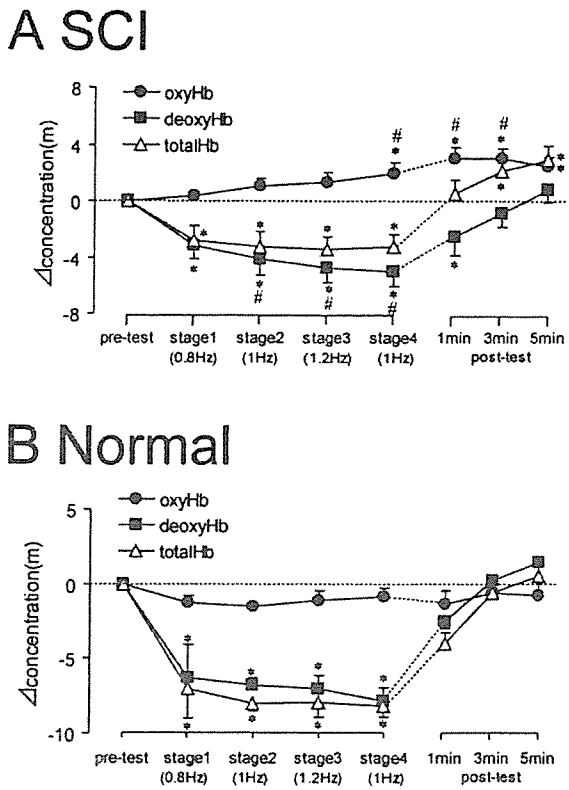

FIGURE 4—Concentration changes in total, oxygenated hemoglobin (oxyHb), and deoxygenated hemoglobin (deoxyHb) throughout the experiment for patients with spinal cord injury (SCI) (A) and normal subjects (B). The error bars indicate the SEM value. * Significant difference ($P < 0.05$) compared with the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

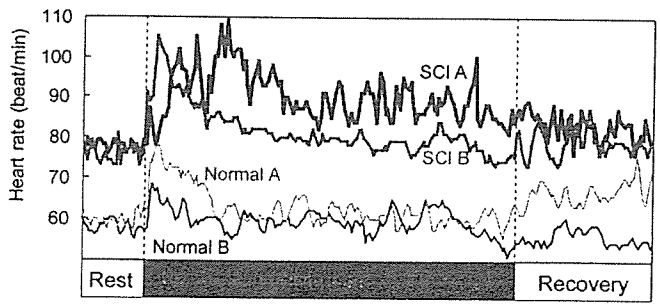

FIGURE 5—HR changes at rest, during passive leg movement, and in the recovery period obtained by two patients with spinal cord injury (SCI) and two normal subjects.

muscle is expressed with size relative to the MVC obtained by normal subjects (average: 417.7 ± 43.24 μV), 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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特集：高齢者の歩行障害

Short Topics

1．歩行の中枢とCPG

中澤　公孝

株式会社　ライフ・サイエンス

特集
高齢者の歩行障害

Geriat. Med. 43(1)：93～98, 2005

Short Topics

1．歩行の中枢とCPG

中澤　公孝*

KEY WORD
歩行中枢
脊髄
CPG
可塑性
リハビリテーション

POINT
- ヒトの脊髄にも歩行のための基本的運動出力を生成する神経機構が存在する．この脊髄神経機構は四足動物などで同定されているパターン発生器(CPG)と共通する性質を有する．
- ヒトのCPGもおそらく脊髄に広範に分布する神経回路からなり，その出力は求心性入力，CPGには可塑性な性質があり，時空間的にパターン化した入力を継続的に受けとることにより，入出力特性が変化する．

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はじめに

人間にとって立位歩行は最も基本的な身体運動の1つである．しかし，これを成立させる神経機序はまだまだ多くのなぞに満ちている．1900年代初頭からめざましい発展を遂げた神経生理学研究によって，ネコなど四足動物の歩行を発現させる神経機序はかなりの部分が明らかになった．

しかしながら，人間の立位歩行がどこまで四足歩行と共通で，どこからどの程度異なるのか，これに関わる問題はほとんど未解決といっても過言ではない．このことは，ヒトを対象とした実験で得られた結果を動物モデルを用いて確認するという生理学研究の正当な手法が，人間固有の機能の研究には適用困難であるという限界に起因する．この限界は依然として付きまとうものの，近年の経頭蓋磁気刺激や神経活動の画像化技術を用いた研究は非侵襲的に人間の神経活動を評価する新たな道を開いたし，脊髄損傷者を対象とした脊髄のパターン発生機構の研究は，臨床的成果を契機として基礎的側面の研究も加速させるに至った．

本稿では，特に近年飛躍的に進展した人間の脊髄歩行パターン発生機構に関わる研究成果についてまとめてみたい．

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ロコモーションパターンを生成する神経機構

歩行に関与する神経機構をRossignol[1]，Orlovskyら[2]のモデルを基に図1にまとめた．高位中枢で決定された歩行開始の司令は中脳歩行誘発野など脳幹の歩行中枢を賦活し，最終的に脊髄へと伝達される．そこから時空間的にパ

