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Oxygen Uptake Responses during Passive Walking-like Exercise in Persons with Spinal Cord Injury

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Abstract

The aim of the present study was to examine the response of pulmonary oxygen uptake ($\dot{V}O_2$) during passive walking-like exercise in the standing position (PWE) in persons with spinal cord injury (PSCI, n = 12). PSCI were divided into groups of those with upper motor neuron lesion (group U) and those with lower motor neuron lesion (group L) according to the presence of muscle spasm. They experienced standing quietly for 6 min and subsequent PWE at 1 Hz for 12 min. In the present study, average $\dot{V}O_2$ for every 6-min interval (sitting, standing, and PWE in the first and second halves) were calculated. In two of the PSCI in group U, the largest $\dot{V}O_2$ was observed during quiet standing position. In six of the PSCI in group U, $\dot{V}O_2$ in the first half of PWE was the largest and was significantly larger than that during sitting position (127 ± 5%). In all four of the PSCI in group L, $\dot{V}O_2$ during quiet standing position and $\dot{V}O_2$ during PWE were not significantly different from that during sitting position. In conclusion, PWE enhances $\dot{V}O_2$ (i.e., energy expenditure) in PSCI, especially in PSCI with an upper motor neuron lesion. This fact suggests that PWE is applicable as a physical exercise for preventing overweight and/or obesity that may arise from sedentary daily life in PSCI.

Key Words : pulmonary oxygen uptake, standing posture, passive exercise, rehabilitation, spinal cord injuries
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1. Introduction

Overweight and obesity are closely linked to various medical disorders, such as cardiovascular disease, hypertension, diabetes, dyslipidemia and metabolic syndrome, and have a negative effect on longevity.[29] Anson and Shepherd[1] reported that nearly 40% of the 309 persons with spinal cord injury (PSCI) in their study were either overweight or obese and that such PSCI tended to have more medical problems than did those in an underweight/ideal weight group. In addition, Buchholz et al.[6] found that 70% (19/27) of PSCI activity level, calculated by dividing total daily in their study were obese and that physical energy expenditure by resting metabolic rate, of the PSCI was low compared to the World Health Organization recommendations. Thus, Buchholz et al.[6] emphasized that PSCI need to engage in structured physical

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activity of increased frequency, intensity and/or duration to offset sedentary activities of daily living.

As a result of a lower-body dysfunctioning, PSCI depend largely on their upper body during almost all activities of daily living. Therefore, it is necessary to enhance physical activity level with less fatigue and without injury to the upper body. That is, methods without the use of the upper body are needed. It has been shown that passive walking-like exercise (PWE) in the standing posture induces increases in double product as an index of myocardial blood flow and oxygen consumption (unpublished data) and in electromyographic activity[8,9] and also an increase in oxygen supply in the paralyzed lower limb muscle.[16] These physiological responses indicate that PWE has the potential to enhance energy expenditure.

Pulmonary oxygen uptake ($\dot{V}O_2$) represents whole-body energy expenditure. To our knowledge, there has been only one case study in which the effect of PWE on $\dot{V}O_2$ was evaluated.[22] In that study, a person with cervical spinal cord injury performed PWE for 40 min, and it was found that average $\dot{V}O_2$ during PWE reached a value 2.4-times larger than that during sitting (50 ml·min^{-1}→118 ml·min^{-1}). Therefore, we reexamined the effect of PWE on $\dot{V}O_2$ in PSCI with various injury levels (thoracic levels 3 to 12).

2. Materials & Methods

Subjects

Twelve PSCI and six able-bodied controls (CON) participated in the present study. Characteristics of the PSCI are shown in Table 1. All of the PSCI had spinal cord lesions of traumatic origin except for subject 7 (spinal cord tumor). The PSCI had no cardiorespiratory risk factors and were not taking any medications. Voluntary consent for participation in this study was obtained from all subjects after they were informed of the purpose of the experiment, the procedure and possible risks. The study was conducted in accordance with the Helsinki Declaration and was approved by the Ethics Committee of the National Rehabilitation Center for Persons with Disabilities in Tokorozawa, Japan.

Protocol

At least 2 hours before the test, the subjects refrained from eating and taking caffeine. During measurements, the temperature in the experimental room was set within the range of 21-23°C.

