

FIGURE 1—A. Experimental setup. This apparatus enables SCI patients to stand securely by immobilizing their trunk and pelvis using front and back pads, and by preventing hyperextension of the knee joint using a kneepad. It also enables them to swing their legs by moving a handle connected to a foot plate. In this study, the experimenter manually moved the handle back and forth in a sinusoidal manner by matching the movement frequency with the sound of a metronome. **B.** Location of the EMG electrode and the near-infrared spectroscopy (NIRS) sensor. Because it was impossible to place both the NIRS sensor and the EMG electrode at the same place, they were placed proximally and distally on the medial side of the muscle.

signal was amplified (Bagnoli-8 EMG System, DelSys, Inc.).

Electrogoniometer. In order to ascertain the similarity of the leg motion throughout the exercise session, the angle of the device was recorded by an electrogoniometer (Goniometer System, Biometrics Ltd., Ladysmith, VA) with sensors placed on the lateral aspect of the apparatus.

Heart rate. To confirm whether central circulation is enhanced by imposing the passive leg movement, HR was continuously measured by using an integrated telemetric monitor (HR meter, Polar, Vantage, Finland) in two SCI patients and two normal subjects.

Data Analysis

During the experiment, all data were continuously monitored by PowerLab software (Chart ver. 4, AD Instruments Inc., Milford, MA) and were digitized at 1 kHz for later analysis. For NIRS data, the average value in the last 30 s at each stage and those at 1, 3, and 5 min postexercise were evaluated for each parameter. The EMG signals were full-wave rectified after subtraction of the DC component. The magnitude of the EMG activity was quantified by the mean amplitude and integrated area of the EMG activity during the last 1 min of each stage.

Statistical Analysis

Values are given as means \pm SD. Statistical differences in the size of EMG value and each Hb value were tested by ANOVA with repeated measures. Tukey's *post hoc* test was applied to identify differences between the conditions. The statistical software SPSS 11.0 was used to carry out all analyses. Significance was accepted at $P < 0.05$.

RESULTS

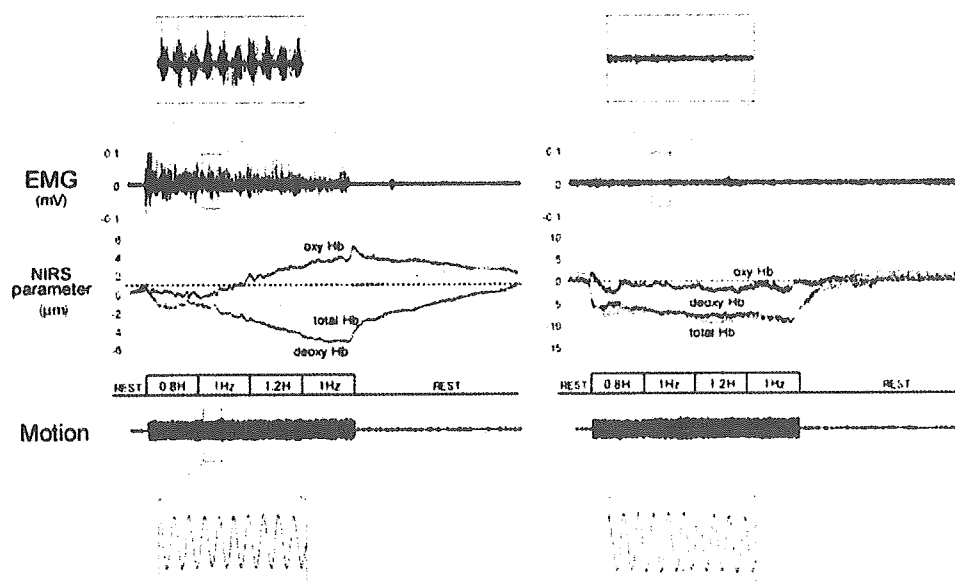
Figure 2 shows a typical example of the EMG activity, NIRS values, and leg motion during an experiment in a SCI patient (Fig. 2A) and a normal subject (Fig. 2B). As clearly shown in this figure, there are remarkable differences in both EMG activity and NIRS parameters between the two groups. It was confirmed that the leg motion was maintained within similar range throughout the exercise in both SCI and normal subjects.

EMG activity. During passive movement, all SCI patients showed EMG activity in the gastrocnemius muscle. The active phase of the EMG activity corresponded to the backward phase of leg movement. Despite the fact that the total (integrated) response area increased with the frequency of the movement, there was no remarkable change in amplitude (Fig. 3). Although the movement frequency in both the second and fourth stages was set at 1 Hz, the EMG amplitude in the fourth stage was significantly lower than that in the second stage (second vs fourth: 42.08 ± 3.73 vs $29.66 \pm 6.19 \mu\text{V}$, $P < 0.05$). In contrast to the SCI patients, normal subjects showed no visible EMG activity in the gastrocnemius muscle at any time during the exercise.

NIRS parameters. In both the SCI and normal groups, the concentrations of total Hb and deoxy-Hb showed rapid decrements following the onset of the exercise and remained at lower levels compared to the resting value while the legs were passively moved. The degree of the decrease of total Hb in the first stage was much smaller in the SCI group than in the normal group (SCI vs normal: 2.79 ± 0.99 vs $7.04 \pm 2.18 \mu\text{m}$). During the exercise period, an increase in oxy-Hb and a decrease in deoxy-Hb, which were independent of the changes in total Hb, were observed in the SCI group but not in the normal group (Figs. 2 and 4). In the recovery stage,

A SCI B Normal

FIGURE 2—Typical example of the EMG activity and concentration changes in each hemoglobin parameter in a patient with motor-complete spinal cord injury (SCI) (A) and a neurologically normal subject (B). Note that there are remarkable differences in both EMG activity and near-infrared spectroscopy (NIRS) parameters between the two subjects. The motion of the apparatus was maintained within a similar range throughout the exercise for both subject groups.



the total Hb level exceeded the resting value in the SCI group, whereas it merely recovered to the pretest level in the normal group.

Heart rate. Figure 5 shows the change of the HR at rest and during passive leg movement and the recovery period obtained from two SCI patients and two normal subjects. As shown in this figure, HR increased just after the onset of passive leg movement in all subjects.

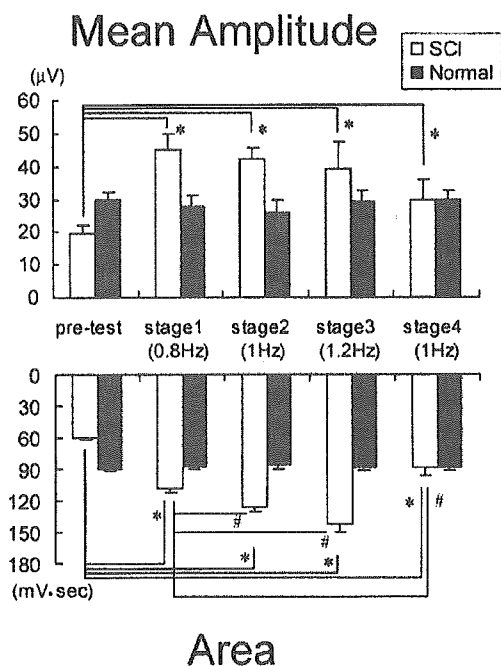


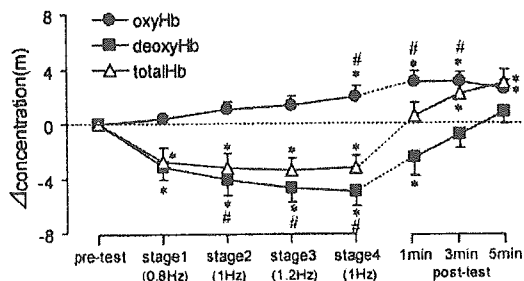
FIGURE 3—Mean amplitude and area of muscle EMG activity in the gastrocnemius muscle. The error bars indicate the SEM value. * Significant difference ($P < 0.05$) compared with the resting value. # Significant difference to the first set value. SCI, spinal cord injury.

DISCUSSION

The present study was designed to examine whether the oxygenation level of the paralyzed muscle is altered with the EMG activity induced by imposed passive leg movement. Our primary observations are the following: (i) during passive movement, all SCI patients showed EMG activity in the gastrocnemius muscle, whereas none of the normal subjects showed such activity; (ii) during the exercise period, an increase in oxy-Hb and a decrease in deoxy-Hb, both of which were independent of changes in total Hb, were observed in the SCI group; and (iii) in the recovery stage, total Hb exceeded the preexercise value in the SCI group. A possible mechanism for these changes in oxygenation level in the SCI patients and its implications for rehabilitation are discussed below.

Muscle activity during passive movement. Despite the motor paralysis in their lower legs, all six SCI subjects showed EMG activity in the paralyzed gastrocnemius muscle during passive movement. On the other hand, no normal subjects showed any EMG activity in the gastrocnemius muscle, even though the applied leg movements were identical to those applied to the SCI patients. In our previous data, it was found that the passive leg movement can also induce EMG activity in other lower leg muscles, for instance, the soleus and biceps femoris muscles (18). It is possible that the observed muscle activity consisted of complex spinal reflexes rather than simple stretch reflex responses induced by rhythmical stretching of the muscle tendon (14,18), and that the lack of EMG activity in normal subjects can be partly explained by the inhibitory neural input from a higher center to the spinal motor neurons (8). We do not discuss any further details of the neural mechanism of this EMG activity here because this article is con-

A SCI



B Normal

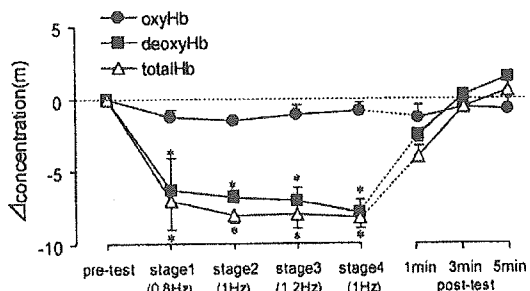


FIGURE 4—Concentration changes in total, oxygenated hemoglobin (oxyHb), and deoxygenated hemoglobin (deoxyHb) throughout the experiment for patients with spinal cord injury (SCI) (A) and normal subjects (B). The error bars indicate the SEM value. * Significant difference ($P < 0.05$) compared with the resting value. # Significant difference to the first set value.

cerned primarily with the relationship between the magnitude of muscular activity and the degree of the Hb value. The neural mechanism underlying this EMG activity has been described in detail in the previous research (for a review, see Harkema (14)).