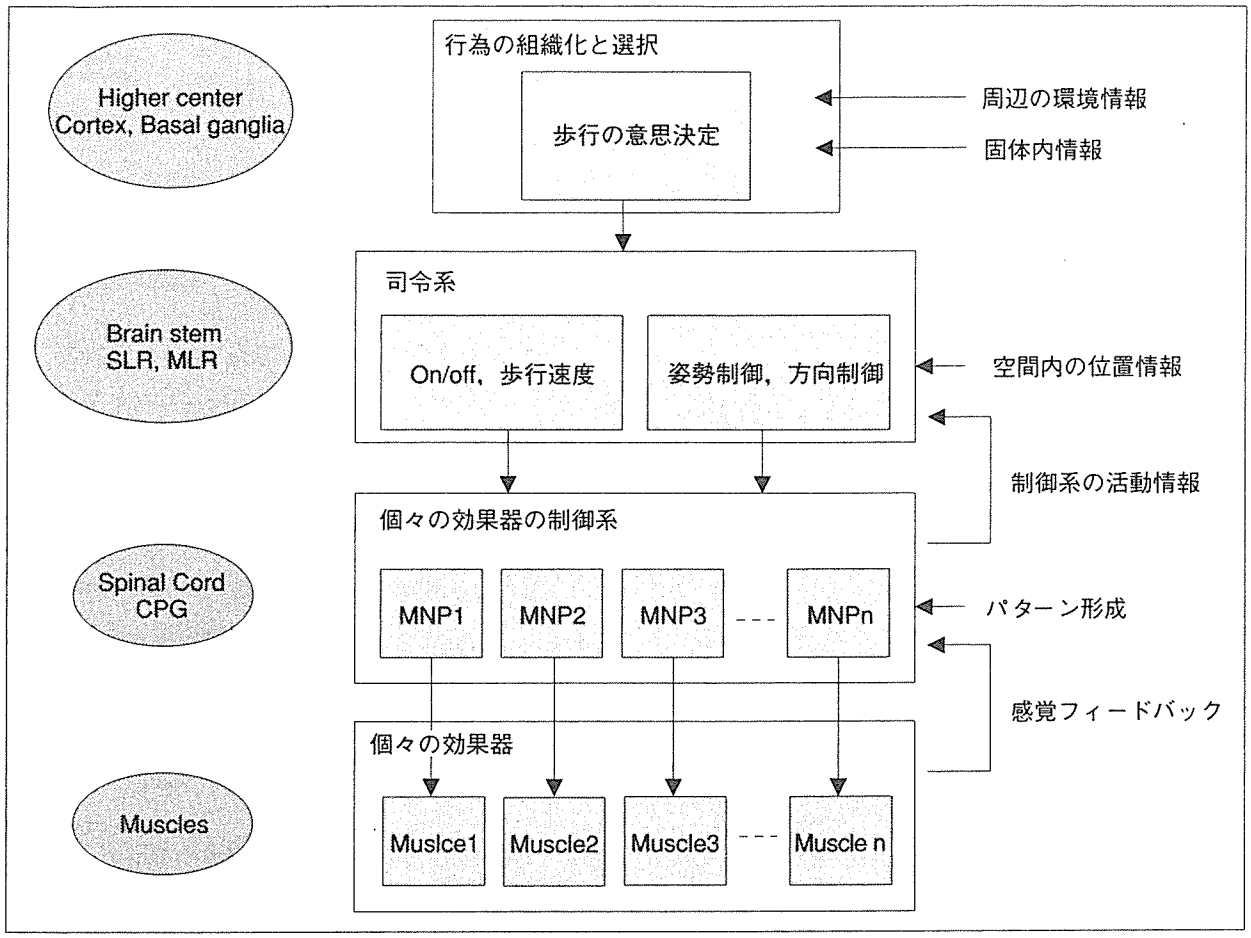

図1　歩行開始に関与する神経機構

Rossignol[1]，Orlovskyら[2]のモデルを基に作成．図中 SLR：subthalamic locomotor region，MLR：mesencephalic locomotor region，CPG：central pattern generator，MNP：motoneuron pool.

ターン化した運動出力が筋へと送られる．この時空間的にパターン化した運動出力を生成する神経機構が，いわゆる脊髄セントラルパターンジェネレーター(central pattern generator；CPG)である．CPG の構造と機能は系統発生学上下等生物である軟体類から高等な哺乳類まで広範にわたって研究されており，本質的な性質はヒトに至るまで共通すると考えられている[2]．しかしながら，脊髄からの歩行出力の自律性は霊長類では四足動物などに比べてかなり低く，それは霊長類において皮質脊髄路の重要性が増大することと関係があると考えられている[3]．

ヒトの脊髄 CPG

四足動物やその他の下等生物の移動運動が CPG 的な神経回路に大きく依存していることは疑いない．しかしヒトでの研究はまだまだその数も少なく，脊髄 CPG の存在自体を示唆する間接的証拠が最近になって多く報告されるようになったにすぎない．とはいえ，いわゆる乳幼児の足ふみ反射(newborn stepping)などの現象は昔から知られており，CPG の原型が生得的に備わっていることを示唆する現象と考えられている．最近，Yang らのグループは乳幼児の原始歩行について系統的に研究しており，ネコなど四足動物で確認されている荷重や股関節からの求心性入力と CPG の相互作用が乳幼児にもあてはまることなどを報告している[4]．

冒頭でも述べたように，ヒトの脊髄 CPG に関する研究の近年の進展は脊髄損傷者の歩行リハビリテーションの臨床的研究の発展と関連し

ている．1990年代初頭のRossignolのグループ，Edgertonのグループによる脊髄ネコの実験成績から，トレッドミルを用いたステッピングトレーニングによって歩行機能が従来考えられていた以上に回復することが示された[5,6]．それらをきっかけに脊髄損傷者をトレッドミル上で他動的にステッピングさせる“免荷式ステッピングトレーニング”が人間でも行われるようになったのである．他動的ステッピングによって，たとえ麻痺領域の随意筋収縮が全くみられない完全対麻痺者であっても，ステッピング周期にあった歩行様の筋活動が誘発されることが明らかとなった[7,8,11,12]．さらに，誘発される歩行様筋活動はステッピング時に下肢に加わる荷重や股関節からの求心性入力に強く依存することが示され[13,14]，その点でネコなど四足動物のCPGの性質と合致した．

このように，対麻痺者のステッピングトレーニングはヒトの脊髄CPGの存在と性質に関わる研究の発展と切っても切れない関係にあるが，CPGの存在を示唆する現象の報告自体はもう少し遡ることができる．Busselらは既に1980年代の後半に対麻痺者のミオクローヌスとFRA(flexor reflex afferents)刺激の関係などを記録し，CPGの存在を示唆していたし(Busselら[15,16]，Calancieら[17]も対麻痺者の不随意性ステッピング運動を詳細に記録し，それが脊髄CPGからの出力である可能性がきわめて高いことを主張した．近年ではDimitrijevicのグループが対麻痺者の痙性治療に用いる脊髄硬膜外電気刺激を応用し，CPGの存在をより直接的方法に近いかたちで示している[10]．彼らは近年の報告[18]で，対麻痺者の脊髄に対し，硬膜外で一定強度の電気刺激を種々の周波数で与え，脊髄からの運動出力との関係を示している(**図2**)．それによると，臨床的完全対麻痺者5名の脊髄腰膨大部近辺を5～15Hzで刺激すると下肢の伸筋群に放電が誘発され，下肢全体を突っ張るような運動が出現したのに対し，それより高い周波数(25～50Hz)で刺激するとステッピングに似た動きが誘発されたという．これらの結果は，脊髄内の神経回路に末梢入力との相互作用