Quiet standing and PWE were carried out using a commercially available device (Easy Stand Glider6000 Altimate Medical, Inc., Morton, USA) as described in detail elsewhere.[15,16] Briefly, this device enables subjects to change their posture from sitting to standing by pulling a built-in hydraulic lever. Standing posture is stabilized by fixing the trunk, pelvis and knees using front and back pads, lateral pelvic pads, and kneepads. Bilateral handles located in front of the trunk are linked to the footplates, thus allowing one leg to move forward while the other moves back by pushing and pulling the handles alternately. Since the footplates are movable only in the horizontal direction and since the knees are stabilized, hip and ankle joints are rotated during PWE. In the present study, an experimenter manually pulled the hydraulic lever and moved the handles.

Before the start of the experiment, moving distance of the footplates was determined as the maximal distance in consideration of joint contractures of the subject (10-32 cm for PSCI and 18-37 cm for CON). After more than 20 min of rest in the sitting position, data recording was started. First, subjects rested in the sitting position for 6 min and then changed to the standing position within 30 sec. The standing posture was maintained for 6 min. Thereafter, they experienced 12-min rhythmic PWE at 1 Hz. In some PSCI, muscle spasm sometimes occurred during PWE, thus restricting the moving distance of the footplate. In such cases, the experimenter narrowed the distance transiently. The experimenter always checked data on moving distance displayed on an oscilloscope to avoid

Table 1 Characteristics of persons with spinal cord injury and able-bodied controls (CON)

Subject No.	Age (years)	Height (cm)	Weight (kg)	Time since injury (months)	Lesion level	Asia score
1	33	185	81	85	T3 -5	A
2	23	175	57	73	T4, 5	B
3	44	153	49	317	T5	A
4	35	173	79	172	T5	A
5	22	170	62	40	T6, 7	A
6	34	174	71	102	T7	A
7	35	163	60	23 3	T9	A
8	21	171	56	48	T10	A
9	36	168	66	85	T11	A
10	42	167	55	276	T11, 12	A
11	34	183	72	106	T12	A
12	33	168	55	160	T12, L1	A
Mean ± SD	33 ± 7	171 ± 8	64 ± 10	141 ± 91		
CON (n = 8)	32 ± 5	170 ± 8	69 ± 15			

large variation. The distance was derived from angle data measured by an electrogoniometer placed at the junction of the handle and footplate. After the PWE, the subjects maintained standing posture for 6 min and then changed to sitting posture within 30 sec. The sitting posture was maintained for 6 min. The subjects were occasionally instructed to relax their upper limbs during the experiment.

Measurements

Data on minute ventilation ($\dot{V}E$) and pulmonary oxygen uptake ($\dot{V}O_2$) were obtained breath-by-breath using a respiratory gas analyzer (AE-300S, Minato Medical Science, Osaka, Japan). $\dot{V}E$ was measured by a hot-wire flow meter, and the flow meter was calibrated with a syringe of known volume (2.0 l). O_2 was measured by a zirconium sensor analyzer. The gas analyzer was calibrated by known standard gas (O_2 15.18%, CO_2 5.06%). These data were measured continuously throughout the experiment. Averages of these data were calculated for each 1-min interval.

Continuous surface electromyograms (EMGs) of the medial gastrocnemius muscle (GM) and tibialis anterior muscle (TA) of the right leg were recorded using bipolar electrodes (DE-2.1, Delsys, Boston, USA). These two muscles were chosen as representative flexor and extensor muscles. Prior to EMG electrode application, the measurement site was prepared by the removal of dead skin by gentle abrasion with abrasive paper. Then the EMG electrodes were attached to the skin in a direction parallel to the muscle fiber orientation with double-sided adhesive tape. The reference electrode was placed over the patella. The electrode contacts were made from two silver bars that were each 10 mm in length and 1 mm in diameter, and they were spaced 10 mm apart. The detected signals were amplified before being sent along a shielded cable to the rest of the EMG system. The EMG signals were then amplified using a main amplifier (Bagnoli-8, Delsys, Boston, USA) with a gain of 1000 fold. The analog signals of EMG were sampled at 1000 Hz by a dedicated computerized data acquisition system and stored on hard disk for later analysis. In the present study, root mean square of the EMG signal (RMS-EMG, μV) for every 1-min interval for each muscle was calculated. RMS-EMG of the GM and that of TA were summed up in order to estimate overall activity of flexor and extensor muscles.