The degree of changes in the NIRS signals should strongly depend on the muscle contraction level. It is therefore important to know how much the muscle activity occurs during passive leg movement. However, it is difficult to evaluate the muscle contraction level using the percentage of the maximal voluntary contraction (%MVC), which is commonly used to normalize and evaluate the muscle contraction level because SCI patients cannot accomplish voluntary contraction. When the EMG activity of the paralyzed

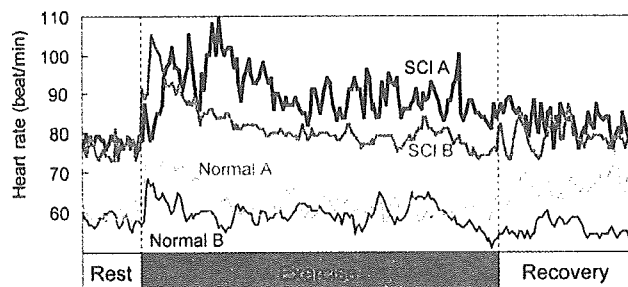


FIGURE 5—HR changes at rest, during passive leg movement, and in the recovery period obtained by two patients with spinal cord injury (SCI) and two normal subjects.

muscle is expressed with size relative to the MVC obtained by normal subjects (average: $417.7 \pm 43.24 \mu V$), it corresponds with approximately 10% MVC. Given the muscle atrophy of the paralyzed muscle (7,19), it can be assumed that the SCI patients have an MVC lower than that in the normal subjects. Therefore, we estimate the contraction level observed in the SCI patients as no less than 10% MVC.

Changes in Hb concentration during exercise. In the present study, both the SCI and normal groups showed a rapid decrease in the total Hb concentration following the onset of exercise and maintained the lower value while the legs were passively moved. We considered venous blood in the calf to be complete, that is, to have reached plateau level, at the beginning of the passive leg movement because subjects were kept in standing posture until the total Hb value stabilized. By imposing passive leg movement, the pooling venous blood might be expelled from the calf because intramuscular pressure is increased due to the imposed length changes in the muscle (28,30), irrespective of the appearance of EMG activity. Therefore, it seems reasonable to assume that the decreased total Hb observed in this study during movement is explained by this expulsion of the pooling venous blood in the calf.

In addition, the degree of the concentration changes in the total Hb was much larger in the normal group than in the SCI group. This result is consistent with the report of van Beekvelt et al. (36) that muscle pump activity induced by imposing electrical stimulation is reduced in SCI subjects compared with healthy subjects. It has been suggested that this reduced muscle pump activity might be explained by the muscle atrophy and low venous capacity found in SCI subjects (17). A possible explanation for this result is that SCI patients have more fat because larger amounts of fat result in lower NIRS signals (37). However, as described later, our results which the oxy- and deoxy-Hb showed in the opposite concentration changes would not be expected from fat for the same reason.

In the present study, a gradual increase in oxy-Hb and decrease in deoxy-Hb that were independent of changes in total Hb were observed in the SCI subjects during the exercise period. On the contrary, there is no obvious concentration change of the oxy-Hb in the normal subjects who showed no EMG activity during passive leg movement. If no muscle oxygen consumption and/or supply was induced by the imposed movement, both the oxy-Hb and the deoxy-Hb should vary in a manner related to the concentration changes in the total Hb. Taken together with the occurrence of EMG activity in the SCI group, this change in the muscle oxygenation level can be attributed to the muscle activity produced by imposing passive leg movements. These results are in good agreement with a recent report by Bhambhani et al. (3), who suggest that changes in the oxygenation level in the paralyzed rectus femoris muscle during cycling movement are generated by functional electrical stimulation.

With respect to muscle oxygenation during exercise, previous studies have reported that continuous muscle contraction at moderate intensity follows the increments of deoxy-Hb because of the oxygen consumption in the acting

muscle (6,34). Although we hypothesized that muscle was "active" during passive leg movement, the present results did not show increments of the deoxy-Hb. According to the general principle, concentration changes in oxy- and deoxy-Hb are dependent on the dynamics of the equilibrium between tissue oxygen demand and supply (2,16). Therefore, a possible reason for our result is that oxygen delivery far exceeds the oxygen extraction in the acting muscle. The enhancement of HR during passive leg motion (Fig. 5) provides evidence to support this notion.

Changes in Hb concentration after exercise. After the cessation of the passive leg movement, the concentration of total Hb in the SCI group exceeded the preexercise level, whereas that in the normal group simply recovered to the preexercise level. Because the total Hb reflects the degree of muscle blood flow (6), these changes may suggest that enhancement of the muscle blood flow occurred in the SCI group, possibly resulting from the muscle contraction and oxygenation during the exercise period. It is likely that the excess total Hb following exercise resulted from the pooling of blood in the calf. Nevertheless, in this study, the subjects were kept in a standing posture on the apparatus before the initiation of the exercise period until the total Hb value reached a constant level; therefore, the above total Hb changes during the recovery stage cannot be explained solely by blood pooling in the calf. These total Hb changes may be due to postexercise hyperemia (35).

HR changes by imposing passive leg movement. As shown in the Figure 5, the HR increased after the onset of the passive leg movement in both SCI and normal subjects. These results provide evidence of the enhancement of central circulation by imposing passive leg motion even in the SCI patients. The simplest explanation is that increments of the venous return due to the muscle pump activity result in the central circulation (29). However, taken together with the results of differences in the EMG activity, there would be different mechanisms underlying the enhanced HR between two groups. In the case of the normal subjects, it is plausible that the enhancement of the HR is induced by the neuronal factor, which is an afferent neural signal from the mechanoreceptor by inducing muscle stretching (12). On the other hand, this neuronal factor is not a suitable explanation for SCI results because of the sensory paralysis. Rather, our results, the appearance of muscular activity and an alteration of the NIRS signals, imply that a metabolic change accompanied by muscle contraction seems to play a primary role in the enhancement of the central circulation. Because we do not still have any direct evidence, further investigations are needed to clarify this point.

Implications for rehabilitation. As mentioned at the beginning, chronic inactivity and hypocirculation of the paralyzed area are crucial factors in secondary impairment

in SCI subjects (25). The present results provide indirect evidence that passive leg movement performed in a standing posture could alter the oxygenation level of the paralyzed muscle and has the potential to facilitate circulation of the paralyzed area. Given that the muscle contraction level during normal walking is about 15% MVC (21), it is considered that the muscle contraction level observed in this study is adequate to facilitate neural activity and circulation of the paralyzed area.

On a practical level, the subjects in the present study did not move their upper limbs and trunk voluntarily, because our aim was to examine whether the oxygenation level of the paralyzed muscle was altered by imposing passive leg movement. In a nonexperimental situation, however, patients would commonly operate the device themselves by manipulating the lever with their upper limbs. It is possible that the additional voluntary upper limb movement could enhance circulation not only in the voluntarily acting area, but also in the paralyzed area.

Although muscular activity in the paralyzed area can also be induced by applying electrical stimulation, as is the case in functional electrical stimulation (FES) (1,23,31), there are essential differences between our method and the FES technique. Previous investigations pointed out that one of major disadvantages of the FES technique is that it is difficult to generate FES-induced continuous muscle contractions without fatigue (for a review, see Stein et al. (32)). The muscle fatigue can be attributed to the fact that the fatigable motor unit is preferentially recruited by imposing electrical stimulation, in that large motor nerves are more easily activated than smaller ones. In contrast, in the case of the passive leg movement produced in the present study, the motor units are presumably recruited according to the size principle (15), because the afferent input was offered from proprioceptors by imposing muscle stretch and body load. Furthermore, passive leg movement is simpler and more practical than FES, and has a lower risk of misuse. Therefore, this type of passive leg movement might be a useful and efficient method for rehabilitation following SCI.

CONCLUSION

The present results demonstrate that passive leg movement can induce not only muscular activity, but also alteration of the muscle oxygenation level in the paralyzed lower limb. There may be increased oxygen consumption, but this could not be ascertained from the measurements in this study. Further study will be needed to clarify this issue.

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Original Article

Cardiorespiratory responses during passive walking-like exercise in quadriplegics

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Study design: Cross-sectional and comparative investigation using quadriplegics (QP) and nondisabled subjects (ND).

Objective: To evaluate cardiorespiratory responses during passive walking-like exercise (PWE) in QP.

Setting: National Rehabilitation Center for Persons with Disabilities in Japan.

Method: The subjects were seven male QP with complete lesion (age: 27.0 ± 5.4 , injured level: C6–C7) and six male ND (age: 26.3 ± 4.5). Cardiorespiratory responses were measured until voluntary fatigue during PWE, the rhythmical activity of paralyzed lower limbs synchronized with arm movements.

