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] Bhambhani 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 exercise[18–20] 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 ($\dot{V}E$), 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 circuratory 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.

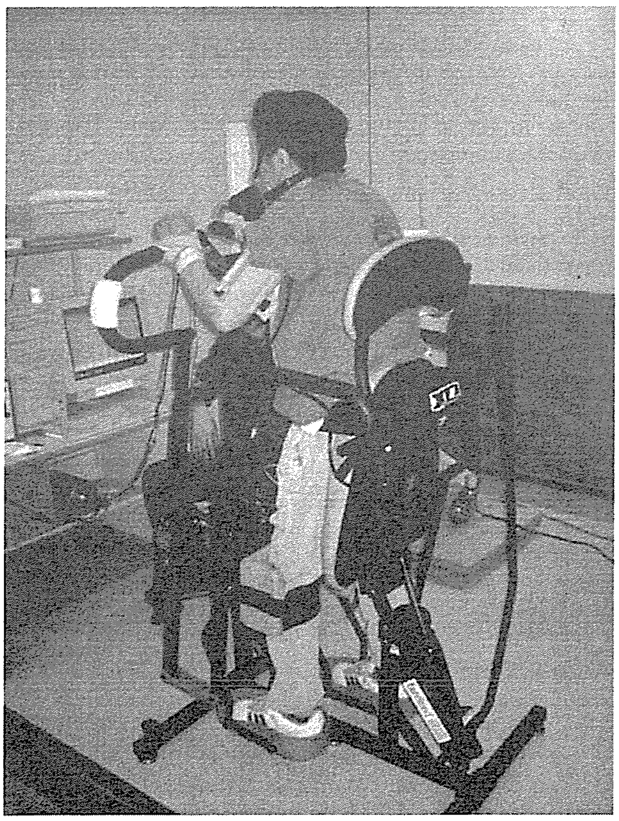

Figure 1 The arm swinging and passive walking machine. When a quadriplegic subject swings his arms back and forth, the paralyzed legs simultaneously move as if walking

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).

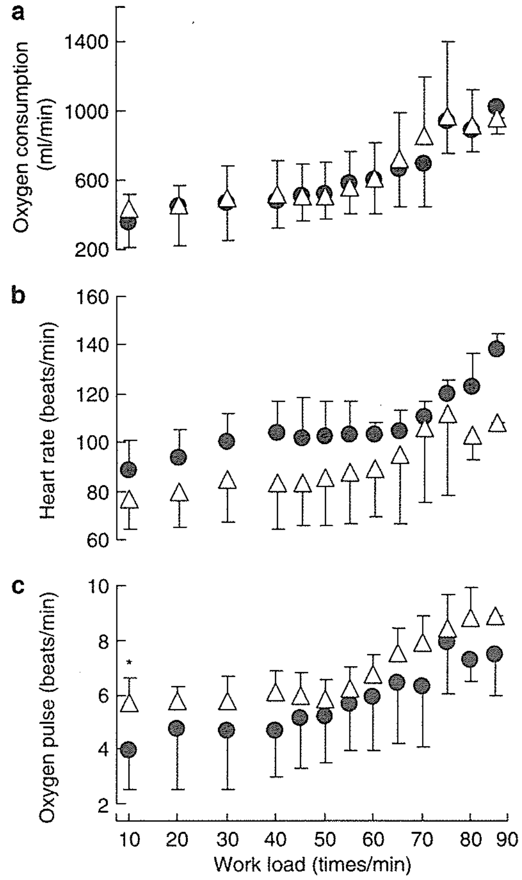

Figure 2 Change in oxygen consumption (**a**), heart rate (**b**), and oxygen pulse (**c**) during passive walking with arm swinging exercise in persons with Quadriplegics (●) and nondisabled (△). Although there was no difference of oxygen consumption between QP and ND, oxygen transportation of QP was 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.

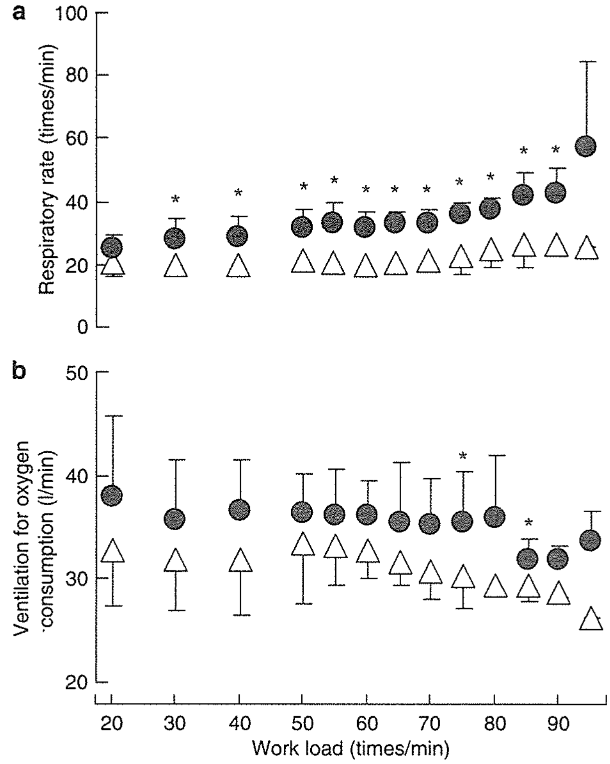

Figure 3 Change in respiratory rate (**a**) and ventilation for oxygen consumption (**b**) during passive walking with arm swinging exercise in persons with Quadriplegics (●) and nondisabled (△). Ventilation efficiency showed significant decrease in QP as compared with ND. $*P<0.05$, $^{\#}P<0.01$: compared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