Statistical analysis

Average $\dot{V}O_2$, average $\dot{V}E$ and average RMS-EMG for every 6-min interval (sitting, standing, and PWE in the first and second halves) were calculated. Ventilatory equivalent for oxygen uptake ($\dot{V}E_{O2}$) was calculated by dividing the average $\dot{V}E$ by the average $\dot{V}O_2$. These average values were used for statistical analysis. One-way analysis of variance for repeated measures was used for comparison of the values during sitting, standing and PWE. If significant F-ratios were found, post hoc analysis was conducted using the Tukey-Kramer test to detect differences in the values between conditions. A value of $P<0.05$ was regarded as statistically significant. All data are presented as means and S.E.M.

3. Results

Fig.1 shows changes in $\dot{V}O_2$ throughout the experiment in PSCI and CON. Since there were three patterns of changes in $\dot{V}O_2$ in PSCI, examples in three PSCI are shown in the figure. For CON, on the other hand, average change in $\dot{V}O_2$ is shown since changes in $\dot{V}O_2$ in all subjects were similar. In subject 7 of PSCI (Fig.1A), $\dot{V}O_2$ increased during standing and increased further after the onset of PWE. After the end of PWE, $\dot{V}O_2$ decreased to the pre-PWE standing level. In subject 1 of PSCI (Fig.1B), $\dot{V}O_2$ showed the largest value during standing and then decreased gradually with time. In contrast to the pattern of change in subject 7, there was no sharp increase in $\dot{V}O_2$ after the onset of PWE, but $\dot{V}O_2$ during PWE remained elevated above the sitting level. In subject 11 of PSCI (Fig.1C), $\dot{V}O_2$ during standing showed larger variation than did $\dot{V}O_2$ during sitting. $\dot{V}O_2$ during PWE was almost the same level as that during sitting. In CON (Fig.1 D), $\dot{V}O_2$ during standing tended to decrease below the sitting level. After the onset of PWE, $\dot{V}O_2$ increased abruptly, and $\dot{V}O_2$ decreased abruptly after the end of PWE. In subjects 1 and 7, who showed increases in $\dot{V}O_2$ during PWE (Fig.1A and B), muscle spasm, represented as increases in RMS-EMG, occurred in the transition from sitting to standing, probably due to muscle stretching. Such muscle activity was observed in eight of the twelve PSCI (thoracic levels 3 to 10, T3 to T10).