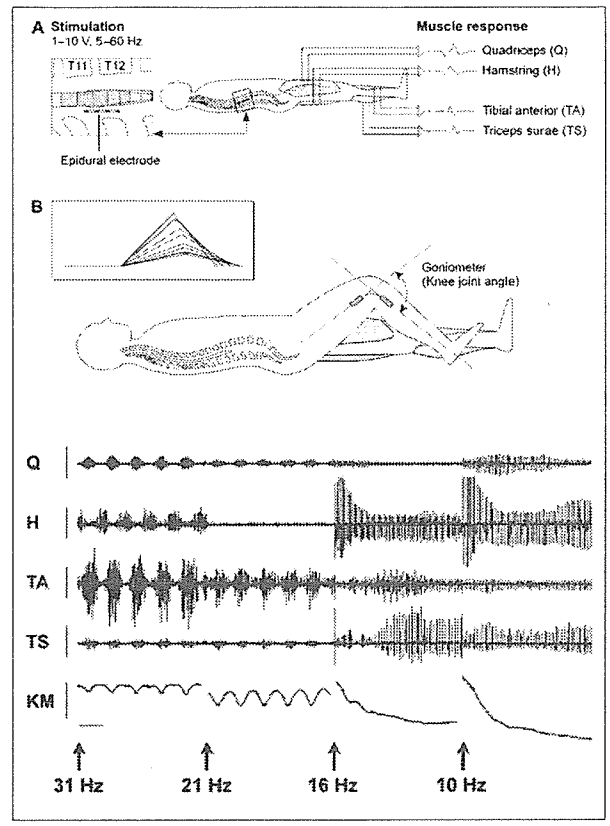

図2　Jilgeら[18]の脊髄硬膜外刺激の様子と，記録された脊髄からの運動出力と刺激周波数との関係(文献18を筆者改変)

によって異なる運動を生成する機能ユニットが存在することを示唆すると解釈されている．さらにGurfinkelら[19]は健常者において，下肢の筋あるいは腱への振動刺激でステッピングが誘発可能であることを示し，やはり求心性入力のみで脊髄からパターン化した出力が誘発可能なことを示した．

ヒトCPGの性質

1．損傷高位と歩行様筋活動

Dietzら[9]は上行性入力によって歩行様の筋出力を発生する脊髄内の神経機構が脊髄内のいずれかの髄節に限局しているのか，それとも脊髄内に広く分布しているのかを明らかにするために，下降性入力が遮断されている完全対麻痺者のみを対象に，脊髄の損傷高位と他動的ステッピングで誘発される歩行様筋活動の強度および波形の関係を分析した．両筋の立脚相，遊脚

相それぞれの放電量と損傷高位との関係は，総じて損傷高位が高い損傷者の方が筋放電量が大きい傾向が，ヒラメ筋では立脚相，前脛骨筋では遊脚相でそれぞれ観察された．両筋は健常者においてこれらの歩行位相で活動することから，損傷高位が高い損傷者の歩行時筋活動の方が健常者に近いことが判明した．さらに筋放電波形の類似度を示す指標でも，全体的に損傷高位の高い損傷者の方が健常者のパターンに近いことが示された．Dietz らは，これらの結果は末梢入力によって歩行様筋活動を発生する神経機構が脊髄のある髄節に限局して存在するのではなく，脊髄内広範にわたって分布していることを示唆するものであり，ムッドパピー[注1]で示された結果[20]に一致するとした．

2．感覚入力との相互作用

Dietz のグループは近年，歩行トレーニングロボット(Lokomat, Hokoma 社，スイス)を開発し[21]，それを使った実験で，脊髄からの歩行様出力にとって股関節と荷重関連受容器(load receptor)からの入力が必須であることを明らかにした．筆者らのグループは，さらに立位で股関節の屈曲・伸展動作が可能な特殊な装置(Easystander, Ultimate 社，USA)を用い，対側からの求心性入力は左右脚が交互に動くときのみ脊髄からの歩行様出力に促通性の効果をもつことを明らかにした[22](図3)．すなわち，歩行様出力を生成する脊髄神経回路には対側からの交叉性経路も含まれており，しかもその経路を介する求心性入力はある位相でのみ促通性となる位相依存性があるらしい．

3．上肢と下肢の協調

近年この分野において注目を集めているのは，上肢と下肢の CPG の連関である．

Zehr のグループは上肢の周期的運動時には H-反射や皮膚反射が位相依存性および課題依存性に修飾されることを観察し，そのような性質は CPG の参画に起因するとした[23, 25]．

上肢 CPG と下肢 CPG の結合に関してもいくつか興味深い報告がなされている．手関節屈筋の H-反射を誘発する際に下肢の周期的な底背屈運動を付加すると，それに応じて H-反射の振幅が変調され[26]，それは脊髄頸膨大部と腰膨大部間を結合する固有ニューロンを介すると考えられる[27]．また，片側下肢への電気あるいは機械的刺激を歩行中に加えると両側上肢筋に応答が誘発されるが，立位時や他の運動課題中には誘発されないことから，上肢と下肢運動ニューロン間の結合は課題依存性に変調し，それぞれの CPG の活動が関係すると考えられている[28]．

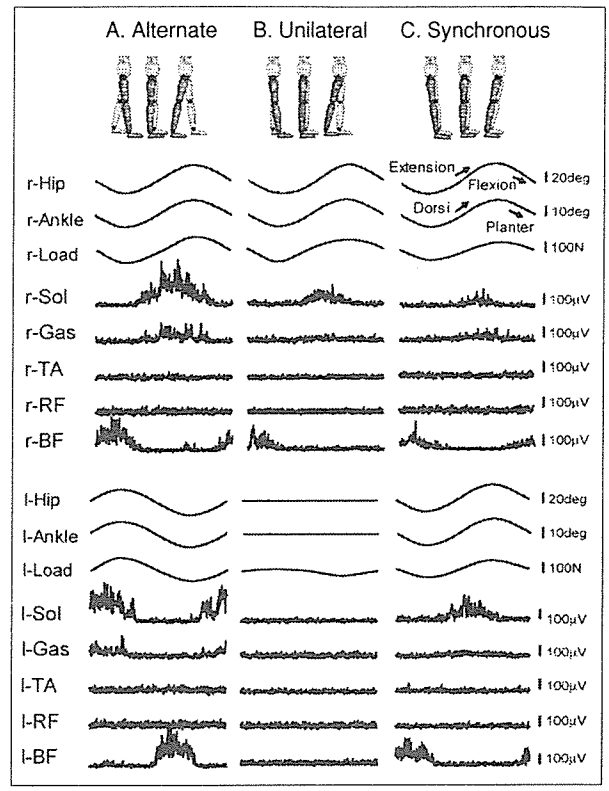

図3　左側脚の動作を変えたときの右側脚他動運動で誘発される歩行様筋活動の変化

左側下肢が交互性(alternate)，同側のみ(unilateral)，左右同位相(synchronous)で他動的に動かされた時の左右下腿筋電図と股関節，足関節の各関節角度，足部荷重の加算平均波形を表示．図中，Hip，股関節角度；Ankle，足関節角度；Load，足部荷重；Sol，ヒラメ筋；Gas，腓腹筋；TA，前脛骨筋；RF，大腿直筋；BF，大腿二頭筋．r-は右側，l-は左側を表す．

