The effect of training on cardiovascular responses to arm exercise in individuals with tetraplegia

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Abstract The aim of this study was to investigate the physiological responses to maximal and submaximal arm-cranking exercise in 21 individuals with tetraplegia (TP) and to evaluate the effect of a 3 and 6-month training period (mean frequency of 1.5 h·week⁻¹, mean intensity at 35% of the training time above 60% of the heart rate reserve) on these physiological responses. The TP were divided into 8 trained subjects (T), 7 untrained subjects (U) who started their training at the beginning of the study, and 6 sedentary subjects (S). All the subjects were tested at the beginning of training and after 6 months, whereas T and U were also tested in between, at 3 months. During maximal exercise, peak power output and peak oxygen uptake per kilogram bodymass were significantly higher in T (49.9 W and 14.2 ml·min⁻¹·kg⁻¹ respectively) compared to U (20.7 W and 8.8 ml·min⁻¹·kg⁻¹ respectively) and S (15.9 W and 7.4 ml·min⁻¹·kg⁻¹ respectively), whereas all other peak responses showed tendencies to be higher in T. This is most likely to have been the result of participation in sport and the effect of it on performance capacity in T, although differences in completeness of the lesion may have influenced the results. No significant differences were found for submaximal and maximal responses after 3 or 6 months of training in either T and U or in S. This may have been due on the one hand to the vulnerability of the subjects to diseases and injuries and on the other hand to the low frequency of training. On an individual basis, however, remarkable improvement was observed during the training period, especially for individuals in the U group. These results would suggest that a 3 or 6-month training period has no measurable positive effect on the fitness level of TP.

Key words Arm-cranking exercise · Cardiac output · Oxygen uptake · Quad rugby · Maximal performance

Introduction

A spinal cord injury (SCI) results in paralysis, loss of sensation and dysfunctioning of the autonomic nervous system below the lesion. In individuals with tetraplegia (TP) the paralysis affects the four extremities and the trunk. Consequently, such individuals are confined to a wheelchair and have only a small muscle mass left for activities. This results in a predominantly sedentary life style, which may lead to a poor capacity for physiological performance. This may in turn lead to a further reduction in activities, causing a decline in physical performance capacity and it has been shown that a debilitating cycle is likely to develop (Hoffman 1986). It has been shown that a sedentary lifestyle may also result in a higher risk of health disorders in SCI people and an increased occurrence of cardiovascular diseases (Le and Price 1982; Haas et al. 1986; Dearwater et al. 1986; Dearwater et al. 1986; Hooker and Wells 1989).

In daily life the activity level is not strenuous enough to maintain or improve cardiovascular fitness in individuals with SCI, therefore, it has been suggested that participation in regular exercise is needed (Hjeltnes and Vokac 1979; Wicks et al. 1983; Figoni 1990; Janssen et al. 1994a). However, in contrast to information which has been published about the amount of exercise and training required to maintain or improve cardiovascular fitness in able-bodied subjects (Haskell 1994), hardly anything is known about the intensity and frequency of exercise needed to maintain or improve
physical capacity in individuals with SCI. In addition, autonomic dysregulation makes it impossible to compare the fitness level and related training recommendations of able-bodied subjects to individuals with SCI. It has been found that the lack of sympathetic innervation below the lesion may disturb vasoregulation, which affects the redistribution of blood (Davis and Shephard 1988; Hopman et al. 1992). Furthermore, individuals with TP have a lack of cardiac sympathetic innervation and the heart will only be innervated by the vagus. Consequently, the heart rate (HR) increase during exercise may depend mainly on vagal withdrawal, which has been suggested as the explanation for the low maximal HR observed in these individuals (Erikson et al. 1988; Coutts et al. 1983).

Previous studies focused on the trainability of individuals with TP have reported conflicting results (DiCarlo et al. 1983; Hooker and Wells 1989). Differences between these studies may be explained by differences in the number of participating subjects (4 versus 11), in training protocols used and by the fact that in one of these studies (Hooker and Wells 1989) individuals with paraplegia were involved. Another study (Erikson et al. 1988) has reported differences in the fitness level between trained and untrained individuals with TP, which suggests the possibility of being able to train these individuals if the training is applied properly. In addition, TP individuals who participate regularly in quad rugby have reported experiencing an improved fitness (personal communication). This, however, has never been evaluated or assessed by exercise tests in the laboratory.