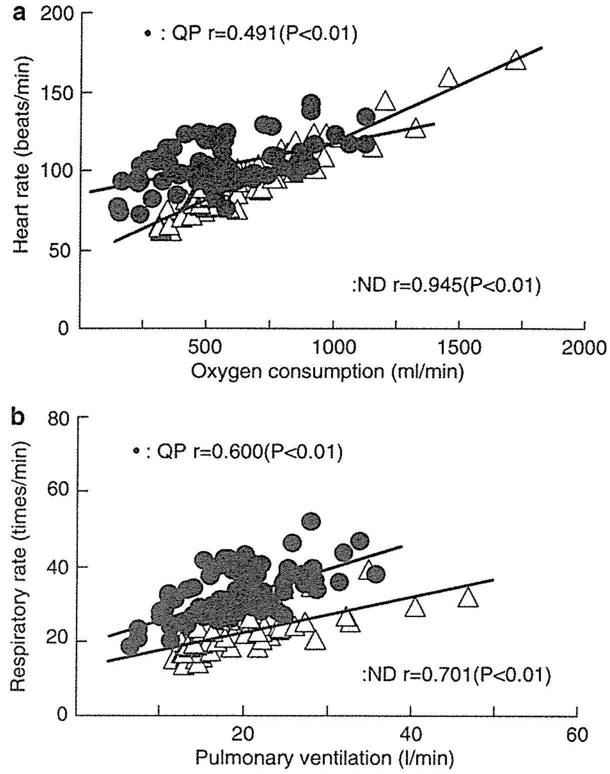

Figure 4 Relationship between oxygen consumption and heart rate (**a**), pulmonary ventilation and respiratory rate (**b**) during passive walking in persons with Quadriplegics (●) and nondisabled (△). QP showed inactive to HR and remarkable increase in RR as compared with ND

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 rest[30] 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 lobotic-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 rhythmical 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.

Green[36] 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 paraplegiac 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 B[11]) 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).

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

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.

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±4.2°C and 58.3±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

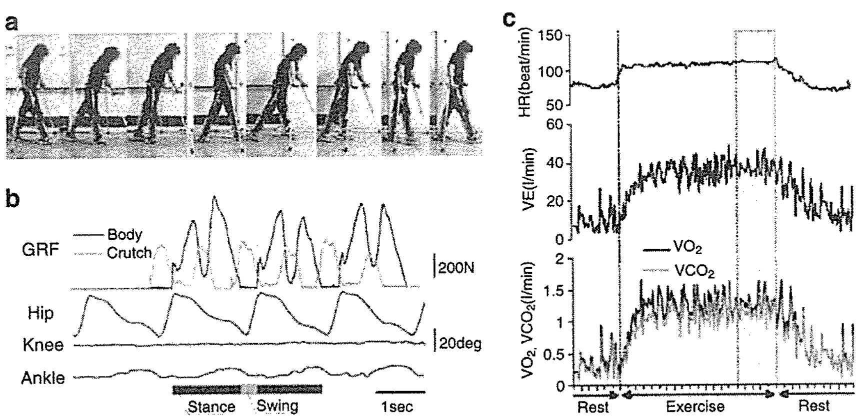

Figure 1 (**a**) Sequential picture of walking with the ARGO in a SCI subject (injured at Th12). (**b**) Time series data of the GRF (body and stick) and joint angle motion (Hip, Knee, and Ankle) obtained by the VICON system. (**c**) Typical example of the changes in the cardiorespiratory parameters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

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

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

where $K = 20.19$ J/ml, since 1 ml $O_2 = 4.825$ cal and 1 cal $= 4.184$ 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 × 450 cm in size, consisted of two 80 × 200 cm plates and four 40 × 250 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 ± 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 ± 5.82 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)		*VO_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