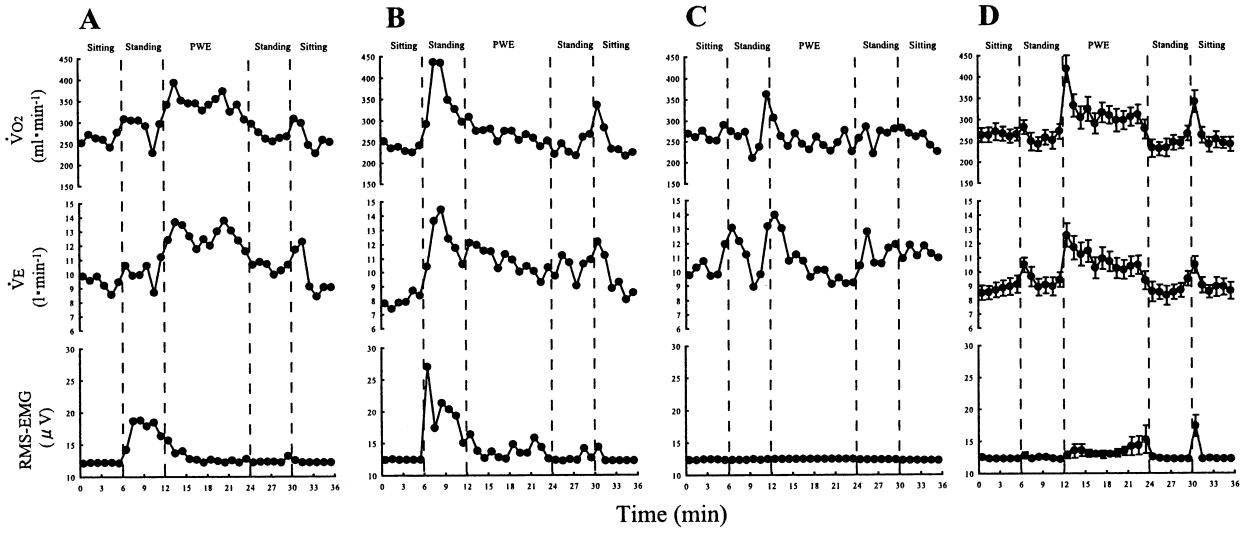

Figure 1. Changes in pulmonary oxygen uptake ($\dot{V}O_2$, upper panels), minute ventilation ($\dot{V}E$, middle panels) and root mean square of electromyographic activity of leg muscles (RMS-EMG, lower panels) throughout the experiment in persons with spinal cord injury (PSCI, A-C) and able-bodied controls (CON, D).
For PSCI, changes in three subjects are shown due to large interindividual differences in $\dot{V}O_2$. In Fig.A and B, $\dot{V}O_2$ increased during PWE compared to the sitting level. In Fig.A, $\dot{V}O_2$ was the largest during PWE, while $\dot{V}O_2$ was the largest during standing in Fig.B. In Fig.C, there was no no apparent increase in $\dot{V}O_2$ during PWE. For CON, average changes (n = 8) are shown due to small interindividual differences.
PWE: passive walking-like exercise.

In subject 11, who showed no apparent increase in $\dot{V}O_2$ during PWE (Fig.1C), there was also no apparent increase in RMS-EMG in the transition from sitting to standing. Four PSCI (T11 to T12) showed no increase in RMS-EMG. For these PSCI, the experimenter tried to evoke muscle spasm by sudden flexion and extension of the ankle joints after the experiment but failed to observe any muscle spasm. In addition, the PSCI reported no occurrence of muscle spasms in daily life. Accordingly, we regarded PSCI with muscle spasm as those with an upper motor neuron lesion (group U) and PSCI without muscle spasm as those with a lower motor neuron lesion (group L).[15] In group U, there were two patterns of $\dot{V}O_2$ responses as shown in Fig.1A and B. Two of the PSCI showed the pattern presented in Fig.1B: $\dot{V}O_2$ was the largest during standing and did not show a sharp increase after the onset of PWE. We suspect that the increased $\dot{V}O_2$ during standing had an influence on $\dot{V}O_2$ during PWE and thus masked a sharp increase in $\dot{V}O_2$ after the onset of PWE. Therefore, in order to analyze the actual $\dot{V}O_2$ responses during PWE, we excluded two of the PSCI from group U and carried out statistical analysis using data obtained from the remaining six PSCI.

Table 2 shows $\dot{V}O_2$ and RMS-EMG during sitting, standing and PWE in each of the PSCI groups U and L and in CON. There was no significant difference between $\dot{V}O_2$ during sitting and that during standing in any of the groups. However, average $\dot{V}O_2$ in each of the PSCI groups increased slightly, whereas that in CON decreased slightly. RMS-EMG was significantly larger during standing than during sitting only in group U. $\dot{V}O_2$ during PWE was larger than that during standing in group U and in CON. In CON, $\dot{V}O_2$ during the latter part of PWE was significantly lower than that during the former part of PWE. In group U, average $\dot{V}O_2$ during the latter part of PWE was lower than that during the former part of PWE, although the difference was not significant. In group L, there was no significant difference between $\dot{V}O_2$ during standing and that during PWE.