The purpose of this study can, therefore, be summarized as:

1. To compare physiological responses to submaximal and maximal arm exercise in trained and untrained individuals with TP and
2. To examine the effect of a 3 and 6-month training period on these responses in comparison to sedentary individuals with TP.

### Methods

#### Subjects

A group of 21 individuals with a cervical SCI (C4 to C8) participated in this study. The subjects were divided into three groups according to their fitness levels, which was based on their participation in sport at the start of the study for [peak oxygen uptake, $VO_{2peak}$, see Table 1]. The trained group (T) had participated in sport twice a week for at least 2 years. The T were all men and 4 had an incomplete and 4 a complete lesion. The untrained group (U) had not participated in any sport for the previous 2 years and started to train regularly at the beginning of the study. The U, 6 man and 1 woman, all had complete lesions. The sedentary group (S), 4 men and 2 women, did not participate in any sport throughout the study. The S had complete lesions, except 1 woman, who had an incomplete lesion. They were interviewed to obtain information about health status, the time since injury and the use of medications. Written informed consent was obtained from each subject. The study was approved by the Faculty Ethics Committee.

#### Protocol

Group T and U were tested three times with 3 months in between, whereas group S was tested only at the beginning and at the end of the study (0 and 6 months). The room in which the tests took place had a constant temperature between 20 and 21 °C with a relative humidity of 38%-43%. Skinfold thickness was determined from four sites: biceps, triceps, subscapular and suprailliac with a calliper (Holstain Ltd, Crymmych, Pemb., UK). The subjects were weighed in a sitting position on a hospital scale. Body height was taken from their medical file. All the subjects abstained from nicotine, caffeine and alcohol for at least 2 h prior to each test.

The submaximal exercise test

Each subject performed exercise on an electrically braked arm-crank ergometer (ACE). The submaximal test consisted of three periods of exercise at 20%, 40% and 60% of an estimated maximal power output ($P_{est}$). The $P_{est}$ for each subject was based on the level of injury, completeness of lesion, training level, sex, and strength of the upper limbs as measured by an isometric strength test prior to the exercise test (Table 1), based on experiences in previous exercise tests with a similar group of individuals. The submaximal test started with 5-min arm cranking at 20% $P_{est}$ followed by a 3-min rest period. The second period consisted of 6-min arm cranking at 40% $P_{est}$.

### Table 1

<table>
<thead>
<tr>
<th>Group U (n = 7)</th>
<th>Group T (n = 8)</th>
<th>Group S (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>26.6 ± 6.9</td>
<td>27.6 ± 6.9</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>77.6 ± 23.4</td>
<td>77.6 ± 23.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>183 ± 9.4</td>
<td>184 ± 9.4</td>
</tr>
<tr>
<td>Skinfold thickness (mm)</td>
<td>51.4 ± 25.3</td>
<td>47.9 ± 24.4</td>
</tr>
<tr>
<td>$VO_{2peak}$ (l·min⁻¹)</td>
<td>0.63 ± 0.24</td>
<td>1.03 ± 0.42</td>
</tr>
<tr>
<td>$P_{peak}$ (W)</td>
<td>20.7 ± 14.8</td>
<td>49.9 ± 28.6</td>
</tr>
<tr>
<td>$P_{est}$ (W)</td>
<td>22.1 ± 13.5</td>
<td>30.0 ± 12.1</td>
</tr>
<tr>
<td>Years post injury (years)</td>
<td>6.6 ± 5.2</td>
<td>8.1 ± 10.3</td>
</tr>
</tbody>
</table>
Each test with a gas mixture analysed using the Scholander technique was performed for 7 min at 60% $P_{\text{est}}$. The cycle frequency was kept between 60 and 70. Cardiac output ($Q_c$) was determined at the end of each submaximal exercise bout by a CO$_2$ rebreathing method (Hopman et al. 1994), while the subjects were still cranking.