注1　Mudpuppy あるいは Waterdog．イモリの仲間．両生類

4．CPGの可塑性

従来，脊髄神経機構に可塑的な性質はないと考えられてきたが，近年脊髄の可塑性を示す実験結果が次々に報告されるようになり，そのような考え方は覆されつつある[29, 30]．特に，脊髄および他の中枢神経系のuse-dependentまたはactivity-dependentな可塑性は，これからのニューロリハビリテーションの理論的枠組みにおいて重要な位置を占めるであろう．これまで，動物の実験成績に比べて，ヒトを対象とした研究報告はまだまだ少ない．主に脊髄損傷によって脳との結合が遮断された条件下で他動的トレーニングを介入し，その結果生じる脊髄神経回路の入出力関係の変化が，use-dependentな可塑性を表す現象ととらえられている．筆者らのグループも，歩行用の装具を用いたトレーニング後に完全対麻痺者の歩行用筋活動が増強する現象を観察しており[31]，CPGを含む脊髄神経回路の入出力関係が可塑的に変化したことを反映すると考えている．

まとめ

本稿では，主に人間の脊髄歩行パターン発生機構に関わる近年の研究成果を概観した．ヒトの二足歩行あるいは直立姿勢の制御機構自体まだまだ不明な点が多く，学術的に興味深い問題を数多く含んでいる．それらの問題は同時にリハビリテーションなどの臨床にも直結する課題であり，今後両面での研究が加速することが望まれる．

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ORIGINAL ARTICLE

Enhanced Stretch Reflex Excitability of the Soleus Muscle in Persons With Incomplete Rather Than Complete Chronic Spinal Cord Injury

Kimitaka Nakazawa, PhD, Noritaka Kawashima, MSc, Masami Akai, MD

ABSTRACT. Nakazawa K, Kawashima N, Akai M. Enhanced stretch reflex excitability of the soleus muscle in persons with incomplete rather than complete chronic spinal cord injury. Arch Phys Med Rehabil 2006;87:71-5.

Objective: To compare excitabilities of spinal stretch reflex among clinically complete spinal cord injury (SCI), incomplete SCI, elderly healthy, and young healthy subjects.

Design: Case comparison.

Setting: Research laboratory.

Participants: Volunteer sample of 12 complete SCI, 10 incomplete SCI, 10 elderly, and 11 young subjects.

Intervention: Mechanically induced stretch reflex, H-reflex, and M response in electromyographic activity of the soleus muscle were recorded in all subjects.

Main Outcome Measures: Absolute peak-to-peak stretch reflex amplitude and maximum H-reflex (Hmax), and those values relative to the maximum M response (Mmax) amplitude (relative peak-to-peak stretch reflex amplitude) and H/M ratio.

Results: Both the absolute and relative peak-to-peak stretch reflex amplitudes showed the greatest values in incomplete SCI among the 4 groups. Although absolute and relative peak-to-peak stretch reflex amplitudes of the incomplete SCI group were greater than those of the complete SCI group, the H/M ratios of both groups were comparable, and were greater than those of the younger and elderly groups.

Conclusions: The results suggest that the greater absolute and relative peak-to-peak stretch reflex amplitudes of incomplete SCI were mostly due to the greater maximum motor potential (Mmax), while the elevated spinal motoneuronal excitability shown by the increased H/M ratio was maintained in the chronic stage after both complete and incomplete SCIs.

Key Words: Reflex, stretch; Rehabilitation; Spinal cord injuries.

AFTER SPINAL CORD INJURY (SCI), reorganization occurs both below and above the lesion sites in the central nervous system (CNS). This reorganization can be facilitated and directed toward functional recovery by imposed activity of paralyzed limbs, especially in cases of incomplete spinal cord lesions.[1] Therefore, learning more about the neuronal reorganization after SCI prepares the way for a better understanding of rehabilitation strategies and for the development of new approaches to restore functional movements in people with SCI.

The spinal stretch reflex is the simplest behavior of the vertebrate CNS. Many studies have attempted to elucidate the effects of SCI on stretch reflex responses in humans, because the stretch reflex excitability of lower-limb muscles is believed to strongly relate to typical physiologic symptoms such as spinal shock or spastic paralysis after injury.[2-12] It is generally accepted that SCI in humans and animals is followed by a spinal shock period with the loss of tendon tap reflexes and flaccid muscle tone. Several weeks to months after SCI, a "spastic syndrome" develops with exaggerated tendon reflexes, increased muscle tone, and muscle spasm.[9] Recently, however, Calancie et al[13] reported that the loss of tendon reflex was not observed even at the acute stage after incomplete SCI, whereas it was commonly observed in people with motor-complete injuries. In addition, while taps to the right patellar tendon elicited electromyographic responses in both the ipsilateral quadriceps muscle and in the contralateral thigh muscles (crossed-adductor response), specifically in most motor-incomplete SCIs, the response was never seen in the motor-complete SCIs. Thus, reorganization of stretch reflex responses after SCI seems to differ between motor-complete and incomplete injuries. The extent to which the reorganization process depends on the severity of SCI in humans is unknown.

In this study, we compared the spinal stretch reflex excitabilities among 4 groups: clinically complete SCI, incomplete SCI, elderly healthy, and young healthy subjects.

METHODS

Participants

Twelve clinically motor-complete and 10 motor-incomplete subjects with SCI participated in the experiment, along with 10 elderly and 11 younger healthy controls. Table 1 summarizes their physical characteristics. All SCI subjects were at least 12 months postinjury and none had taken any antispasticity medication for at least 6 months before the testing. The subjects gave written informed consent to the experimental procedures, which were approved by the ethics committee of Japan's National Rehabilitation Center for Persons with Disabilities, Japan.

Experiments

We recorded mechanically induced stretch reflex electromyographic responses, H-reflexes, and M responses from the right soleus muscles of all subjects.

