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Cardiac output in paraplegic subjects at high exercise intensities

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Summary. The purpose of this investigation was to compare cardiac output ($\dot{Q}_c$) in paraplegic subjects (P) with wheelchair-confined control subjects (C) at high intensities of arm exercise. At low and moderate exercise intensity $\dot{Q}_c$ was the same at a given oxygen uptake ($\dot{V}O_2$) in P and C. A group of 11 athletic male P with complete spinal-cord lesions between T6 and T12 and a group of 5 well-matched athletic male C performed maximal arm-cranking exercise and submaximal exercise at 50%, 70% and 80% of each individual's maximal power output (W_{max}). Maximal $\dot{V}O_2$ ($\dot{V}O_{2max}$) was significantly lower, $\dot{V}O_{2max}$ per kilogram body mass was equal and maximal heart rate (f_c) was significantly higher in P compared to C. At $\dot{V}O_2$ of 1.3, 1.5 and 1.7 $l \cdot min^{-1}$, and for P 65%–90% of the $\dot{V}O_{2max}$, $\dot{Q}_c$ was not significantly different between the groups, although, $\dot{Q}_c$ in P was achieved with a significantly lower stroke volume (SV) and a significantly higher f_c. Although the SV was lower in P, it followed the same pattern as SV in C during incremental exercise, i.e. an increase in SV until about 45% W_{max} and thereafter a stable SV. The similar $\dot{Q}_c$ at a given $\dot{V}O_2$ in both groups indicated that, even at high exercise intensities, circulation in P can be considered isokinetic with a complete compensation by f_c for a lower SV.

Key words: Spinal cord injury – CO_2 rebreathing method – Hypokinetic/isokinetic circulation – Stroke volume – Arm-cranking exercise

Introduction

During exercise in able-bodied subjects (ABS), a redistribution of blood takes place to elevate mean ventricular filling pressure, augment cardiac output ($\dot{Q}_c$) and supply the exercising muscles with blood (Sawka 1986; Rowell and O'Leary 1990). In paraplegic subjects (P), however, the redistribution of blood during arm exercise has been reported to be disturbed, probably due to the lack of sympathetic innervation below the level of the spinal-cord lesion and the inability to activate the muscle pump in the legs (Hjeltnes 1977; Davis and Shepard 1988; Kinzer and Convertino 1989; Hopman et al. 1992a). It has been suggested that this may result in a smaller increase in end-diastolic ventricular volume and, according to the Frank-Starling mechanism, in a lower stroke volume (SV) compared to ABS (Hopman et al. 1992a).

In compensation for the lower SV, heart rate (f_c) has been shown to increase rapidly in P. This compensation by a change in f_c, however, has been reported to be either incomplete, complete or even overcompensatory, which may result in a lower, an equal or a higher $\dot{Q}_c$ in P compared to ABS at a given oxygen uptake ($\dot{V}O_2$) (the so-called hypo-, iso- and hyperkinetic circulation, respectively), as referred to by Hopman et al. (1992a). This variation in $\dot{Q}_c$ found by different investigators could be the result of differences in the level and completeness of spinal cord lesions, in the physical fitness of P and in the methods used to determine $\dot{Q}_c$. Moreover, the different intensities of exercise used in these studies may have played an important role in the variation in $\dot{Q}_c$. Since it is conceivable that at low and moderate exercise intensities f_c can completely compensate for the lower SV, it has been found that an equal $\dot{Q}_c$ can be measured in P compared to ABS (De Bruin and Binkhorst 1984; Kinzer and Convertino 1989; Hopman et al. 1992a). At higher exercise intensities, however, f_c may fail to compensate completely for SV and, therefore, cause a hypokinetic circulation as has been reported by Hjeltnes (1977) and Davis and Shephard (1988). These investigators have compared $\dot{Q}_c$ in P at high exercise intensities with $\dot{Q}_c$ in ABS from the literature. Due to the absence of a control group, it is not known whether the lower $\dot{Q}_c$ resulted from the use of different methods in determining $\dot{Q}_c$ or from P subjects actually possessing a lower $\dot{Q}_c$ at high exercise intensities in comparison with ABS.

The purpose of the present study was, therefore, to compare $\dot{Q}_c$ at high exercise intensities in an exactly

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defined group of P with complete spinal cord lesions between T6 and T12 with that in a well-matched group of wheelchair-confined controls (C). It may be hypothesised that at high exercise intensities, at a given $\dot{V}O_2$, $\dot{Q}_c$ is lower in P than in ABS, as result of an incomplete compensation by f_c for a lower SV.

