ORIGINAL ARTICLE

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Cardiovascular responses at the onset of passive leg cycle exercise in paraplegics with spinal cord injury

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Abstract The purpose of this study was to examine the cardiovascular responses at the onset of passive leg cycle exercise (PLCE) in paraplegics with spinal cord injury (SCI) to investigate the increase in venous return from the paralyzed lower limbs during PLCE. Six male SCI patients with lesions at levels ranging from T8 to L1 and five able-bodied subjects (ABS) participated in this study. The subjects performed PLCE at pedalling frequencies of 40 rpm for 6 min. Cardiac output (Qc), stroke volume (SV) and heart rate (fH) were measured before and during PLCE. In the steady state (4th and 5th min) of PLCE, both SCI and ABS showed a significant increase in Qc. At the onset of PLCE, however, clear differences in the cardiovascular response were found between SCI and ABS. The ABS showed a rapid and marked increase in fH and consequently Qc within 20 s of the onset of PLCE. On the other hand, in SCI, the Qc increased more slowly, compared with that in ABS, because of a smaller increase in fH and a delayed increase in SV. The observed delay in the increases of Qc and SV at the onset of PLCE in SCI was presumably due to the absence of afferent reflexes from the lower limbs, and to the additional time needed for venous return to arrive at the heart from the passively moved muscles.

Key words Spinal cord injury · Passive exercise · Cardiovascular response · Venous return · Paralyzed lower limbs

Introduction

We have previously investigated cardiovascular responses during passive cycling exercise of the paralyzed lower limbs in paraplegics with spinal cord injury (SCI) (Muraki et al. 1996). This study demonstrated significant increases in cardiac output (Qc) and stroke volume (SV) with no increase in heart rate (fH) during passive exercise at a steady state in SCI. Based on these results, we have speculated that rhythmic lengthening and shortening of the paralyzed muscles by passive exercise would promote venous return from the muscles.

It has been demonstrated that in able-bodied subjects (ABS), cardiovascular responses are changed by passive exercise (Benjamin and Peyser 1964; Nakazono and Miyamoto 1985; Nobrega et al. 1994; Nurhayati and Boucher 1998). It has been considered that these changes are mainly due to peripheral afferent reflexes (Nobrega and Araujo 1993; Nobrega et al. 1994) and to the increased venous return from passively moved muscles (Nakazono and Miyamoto 1985; Nobrega et al. 1994). In particular, rapid changes at the onset of exercise have been assumed to be controlled by afferent reflexes from the mechanoreceptors in the moved muscles (De Meersman et al. 1998; Mitchell 1990; Nobrega and Araujo 1993; Williamson et al. 1995). On the other hand, SCI patients lose no afferent reflexes from the paralyzed lower limbs, since the afferent pathway is interrupted by the spinal cord injury. Therefore, if increases in Qc and SV during passive leg exercise were to be mainly due to increased venous return from the paralyzed lower limbs, the beginning of these increases may be delayed in SCI compared with ABS.

Cardiovascular responses at the onset of passive leg exercise in SCI have previously been reported by Morikawa et al. (1989) who failed to show changes in cardiovascular responses at the onset of exercise in ABS as well as SCI. Nobrega et al. (1994) have observed significant changes in the cardiovascular response during passive leg exercise using cycling movement in ABS, and
have suggested that the passive knee extension used by Morikawa et al. (1989) was less effective in increasing venous return compared with cycling movements. Thus, passive cycling movements may be more effective in promoting circulation in passively moved muscles, compared with knee extension movements. To the best of our knowledge, however, no studies have examined the cardiovascular response at the onset of passive exercise using leg cycling movement in PSCI.

The purpose of this study, therefore, was to compare cardiovascular responses at the onset of passive leg cycle exercise (PLCE) between PSCI and ABS, to investigate any differences in the rate of increase in venous return from the paralyzed lower limbs during PLCE.

**Methods**

**Subjects**

Six male PSCI with complete lesions of the spinal cord and five male ABS volunteered to participate in this study. Their physical characteristics are listed in Table 1. The level of spinal cord injury in PSCI was located between T8 to L1. Therefore, cardiac sympathetic innervation was not damaged in this group, suggesting that cardiac regulation was essentially normal. No subject had a history of cardiac, metabolic, or pulmonary disease. Written informed consent was obtained from all of the subjects before starting the study.