Stretch Reflex Test

We used a specially designed machine[4] (fig 1) to elicit stretch reflex responses in the soleus muscle with subjects in a

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Table 1: Physical Characteristics of All Subjects

Subject*†	Age	Sex	Level Lesion	ASIA Grade	Time Since Injury (mo)
a	53	F	C8	D	475
b	66	M	C3	C	38
c	63	M	C6	D	223
d	52	M	C7	D	90
e	52	M	C5	D	438
f	45	M	C4	C	17
g	44	M	C4	D	15
h	32	M	C4	D	14
i	42	M	T12	C	12
j	30	M	T12	D	19
k	22	M	T8	A	19
l	28	M	T8	A	15
m	30	M	C7	B	54
n	32	F	C8	B	135
o	41	F	C7	B	297
p	35	M	C7	A	160
q	20	M	T4	B	28
r	48	M	T12	A	27
s	30	M	C6	A	45
t	20	M	C6	A	23
u	30	M	T11	A	12
v	27	M	T12	A	16

Abbreviations: ASIA, American Spinal Injury Association; F, female; M, male.
*Elderly (8 men, 2 women; mean age ± standard deviation [SD], 62.5±3.2y).
†Younger (8 men, 3 women; mean age ± SD, 25.7±5.2y).

sitting position. This machine can apply quick rotations at ankle joints in various postures, from standing upright to sitting. In this study, we induced stretch reflexes in the plantarflexor muscles as subjects sat with the hip and knee angles fixed at 70° and 60° (anatomic position is 0°), respectively. Subjects were seated comfortably in the machine with the right leg fixed to a footplate connected to a servo-controlled torque motor, which could generate quick rotations of the plates at various velocities and amplitudes with commands from a computer. We adjusted the axis of rotation of the footplate to the center of the ankle joint. To elicit stretch reflexes in the soleus muscle, the machine provided quick stretch stimuli to the ankle joint in the dorsiflexion direction at an angular velocity of approximately 400°/s; this stretch was repeated 5 times, with an interstimulus interval of 10 seconds.

For the electromyographic recording, we used bipolar Ag-AgCl surface electrodes (diameter, 7mm) placed 2cm distal to the endpoint of the medial head of the gastrocnemius muscle, with an interelectrode distance (center to center) of 15mm. A conventional bioamplifier[b] amplified and band-pass filtered (low-high cut, 20–1kHz) the electromyographic signals. The electromyographic, torque, and angle signals were all digitized at a sampling rate of 1kHz and stored for later analysis.

H-Reflex and M-Response Test

We elicited the H-reflexes and M responses in the soleus muscle by applying rectangular pulses of 1-ms duration to the posterior tibial nerve in the popliteal fossa with a constant voltage stimulator.[c] The postural condition was identical to that in the stretch reflex test, with the subjects sitting in the same machine. On the basis of the threshold intensity of the H-reflex, we increased the stimulus intensity from a level sufficiently below the threshold intensity until the M response reached a plateau; the same intensity was repeated 5 times. The sampling frequency for electromyographic signals and pulses were 5kHz.

Data Analysis

For the stretch reflex electromyographic response, we defined the background electromyographic level over 100ms before the onset of the ankle joint stretch, and the onset of the stretch reflex response as the moment when the electromyographic activity levels reached levels higher than the mean background electromyographic level plus 3 times its standard deviation. The stretch reflex electromyographic response was defined as the peak-to-peak electromyographic amplitude for 30ms after the onset of the stretch reflex electromyographic response.

For the H and M waves, we evaluated peak-to-peak values within time windows from 30 to 50ms and from 5 to 25ms after the electric stimulation, respectively. From the H-M recruitment curve, we determined the maximum H-reflex, M response, and H/M ratio for each subject.

Statistical Test

We used analysis of variance (ANOVA) with a post hoc test (Scheffé) to test for statistically significant differences in stretch reflex electromyographic responses, H-reflexes, and M responses among the 4 subject groups. Data are presented as the mean and standard error of the mean (SEM). Significance was accepted at P less than .05.

RESULTS

Stretch Reflex

Figure 1 illustrates a typical example of the stretch reflex electromyographic response. Typically, single twitch-like bursts appeared in electromyographic activity after mechanical stretches to the soleus muscle. We evaluated the peak-to-peak amplitudes of reflex electromyographic responses both as absolute values and as values relative to the maximum motor potential (Mmax) obtained in the H-reflex and M response tests. The mean latency of the reflex electromyographic response ± SEM was 47.22±0.73ms from the stretch onset, indicating that the responses were short-latency spinally mediated reflexes. Figure 2A compares the stretch reflex amplitudes among the different subject groups. The ANOVA comparison

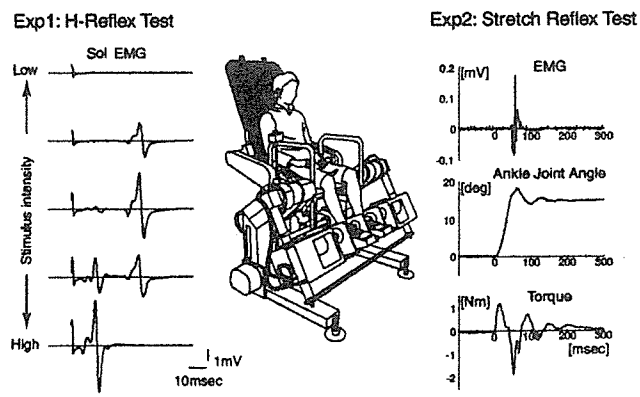

Fig 1. The machine used in both the stretch reflex and H-reflex experiments, and representative raw electromyograms (EMGs), joint angles, and torque signal waveforms in both tests. Abbreviations: Exp1, experiment 1; Exp2, experiment 2; Sol, soleus.