Methods

Subjects. A group of 11 male P and a group of 5 male C participated in this study after giving their written informed consent. The study was approved by the Faculty Ethics Committee. The P had complete spinal cord lesions between T6 and T12 (2 subjects T7; 3 subjects T8, T9 and T10, respectively; 6 subjects T12). It was probable, therefore, that cardiac sympathetic innervation would not have been affected, suggesting a basically normal cardiac regulation. The C group consisted of wheelchair-dependent subjects, due to permanent knee or hip injuries. These subjects used their wheelchair, similarly to P subjects, in their daily lives and for sport. Both P and C subjects were well-trained wheelchair athletes with almost identical training levels and were matched for age (Table 1). The disabilities in both groups had existed for at least 2 years. In other words, P and C subjects were well-matched with the exception of the spinal cord lesion. The subjects underwent a medical examination, including a 12-lead electrocardiogram (ECG), cardiac and pulymonary auscultation and for P subjects a neurological examination to establish the level and completeness of the spinal cord lesion. None of the subjects had any cardiopulmonary disease or used medicines likely to affect the results.

Protocol. This investigation included two arm-cranking tests, first a maximal exercise test and within a week a submaximal exercise test. During both occasions, temperature and relative humidity in the experimental room were maintained constant, 20–22° C and 45%–50%, respectively. The subjects abstained from nicotine, caffeine and alcohol for at least 2 h prior to the test. Arm-cranking exercise was performed using an electro-magnetic arm-crank ergometer (modified cycle ergometer, Lode, Groningen, The Netherlands), sitting in a wheelchair. The crankshaft was adjusted to shoulder height and during cranking the elbow was at no time fully extended. The lower-limb position was standardised on both occasions, so that the knees were bent in 90° flexion with the feet on the ground.

On the first occasion, the subjects were weighed in a sitting position on a hospital scale and skinfold-thickness was measured as the sum of four skinfolds (biceps, triceps, subscapular, suprailiac sites; Holtain Ltd., Crymmych, Pembs., U.K.). Body height was taken from the medical files. Each subject performed maximal exercise, using a continuous multistage-increasing protocol (10 $W \cdot min^{-1}$) with a crank frequency of 60 rpm (Washburn and Seals 1983). To determine the maximal power output (W_{max}) during the last minute and the maximal oxygen uptake ($\dot{V}O_{2max}$), taken as the mean $\dot{V}O_2$ during the last minute, the test was terminated when, even after verbal encouragement by the examiner, the subject was unable to maintain the frequency of 60 rpm. A f_c greater than 170 beats $\cdot min^{-1}$, base excess (BE) less than -10 mmol $\cdot l^{-1}$ and respiratory exchange ratio greater than 1.00 were used as objective criteria for maximal exercise (Sawka 1986). Each subject had to meet at least two out of these three criteria.

During the second visits, the subjects performed arm-cranking exercise at 50%, 70% and 80% of their individual W_{max}. Each exercise session lasted 6 min, 2 min to increase the power output in four steps to the specified exercise intensity and 4 min to achieve steady-state conditions. Steady-state exercise during the last 2 min of each exercise intensity was verified by $\dot{V}O_2$ carbon dioxide output ($\dot{V}CO_2$) and f_c. Between sessions, the subjects had a 7-min recovery period.

Measurements. During both tests, $\dot{V}O_2$, $\dot{V}CO_2$ and pulmonary ventilation were measured continuously and averaged over 30-s intervals, by an automatic gas analyser (Oxycon IV, Mijnhardt, Bunnik, The Netherlands). The gas analyser uses a paramagnetic O_2 analyser and an infrared CO_2 analyser to measure the $\%O_2$ and $\%CO_2$ in the expired air. The gas analyser was calibrated daily with a gas mixture analysed by the Scholander technique.

The f_c and ECG were monitored continuously and recorded every 30 s during both tests using a cardiotachometer.

Blood gases were measured 3 min after termination of maximal exercise and during the last minute of each submaximal exercise level. An arterialized ear-lobe blood sample was obtained after rubbing the ear-lobe with a vasodilatating ointment (IL Blood Gas Analyser, model 1312, Instrumentation Laboratory, Lexington, Mass.).