The maximal exercise test

Following the submaximal test subjects rested for at least 10 min and then performed a peak exercise test. This test used a continuously increasing protocol which started at a level of 20% $P_{\text{est}}$ and increased every 30 s by 20% until 80% $P_{\text{est}}$ was reached. Thereafter power output increased every minute by approximately 10% of $P_{\text{est}}$ until exhaustion or until the cycle frequency dropped below 60 rpm. Peak power output ($P_{\text{peak}}$) was defined as the highest power output that was maintained for 1 min. The subjects were verbally encouraged during the whole test. Time to exhaustion varied between 6 and 14 min (mean 9.6 min).

Materials

Arm-cranking was performed on an electro-magnetic arm-crank ergometer (modified cycle ergometer, Lode, Groningen, The Netherlands). The subjects sat in their wheelchairs, which were fixed to the ground. The axis of the arm-crank was at shoulder level. The distance between the subjects and the ACE prevented the subjects from fully extending their arms. If the subject had no intrinsic hand function, specially designed mitts were used to fix the hands to the handle-bars.

Measurements

Oxygen uptake ($V'O_2$), carbon dioxide output ($V'CO_2$), respiratory exchange ratio ($R$), expired air ventilation ($V_d$) and breathing frequency ($f_d$) were measured continuously and averaged over 30-s periods using an automatic gas analyser (Oxycon IV, Mijnhardt, Bunnik, The Netherlands). The gas analyser used 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 prior to each test with a gas mixture analysed using the Scholander technique. During submaximal exercise, variables were calculated as the average over the last 2 min. During the maximal test the mean of the last minute was taken as the peak value for all variables. During the whole test electrocardiograms (ECG) and HR were recorded using a cardiotachometer, and HR was averaged over 30-s intervals.

Capillary blood samples were taken from the ear-lobe, after rubbing it with a vasodilating ointment, before the test started, at the end of each submaximal bout and 2 min after the peak test. Base excess in millimoles per liter was calculated from the measured hydrogen ion activity (pH), partial pressure of carbon dioxide (PCO$_2$) and pressure of oxygen partial (PO$_2$) of the blood sample (IL Blood Gas Analyser, model 1312).

At each submaximal level $Q_c$ was determined by the Collier CO$_2$-rebreathing method (Hopman et al. 1994). A Hans-Rudolph valve was connected to a three-way stop-cock for switching from outdoor air to the rebreathing bag. To obtain mixed venous carbon dioxide tension ($P_{\text{CO}}$$_2$) a rapid and linear (within the measuring range) CO$_2$ analyser (Capnograph, Godart type MO, de Bilt, The Netherlands) was used to measure the CO$_2$ plateau during rebreathing. A correction for alveolar-capillary PCO$_2$ differences was applied (Hopman et al. 1994). With the modified Bohr-equation for physiologically dead-space the arterial carbon dioxide tension ($P_{\text{CO}}$$_2$) was calculated. The CO$_2$ tensions were converted into concentrations using the CO$_2$ dissociation-curve (Van Herwaarden et al. 1980). A computer program with an algorithm was used to determine the quality of the plateau (Hopman et al. 1994). Stroke volume (SV) was calculated by dividing $Q_c$ into HR.

Training

The training was the team sport quad rugby, a game specially created for individuals with TP. The training consisted of endurance, sprint and skill training. The U and T trained once a week and played two games once a month. Sport Testers (PE 3000, Polar Electro, Finland) were used to determine the intensity of the training. Every 5 s the Sport Tester stored and calculated the mean HR using the last 15 R-R intervals. The percentage heart rate reserve (%HRr) (Karvonen and Vuorimaa 1988; Janssen et al. 1994b) was calculated, according to the equation: %HRr = ([HR$_{\text{recorded}}$ - HR$_{\text{rest}}$]/HR$_{\text{peak}}$ - HR$_{\text{rest}}$) x 100%, with HR$_{\text{peak}}$ being the maximal HR as measured during the arm-cranking test and HR$_{\text{rest}}$ being the lowest recorded HR at rest. The HRr was categorised in 10% intervals and the percentage time for 1 h of training was calculated for both U and T (Janssen et al. 1994a). (Fig. 5)

Statistical analyses

A one-way ANOVA was used to assess differences in the physical characteristics and in responses to maximal exercise at the beginning of training among T, U and S. A paired Student's t-test was used to determine the absolute and relative differences in maximal and submaximal responses in the first, second and third sessions between T and U and a Student's t-test was used to assess differences in changes between U and T. A two-tailed probability of $P < 0.05$ was considered to be statistically significant.