Results: There were no significant differences in oxygen consumption ($\dot{V}O_2$), pulmonary ventilation ($\dot{V}E$), heart rate (HR) and oxygen pulse (O_2 pulse) between QP and ND during PWE. ND showed increased ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$ ratio) during exercise, while QP showed a significantly greater respiratory rate (RR) during exercise than ND ($P < 0.05$).

Conclusion: PWE elicited an increase in $\dot{V}O_2$ with workload increment in QP similar to ND. However, higher RR suggested the intrinsic dysfunction of RR control during submaximal exercise in QP. From these results, it was thought that respiratory response would be the restriction factor of efficient oxygen transportation during PWE in QP.

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Keywords: quadriplegics; passive walking-like exercise; cardiorespiratory response

Introduction

For individuals with spinal cord injury (SCI), it is difficult to improve their cardiorespiratory function and to activate the oxygen supply function by exercise training such as wheelchair or arm swinging exercise because of the characteristics of the obstacles.^{1,2} In general, the peak oxygen uptake (peak $\dot{V}O_2$) is dependent on the level of spinal cord injury (SCI), and quadriplegics (QP) and high lesion paraplegics (PP) show a lower peak $\dot{V}O_2$ than nondisabled (ND) or low lesion PP.^{3–5} In addition, during submaximal exercise, QP and high lesion PP showed the reduced ventilation efficiency, stroke volume, venous return and sympathetic activity in comparison to ND or low lesion PP.^{5,6}

For such SCI with low physiological responses to exercise, the exercise posture is an important factor enhancing the effect of aerobic training. McLean *et al*⁷ investigated the influence of body posture in training on aerobic capacity in SCI and indicated that although improvements in aerobic capacity could be achieved by training in either a supine or a sitting posture, the supine posture had more effect on aerobic training than the sitting posture. One reason is that the supine posture is advantageous to SCI circulation, because there is small amount of blood in the paralyzed leg compared to the sitting posture due to the lower effect of gravity.

In contrast, in a sitting posture during exercise, venous blood pools in the legs and abdomen, causing reduced filling pressure and diminished ventricular volume. Therefore, when QP and PP perform exercise in a sitting posture, blood is not efficiently redistributed

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to the working muscles.⁸⁻¹¹ To improve venous blood pooling during exercise in a sitting posture, some researchers have investigated the effect of passive leg exercise on circulation.^{9,12} This exercise passively moves the paralyzed lower limbs, the blood stored in lower limbs returns to the heart, and stroke volume increases conjointly with the law of Frank-Starling.^{9,12}

It is reported that functional electrical stimulation (FES) is also a useful method to decrease venous blood pooling in the legs, because FES increases the activity of muscle pumping and vasoconstriction in the legs.¹³⁻¹⁵ Bhamhani *et al*¹⁶ measured the deoxygenation of the quadriceps muscle during FES using QP and PP. They indicated that the muscle deoxygenation of paralyzed muscles occurred more quickly with metabolic responses in the paralyzed muscles. Mutton *et al*¹⁷ reported a significant increase in peak $\dot{V}O_2$ during hybrid exercise¹⁸⁻²⁰ that combined upper arm exercise with FES in QP and PP when compared with only FES. Moreover, Raymond *et al*¹⁸ showed a higher oxygen intake and lower heart rate (HR) during hybrid exercise including arm-swinging exercise at a workload of 65% maximal oxygen uptake in PP. In addition, Hooker *et al*²¹ compared the respiratory and circulatory responses during hybrid exercise with those during submaximal arm-swinging exercise and FES leg-cycle exercise in QP. They showed higher pulmonary ventilation (VE), oxygen uptake ($\dot{V}O_2$) and carbon dioxide elimination during hybrid exercise than the other two exercises, but a higher stroke volume than only arm-swinging exercise. They concluded that hybrid exercise using whole-body exercise, including the paralyzed muscles, is effective in improving the cardiorespiratory function of ISCI.

More recently, it has been reported that standing gait exercise with orthoses has a good influence on cardiorespiratory function in ISCI.²² Faghri *et al*^{13,23} investigated the physiological reaction of a standing posture on ISCI with and without FES. They showed stable cardiac output, stroke volume and total peripheral resistance (TPR) during 30 min standing with FES in both QP and PP. In contrast, during passive standing without FES, QP demonstrated significantly higher TPR and

significantly lower systolic blood pressure and mean arterial pressure than PP. Faghri *et al*^{13,23} also indicated that standing without FES was disadvantageous to the regulation of hemodynamics during posture change in QP.

When ISCI passively walked on a treadmill using body weight support equipment, they showed a similar electromyographic pattern in the paralyzed muscles to ND.²⁴ Furthermore, Colombo *et al*²⁵ obtained the same result during passive stepping using driven gait orthosis for C3 (incomplete) and C5 (complete). It is naturally expected that passive walking with arm exercise increases energy expenditure and oxygen supply to the arm is elevated. However, as far as we know, there are no studies investigating the cardiorespiratory responses of QP during passive walking-like exercise (PWE) when standing.

The purpose of this study, therefore, was to clarify respiratory and circulatory responses during PWE by a stepwise incremental method and to compare the results of QP with those of ND.

Methods

Subjects

Seven male patients with complete chronic QP and six ND male subjects volunteered to participate in this study. Table 1 lists their physical characteristics. The lesion in SCI was located between C6 and C7. All subjects regularly performed wheelchair sports, such as twin basketball, quad rugby and distance running, for more than 60 min a day and more than twice a week. No subject had a history of cardiovascular, metabolic, or pulmonary disease. Informed consent was obtained from all subjects before their participation in this study. The subject refrained from food, caffeine and nicotine for at least 3 h before testing. The study was approved by the Ethical Research Committee in the National Rehabilitation Center for Persons with Disabilities.

Testing protocols

All subjects performed an incremental exercise test on an Easy Stand Glider 6000 (Altimate Medical Int.,

Table 1 Characteristics of the subjects

No	Sex	Height (cm)	Weight (kg)	Age (years)	Level of injury	Zancolli	ASIA	Times of injury (month)	Sports
a	M	163.0	57.3	34	C6	2B3	A	153	Twin basketball
b	M	168.0	55.7	23	C6	2B1	A	32	Twin basketball
c	M	174.0	55.1	29	C6	2B2	A	27	Twin basketball
d	M	166.0	52.3	29	C6	2B1	A	81	Distance running
e	M	160.0	41.3	20	C7	3A	A	25	Twin basketball
f	M	177.0	69.8	22	C7	3A	B	54	Quad rugby
g	M	172.0	66.6	32	C6	2B1	A	107	Quad rugby
h	M	168.0	59.7	25	ND				Track and field
i	M	180.0	62.2	26	ND				Soccer
j	M	172.0	62.5	35	ND				Track and field
k	M	175.0	68.4	23	ND				Track and field
l	M	168.0	65.0	26	ND				Baseball
m	M	170.0	54.0	23	ND				Skiing

Morton, MN, USA). The Easy Stand Glider 6000 is designed to strengthen both the upper and lower extremities while standing. It has a safety belt around the waist, a chest pad, hip guide and knee support. When the subject swings his arms back and forth, his legs simultaneously move passively just like walking. Tests were performed with the push and pull handle horizontal to the level of the shoulder joint and elbows slightly flexed at the point of maximal arm extension (Figure 1).

The subjects remained seated in a wheelchair or chair for at least 30 min. Baseline physiological measurements were recorded for the last 5 min during seating. They were subsequently guided with a metronome for reciprocal movement of the arms and legs while standing. Exercise commenced by swinging the arms back and forth at 20 times/min for 2 min. The swings were then increased to 10 times/min every 2 min until 50 times/min, and then 5 times/min every 2 min until exhaustion. The incremental exercise test was terminated when voluntary fatigue was attained. Voluntary fatigue was defined as the point at which the subject could no longer keep pace and his RPE was over 15. The experiment was carried out in a room with ambient temperature and relative humidity maintained at 22–25°C and 30–50%, respectively.

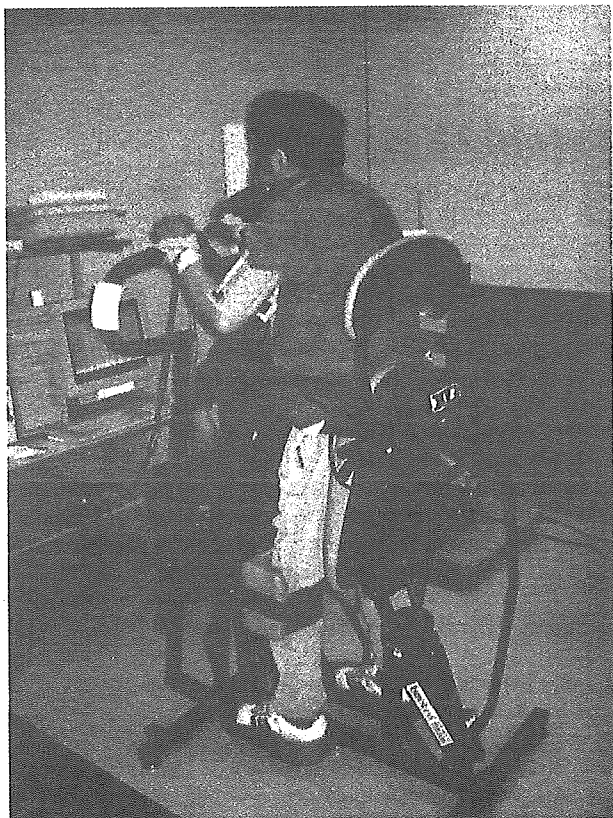


Figure 1 The arm swinging and passive walking machine. When a quadriplegic subject swings his arms back and forth, the paralyzed legs simultaneously move as if walking

Cardiorespiratory measurements

Cardiorespiratory measurements were continuously monitored during the test using the gas analyzer of the metabolic system (Model AT-3000, Anima, Tokyo, Japan). The gas analyzer was calibrated using standard gas concentrations (16.1% oxygen, 5.01% carbon dioxide). The volume transducer was calibrated using a syringe calibrated to 2 l. The gas analyzer was programmed to present the following results: absolute $\dot{V}O_2$ (l/min), relative $\dot{V}O_2$ (ml/kg/min), respiratory rate (RR, times/min) and $\dot{V}E$ (l/min). The following variables were calculated from the oxygen pulse (O_2 pulse, ml/beat) as the ratio between absolute $\dot{V}O_2$ and HR (beats/min), and the ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$ ratio, l/ml) as the ratio between $\dot{V}E$ and absolute $\dot{V}O_2$.