Table 2 Pulmonary oxygen uptake ($\dot{V}O_2$), minute ventilation ($\dot{V}E$) and root mean square of electromyogram of leg muscles (RMS-EMG) during sitting, standing and passive walking-like exercise (PWE)

			Sitt ing	Standing	PWE (1-6 min)	PWE (7-12 min)
PSCI with upper motor neuron lesion (n = 6)	$\dot{V}O_2$	($ml \cdot min^{-1}$)	229 ± 10	248 ± 14	292 ± 19 *,†	274 ± 21 *
	$\dot{V}E$	($l \cdot min^{-1}$)	8.5 ± 0.5	9.4 ± 0.6	10.4 ± 0.7 *	10.0 ± 0.9 *
	RMS-EMG	(μV)	13.0 ± 0.5	15.3 ± 0.7 *	14.3 ± 07	12.7 ± 0.2 †
PSCI with lower motor neuron lesion (n = 4)	$\dot{V}O_2$	($ml \cdot min^{-1}$)	243 ± 14	256 ± 13	265 ± 9	248 ± 14
	$\dot{V}E$	($l \cdot min^{-1}$)	9.1 ± 1.0	9.8 ± 0.8	10.2 ± 0.5	9.5 ± 0.4
	RMS-EMG	(μV)	12.5 ± 0.2	12.4 ± 0.2	12.5 ± 0.2	12.4 ± 0.2
CON (n = 8)	$\dot{V}O_2$	($ml \cdot min^{-1}$)	265 ± 17	258 ± 16	330 ± 26 *,†	299 ± 22 *,†,‡
	$\dot{V}E$	($l \cdot min^{-1}$)	8.5 ± 0.5	9.1 ± 0.5 *	11.3 ± 0.8 *,†	10.2 ± 0.6 *,†,‡
	RMS-EMG	(μV)	12.4 ± 0.2	12.5 ± 0.2	13.2 ± 0.7	13.9 ± 1.1

Averages for every 6-min interval (sitting, standing, and PWE in the first half and second half, respectively) were calculated and are shown in the table.

*, †, ‡ denote significant difference compared to the value during sitting, standing and PWE in the first half, respectively.

In CON, average RMS-EMG during PWE was larger than that during standing, although the difference was not significant. In group U, RMS-EMG during PWE tended to be lower than that during standing and RMS-EMG during the latter part of PWE was significantly lower than that during standing. In group L, there was no significant difference between RMS-EMG during standing and that during PWE.

Fig. 2 shows the raw EMG signals during PWE. EMG activity observed in subject 4 is shown in Fig.2A, and EMG activity observed in subject 8 in the additional experiment is shown in Fig.2B. The occurrence of the former EMG activity was not synchronized with leg motion, whereas the occurrence of the latter EMG activity was synchronized with leg motion.

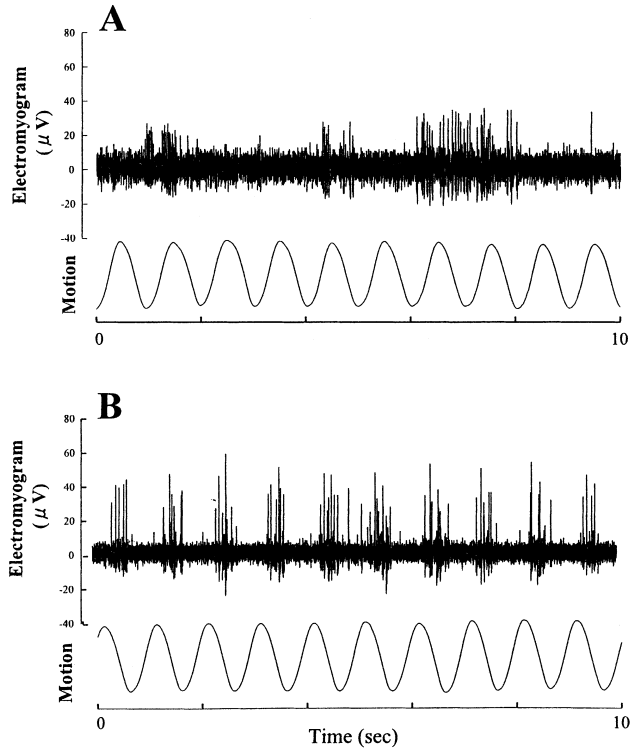

Figure 2. Raw electromyogram of the gastrocnemius muscle of two persons with spinal cord injury. A: spasmodic electromyographic activity. B: electromyographic activity that is synchronized with leg motion.