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

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

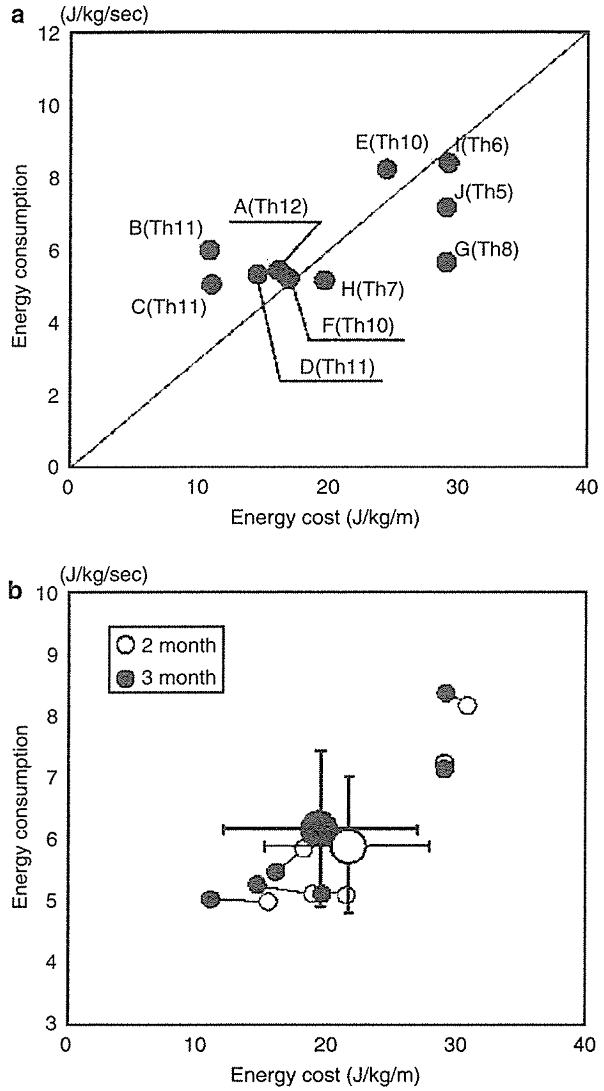

Figure 2 (a) The relationship between energy consumption and cost in each investigation. The slope of each line from zero to each plot reflects the walking speed. The subjects represented by points plotted on the upper and left side of the figure can be considered to have adequate aerobic conditioning. (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

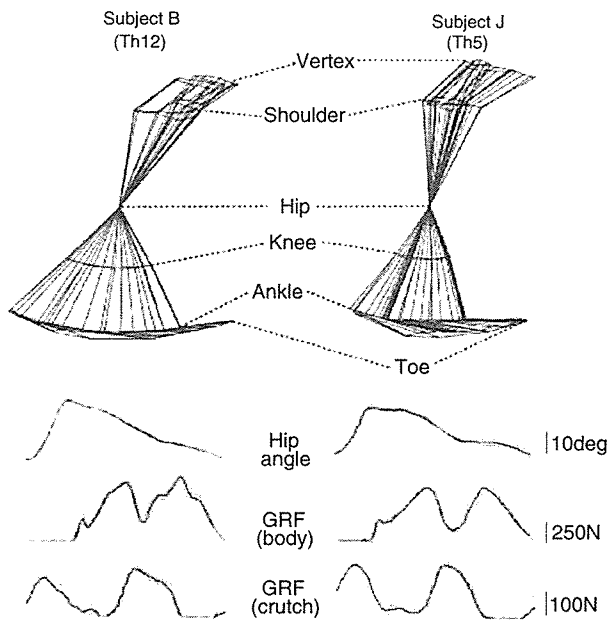

Figure 3 Differences in the gait motion between SCI subjects injured at a lower (left; Th12) and higher level (right; Th5). The stick pictures represented were standardized by the marker on the GTR

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*

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

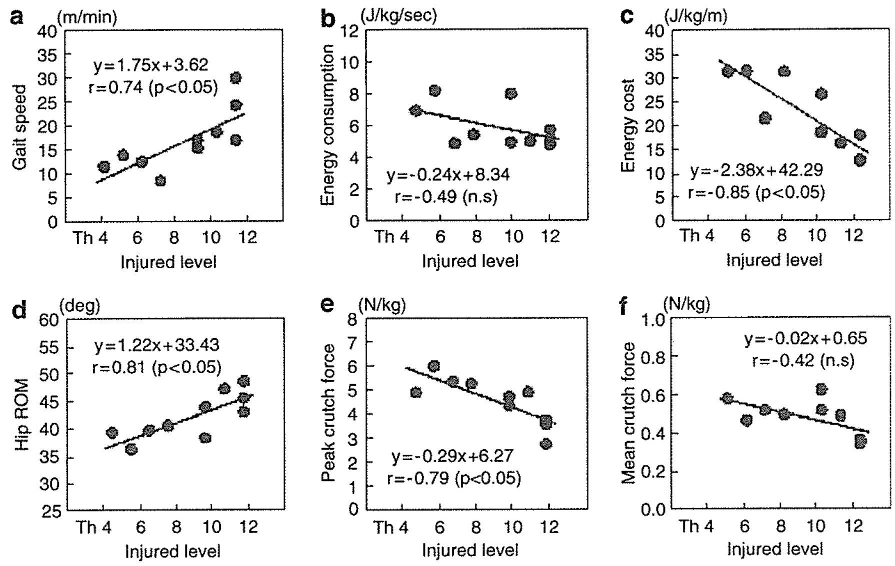

Figure 4 Effects of the level of injury on gait speed, cadence, stride length, energy consumption, energy cost, and hip range of motion. The lines indicate the linear regression lines of the level of injury against each parameter

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
Normal subject					
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*[15] 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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