HR was recorded during the last 10 s at each work stage using a wireless monitor (Life Scope 8/Two, Nihon Koden, Tokyo, Japan). Blood was sampled from the earlobe during rest and exercise and blood lactate accumulation (LA, mmol/ml) was measured using a simplified blood lactate test meter (Lactate Pro™ LT-1710, Arckly, Inc., Kyoto, Japan). Blood sampling was conducted immediately after rest and within 30 s after each workload. The sampling time was within 20 s.

Statistical analysis

All variables were expressed as the mean \pm SD. Two-way repeated analysis of variance was used to compare the difference between groups. *P*-values <0.05 were considered significant.

Results

Figure 2a shows the relationship between $\dot{V}O_2$ and workload in QP and ND. $\dot{V}O_2$ within the workload of 60 times/min varied little and almost no rise was observed. At a workload of 65 times/min, $\dot{V}O_2$ started to increase rapidly. At any workload, there was no significant difference between the groups. Changes in $\dot{V}E$ and LA over time during exercise were similar to those in $\dot{V}O_2$. No significant differences were found in $\dot{V}E$ and LA between QP and ND.

HR increased with the workload increment in both groups (Figure 2b). When the workloads were between 40 and 70 times/min, there was little increase in HR for QP and ND. Although QP showed a higher HR than ND during rest and exercise, significant difference was only found at a workload of 30 times/min. Figure 2c indicates the O_2 pulse over the time course of exercise. In ND, the O_2 pulse remained unchanged from the beginning of exercise to a workload of 60 times/min. Subsequently, the O_2 pulse of QP increased rapidly. In contrast, QP showed a gradual increase in the O_2 pulse linearly with the workload. The O_2 pulse of QP was higher than that of ND at any workload and significant difference was only found at the beginning of exercise (20 times/min).

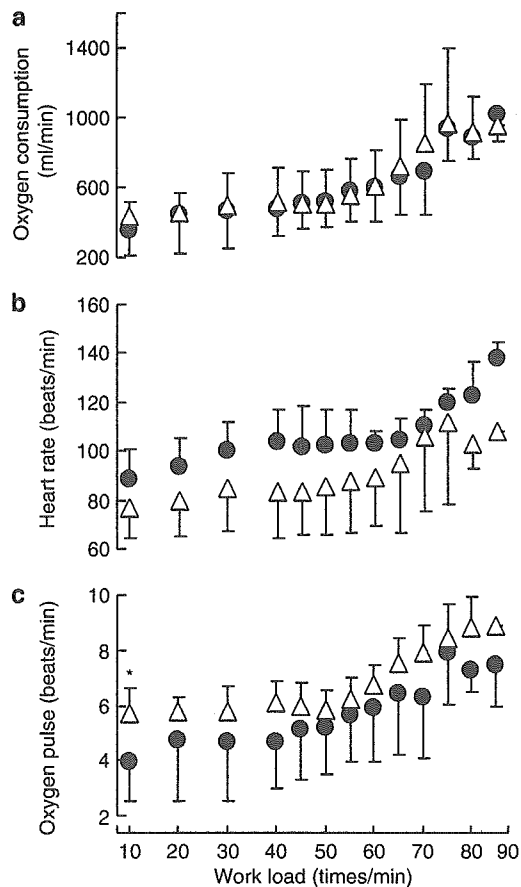


Figure 2 Change in oxygen consumption (a), heart rate (b), and oxygen pulse (c) during passive walking with arm swinging exercise in persons with Quadriplegics (●) and nondisabled (Δ). Although there was no difference of oxygen consumption between QP and ND, oxygen transportation of QP was inferior to ND during the exercise. * $P < 0.05$: compared with nondisabled subjects

Figure 4a illustrates the relationship of HR to $\dot{V}O_2$ in QP and ND. With increasing $\dot{V}O_2$, HR linearly and significantly increased in both groups. When $\dot{V}O_2$ was around 500 ml, HR of QP was apparently greater than ND.

There existed a great difference in RR between QP and ND during exercise (Figure 3a). ND had almost unchanged RR during exercise. In contrast to ND, QP showed increased RR over the time course of exercise. There were significant differences in RR between the groups at any workload except the lowest (20 times/min) and the highest (95 times/min). Figure 3b shows the $\dot{V}E/\dot{V}O_2$ ratio during the incremental exercise test in QP and ND. The $\dot{V}E/\dot{V}O_2$ ratio of QP was higher than that of ND at any workload and significant differences were found at higher workloads of 75 and 85 times/min.

The relationship of RR to $\dot{V}E$ is illustrated in Figure 4b with regression lines. The regression line of QP shifted to the upper side of ND, indicating that QP required more RR to achieve the same $\dot{V}E$ as ND.

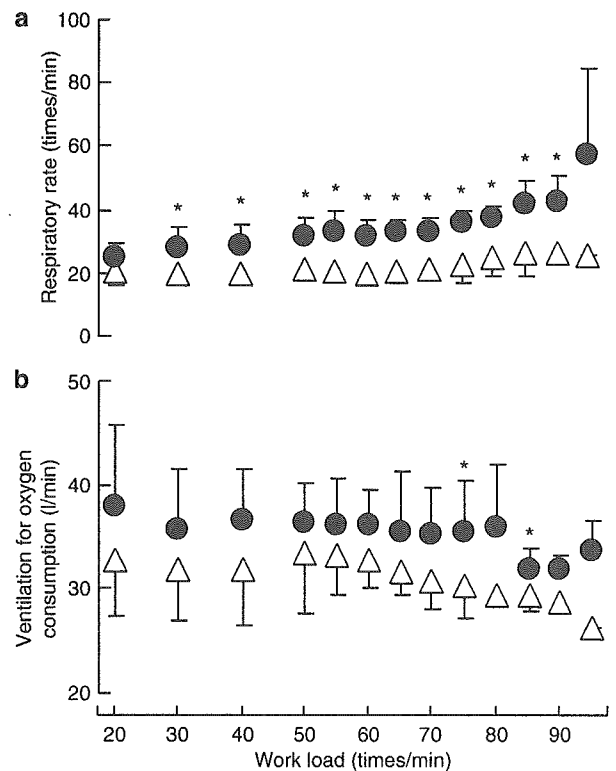


Figure 3 Change in respiratory rate (a) and ventilation for oxygen consumption (b) during passive walking with arm swinging exercise in persons with Quadriplegics (●) and nondisabled (Δ). Ventilation efficiency showed significant decrease in QP as compared with ND. * $P < 0.05$, # $P < 0.01$: compared with nondisabled subjects

Discussion

In this study, there were no significant differences in $\dot{V}O_2$ and $\dot{V}E$ during standing exercise between QP and ND (Figure 2a). Some investigators showed that the cardiorespiratory responses of QP during arm exercise were relatively lower than ND and PP,^{4,5,26} suggesting that the cardiorespiratory responses of ISCI are largely dependent on the level of SCI.^{3,6,27} These results were obtained from arm-swinging exercise or wheelchair ergometer exercise requiring mainly upper limb activity in a sitting posture. It may be considered that the exercise while sitting, using only the upper limbs, influences the $\dot{V}O_2$ of ISCI. Hopman *et al*²⁸ demonstrated that peak $\dot{V}O_2$ significantly increased in a supine posture during maximal arm-swinging exercise in comparison with a sitting posture. In addition, McLean *et al*⁷ compared the power output (PO) of QP during an intermittent progressive peak exercise test in a sitting posture with that in a supine posture. As a result, they reported higher PO in a sitting than supine posture. These investigations suggest that cardiorespiratory responses during exercise are affected by exercise postures in QP.