Table 2 also shows $\dot{V}E$ during sitting, standing and PWE. In CON, $\dot{V}E$ during standing was significantly larger than that during sitting, and $\dot{V}E$ during PWE was significantly larger than that during standing. In both PSCI groups, average $\dot{V}E$ during standing was larger than that during sitting, although the difference was not significant. Furthermore, there was no significant difference between $\dot{V}E$ during standing and that during PWE in either of the PSCI groups. However, in group U, $\dot{V}E$ during PWE was significantly larger than that during sitting.

Fig. 3 shows changes in $\dot{V}E_{O2}$ in each of the PSCI groups and in CON. In CON, $\dot{V}E_{O2}$ during standing was significantly larger than that during sitting and $\dot{V}E_{O2}$ during PWE was significantly lower than that during standing. In group U, $\dot{V}E_{O2}$ during standing was not significantly different from that during sitting. Average $\dot{V}E_{O2}$ during PWE was lower than that during standing, although the difference was not significant. In group L, there was no significant difference in $\dot{V}E_{O2}$ between each pair of conditions.

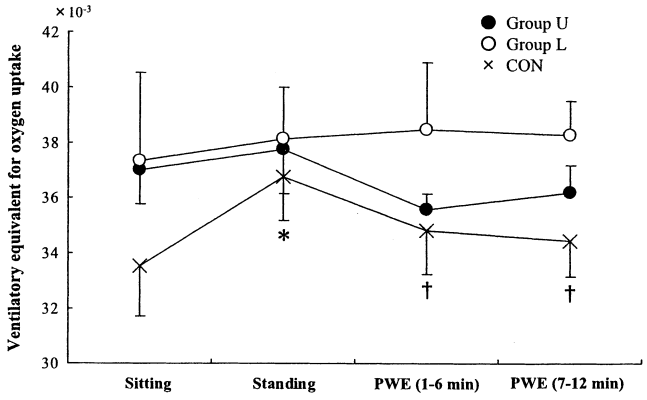

Figure 3. Ventilatory equivalent for oxygen uptake during sitting, standing and passive walking-like exercise in persons with spinal cord injury (PSCI) and able-bodied controls (CON). PSCI were divided into one group with upper motor neuron lesions (group U) and one group with lower motor neuron lesions (group L).
*, † denote significant difference compared to the value during sitting and standing, respectively. Significant differences were observed only in CON.

4. Discussion

The main finding of the present study was that $\dot{V}O_2$ increased during PWE in PSCI with an upper motor neuron lesion but not in PSCI with a lower motor neuron lesion. We discuss the possible physiological mechanisms underlying changes in $\dot{V}O_2$ and the clinical implications of our results below.

During standing

In CON, $\dot{V}O_2$ decreased slightly during standing. Miyamoto et al.[18] found that after tilting from the supine position to a 60° head-up position, $\dot{V}O_2$ decreased transiently to 50-60% of the value in the supine position, these values being restored to the pre-tilting value during the first 5 min. They also found that the decrease in $\dot{V}O_2$ was accompanied by a decrease in cardiac output. Based on these findings, they speculated that the decrease in cardiac output caused a reduction of O_2 transfer from the lungs to blood, resulting in a decrease in $\dot{V}O_2$. Cardiac output has also been found to be decreased by postural change from sitting to standing.[11] Thus, the decrease in $\dot{V}O_2$ during standing would have been caused by a decrease in cardiac output.

Contrary to what was shown in CON, $\dot{V}O_2$ increased slightly during standing in groups U and L. In group U, $\dot{V}O_2$ would have been increased partly by muscle activity of the paralyzed lower limb muscles. However, this is unlikely in group L. Considering that PSCI are unaccustomed to the standing posture, some postural trunk muscle might have been activated and increased $\dot{V}O_2$ in PSCI.

$\dot{V}E$ increased during standing in CON and PSCI. Chang et al.[7] found that in normal subjects, $\dot{V}E$ was increased by postural change from the supine position to a 70° head-up position and that the increase in $\dot{V}E$ was not accompanied by any increase in $\dot{V}O_2$. These responses of $\dot{V}E$ and $\dot{V}O_2$ are similar to those observed in the transition from standing to sitting in CON in the present study. Chang et al.[7] suggested that the increase in $\dot{V}E$ was caused by increased compliance of the lungs during head-up position. In the present study, however, since PSCI showed a slight increase in $\dot{V}O_2$ during standing, the increase would have caused a certain amount of increase in $\dot{V}E$ during standing.