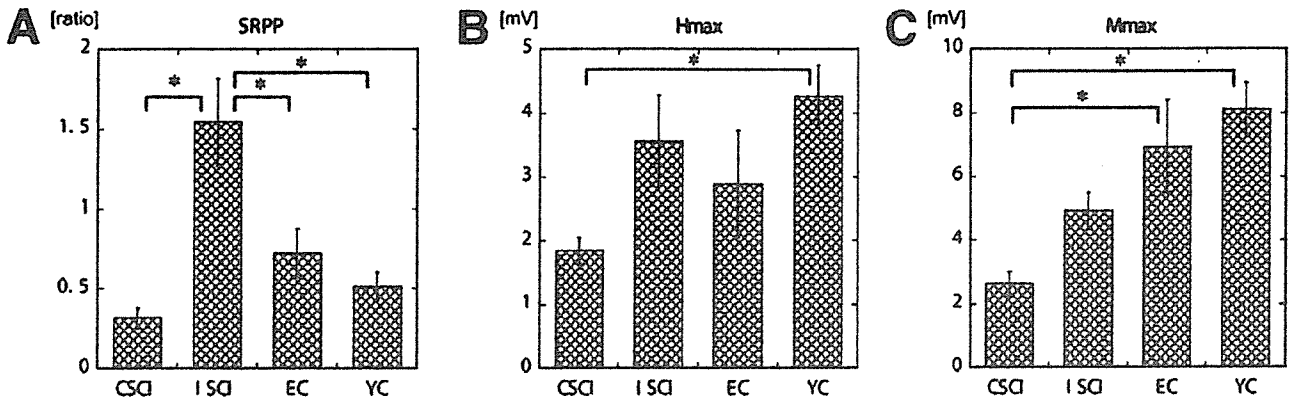

Fig 2. Summary of absolute peak-to-peak reflex electromyographic amplitude (SRPP), absolute Hmax value, and Mmax of each subject group. Bar heights show mean and error bars represent the SEM. ANOVA indicated there were statistically significant intergroup differences in the absolute reflex electromyographic amplitude, Hmax, and Mmax. Abbreviations: CSCI, complete spinal cord injury; EC, elderly healthy subjects; ISCI, incomplete spinal cord injury; YC, young health subjects. *Significant between-group differences revealed by Scheffé post hoc comparison ($P<.05$).

showed that there were statistically significant differences in the group means of the stretch reflex amplitudes among the 4 groups ($F=12.735$, $P<.05$). Scheffé post hoc multiple comparisons further revealed that the stretch reflex of the incomplete SCI group was significantly greater ($P<.05$) than those of the other groups, indicating that the absolute reflex electromyographic amplitude was the greatest in the motor-incomplete SCI group.

Maximum H-Reflex Amplitude

Figure 1 shows typical examples of H-reflex and M response. The mean latencies of H-reflex and M responses ± SEM were 11.52±0.15ms and 30.83±0.50ms, respectively.

The maximum H-reflex amplitude (Hmax) differed significantly among the groups ($F=3.653$, $P<.05$). The group mean of Hmax was greatest in the young healthy group, whereas the stretch reflex amplitude was greatest in the incomplete SCI group. There was a significant difference in the group means of Hmax between complete SCI and young healthy ($P<.05$).

Maximum Motor Potential

The Mmax differed significantly among the groups ($F=8.843$, $P<.05$; fig 2C). Mmax values were generally larger in the younger and elderly subject groups, indicating greater maximum motor potentials in the non-SCI groups. Statistically, the Mmax of the complete SCI group was significantly smaller than those of the elderly and younger subject groups ($P<.05$).

Relative Stretch Reflex and Hmax Sizes to the Mmax

To test whether the reflex electromyographic response relative to the Mmax is different among the 4 groups, the stretch reflex and Hmax were normalized to the Mmax. Figure 3 summarizes those results. There were still statistically significant differences in the relative sizes of the stretch reflex electromyographic amplitude among the groups ($F=5.900$, $P<.05$), with that of incomplete SCI showing the highest value (see fig 3A). The intergroup relation of the H/M size represented in figure 3B was not identical to that of the stretch reflex (see fig 3A). The H/M ratios were greater in the SCI groups, suggesting an enhanced H-reflex excitability in both the complete and incomplete SCI groups. Statistically, there were significant differences among the group means of the H/M ratio ($F=3.920$, $P<.05$), although the post hoc test indicated no significant differences between any pairs of groups.

Effects of Postinjury Time on Stretch Reflex Excitability

There was substantial variability in postinjury time in the subjects with SCI (see table 1). Therefore we tested whether the postinjury time had a significant influence on the stretch reflex and H-reflex variables.

Figure 4 depicts the relationships between the reflex-related variables and postinjury time for both the complete and incomplete SCI subjects. There were no statistically significant correlations in the relations of the Hmax and Mmax and postinjury time only in the incomplete SCI subjects (Hmax: $r=.723$, $P<.05$; Mmax: $r=.671$, $P<.05$). Both the Hmax and Mmax increased with postinjury time in the incomplete SCI subjects, whereas in the complete SCI group there were no such relations. For the other variables there were no statistically significant relations with postinjury time in the both subject groups.

DISCUSSION

In this study, we compared both mechanically and electrically induced reflex electromyographic responses among 4 different subject groups: complete and incomplete SCI subjects, and elderly and young healthy subjects. The main findings from the intergroup comparisons were: (1) the absolute stretch reflex electromyographic amplitude and its relative value to the Mmax were markedly greater in the incomplete SCI group; and

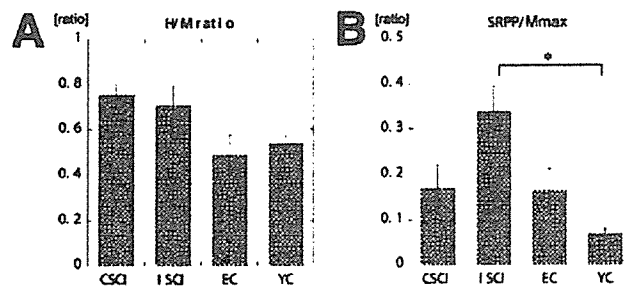

Fig 3. Summary of relative peak-to-peak stretch reflex sizes and H/M ratios for each subject group. Bar heights show mean and error bars represent the SEM. There were statistically significant intergroup differences in the relative peak-to-peak stretch reflex sizes and H/M. *Significant ($P<.05$) between-group differences revealed by Scheffé post hoc comparison ($P<.05$).