Results

The physical characteristics of U, T and S at the beginning of training are presented in Table 1. No significant differences for age, body mass, height, skinfold thickness and years-post-injury existed among groups.

Groups at the beginning of training

The maximal exercise test revealed a significantly higher peak oxygen uptake per kilogram body mass ($V'O_2$peak kg$^{-1}$) and $P_{\text{peak}}$ for T compared to U and S (Fig. 1). Although no significant differences were found in the other responses, a trend could be observed towards higher values for all peak responses in T compared to U and S.

The relationship between $Q_c$, SV, HR and $V'O_2$, during submaximal exercise in U, T and S, are graphically presented in Figs. 2, 3 and 4, respectively. In these figures data for individuals with paraplegia and able-bodied subjects from previous studies using the same methods and protocol have been added (Hopman et al. 1992, 1993). Since the submaximal exercise levels for U and S were low and the range was very small, no statistical analyses were performed and the results for the different groups compared in a descriptive way.
Changes in physiological responses
over 3 and 6 months

No significant differences were found in either absolute or relative changes in the physiological responses to arm exercise for submaximal and maximal exercise over 3 or 6 months in U, T and S (Table 2). In addition, no significant differences were found among the groups in any of the changes in responses.
Fig. 2 Cardiac output ($Q$) versus oxygen uptake ($\dot{V}O_2$) as reported in this investigation and a study by Hopman et al. (1992). Both studies used the same exercise protocol and methods. $U$ Untrained subjects (this study), $T$ trained subjects (this study), $S$ sedentary subjects (this study), $AB$ able-bodied subjects as reported by Hopman et al. (1992), $PP$ individuals with paraplegia as reported by Hopman et al. (1992).

Fig. 3 Stroke volume ($SV$) versus oxygen uptake ($\dot{V}O_2$) as reported in this investigation and a study by Hopman et al. (1992). Both studies used the same exercise protocol and methods. $U$ Untrained subjects (this study), $T$ trained subjects (this study), $S$ sedentary subjects (this study), $AB$ able-bodied subjects as reported by Hopman et al. (1992), $PP$ individuals with paraplegia as reported by Hopman et al. (1992).

Fig. 4 Heart rate (HR) versus oxygen uptake ($\dot{V}O_2$) as reported in this investigation and a study by Hopman et al. (1992). Both studies used the same exercise protocol and methods. $U$ Untrained subjects (this study), $T$ trained subjects (this study), $S$ sedentary subjects (this study), $AB$ able-bodied subjects as reported by Hopman et al. (1992), $PP$ individuals with paraplegia as reported by Hopman et al. (1992).

Training

The $T$ trained 2 h and 15 min each week, while $U$ trained 1 h each week. Over the 6 months the number of followed training sessions ranged among subjects from 19 to 25, due to injuries or illnesses. The $U$ and $T$ played two games once a month, both games were played on the same day and each game lasted 90 min. Figure 5 shows the amount of relative time at different physical strain levels (%HRr) during 1-h quad rugby training sessions for $U$ and $T$. The HRr was divided in 10% intervals. The $U$ trained 42.2 min (56.4% of the total training time) and $T$ 21.3 min (28.6% of the total training time) above 60% HRr.