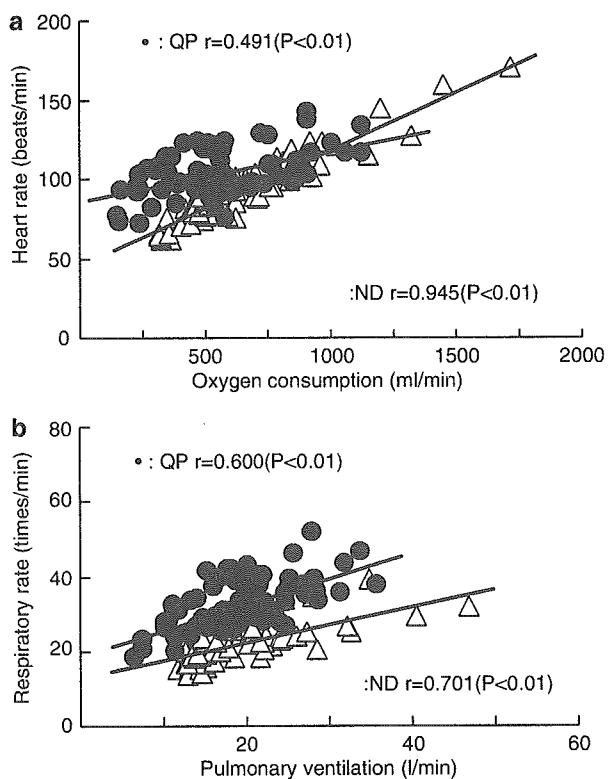


Figure 4 Relationship between oxygen consumption and heart rate (a), pulmonary ventilation and respiratory rate (b) during passive walking in persons with Quadriplegics (●) and nondisabled (Δ). QP showed inactive to HR and remarkable increase in RR as compared with ND

Cardiorespiratory responses during exercise in QP change with passive exercise by the paralyzed limbs in addition to the exercise posture. Pitetti *et al*²⁹ carried out arm-swinging exercise in ISCI and ND with lower body positive pressure (LBPP). They found a significant increase in $\dot{V}O_2$, $\dot{V}E$ and work rate during arm-swinging exercise with LBPP compared to without LBPP. Furthermore, there were no differences in $\dot{V}O_2$, $\dot{V}E$ and work rate between ISCI and ND during exercise with LBPP. From these results, Pitetti *et al*²⁹ suggested that for ISCI, LBPP augmented the exercise capacity by preventing the redistribution of blood to the lower extremities. Hopman *et al*^{27,28} investigated the effects of exercise posture, wearing an antigravity suit (anti-G suit), elastic stocking and abdominal binder, and FES on blood redistribution and circulatory responses in QP and PP. They demonstrated that $\dot{V}O_2$ and HR decreased by wearing an anti-G suit and increased by FES, and increased by wearing elastic stockings and FES during submaximal exercise. In contrast, during maximal exercise, only FES increased $\dot{V}O_2$ and HR. From these results, Hopman *et al*^{27,28} suggested that these methods of circulatory redistribution have different working mechanisms and the effects are dependent on the SCI level probably because of differences in active muscle

mass, sympathetic impairment and blood pressure values. Furthermore, $\dot{V}O_2$ increased significantly during FES exercise of lower limbs in comparison with at rest³⁰ and $\dot{V}O_2$ during hybrid exercise was higher than that during arm-swinging exercise or leg cycle exercise by FES.^{17,21,31}

The findings using the passive activity of paralyzed lower limbs and FES in addition to arm exercise clearly demonstrated good effects on cardiorespiratory responses and improving the efficiency of their oxygen utilization in ISCI. However, these studies were mostly performed in a sitting posture. If ISCI perform exercise in a standing posture, the cardiorespiratory responses may be different from those in a sitting posture. Nash *et al*³² showed significant increases in $\dot{V}O_2$, $\dot{V}E$ and HR during PWE when standing by using robotic-assisted locomotion in QP with a lesion level of C3–C4. In addition, Dietz *et al*²⁴ identified electromyographic activities of the musculus tibialis anterior and musculus soleus during passive walking on a treadmill in QP and PP. These investigations suggested that PWE in a standing posture with arm exercise in QP facilitated cardiorespiratory responses. Our study showed the same $\dot{V}O_2$ between QP and ND, indicating that the rhythmic activity of paralyzed limb increased $\dot{V}O_2$.

In the present study, QP showed a significantly higher RR from 30 to 90 times/min (Figure 3b), and QP increased RR to the equivalent $\dot{V}E$ of the ND (Figure 4b). Coutts *et al*⁶ found lower respiratory parameters such as $\dot{V}E$ and ventilation equivalent in QP than in PP during submaximal arm-swinging exercise. The ventilation equivalent is generally considered to be a measure of breathing efficiency, and decreases during submaximal exercise are associated with increased tidal volume and relative decreases in dead space ventilation.⁶ Bhambhani *et al*¹⁶ demonstrated that the $\dot{V}E/\dot{V}O_2$ ratio in ISCI, including QP, is lower during FES cycle exercise than ND, indicating that the ventilatory efficiency of ISCI is inferior to that of ND. In good agreement with the data of Bhambhani *et al*,¹⁶ we found a lower $\dot{V}E/\dot{V}O_2$ ratio of QP in comparison with ND. There was no difference in LA between QP (2.9 ± 1.1 mmol/l) and ND (3.0 ± 1.6 mmol/l) during peak exercise, indicating that the increase in RR was not related to metabolic factors, because LA stimulates the respiratory center and consequently increases the elimination of carbon dioxide. Furthermore, it has been reported that expiratory muscle contraction is influenced by sympathetic nerve activity more than muscle metaboreflex.³³

In AB, the impulse from the motor area of the cerebral cortex via the center of breathing adjusts the ventilation equivalent to the exercise intensity during exercise.³⁴ In addition, it has been demonstrated that respiration is regulated by transmitting the afferent information from activity muscles to the center.³⁵ In QP, the afferent information from the agonist of the upper limbs was transmitted to the center during exercise; however, the nerve impulse of the ventilatory regulation corresponding to the workload is not sent to the

respiratory muscles. That is, it is believed that the afferent information to the center increased excessively in our tests. Restrictive ventilatory impairment may disturb respiratory regulation during exercise in QP.

Green³⁶ found that excitations of the stretch receptors stimulated by the stretch reflex (the Hering–Breuer reflex) in the lung were relayed via the vagus nerve to the medullary respiratory center, leading to a reflex decrease in tidal volume. This reflex was not found in normal adults and in babies with undeveloped respiratory-related muscles and in some animals.³⁶

We hypothesized that QP with restrictive ventilatory impairment might show a condition similar to that of the babies in Green's study. To compensate for the decrease in tidal volume by the Hering–Breuer reflex and to achieve the same ventilation as ND, QP increased RR from the commencement of exercise. Specifically, the increase in RR in QP could be a result of the increased afferent information from the agonist to the respiratory center and the increased reflex induced by restrictive ventilatory impairment.

Some investigators have shown a lower maximal HR below 110 beats/min in QP than that of PP and ND.^{4,37} Bhambhani *et al*¹⁶ found no significant difference of HR in ISCI including QP during FES exercise from that at rest. A lower HR during exercise is naturally expected in QP because of sympathetic activity dysfunction controlling the heart. In this study, however, during peak exercise, the HR of QP was higher than the data of Bhambhani *et al*,¹⁶ and there were no significant differences of HR between QP and ND similar to $\dot{V}O_2$ (Figure 2b). Muraki *et al*¹² reported a significant increase in stroke volume and cardiac output without a rise of HR during passive leg cycle exercise in PP. They suggested the promotion of venous return related to the lengthening and shortening of the paralyzed muscle without tension in the lower limbs.

In this study, HR in QP increased during PWE. This is not consistent with the findings of Faghri *et al*,²³ who found no increase of HR in QP during FES when standing. On the other hand, they reported increased TPR, which could be a compensatory mechanism to control the significant drop in blood pressure occurring during standing in QP. Hooker *et al*²¹ investigated cardiorespiratory responses during arm-swinging exercise, FES leg-cycle exercise and hybrid exercise in QP, and revealed that $\dot{V}O_2$, $\dot{V}E$ and HR were higher during hybrid exercise than the other two exercises and there was no significant difference in stroke volume between the hybrid exercise and FES leg-cycle exercise. Dela *et al*¹⁴ reported that although HR increased immediately after the commencement of FES and attained a steady state in ND, QP showed a delay in the HR increment. HR responses in QP may be attributable to arterial baroreceptors that elevate HR in QP with the lower blood pressure developed during exercise.¹⁴

In this study, there was no significant difference in HR between QP and ND during peak passive walking. On the other hand, during submaximal exercise, a clear difference in HR was found between QP and ND. In

ND, HR increased linearly with workload increment, while it increased in QP from the commencement of exercise to 40 times/min and HR increased gradually, showed a steady state between 50 and 75 times/min, increasing remarkably after 80 times/min (Figure 2b). It could be expected that HR in QP increased by the activation of arterial baroreceptors in compensation for deficient blood distribution from the beginning of exercise to 40 times/min, while between 50 and 75 times/min, HR showed a steady state because blood was distributed sufficiently to agonists (Figure 2c).

In conclusion, PWE, the rhythmical activity of paralyzed lower limbs synchronized with arm movements, elicited an increase in $\dot{V}O_2$ in QP similar to ND. However, higher RR suggested the intrinsic dysfunction of RR control during submaximal exercise in QP. From these results, it was thought that respiratory responses would restrict the efficiency of oxygen transportation during PWE in QP.

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Original Article

Effect of lesion level on the orthotic gait performance in individuals with complete paraplegia

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Study design: Cross-sectional, experimental research.

Objectives: To clarify the effect of lesion level on cardio-respiratory responses and biomechanical characteristics of walking with a reciprocating gait orthosis in complete paraplegia with spinal cord injury (SCI).

Setting: National Rehabilitation Center for Persons with Disabilities, Japan.

Methods: Ten SCI individuals (age: 20–34 years, injured level: Th5–12) who experienced orthotic gait training at least for 10 weeks participated in two experiments: (1) measurement of the cardiorespiratory responses during 20 min of orthotic gait exercise; and (2) three-dimensional motion analysis and ground reaction force measurement using the VICON system. We calculated the following parameters: pulmonary ventilation, oxygen consumption ($\dot{V}O_2$), heart rate (HR), gait speed, cadence, stride length, crutch force (CF), hip range of motion (ROM), and hip angular velocity (VEL). Further, energy consumption and energy cost were calculated using the steady-state value of $\dot{V}O_2$ and gait speed.

Results: The steady-state value of the $\dot{V}O_2$ (18.2 ± 3.80 ml/kg) and HR (133.0 ± 21.63 b/min) tended to be larger in higher thoracic SCI subjects. There were strong positive correlations between the lesion level and walking speed ($r=0.74$), energy cost ($r=0.85$), and hip ROM ($r=0.78$). On the other hand, negative correlation between the lesion level and peak CF ($r=-0.78$) was clarified.