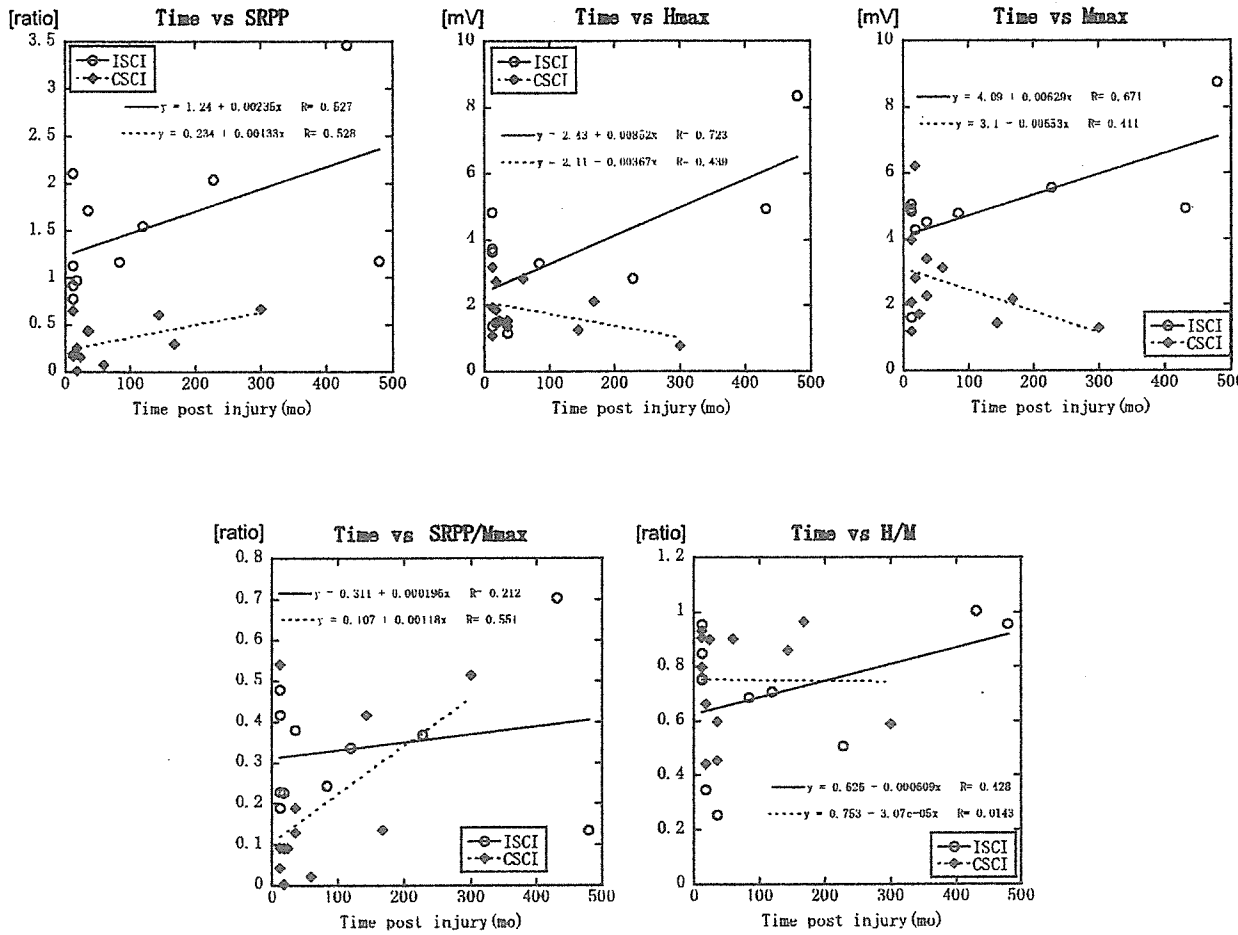

Fig 4. Relations between postinjury time and reflex-related variables evaluated in incomplete SCI and complete SCI subjects. Plots represent individual data from all SCI subjects.

(2) no significant differences were found in the H/M ratios in any pairs of groups, although those of the SCI groups were greater than those of the healthy groups. Possible neural mechanisms explaining the results are discussed below.

H-Reflex Excitability

The H/M ratios of both SCI groups and those of the able-bodied subjects were, respectively, within the ranges of previously reported values.[3,4,7,8,10,14] Noteworthy was that both SCI groups showed comparable values, indicating that at least among those incomplete SCI subjects whose American Spinal Injury Association grades were C and D, H/M ratios did not differ significantly from the ratios of complete SCI subjects. These results suggest that the fraction of motoneurons that can be activated via Ia pathways, which is evaluated using the H/M ratio, is not markedly altered in incomplete SCI subjects as compared with complete SCI subjects. The similarity in H/M ratios between the 2 SCI groups leads us to conclude that the supposed differences in descending inhibitory inputs associated with complete and incomplete spinal cord lesions have no detectable effects on H/M ratios. In other words, the marked differences in motor functions between the complete and incomplete SCI groups were not associated with the difference in H-reflex excitability assessed using H/M ratios.

On the Difference in Stretch Reflex Excitability Between the 2 SCI Groups

The results of mechanically induced stretch reflex indicated that stretch reflex excitability assessed in both absolute and relative terms was markedly enhanced in the incomplete SCI subjects. Also, the relative stretch reflex of the complete SCI subjects was higher than that of the younger subjects, although the difference was not statistically significant. The greater stretch reflex of the motor-incomplete subjects is consistent with the larger muscle responses to patellar and Achilles' tendon taps reported by Calancie et al.[13] They noted that "At a more chronic stage, persons with incomplete SCI tend to have enhanced spinal cord excitability, characterized by frequent spasms, enlarged tendon response amplitude, and recruitment of heteronymous muscles following tendon taps, compared to subjects with complete injury."[13(p2359)] No other study has reported quantitatively the larger stretch reflex responses of incomplete SCI persons.

Why, then, was the mechanically induced stretch reflex more facilitated in the motor-incomplete SCI subjects in this study? Our results alone cannot provide a definitive answer to this question. However, considering reported results, it is possible that differences in descending neural tracts severed[13,15] and/or alteration in mechanical property of muscles and tendons[7,16] are re-

lated to the elevated stretch reflex in the motor-incomplete SCI subjects.

In short, lesion of the lateral corticospinal tract is suggested to play a critical role in elevating the reflex excitability of motor-incomplete SCI[13] as a neural factor. As a mechanical factor, alteration in muscles and tendons after SCI also possibly explains the higher stretch reflex in the motor-incomplete SCI subjects. After SCI, a significant amount of muscle atrophy takes place.[16-19] This decrease in muscle fiber size might be more prominent in complete SCI subjects because of disuse, as suggested by the relation between the Mmax size and postinjury time (see fig 4). This hypothesis is supported by the fact that we found no such decrease with postinjury time for the Mmax of incomplete SCI subjects. Together with the H/M result that demonstrated no large difference in reflex excitability at the spinal motoneuron level, it is possible that alteration in the muscle proprioceptors and in the muscle mechanical properties played the major role in the higher relative stretch reflex amplitudes of both SCI groups. The relatively higher stretch reflex and similar H/M ratio of the elderly subjects in comparison with those of the younger subjects also support this idea (see figs 3A, 3B).

CONCLUSIONS

Our results in this study demonstrated that stretch reflex excitability after SCI differed markedly in chronically motor-complete and incomplete SCI subjects. Regardless of the underlying neuronal mechanisms, the difference most probably reflects unique neurologic and histologic reorganization processes taking place after complete or incomplete SCI. Specifically, the elevated reflex excitability in people with incomplete SCI is said to be closely related to their motor recovery with and without training such as unloaded body weight–supported treadmill (BWST) training.[20] Future studies will focus on the effects of sensorimotor interventions such as BWST on the competitive relation between central inputs via spared pathways and peripheral afferents mediated by reflex pathways at spinal motoneurons.