Table 2 Mean changes (A) in physiological responses after 3 (0-3 months) and 6 (0-6 months) months of training in previously untrained individuals ($U$) and in trained individuals ($T$) and after 6 months in sedentary individuals ($S$). $P_{peak}$ Peak power output, $\dot{V}O_2$ oxygen uptake, $Q$, cardiac output, $SV$ stroke volume at 20%, 40% and 60% exercise intensity as percentage of maximal

<table>
<thead>
<tr>
<th></th>
<th>T (0-3 months)</th>
<th>T (0-6 months)</th>
<th>U (0-3 months)</th>
<th>U (0-6 months)</th>
<th>S (0-6 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta P_{peak}$ (W)</td>
<td>-0.2</td>
<td>-0.8</td>
<td>2.9</td>
<td>3.6</td>
<td>0</td>
</tr>
<tr>
<td>$\Delta \dot{V}O_{peak}$ (l·min$^{-1}$)</td>
<td>-0.02</td>
<td>-0.08</td>
<td>0.00</td>
<td>0.00</td>
<td>0.03</td>
</tr>
<tr>
<td>$\Delta \dot{V}O_2$ 60% (l·min$^{-1}$)</td>
<td>-0.04</td>
<td>-0.05</td>
<td>0.00</td>
<td>0.00</td>
<td>-0.03</td>
</tr>
<tr>
<td>$\Delta \dot{V}O_2$ 40% (l·min$^{-1}$)</td>
<td>-0.04</td>
<td>-0.04</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>$\Delta \dot{V}O_2$ 20% (l·min$^{-1}$)</td>
<td>0.01</td>
<td>0.01</td>
<td>0.03</td>
<td>0.03</td>
<td>-0.07</td>
</tr>
<tr>
<td>$\Delta Q$ 60% (l·min$^{-1}$)</td>
<td>-0.4</td>
<td>1.0</td>
<td>0.0</td>
<td>-0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>$\Delta Q$ 40% (l·min$^{-1}$)</td>
<td>-0.5</td>
<td>0.6</td>
<td>-0.3</td>
<td>-0.6</td>
<td>0.7</td>
</tr>
<tr>
<td>$\Delta Q$ 20% (l·min$^{-1}$)</td>
<td>-0.3</td>
<td>-0.9</td>
<td>-0.2</td>
<td>-0.5</td>
<td>-1.8</td>
</tr>
<tr>
<td>$\Delta SV$ 60% (ml)</td>
<td>0.4</td>
<td>-4.6</td>
<td>2.4</td>
<td>-2.7</td>
<td>-1.5</td>
</tr>
<tr>
<td>$\Delta SV$ 40% (ml)</td>
<td>-10.4</td>
<td>-8.9</td>
<td>0.3</td>
<td>-5.5</td>
<td>-9.5</td>
</tr>
<tr>
<td>$\Delta SV$ 20% (ml)</td>
<td>-3.2</td>
<td>-6.0</td>
<td>-1.7</td>
<td>-2.3</td>
<td>-23.5</td>
</tr>
<tr>
<td>$\Delta HR$ 60% (beats·min$^{-1}$)</td>
<td>-6.5</td>
<td>-6.0</td>
<td>-6.7</td>
<td>-1.4</td>
<td>4.3</td>
</tr>
<tr>
<td>$\Delta HR$ 40% (beats·min$^{-1}$)</td>
<td>6.7</td>
<td>1.5</td>
<td>-6.3</td>
<td>-1.0</td>
<td>2.3</td>
</tr>
<tr>
<td>$\Delta HR$ 20% (beats·min$^{-1}$)</td>
<td>0.7</td>
<td>-5.1</td>
<td>-2.6</td>
<td>-2.0</td>
<td>2.0</td>
</tr>
</tbody>
</table>
The aim of the study was to determine whether or not regular exercise (participation in a team sport) could be a tool to break the debilitating cycle that may exist in TP individuals and could lead to improvement in the physical capacity of these individuals.

Maximal exercise responses

The higher \( P_{\text{peak}} \) and \( \dot{V}O_2 \) in T compared to U and S as found in this study is in agreement with results for trained and untrained TP that have been found by DiCarlo et al. (1983), Coutts et al. (1983, 1985) and Erikson et al. (1988). This confirms the higher fitness level of T compared to U and S, which was likely to have been the effect of regular sport participation. However, there may be other reasons for the higher fitness level in T:

1. The T included 4 individuals with an incomplete lesion, while in U all the subjects had complete lesions and in S only 1 subject had an incomplete lesion. According to Erikson et al. (1988) untrained individuals with incomplete lesions have been found to have a higher \( \dot{V}O_2 \) and \( HR_{\text{peak}} \) than counterparts with complete lesions.