At the end of the last minute of each submaximal exercise intensity $\dot{Q}_c$ was determined by the CO_2 rebreathing method according to Collier (1956) and as described by Van Herwaarden et al. (1980). The values of arterial carbon dioxide content (C_aCO_2) and the mixed venous carbon dioxide content ($C_{\bar{v}}CO_2$) were obtained by translating the partial pressure of carbon dioxide in arterial blood (P_aCO_2) and the partial pressure of carbon dioxide in mixed blood venous ($P_{\bar{v}}CO_2$) into C_aCO_2 and $C_{\bar{v}}CO_2$ respectively, using the CO_2 dissociation curve, corrected for pH and BE. The P_aCO_2 was calculated using the modified Bohr formula for physiological dead space (Van Herwaarden et al. 1980). The $P_{\bar{v}}CO_2$ was obtained from the CO_2 plateau during rebreathing with an additional correction for alveolar-arterial PCO_2 differences (Jones et al. 1969). The CO_2 plateau was measured by a rapid and linear CO_2 analyser (Capnograph Godart type MO, de Bilt, The Netherlands), whereas the quality of the CO_2 plateau was verified using a computer algorithm in which variation of the signal as well as the length of the plateau were compared with predetermined values (Van Herwaarden et al. 1980). The CO_2 plateau had to be present for 10 to 15 s before PCO_2 increased as a result of recirculation. The SV was calculated by dividing f_c into $\dot{Q}_c$.

Statistical analysis. A Student's *t*-test was applied to determine differences between P and C in physical characteristics, in responses to maximal exercise and in responses to submaximal exercise at 50%, 70% and 80% of W_{max} and at given $\dot{V}O_2$ (1.3, 1.5 and 1.1 $l \cdot min^{-1}$). The latter was obtained by restricted inter- and extrapolation of $\dot{Q}_c$ and $\dot{V}O_2$ for each subject. Statistical significance was accepted with a two-tailed probability at $P<0.05$.

Table 1. Physical characteristics of paraplegic and control subjects

	Paraplegic subjects ($n=11$)		Control subjects ($n=5$)	
	mean	SD	mean	SD
Age (year)	33.7	8.0	35.4	4.3
Height (cm)	181	4	183	9
Body mass (kg)	66.1	10.7*	79.0	7.8
Skinfold-thickness (sum of $n=4$) (mm)	29.9	8.6	28.8	7.2
Sports activities ($h \cdot week^{-1}$)	6	2	8	2

* $P<0.05$

Results

The physical characteristics of the subjects are summarised in Table 1. Age, skinfold-thickness, height and sports activities were not significantly different between the groups, whereas body mass was 17% lower in P ($P<0.05$) compared to C (Table 1).

In Table 2 the results of the maximal exercise test are shown. Each subject met at least two of the three objective criteria for maximal exercise, indicating that their efforts can be considered maximal. The W_{max} and $\dot{V}O_{2max}$ were significantly lower in P, by 77% and 73% respectively, whereas $\dot{V}O_{2max}$ per kilogram body mass was equal for both groups. The f_c was 6% higher in P than in C ($P<0.05$) at the end of the maximal test (Table 2).

Table 3 gives the physiological responses to submaximal exercise at 50%, 70% and 80% of the W_{max}. The $\dot{V}O_2$, $\dot{V}CO_2$ and f_c showed slight increases during the 5th and 6th min of each exercise session (less than 3%, 4% and 2%, respectively), but still within the range for steady-state conditions. The $\dot{Q}_c$ and $\dot{V}O_2$ at 50%, 70% and 80% of the W_{max} were significantly lower in P compared to C. Whereas at given $\dot{V}O_2$ of 1.3, 1.5, 1.7 $l \cdot min^{-1}$ (for P between 65% and 90% of their $\dot{V}O_{2max}$) there were no siginificant differences in $\dot{Q}_c$ (Fig. 1), SV was 30% lower and f_c 35% higher in P compared to C ($P<0.05$).

No significant differences were found in blood gases between the groups during maximal or submaximal exercise.

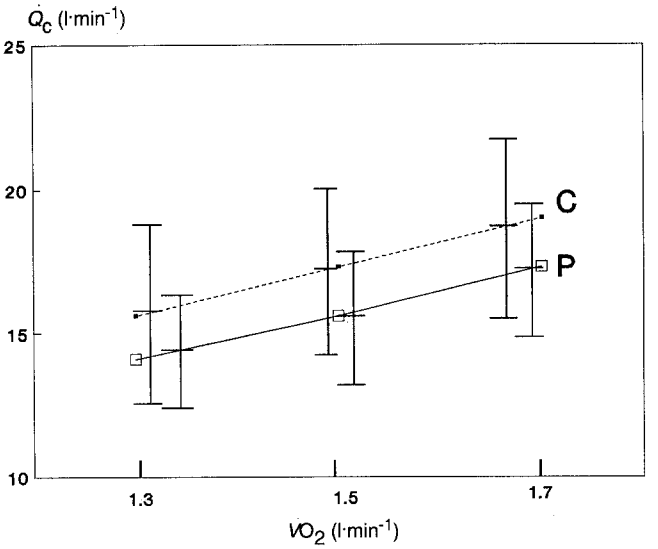

Fig. 1. Cardiac output ($\dot{Q}_c$) at a given oxygen uptake ($\dot{V}O_2$) in paraplegic subjects (P ——) and control subjects (C ----)