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Suppliers

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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

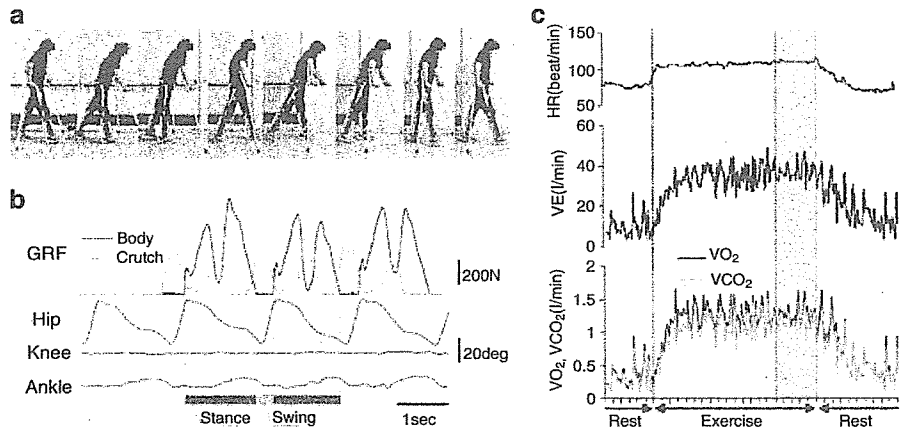

Figure 1 (**a**) Sequential picture of walking with the ARGO in a SCI subject (injured at Th12). (**b**) Time series data of the GRF (body and stick) and joint angle motion (Hip, Knee, and Ankle) obtained by the VICON system. (**c**) Typical example of the changes in the cardiorespiratory parameters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

$$\text{Energy consumption (J/kg/s)} = \frac{\text{Ambulatory min } VO_2 \text{ (ml/min)}}{\text{Weight (kg)} \times 60} \times K$$

$$\text{Energy cost (J/kg/m)} = \frac{\text{Ambulatory min } VO_2 \text{ (ml/min)}}{\text{Speed (m/min)} \times 60} \times K$$

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, VO_2 reached a plateau level about 3–4 min after the beginning of exercise. The steady-state value of VO_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, VO_2, and HR at rest and during orthotic gait exercise

	VE (ml/kg)		*VO₂ (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

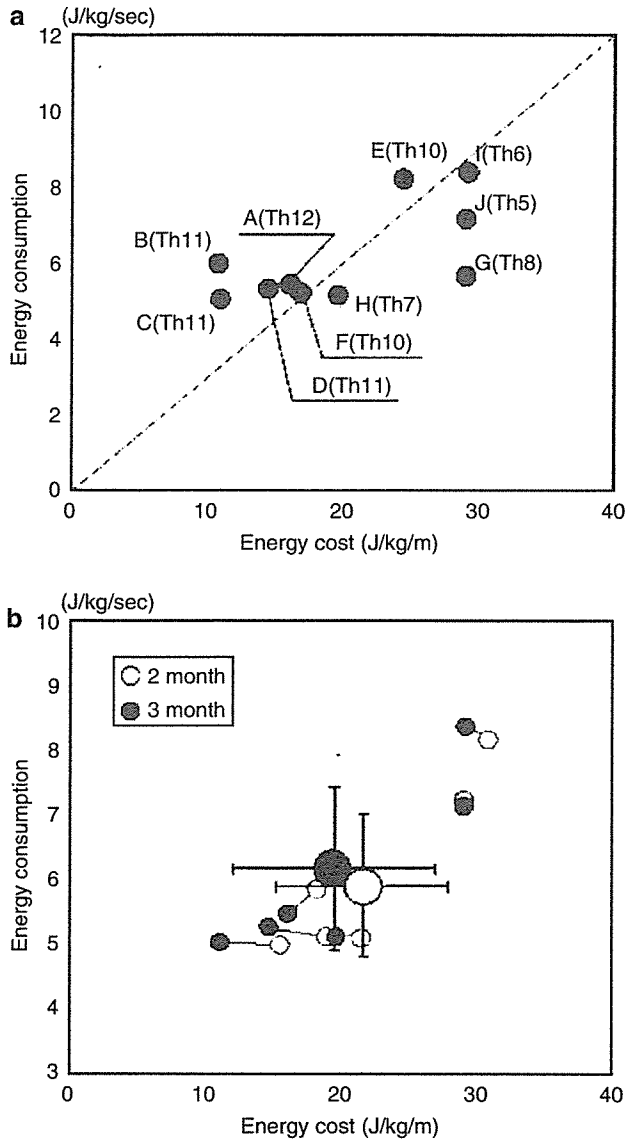

Figure 2 (**a**) The relationship between energy consumption and cost in each investigation. The slope of each line from zero to each plot reflects the walking speed. The subjects represented by points plotted on the upper and left side of the figure can be considered to have adequate aerobic conditioning.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

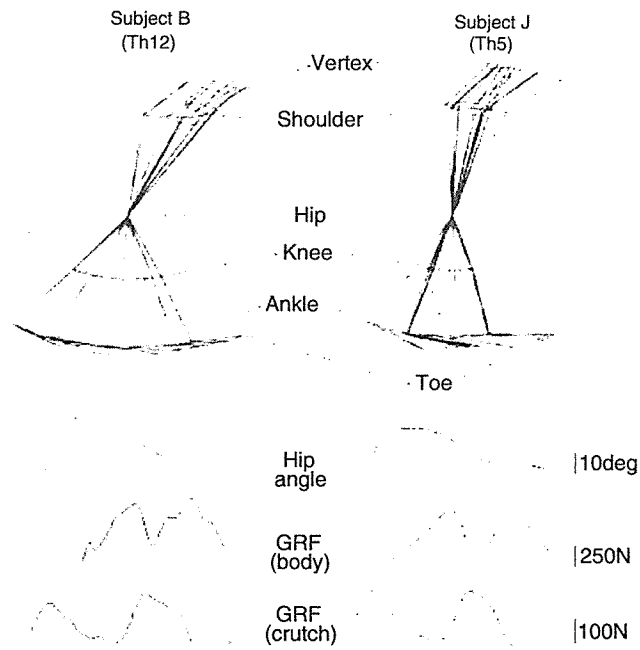

Figure 3 Differences in the gait motion between SCI subjects injured at a lower (left; Th12) and higher level (right; Th5). The stick pictures represented were standardized by the marker on the GTR

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*

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