Conclusions: The physiological intensity of the orthotic gait strongly depended on the level of lesion. It seems likely that a limited hip range of motion and excess upper limb load result in the low energy cost of orthotic gait for the higher thoracic level of paraplegic patients.

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Keywords: spinal cord injury; orthotic gait; energy consumption; motion analysis; cardio-respiratory response

Introduction

Orthotic gait exercise is usually prescribed for patients with spinal cord injury (SCI) in their therapeutic phase to promote their general health. Although the effectiveness of orthotic gait exercise is well recognized, there are several obstacles to achieve walking for complete paraplegic persons, in particular the high energy cost of the orthotic gait.^{1–5} SCI persons inevitably require larger energy expenditure for orthotic gait because they need to produce complementary upper limb and trunk motion in order to swing their paralyzed lower limb.^{6–9} Further, it can be pointed out that the neurological level

of paralysis considerably influences the achievement of orthotic gait motion and energy expenditure.

We have evaluated the energy expenditure during orthotic gait of thoracic level of SCI paraplegics, and suggest that, even with the higher level of lesion, the physiological intensity required was in a feasible range for cardiorespiratory function.¹⁰ We have also found that subjects who had higher levels of lesion demonstrated relatively slower gait speed and a relatively higher physiological intensity. These findings confirm the clinical impression that higher thoracic SCI subjects have some difficulties in performing orthotic walking. Although physicians and therapists already know this because of their clinical experience, it is not clear to what extent the motor paralysis influences orthotic gait performance, and what is the primary reason for limited

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gait performance for higher level SCI subjects. In the present study, we aimed to clarify the effect of injured level on the physiological intensity and biomechanical characteristics of orthotic gait on the complete paraplegic persons.

Methods

Subjects

Ten subjects with the thoracic level of SCI participated in this study. All subjects had complete motor paralysis in the lower limb muscles (ASIA classification; grade A or B¹¹) and no history of cardiorespiratory disease. Criteria for participation of this study were (1) judged to be better general health condition and have adequate exercise tolerance at the health check, (2) no cardiovascular disease, and (3) had past at least a half year after injury. The characteristics of the subjects in detail are summarized in Table 1. All subjects had participated in the basic rehabilitation process, and had undergone at least 10 weeks of orthotic gait training using the advanced reciprocating gait orthosis[®] (ARGO). Each

subject gave written informed consent for the experimental procedure, which was approved by the local biological ethics committee of the National Rehabilitation Center for Persons with Disabilities (NRCD).

Orthotic gait

Sequential pictures of walking with the ARGO are shown in Figure 1a. The ARGO has a single cable which connects both sides of the leg frame. With this device, a torque exerted by the right (left) hip joint is mechanically transmitted to the left (right) hip joint, resulting in the torque to the opposite direction exerted by the left (right) hip joint. Although there is individual variation, in many cases, paraplegic patients with injury to a lower thoracic level could walk after 10 weeks of gait training independently, while patients with injury at a higher thoracic level needed additional practice. After the training period, each subject could perform the orthotic gait (subjects F, H, and J still required light support to avoid falling) independently, and were able to walk continuously for at least 20 min.

All subjects participated in two experiments on the separate day; one was the measurement of cardiorespiratory responses during 20 min of orthotic gait exercise, and another was the three-dimensional motion analysis with the use of the VICON system.

Table 1 Characteristics of the SCI subjects

	Sex	Age (years)	Weight (kg)	Lesion level	Grade of ASIA	Duration of paraplegia (months)
A	M	30	67	Th12	A	32
B	M	25	79	Th12	A	28
C	M	26	80	Th12	A	16
D	M	29	72	Th11	B	12
E	F	27	45	Th10	A	18
F	M	32	74	Th10	A	10
G	M	30	74	Th8	A	22
H	M	22	65	Th7	A	30
I	M	34	54	Th6	A	36
J	M	20	53	Th5	B	28

Measurement of the cardiorespiratory responses

Subjects were asked to abstain from alcohol and caffeine for at least 12 h before the experiment. The temperature and humidity during the experiment were $23.5 \pm 4.2^\circ\text{C}$ and $58.3 \pm 3.3\%$, respectively. The experimental procedure was as follows: 3 min of rest in the standing position followed by 20 min of continuous walking at the most comfortable speed. The cardiorespiratory responses at rest and during walking were measured continuously with a telemetric device (K4-RQ Cosmed

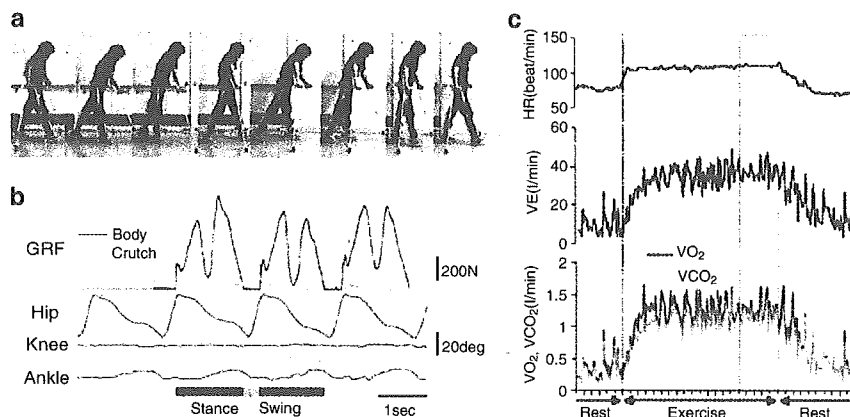


Figure 1 (a) Sequential picture of walking with the ARGO in a SCI subject (injured at Th12). (b) Time series data of the GRF (body and stick) and joint angle motion (Hip, Knee, and Ankle) obtained by the VICON system. (c) Typical example of the changes in the cardiorespiratory parameters during orthotic gait exercise

s.r.l., Rome, Italy) and were analyzed in real time. The telemetric device consisted of a transmitting unit, a facemask for sampling the expired gas, a heart rate (HR) chest strap, a battery, and a receiving unit. The following cardiorespiratory parameters were obtained: pulmonary ventilation (VE), oxygen uptake ($\dot{V}O_2$), and HR. Typical example of the changes in the cardiorespiratory parameters during orthotic gait exercise was shown in the Figure 1b. The amount of time required to walk 10 m was recorded during the exercise period, and gait speed was calculated after the experiment. After the experiments, the energy consumption and energy cost were calculated. The terms adopted were those of Nene and Patrick,¹² and calculations were performed according to their protocol:

$$\begin{aligned} & \text{Energy consumption (J/kg/s)} \\ &= \frac{\text{Ambulatory min } \dot{V}O_2 \text{ (ml/min)}}{\text{Weight (kg)} \times 60} \times K \end{aligned}$$

$$\begin{aligned} & \text{Energy cost (J/kg/m)} \\ &= \frac{\text{Ambulatory min } \dot{V}O_2 \text{ (ml/min)}}{\text{Speed (m/min)} \times 60} \times K \end{aligned}$$

where $K = 20.19 \text{ J/ml}$, since $1 \text{ ml } O_2 = 4.825 \text{ cal}$ and $1 \text{ cal} = 4.184 \text{ J}$.

Motion analysis

Subjects performed orthotic walking along a 10-m walkway in the laboratory at least five times at a comfortable (self-determined) speed. In order to obtain the kinematics and kinetics variables of the orthotic gait, the gait motion was measured with a three-dimensional motion-analysis system (VICON 370, Vicon Motion Systems Ltd, Oxford, UK). The motion-analysis system consisted of a conventional video-analysis system with seven cameras and force plates (Kistler, Switzerland). The force plates, $160 \times 450 \text{ cm}$ in size, consisted of two $80 \times 200 \text{ cm}$ plates and four $40 \times 250 \text{ cm}$ plates. These separate force plates enabled us to measure ground reaction forces (GRF) under the feet and canes on both sides, separately. A total of 17 markers were attached to the orthosis and to the body of the subject on the skin overlying the following marks: the vertex, both sides of the acromium (SHO), the lateral aspects of the hip (HIP), knee (KNE), and ankle (AKL) joints of the orthosis, the top of the great toe (TOE), the protrusion of the ulna at the elbow and wrist joint, and the tip of the crutch. We defined the hip angle as the angle formed by the SHO, HIP, and KNE, and the ankle angle as that formed by the KNE, AKL, and TOE, respectively. Typical time series data of the GRF (body and stick) and joint angle motion (Hip, Knee, and Ankle) obtained by the VICON system was shown in the Figure 1c.

We sampled 10-step cycles for the analysis. The following kinematic and kinetic variables were evaluated on the basis of the motion analysis: cadence, stride length, hip joint range of motion (ROM), hip angular

velocity (VEL), and crutch force (CF) (peak crutch force (PCF) and mean crutch force (MCF)). Cadence was calculated as the time required between heel contacts detected by the body GRF. Stride length was calculated as the distance of the toe marker between two consecutive gait cycles.

Statistics

Values were given as means \pm SEM. Pearson's product moment correlation coefficient was used to examine the relationship between the level of lesion and the kinematic and kinetic variables. Moreover, since the injured level is not strictly regarded as a parametric variable, we also examined this relationship using the Spearman rank-correlation coefficient. For this analysis, the parameters used were the average value for each subject. Significance was accepted at $P < 0.01$ and $P < 0.05$.