During PWE

In CON, $\dot{V}O_2$ increased significantly and average RMS-EMG increased during PWE. It has been found that $\dot{V}O_2$ increases during passive leg cycling in normal subjects.[2,4,17,19,23,28] In addition, the increase in $\dot{V}O_2$ was found to be accompanied by some EMG activity, although the amplitude and duration of the bursts were markedly less during passive leg cycling than during unloaded active cycling.[23] The EMG activity observed during passive cycling is thought to be produced by unconscious activation of descending motor pathways or by spinal reflex evoked by the passive stretching of muscles.[23] The increase in $\dot{V}O_2$ during PWE might have been related to the EMG activity associated with mechanism(s) described above.

In CON, $\dot{V}E$ also increased significantly during PWE. It has been found that $\dot{V}E$ increases during passive leg cycling.[19,28] Hida et al.[14] compared the ventilatory response to passive movement of hindlimb muscles in anesthetized dogs with and without rhizotomy. They found that $\dot{V}E$ was increased by passive movement in spinal intact dogs but not in dogs with rhizotomy. In addition, Morikawa et al.[19] compared the ventilatory response to passive leg cycling in normal and spinal-cord-injured persons. They found that $\dot{V}E$ increased during passive leg cycling in normal persons but not in persons with spinal cord injury. From these findings, the investigators reached the same conclusion that the ventilatory response during passive leg movement is related to afferent drive from exercising limbs.

Morikawa et al.[19] found that an increase in $\dot{V}O_2$ during passive leg cycling was accompanied by an increase in $\dot{V}E$ but not by any increase in EMG activities of the leg muscles. In addition, Waisbern et al.[28] demonstrated that an increase in $\dot{V}O_2$ during passive leg cycling was accompanied by an increase in $\dot{V}E_{O2}$. These findings suggest the

possibility that $\dot{V}O_2$ increases solely depending on an increase in $\dot{V}E$ during passive leg exercise. However, in the present study, this possibility is unlikely due to the fact that $\dot{V}E_{O2}$ tended to decrease and the fact that EMG activity occurred during PWE.

$\dot{V}O_2$ increased during PWE in group U but not in group L. The contribution of unconscious activation of descending motor pathways cannot account for the difference between groups U and L because the descending pathways are disturbed by spinal cord injury in both groups. Since PSCI with an upper motor neuron lesion have intact nerve endings and spinal loops,[12] stretch-reflex-induced muscle activity will occur during PWE in group U but not in group L, resulting in an increase in $\dot{V}O_2$ only in group U. Another explanation is that the occurrence of muscle spasms in daily life in PSCI in group U might have enhanced muscle mass of the paralyzed leg muscles, resulting in the larger increase in $\dot{V}O_2$ during PWE.

EMG activity synchronized with leg motion, referred to as locomotor-like EMG activity, was observed in one subject in group U. Similar EMG activity was observed previously.[8,9] Dietz et al.[8] concluded that afferent input from hip joints, in combination with that from load receptors, plays a crucial role in the generation of locomotor activity in the isolated human spinal cord. In the present study, the locomotor-like EMG activity would have also contributed to the increase in $\dot{V}O_2$ during PWE.

In group U, $\dot{V}O_2$ during the latter part of PWE was lower than that during the former part of PWE. This corresponded to the fact that RMS-EMG during the latter part of PWE was lower than that during the former part of PWE. However, the RMS-EMG during PWE was lower than that during standing despite the fact that $\dot{V}O_2$ during PWE was larger than that during standing. Since only the EMG activities of TA and GM were recorded in the present study, an exact comparison between $\dot{V}O_2$ and RMS-EMG could not be made. However, we recently demonstrated that the double product, as an index of myocardial oxygen consumption, increases by imposing PWE, particularly in PSCI with low lesion levels (below T7) (unpublished data). Therefore, even if the level of muscle activity of the paralyzed leg muscles decreases during PWE, $\dot{V}O_2$ might increase due to increases in oxygen consumption in other organs.