**Experiment procedures**

Each subject, wearing shorts, remained seated in their wheelchair or a chair for at least 30 min before each experiment. Then, each subject sat on the chair of the passive leg cycle ergometer and his feet were placed in the shoes on the pedals. The passive leg cycle ergometer has previously been described in detail by Morikawa et al. (1996). Before the tests, each subject was given time to become accustomed to the ergometer and thereafter rested for 10 min. Each subject performed PLCE twice at 40 revolutions each min for 6 min. An interval of at least 10 min was allowed between determinations. The direction of pedal revolution on the ergometer was the same as that of a normal cycle ergometer. The experiment was carried out in a room with ambient temperature and relative humidity maintained at 24–26°C and 40%–60%, respectively.

**Experiment measurements**

During rest for 30 s immediately before PLCE and during PLCE, SV was measured using noninvasive thoracic impedance cardiography every 10 s. Four steel strain electrodes (leads 1–4) of 5-mm width and 0.1-mm thickness constituted the tetrapolar electrode system of the impedance cardiograph and were arranged on the chest wall and neck as described by Kubicek et al. (1970). A constant, sinusoidal alternating current equal to or less than 450 µA root mean square at a frequency of 50 kHz was generated between leads 1 and 4. The chest cavity impedance voltage (Z0) was transmitted via the leads 2 and 3, amplified, and processed into the impedance cardiograph (NEC-Sanda, Impedance plethysmograph 4134, Japan). The SV was calculated from the equation of Kubicek et al. (1970) as follows:

\[ SV = \rho rh0 \cdot (I^2/Z0^2) \cdot (dZ/dt^{-1}) \cdot t \]

where \( \rho \) is the blood resistivity (135 ohms · cm), \( I \) is the current (centimetres) between leads 2 and 3, \( Z0 \) is the mean blood impedance (ohms) between leads 2 and 3, \( dZ/dt^{-1} \) is the first derivative of change in thoracic impedance (ohms per second during a single cardiac cycle), and \( t \) is the left ventricular ejection time (seconds).

A MacLab system (AD Instruments Pty Ltd, Macquarie Park, Australia) equipped with an analogue to digital converter was used to sample \( Z0, dZ/dt^{-1}, \) electrocardiograph (ECG), and plethysmograph (PCG) signals at 200 Hz. The wave of impulsion \( dZ/dt^{-1} \), ECG, and PCG were displayed, and the points corresponding to aortic valve opening, closure, and the peak of derivative were identified. The \( dZ/dt^{-1} \) and \( t \) were measured from these three points. The \( f_c \) was determined from the R-R intervals of the ECG. The \( Q_c \) was expressed as the product of SV and \( f_c \).

**Statistical analysis**

For each variable, the average values from two trials was used for analysis. A paired Student’s t-test was used to compare the difference between rest and exercise in each variable within PSCI and within ABS. Statistical significance was accepted at \( P < 0.05 \) using two-tailed probability.

**Results**

Figure 1 illustrates the responses of \( Q_c, SV \) and \( f_c \) during PLCE in PSCI and ABS. In both groups, \( Q_c \) and \( SV \) were increased during PLCE. In the steady state (4th and 5th min) of PLCE, \( Q_c \) and \( SV \) in PSCI and \( Q_c \) in ABS significantly differed from those at rest before PLCE (Table 2). However, the change in \( f_c \) differed markedly between PSCI and ABS. In ABS, \( f_c \) was elevated at the onset of PLCE and subsequently returned slowly to resting level until the end of PLCE. In contrast, PSCI showed smaller changes in \( f_c \) during PLCE, compared with ABS.