Results

Figure 1a shows a sequential picture of walking with the ARGO, the typical waveform of the GRF of each body and of the opposite side of the crutch, joint motion (Hip, Knee, and Ankle) (Figure 1b), and the typical data of the cardiorespiratory responses (Figure 1c). As shown in this figure, dynamic hip joint motion and periodic load application appeared during orthotic gait. In the orthotic gait, in contrast with normal walking, the knee joint was held in an extended position throughout the locomotion cycle. Although the ankle was held by the plastic socket, angle changes were observed to some extent in the stance phase.

Gait speed

The average walking speeds calculated during field walking (during cardiorespiratory measurement) and laboratory walking (during motion analysis) were 19.8 ± 6.16 and $21.3 \pm 5.82 \text{ m/min}$, respectively. There was a strong relationship between these variables ($r = 0.87$, $P < 0.01$). Therefore, in this study, the former value was used as the gait speed. Eight of 10 subjects were able to walk continuously for 20 min without a long break. Subjects H and J, who had relatively higher levels of injury, required rest intervals due to arm fatigue and a pressure on the heels of hands.

Cardiorespiratory responses during orthotic gait

Table 2 shows the cardiorespiratory responses at rest and the steady-state value during orthotic walking. In the subjects H and J, the resting value of the HR was much higher than in other subjects. This is presumably caused by a disorder of the autonomic nervous functions. As clearly shown in Figure 1c, cardiorespiratory parameters rapidly increased after the beginning of the walking exercise. HR reached a plateau level in the first few minutes, whereas some subjects with a higher level of lesion (subjects I and H) showed further

increases of HR during prolongation of the exercise. The steady-state value of HR ranged from 99.2 to 166.4 b/min (average value: 130.0 ± 21.63 b/min). As with HR, $\dot{V}O_2$ reached a plateau level about 3–4 min after the beginning of exercise. The steady-state value of $\dot{V}O_2$ ranged from 14.91 to 24.83 ml/kg (18.17 ± 3.80 ml/kg), and this value is approximately 3–4 times the resting level.

The energy consumption and energy cost during walking were 6.11 ± 1.28 J/kg/s and 20.12 ± 7.35 J/kg/m, respectively (Table 3). Figure 2a shows the relationship between energy consumption (y-axis) and energy cost (x-axis) in each subject. As the walking speed was determined by dividing the energy consumption by the energy cost, the slope of the line from zero to each point plotted reflects the walking speed of each subject. It was found that the plots of persons with higher level injuries tended to shift to the right side, which signifies a relatively slower gait speed and a higher energy cost.

Table 2 VE, $\dot{V}O_2$, and HR at rest and during orthotic gait exercise

	VE (ml/kg)		$\dot{V}O_2$ (ml/kg)		HR (beat/min)	
	Rest	Exercise	Rest	Exercise	Rest	Exercise
A	194.0	437.3	6.70	16.01	60.1	99.2
B	171.5	525.1	4.48	17.63	70.4	114.3
C	139.2	574.2	4.29	14.91	87.6	140.2
D	172.9	664.9	4.66	15.62	92.1	129.5
E	185.8	624.2	6.78	24.20	62.1	132.5
F	175.3	592.9	6.62	15.41	78.3	131.5
G	155.0	634.4	4.43	16.75	77.9	110.1
H	221.8	560.6	5.80	15.19	107.8	163.0
I	277.9	660.5	9.75	24.83	81.0	143.7
J	265.1	687.7	8.29	21.14	144.1	166.4
Mean	195.8	596.2	6.18	18.17	86.13	133.0
SD	45.61	75.67	1.83	3.80	24.78	21.63

Table 3 All parameters calculated in this study

	Gait speed (m/min)	E consmp. (J/kg/s)	E cost (J/kg/m)	Cadence (step/min)	Stride length (cm)	Peak CF (N/kg)	Mean CF (N/kg/s)	Hip ROM (deg)	Hip fVEL (deg/s)	Hip eVEL (deg/s)
A	20.06	5.39	16.12	40.86	106.6	1.81	0.32	45.44	110.2	24.35
B	32.58	5.93	10.93	56.92	122.5	2.81	0.34	51.04	142.6	38.90
C	27.22	5.02	11.06	47.62	107.7	2.62	0.33	47.95	126.0	23.91
D	21.55	5.26	14.63	40.59	118.8	3.93	0.46	49.52	127.6	24.17
E	19.99	8.14	24.44	36.34	79.9	3.73	0.49	40.91	119.0	13.36
F	18.35	5.19	16.95	47.10	102.9	3.37	0.60	46.35	131.0	22.20
G	11.64	5.64	29.05	36.81	83.8	4.31	0.47	43.08	110.0	15.78
H	15.58	5.11	19.67	42.25	89.8	4.39	0.50	42.23	132.9	18.00
I	17.09	8.35	29.33	48.98	77.5	5.02	0.45	38.83	133.9	18.75
J	14.69	7.11	29.06	42.01	84.8	3.92	0.55	41.92	109.7	17.79
Mean	19.88	6.11	20.12	43.95	97.4	3.59	0.45	44.73	124.3	21.72
SD	6.16	1.28	7.35	6.27	16.36	0.96	0.09	3.98	11.56	7.12

In order to confirm the data reproducibility, we compared the energy consumption and energy cost evaluated at 2 and 3 months after the beginning of the training in six of 10 subjects (Figure 2b). Although these data include the training effect (improvement of the energy cost), data reproducibility can be well recognized from this figure.

Kinematics and kinetics

The parameters obtained from motion analysis are summarized in Table 3. Cadence and stride length were 44.0 ± 6.27 step/min and 97.4 ± 16.36 cm, respectively. Hip ROM was 44.1 ± 5.20 deg. The angular velocities of the flexion and extension phases were 124.3 ± 11.56 and 21.7 ± 7.12 deg/s, respectively. Figure 3 shows the stick picture (top) and ensemble averaged waveform of the hip angle and GRF for both body and crutch during orthotic gait obtained from subjects A (injured at Th12) and J (Th5). As clearly shown in this figure, there are remarkable differences in the gait motion between the SCI subjects injured at a lower and higher level. This figure also shows good reproducibility of both hip motion and GRF during orthotic gait.

Relationship between injury level and each parameter

Table 4 summarizes the Pearson's product moment correlation coefficient (r_p) and the Spearman rank-correlation coefficient (r_s) between the injured level and each parameter obtained in this study. Figure 4 also shows the correlation diagram between the level of injury and with gait speed, energy consumption, energy cost, hip ROM, and mean GRF of the body and crutch. The parameters that showed strong relevance to the injury level were gait speed ($r_p = 0.74$; $P < 0.01$, $r_s = 0.89$; $P < 0.01$), energy cost ($r_p = -0.88$; $P < 0.01$, $r_s = -0.92$; $P < 0.01$), stride length ($r_p = 0.79$; $P < 0.01$, $r_s = 0.78$; $P < 0.01$), peak CF ($r_p = -0.78$; $P < 0.01$, $r_s = 0.80$; $P < 0.01$), and hip ROM ($r_p = 0.78$; $P < 0.01$, $r_s = 0.77$; $P < 0.01$). By evaluating the Spearman rank-correlation

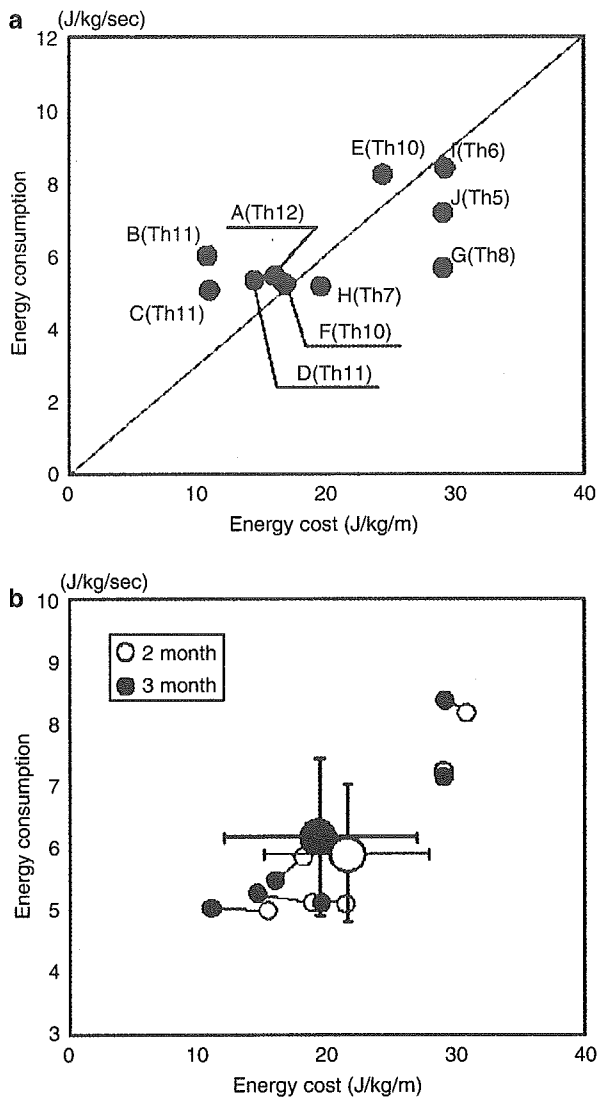


Figure 2 (a) The relationship between energy consumption and cost in each investigation. The slope of each line from zero to each plot reflects the walking speed. The subjects represented by points plotted on the upper and left side of the figure can be considered to have adequate aerobic conditioning. (b) Differences of the energy consumption and cost between 2 and 3 months after the beginning of the orthotic gait training. Gray and black markers indicate each subject's data and averaged data, respectively. Error bar indicates standard deviation

coefficient, mean CF ($r_s = -0.67$; $P < 0.05$) and hip extension VEL ($r_s = 0.74$; $P < 0.05$) also showed a strong relevance to the injury level.