Table 2. Physiological responses to maximal arm-cranking exercise in paraplegic and control subjects

	Paraplegic subjects ($n=11$)		Control subjects ($n=5$)	
	mean	SD	mean	SD
Maximal power output (W)	123	20*	160	24
Maximal oxygen uptake ($l \cdot min^{-1}$)	1.94	0.41*	2.67	0.31
Maximal oxygen uptake/ body mass ($ml \cdot min^{-1} \cdot kg^{-1}$)	30.0	7.4	34.3	7.3
Maximal heart rate (beat·min^{-1})	185	7*	175	6
Base excess ($mmol \cdot l^{-1}$)	−11.1	1.6	−11.5	1.6
Respiratory exchange ratio	1.25	0.12	1.23	0.05

* $P<0.05$

Discussion

This investigation compared cardiovascular responses to high intensities of submaximal arm exercise between a specifically defined group of P and a well-matched wheelchair-confined gorup of C with almost identical physical characteristics. Since physiological responses

Table 3. Physiological responses to submaximal arm-cranking exercise at 50%, 70% and 80% of maximal power output (W_{max}) in paraplegic and control subjects

	% W_{max}	Paraplegic subjects ($n=11$)		Control subjects ($n=5$)	
		mean	SD	mean	SD
Oxygen uptake ($l \cdot min^{-1}$)	50%	1.14	0.15	1.53	0.17
	70%	1.48	0.23	2.05	0.27*
	80%	1.71	0.25	2.30	0.26*
Cardiac output ($l \cdot min^{-1}$)	50%	12.8	2.5	18.8	4.2*
	70%	15.7	3.2	22.8	6.1*
	80%	16.8	2.7	22.6	3.3*
Stroke volume (ml)	50%	99	20	164	36*
	70%	99	22	165	51*
	80%	99	17	146	28*
Heart rate (beat·min^{-1})	50%	130	10	115	6*
	70%	159	10	140	7*
	80%	173	7	156	9*

* $P<0.05$

to exercise in C are the same as those in ABS (Hopman et al. 1992a) and, in addition, C had sports and daily living activities comparable to P, these C subjects acted as an excellently matched control group to P subjects, differing only in the spinal cord injury.

Maximal exercise test

Since height and skinfold-thickness were equal in the P and C groups, it seems reasonable to conclude that the reported difference in body mass was mainly a result of the muscle atrophy of the lower limbs in P subjects. This loss of active muscle mass may have also explained the lower W_{max} and $\dot{V}O_{2max}$ during maximal exercise in P compared to C, because the C group would have used their leg and trunk muscles for stabilisation and as a fulcrum to increase their maximal performance. This explanation was supported by an equal $\dot{V}O_{2max}$ for P and C when corrected for body mass differences, i.e. $\dot{V}O_{2max} \cdot kg^{-1}$, indicating a comparable status of muscle training for both groups.

The values of $\dot{V}O_{2max}$ of the P subjects found in this study were in agreement with results of previous investigators who tested comparable P subjects during arm-cranking exercise (Zwiren and Bar-Or 1975; Gass and Camp 1984; Hopman et al. 1992a). The significantly higher $f_{c,max}$, in P subjects could have been the effect of the venous blood pooling below the lesion in P subjects, resulting in a reduced SV and a compensatory tachycardia, the latter resulting from an enhanced sympathetic activity. At maximal exercise intensities the higher sympathetic activity in P may have explained the higher $f_{c,max}$ (Kinzer and Convertino 1989; Hopman et al. 1992a).

Submaximal exercise test

Due to the lower W_{max} and $\dot{V}O_{2max}$ in P, values of $\dot{V}O_2$ and $\dot{Q}_c$ during submaximal exercise at 50%, 70% and 80% W_{max} were lower in P compared to C.