Figure 2 shows the time course of the ratio of values during PLCE to resting values of \( Q_c, SV \) and \( f_c \). For 60 s at the onset of PLCE in each group. In ABS, \( Q_c \) and \( SV \) and \( f_c \) increased rapidly and attained a plateau level within 20 s at the onset of PLCE. For 10 s at the onset of PLCE, \( Q_c \) significantly increased, compared with the resting level, because of increases in \( SV \) and \( f_c \). However, in PSCI, \( Q_c \) and \( SV \) increased more slowly at the onset of PLCE: for 10 s \( Q_c \) was not significantly increased, because \( SV \) was unchanged. However, significant increases in \( Q_c \) were found 20 s after the onset of PLCE because \( SV \) began to increase. The \( f_c \) in PSCI showed a smaller change compared with that in ABS, but there was a
significant difference in \( f_c \) for 10 s after the onset of PLCE.

**Discussion**

This study showed increases in \( \dot{Q}_e \) and SV at the onset and during the steady state of PLCE in both groups, although the dynamics differed between the groups. These results were unlike those of Morikawa et al. (1989) who used passive knee extension movement. The differences in results between these studies may be explained by differences in the type of passive movement. In the steady state, previous studies (Figoni et al. 1990; Muraki et al. 1996) using leg cycling movement in PSCI have reported increased \( \dot{Q}_e \) and SV without increased \( f_c \), which was consistent with our findings. However, Thomas et al. (1997) using passive knee extension movement have recently found no increase in \( \dot{Q}_e \) or SV in both PSCI and
ABS. Morikawa et al. (1989) have reported similar findings. Thus, passive cycling movement may be more suitable in promoting circulation in passively moved muscles.

At the onset of PLCE, the $f_c$ response in PSCI differed markedly from that of ABS. The $f_c$ in ABS elevated rapidly and markedly within 20 s from the onset of PLCE, resulting in an increase in $Q_e$, which has been found by previous studies (Nobrega and Araujo 1993; Waisbren et al. 1990; Williamson et al. 1995). In contrast, $f_c$ responses as seen in ABS were not found in PSCI, although a slight elevation of $f_c$ was observed at the onset. It has been considered that in ABS, the increase in $f_c$ elicited at the onset of passive exercise is produced mainly by peripheral afferent reflexes from exercising muscles (De Meurs et al. 1998, Nobrega and Araujo 1993; Williamson et al. 1995). On the other hand, in PSCI, afferent reflexes from the paralyzed lower limbs during PLCE are dysfunctioning because of the interruption of afferent pathways. Therefore, dysfunction of peripheral afferent reflexes may inhibit the increase in $f_c$ at the onset of PLCE. These findings supported the theory that the increase in $f_c$ at the onset of passive exercise in ABS is due mainly to peripheral afferent reflexes from the lower limbs.

In both PSCI and ABS, the significant increase in $Q_e$ during the steady state of PLCE was dependent on the increase in SV. This finding was consistent with previous studies (Figoni et al. 1990; Nakazono and Miyamoto 1985; Nobrega et al. 1994). Nobrega et al. (1994) have proposed that the increase in SV during the steady state of PLCE was induced by an increase in venous return from the passively moved limbs and/or by a muscle mechanoreceptor-evoked increase in myocardial contractility in ABS. In PSCI, however, afferent signals from the muscle receptors including the mechanoreceptor in the paralyzed lower limbs cannot have played a role, as previously described. Accordingly, the increase in SV may have been due mainly to an increase in venous return resulting from the rhythmic shortening and lengthening of the paralyzed muscles in the lower limbs.

Furthermore, it should be noted that the beginning of the increase in SV was delayed in PSCI, compared with ABS, since it remained unchanged during the 10 s from the onset of PLCE. This delay may have been due to the circulation time needed for the additional venous return to arrive at the heart from the lower limbs. Unfortunately, no studies have reported the circulation time from the lower limbs to the heart. However, circulation time is clearly at least several seconds, according to studies of the venous blood velocity in the lower limbs during passive movement of the foot that have been reported by Staubesand et al. (1995) and the circulation time from the lower limbs to other regions by Katori (1997). Thus, the delayed increase in SV supported the promotion of venous return from the paralyzed lower limbs during PLCE in PSCI.

In conclusion, this study showed a delayed increase in SV at the onset of PLCE in PSCI. The findings demonstrated that rhythmic shortening and lengthening of the paralyzed muscles by passive exercise promote venous return from the muscles.

References