2. The T had lower lesion levels than U and S. A lower lesion level mostly resulted in a lower \( \dot{V}O_2 \), which has been suggested may be caused by a greater reduction in active muscle mass and strength and a more severe autonomic disturbance (Wicks et al. 1983; Van Loan et al. 1987; Burkett et al. 1990).

Submaximal exercise responses

The values found in the submaximal test were compared using the relationship between \( \dot{V}O_2 \) with \( Q_c \), SV and HR (Figs 2, 3, 4). These figures also show results found by Hopman et al. (1992, 1993), who have used the same protocol and \( CO_2 \) rebreathing method in individuals with paraplegia and able-bodied subjects.

Some authors have suggested that a hypokinetic (Davis and Shephard 1988) or a hyperkinetic circulation (Jehl et al. 1991) exists in SCI individuals. Results of the present study would suggest that the circulation was isokinetic in the individuals with TP, which is in accordance with results that have been reported in paraplegics and able-bodied subjects by other authors (Sawka 1986; Hopman et al. 1992, 1993).

Although the ratio \( Q_c \times \dot{V}O_2 \) is comparable for TP and able-bodied subjects or paraplegics, data from this study clearly showed markedly lower values for \( Q_c \) and SV in TP compared to the other groups. Hardly any data are available on \( Q_c \) and SV in individuals with TP. Van Loan et al. (1987) have studied \( Q_c \) and SV during a maximal test and found values comparable (52.0 ml and 5.7 l·min\(^{-1}\), respectively) to the results found in U and S (Figs. 2, 3).

The lower SV in TP compared to able-bodied subjects and paraplegics has been confirmed by a left ventricular atrophy reported by Kessler et al. (1986) using the Echo Doppler method. This is most likely due to a decrease in left ventricular wall stress, i.e. lack of sufficient cardiac load as a result of the sedentary life style in TP and the limited amount of active muscle mass available for exercise. It is not known whether or not this atrophy of the left ventricle is reversible by training individuals with TP. Although Fig. 3 shows a higher SV for T compared to U and S, it is too early to conclude that this was the effect of regular participation in sport on cardiac muscle atrophy.

The \( HR_{\text{peak}} \) was lower in TP (around 120 beats·min\(^{-1}\)) than in able-bodied subjects (Fig. 4) as a result of the loss of sympathetic innervation of the heart. In addition, the smaller active muscle mass and the lower metabolic rate may also have contributed to a lower \( HR_{\text{peak}} \) in TP (see Van Loan et al. 1987).
Trainability

The fact that the physiological responses to maximal and submaximal exercise had not changed significantly after 3 or 6 months of training in both groups (T and U) seems to indicate that participation in quad rugby had no effect on their physical capacity. Although the intensity of the training, expressed in minutes above 60% of the HRs, met the requirements for gaining benefits from training, it has been thought that a low frequency of training may account for an absence of a measurable training effect (Haskell 1994).

It has been suggested that another explanation for the lack of a training effect may be the vulnerability of TP to illness and injury (Millar and Ward 1983), leading to a reduction of the training sessions and, consequently, to any training effect eventually gained being lost. Furthermore, the occurrence of illness or disease may lead to drop outs, and a natural selection will happen as a result. In other words, sport participation and its continuation seem to depend on the degree of vulnerability in these individuals.

Of note, however, is the fact that individually remarkable improvements were observed after 3 and 6 months of training, especially for individuals in the U group. This would suggest that training can improve the fitness in TP if the training programme is efficacious and the medical status of the subjects is stable.

Additional research is needed to elucidate the effect that training has on the fitness and activities in daily life in this population. Special attention has to be focused on the minimal training frequency and intensity needed to improve the fitness level without causing any injuries. Furthermore, the prevention of diseases such as decubitus, pneumonia and bladder infections needs higher priority in training programmes.

Conclusion

The results suggested that a 3 or 6-month training period had no measurable positive effect on the fitness of the group of individuals with TP, although individually marked improvements in fitness were achieved.

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