At a given $\dot{V}O_2$ during high exercise intensities, however, $\dot{Q}_c$ in the P group was not significantly different from the C group (Fig. 1), which implies that the circulation of P subjects, even at high exercise intensities could be considered isokinetic in comparison with the circulation of C subjects. This was in contrast to the results of Hjeltnes (1977), Davis and Shephard (1988) and Jehl et al. (1991; Fig. 2). At exercise levels comparable to the present study, Hjeltnes (1977) and Davis and Shephard (1988) have reported a hypokinetic circulation. Both studies compared $\dot{Q}_c$ of the P subjects with data of ABS in the literature. It is, therefore, not quite clear whether $\dot{Q}_c$ was lower in P or that the method used to determine $\dot{Q}_c$ yielded an underestimation. An indication that the latter was indeed the case was the low SV values (calculated by dividing f_c into $\dot{Q}_c$) of the P subjects, given their physical fitness status (Davis and Shephard 1988). Jehl et al. (1991) have reported a hyperkinetic circulation in P compared to ABS at high

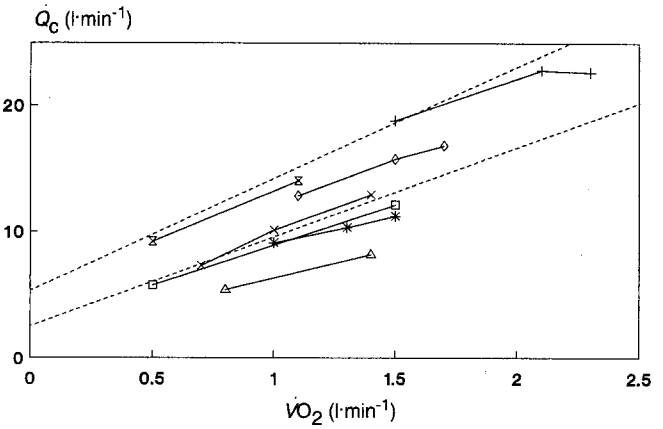

Fig. 2. Relationship between cardiac ouput ($\dot{Q}_c$) and oxygen uptake ($\dot{V}O_2$) during arm exercise as reported by several investigators. The area between the two *dashed lines* represents the range of $\dot{Q}_c$ in able bodies subjects during arm exercise (Åstrand et al. 1964; Sawka 1986). C, control subjects; P, paraplegic subjects. +, control subjects of this study; ◇, paraplegic subjects of this study; ×, paraplegic subjects of the study of Hopman et al. 1992a; *, inactive paraplegics of the study of Davis and Shephard (1988); △, paraplegic subjects of the study of Hjeltnes (1977); ⧖, paraplegic subjects of the study of Jehl et al. (1991); □, paraplegic subjects De Bruin and Binkhorst (1984)

exercise intensites. However, since these investigators tested P subjects with higher levels of spinal cord lesions (between T1–T6) than the present study, the probability exists that the subjects in the Jehl et al. (1991) study suffered from disturbed cardiac sympathetic innervation. Furthermore, the isokinetic circulation in P at high exercise intensities as found in the present study is in agreement with the results reported at low and moderate exercise intensities (Sawka et al. 1980; De Bruin and Binkhorst 1984; Kinzer and Convertino 1989; Hopman et al. 1992a; Fig. 2).

Although $\dot{Q}_c$ was the same in P and C at a given $\dot{V}O_2$, $\dot{Q}_c$ was achieved by a marked difference in the SV-f_c relationship. It is very interesting that although SV was lower in P, probably due to pooling of venous blood below the lesion, it followed the same pattern as SV in ABS. It is well known that SV in ABS increases up to about 45% of the W_{max}, after which a stable SV is achieved with even a decrease in SV near maximal exercise (Åstrand et al. 1964). For P, an increase in SV has been reported up to exercise intensities of 40%–50% W_{max} (Hopman et al. 1992a, Hopman et al. 1992b), while the present study demonstrated that SV did not increase further between 50% and 80% W_{max}. In spite of the low SV and lack of any further increase above 50% W_{max}, P subjects were still able to compensate by an increase in f_c, which at $\dot{V}O_2$ up to 90% of the $\dot{V}O_{2max}$, resulted in $\dot{Q}_c$ equal to that of C subjects. In accordance data in the literature of SV near maximal intensity (Åstrand et al. 1964), C demonstrated a decrease in SV at 80% of W_{max}.

The present study provided important additional information concerning circulatory behaviour in P subjects at high exercise intensities.

Conclusion

This study has demonstrated that even at high intensities of exercise P subjects with low thoracic lesions are able to compensate completely for a lower SV by an increase in f_c and, therefore, can still achieve the same $\dot{Q}_c$ at a given $\dot{V}O_2$ as a well-matched C group. In addition, this study showed that SV in P followed the same pattern as in C and in ABS (literature). Probably as a consequence of the low SV, tachycardia was observed at maximal exercise intensity.

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