Discussion

The present results clearly show the significant relationship between the level of neurological lesion and orthotic gait performance (Figure 4 and Table 4). The injured segment of the spinal cord in our subjects ranged from Th5 to 12. This included the anatomical levels of those innervating muscles that move the trunk, for example, the volitional control of the abdominal and iliopsoas muscle. This was intact in those injured at Th12, but not in those injured at Th5. It is therefore likely that these results are attributable to the degree of residual motor function around the trunk.

Physiological intensity of orthotic gait exercise

In the present study, the steady-state values of the $\dot{V}O_2$ and HR during orthotic gait were 18.2 ± 3.80 ml/kg and

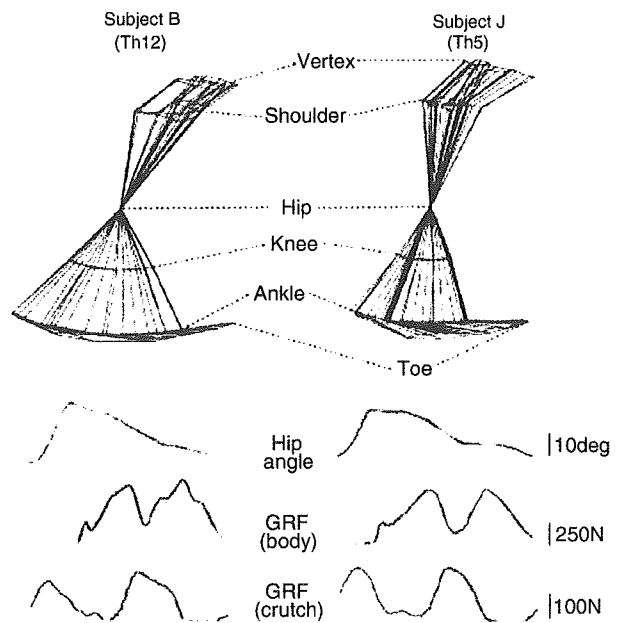


Figure 3 Differences in the gait motion between SCI subjects injured at a lower (left; Th12) and higher level (right; Th5). The stick pictures represented were standardized by the marker on the GTR

Table 4 Pearson's product moment correlation coefficient and Spearman rank-correlation coefficient between injured level and each parameter

Level versus	Gait speed	E consmp.	E cost	Cadence	Stride length	Peak CF	Mean CF	Hip ROM	Hip fVEL	Hip eVEL
Pearson	0.74**	-0.54	-0.88**	0.22	0.79**	-0.78**	-0.60	0.78**	0.23	0.60
Spearman	0.89**	-0.44	-0.92**	0.14	0.78**	-0.80**	-0.67*	0.77**	0.20	0.74*

* $P < 0.05$, ** $P < 0.01$

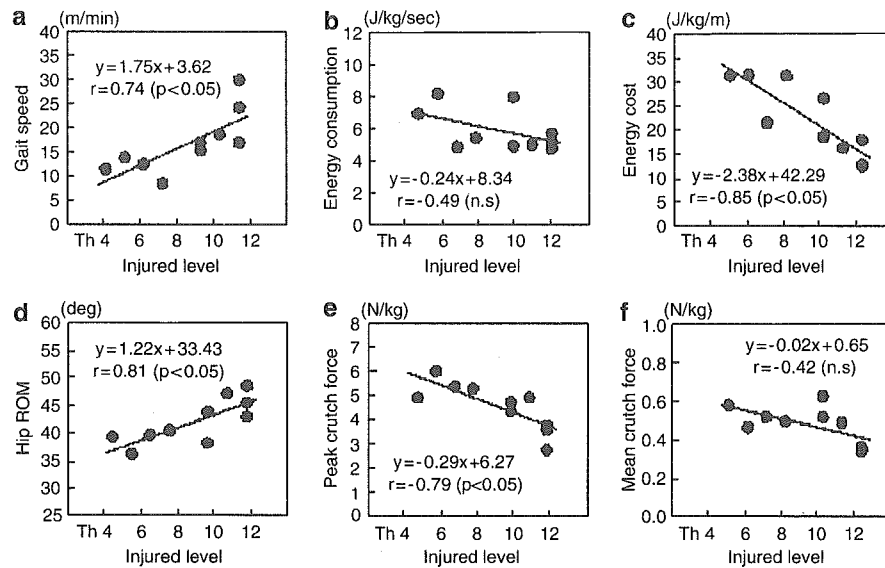


Figure 4 Effects of the level of injury on gait speed, cadence, stride length, energy consumption, energy cost, and hip range of motion. The lines indicate the linear regression lines of the level of injury against each parameter

Table 5 Energy consumption, cost and gait speed with different orthoses in previous studies and our present study

Series	Number of subjects	E. consump (J/kg/s)	E. cost (J/kg/m)	Gait speed (m/min)	Level and aid
Hirokawa et al ⁵	6	4.18	21	12.48	T1-T10 RGO
Winchester et al ¹³	4	4.37	19.44	13.5	T5-T10 RGO
Bernardi et al ⁴	10	4.3	20	12.78	T4-12 RGO
Felici et al ¹⁴	6	8.26	32.3	15.34	T5-L1 RGO, ARGO
Massucci et al ¹⁵	6	4.64	29	9.6	T3-T12 ARGO
Ijzerman et al ⁸	10	5.92	28.20	12.6	T4-12 ARGO
Merati et al ¹⁶	6	4.64	24.87	11.2	T3-T11 RGO
Present study	10	6.11	20.12	19.88	T5-12 ARGO
<i>Normal subject</i>					
Blessey ¹⁷		4.35	3.25	82.2	
Bernardi et al ¹⁸	18	4.52	3.53	76.8	

All values are expressed as averages

133.0 ± 21.63 b/min, respectively (Table 2), approximately a three-fold increase over the respective resting values. We note that the physiological intensity presumed by these valuables tended to be larger in the higher thoracic SCI subjects. This is clearly shown in Figure 2, where we show that the energy consumption in the higher thoracic SCI subjects is relatively larger, while that in the lower thoracic SCI subjects remained at the same level as that of normal walking. As the subjects were asked to walk at their preferred speed, this result suggests that higher level SCI persons cannot walk comfortably because of their larger area of motor paralysis.

Although the physiological intensity of orthotic gait is in a feasible range to allow safe walking because these

patients have sufficient aerobic capacity even in the higher thoracic SCI subjects, the excess energy expenditure and burden on their upper limbs make it impossible to achieve suitable exercise intensity for promoting general health. Therefore, it is important to discover ways to reduce this excess physiological load. In the following section, we will discuss the reason for limited orthotic gait performance in higher thoracic level of SCI patients based on our results of motion analysis.

Relevance between residual motor function and orthotic gait mechanics

During orthotic gait with the ARGO, in order to swing the paralyzed lower limb, the user first puts his or her

weight onto one foot, and rises up the upper body by coincidentally pushing the ground throughout the crutch. By inducing this upper body motion, the reciprocating device mounted on the hip joint functions to swing opposite side of the leg.^{7,8} Therefore, the trunk muscle contraction and compensatory upper limb motion are important for creating the hip swing motion. The mechanics of orthotic gait are the key to explaining our results. As clearly shown in Figures 3 and 4d, subjects with higher thoracic injuries showed a remarkably small hip ROM as compared to that in lower thoracic SCI subjects. This result might imply that the upper body motion was insufficient to produce the leg swing in higher level SCI subjects because of their trunk and hip muscle paralysis. Concerning the CF, as is clearly shown in Figure 4e, PCF shows an inverse relationship to the injury level. This result indicates an additional upper limb burden in the compensation for the trunk paralysis in the higher thoracic SCI subjects. It is therefore considered that the slower gait speed and higher energy cost of higher thoracic SCI subjects may be attributed to the limited hip motion and excess upper limb load.

Comparison with previous results

As compared to the other reports^{4,5,13-16} where the energy expenditure of orthotic gait was measured in reciprocating gait orthosis (RGO and ARGO), our data shows a faster gait speed with similar energy expenditures (Tables 4 and 5). The gait speed reported by Massucci *et al*⁵ and IJzerman *et al*⁸ both of which used ARGO, was remarkably slower than that shown in our data. Taken together with the similarity in the injured level of the subjects between these two studies, this result may indicate that our degree of training especially in time spent practicing, may have influenced the gait speed. Our subjects initially showed a slower gait speed, and reached exhaustion in only a few minutes. However, throughout the training period, they had acquired gait skills and thus improved their energy cost when performing the orthotic gait.

When the present results for energy expenditure during orthotic gait are compared to those for energy expenditure during walking in neurologically normal persons,^{17,18} although the levels of energy consumption are within a similar range, the levels of energy cost are considerably worse (approximately six times) during orthotic gait.

Implication for rehabilitation

Owing to the physiological need of gait training for SCI patients, it is important to find a way to reduce the excess physiological load during orthotic gait movement. Many researchers have paid attention to this issue, and in some studies they have attempted to accomplish effective orthotic gait performance with the use of functional electrical stimulation (FES). Although

some studies suggested the effectiveness of FES based on the additional muscle contraction of the paralyzed area in, for instance, the reduced energy cost (Hirokawa *et al*⁵), it is obvious that the FES technique is insufficient as a solution to the above problem.

In addition to the higher energy cost of orthotic gait movement, the present results clearly show the relationship between orthotic gait performance and the thoracic level of lesion in SCI patients. Further, the results of the motion analysis have revealed that the slower gait speed and higher energy cost of higher thoracic SCI subjects can be attributed to their limited hip motion and presumed excess upper limb load. Our results indicate that higher thoracic SCI patients need some way of reducing the excess physiological load to acquire the suitable exercise intensity.

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