The increase in $\dot{V}O_2$ during PWE observed in group U is unlikely to be related to the increase in $\dot{V}E$ per se, because the afferent drives from lower limb muscles were absent or small and because $\dot{V}E_{O2}$ tended to decrease during PWE.

There have been conflicting findings regarding $\dot{V}O_2$ responses during passive leg cycling in PSCI: Muraki et al.[20] found a significant increase in $\dot{V}O_2$, whereas other researchers failed to find any significant increases in $\dot{V}O_2$ during passive leg cycling.[10,19,21] Our findings suggest that the diversity of previous findings is related to the presence of muscle spasm and/or to the difference in the magnitude and/or type of stimuli to the paralyzed leg during exercise.

Clinical implications

As mentioned in the introduction section, overweight and obesity and their subsequent medical disorders are a problem for PSCI and these are thought to be attributed to less energy expenditure due to sedentary daily living. In the present study, $\dot{V}O_2$ during PWE was found to increase energy expenditure especially in group U. Thus, PWE is applicable as a physical exercise for enhancement of energy expenditure. However, $\dot{V}O_2$ during PWE increased to no more than 300 ml · min^{-1} (Table 2). In terms of enhancement of energy expenditure, it has been shown that $\dot{V}O_2$ during functional electrical stimulation (FES) leg cycling exercise increased up to about 1.27 l · min^{-1},[3] indicating that muscle electrical stimulation is a more effective means than PWE to increase $\dot{V}O_2$. However, electrical stimulation has some disadvantages for use in paralyzed muscles. The reason for this is as follows. Human studies have suggested that electrical stimulation of a muscle can alter the recruitment order of motor units from fast fatigable to slow fatigue-resistant ones.[13, 27]

Following spinal cord injury, the percentage of fast fibers increases over time, and therefore fatigue resistance to electrical stimulation becomes lower in PSCI than in normal persons .[26] In addition, Bickel et al.[5] suggested that it takes more time to restore electrical-stimulation-induced muscle damage, as determined by T2 signal measured by magnetic resonance imaging, in PSCI than in normal persons. Therefore, it is preferable not to use electrical stimulation for paralyzed muscles for a long time and frequently. In contrast, it is thought that during PWE, the motor units are recruited in order from slow fatigue-resistant motor units to fast fatigable motor units according to the size principle since the afferent inputs were derived from proprioceptors by imposing muscle stretching and body load.[16] Therefore, we believe that PWE can be performed with less fatigue, thus enabling PSCI to perform PWE for a long time and frequently.

In a study by Higuchi et al.,[15] PSCI performed combined PWE and upper body exercise. Combined exercise is advantageous, because $\dot{V}O_2$ derived from PWE is added on that derived from upper body exercise even though PSCI perform upper body exercise at the same workload. In addition to these positive effects, combined exercise may enhance $\dot{V}O_2$ derived from upper body exercise. The reason for this is as follows. In a study by Phillips and Burkett,[24] PSCI performed upper body exercise with and without FES of the paralyzed leg muscles, and $\dot{V}O_2$ derived from upper body exercise was compared by subtracting $\dot{V}O_2$ at rest and FES-generated $\dot{V}O_2$ from whole body $\dot{V}O_2$. They demonstrated small and non-significant increases in upper body $\dot{V}O_2$. They attributed the increase in upper body $\dot{V}O_2$ to FES-enhanced blood flow in the upper body. In our recent study (unpublished data), PWE was found to raise arterial blood pressure. In addition, Kawashima et al.[16] found that PWE reduces blood volume pooled in the paralyzed limb, suggesting an increase in blood volume available in the upper limb musculature. Thus, PWE may enhance blood flow (i.e., oxygen supply) and secondary to oxygen consumption in the upper limb musculature during combined PWE and upper body exercise. Further study is needed to clarify this hypothesis.

5. Conclusions

During passive walking-like exercise in the standing posture, pulmonary oxygen uptake increases in spinal-cord-injured persons with upper motor neuron lesions, but the magnitude of increase is small. Passive walking-like exercise may be effective for the purpose of preventing and ameliorating the status of overweight and/or obesity if this exercise is used in combination with other exercise interventions such as electrical stimulation and upper body exercise.

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