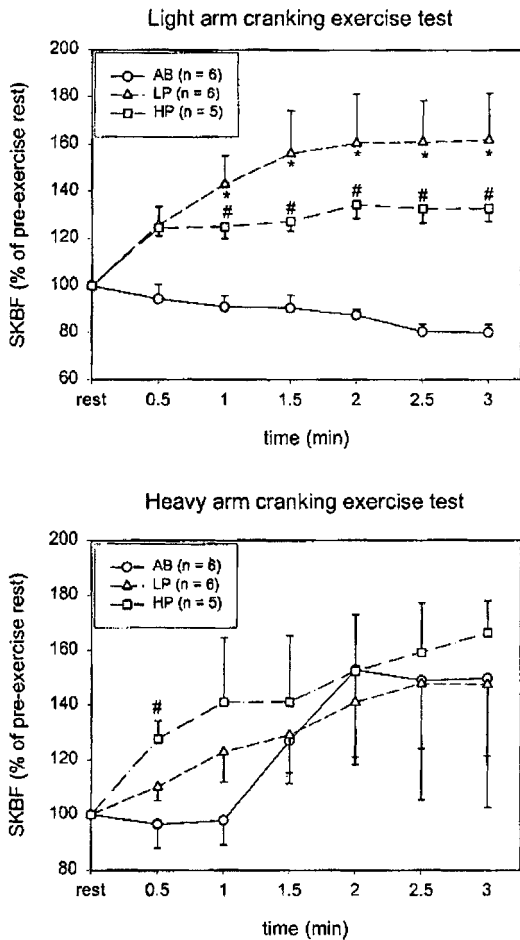Heavy arm cranking exercise test

Fig. 1. Skin blood flow (SKBF) results obtained from able-bodied persons (AB), and individuals with low-level (LP) and high-level paraplegia (HP) during the light (~15% of maximal exercise capacity) and heavy (~80% of maximal exercise capacity) exercise bout. *LP significantly different from AB; #HP significantly different from AB ($p < 0.05$). Data are presented as means ± S.E.M.

may have masked the skin vasoconstriction in this position. However, such an adaptation was detected during the light exercise protocol (~15% of maximal power output) (Fig. 1, upper graph), where the possible effect of an increased cardiac output was comparatively low. Thus, vasoconstriction was noted in the AB group, but not in the two SCI groups.

Few studies have analysed lower limb blood flow in persons with SCI during upper body exercise. Bidart & Maury (20) studied arteriolar motricity with strain gauge plethysmography applied to the foot and found that during exercise, blood flow decreased in AB participants whereas it increased in SCI participants. This confirms the present results of leg skin blood flow obtained during the light exercise bout, as well as for the HP participants at the beginning of the heavy arm-cranking exercise test. Close comparison with the previous study is difficult, however, since the methods were not explicitly described.

Similarly to the present study, Kinzer & Convertino (9) observed a decrease in fluid accumulation (impedance plethysmography) in the legs of AB participants during a 35 W arm-cranking exercise, whereas individuals with T6–T11 paraplegia ($n = 5$) showed a significant increase. The investigators concluded that these discrepancies were due to the absence of normal muscle pump action in the lower limbs, which could act as a reservoir for "significant blood pooling" and thus limit the venous return and cardiac performance of persons with SCI. However, long-term muscle inactivity results in significant fibre type changes, a proportional increase in intramuscular connective tissue and a decrease in intramuscular capillary supply (21, 22). Lower limb paralysis has also been associated with a hypotrophy of the common femoral artery (23) and reduced venous volume variations and venous capacity (24). Thus, the vascular bed in the paralysed legs of participants with long-term SCI may be largely reduced (3), and the absence of the muscle venous pump is probably secondary as an explanation to impaired blood flow adaptations in individuals with paraplegia.

Compared with the findings of Hopman et al. (10), based on strain gauge plethysmography, the present results suggest that the sympathetic nervous system is probably a key factor in deficient blood flow adaptations of SCI participants. Hopman et al. found small or no leg volume decreases in individuals with paraplegia during exercise, in contrast to the marked volume decreases found in AB participants. However, strain gauge plethysmography evaluates total limb blood flow, including muscle and skin, whereas LDF used here is more specific, measuring blood flow confined exclusively to the skin.

In the present study, vasomotor responses were not related to the spinal lesion level. The skin blood flow results of the two SCI groups were not significantly different from each other, although the subdivision of the participants was based on their neurological injury level. It was expected that the LP group would benefit from a partially intact sympathetic innervation in the lower limb and would show more appropriate skin blood flow adaptations, whereas the HP would have no supramedullar control of this area. To analyse further the possible influence of the lesion level on skin blood flow variations, the relationship between the maximal skin blood flow increase during exercise and the lesion level was assessed. The correlations found were low and insignificant, suggesting that the lesion level had no influence on the present results.

In conclusion, this investigation presents new arguments for impaired vascular adaptations in the paralysed lower limbs of individuals with paraplegia during an arm-cranking exercise. The deficient blood flow adaptations may provide an explanation for less efficient cardiac work performance in individuals with paraplegia.

ACKNOWLEDGMENTS

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