SHORT REPORT

Long term effects of locomotor training in spinal humans

M Wirz, G Colombo, V Dietz

Abstract

The long term effects of locomotor training in patients with spinal cord injury (SCI) were studied. In patients with complete or incomplete SCI coordinated stepping movements were induced and trained by bodyweight support and standing on a moving treadmill. The leg extensor muscle EMG activity in both groups of patients increased significantly over the training period, associated with improved locomotor ability in those with incomplete SCI. During a period of more than 3 years after training, the level of leg extensor EMG remained about constant in incomplete SCI in those who regularly maintained locomotor activity. By contrast the EMG significantly fell in those with complete SCI. The results suggest a training induced plasticity of neuronal centres in the isolated spinal cord which may be of relevance for future interventional therapies.

(*J Neurol Neurosurg Psychiatry* 2001;**71**:93–96)

Keywords: locomotion; spinal cord injury; leg muscle EMG activity; motor learning

ParaCare, Paraplegic Centre of the University Hospital Balgrist, Forchstrasse 340, CH—8008 Zurich, Switzerland
M Wirz
G Colombo
V Dietz

Correspondence to:
Professor V Dietz
dietz@balgrist.unizh.ch

Received 21 August 2000
and in revised form
7 December 2000
Accepted 11 December 2000

Table 1 Patients with SCI included in the study

Patient No	*Sex*	*Age (y)*	*Level of lesion*	*ASIA score at beginning of treadmill training*	*ASIA score at end of treadmill training*	*Duration of treadmill training (days)*
Complete SCI:						
1	M	46	C5	A	A	343
2	M	23	C6	A	A	219
3	M	28	C6	B	B	158
4	M	42	C7	A	A	101
5	M	21	C7	A	A	210
6	M	42	T1	A	A	139
7	M	41	T1	A	A	85
8	F	19	T1	A	A	112
9	M	53	T1	B	B	148
10	M	27	T1	A	A	71
11	M	35	T10	A	A	90
12	F	14	T3	A	A	120
13	M	50	T4	A	A	273
14	M	33	T6	A	A	84
15	M	26	T6	A	A	103
16	M	53	T8	B	B	113
					Mean:	148
Incomplete SCI:						
17	M	24	C4	C	C	288
18	M	51	C4	C	D	126
19	M	47	C4	D	D	107
20	M	72	C6	D	D	174
21	M	13	C7	D	D	147
22	M	20	C8	D	D	165
23	M	33	L3	D	D	98
24	M	37	T10	C	D	36
25	M	30	T11	C	D	49
26	M	32	T11	D	D	91
27	F	29	T11	C	D	175
28	M	39	T11	D	D	35
29	M	31	T12	C	D	196
30	M	23	T12	C	D	103
31	M	59	T7	D	D	154
32	M	11	T7	C	C	60
					Mean:	125

SCI=Spinal cord injury.

It has been known for several years that patients with spinal cord injury (SCI) profit from specific locomotor training experiments,[1–3] based on cat studies. In the spinal cat the generation of coordinated leg muscle EMG activity on a moving treadmill was first described in 1980.[4] Similar step-like automatic movements are present at birth and in anencephalic children.[5] Evidence arose that in adult humans also, spinal interneuronal circuits exist which are involved in the generation of locomotor activity.[2 6] The locomotor function of incomplete paraplegic patients was shown to profit by specific training on a treadmill with partial body weight support.[3 7] Certain guidelines must be followed to make the training effective.[2 6 7] The beneficial effect of this training critically depends on a physiological proprioceptive afferent input—for example, from load receptors[8]—to the spinal locomotor centres. Even in patients with a complete SCI, a locomotor pattern could be induced.[1] The aim of this study was to investigate the course of locomotor EMG activity after the end of locomotor training in patients with complete and incomplete SCI.

Patients and methods

GENERAL PROCEDURES

Consent was obtained from patients and the local ethics committee to make recordings in 32 patients with SCI. The clinical diagnosis of spinal cord lesion was confirmed by electrophysiological[9] and radiological[10] examinations. Patients with peripheral nerve damage were excluded.

Thirty two patients had either a complete motor SCI according to the American Spinal Cord Injury Association (ASIA) classification[11] (A or B (n=16; mean age 35 (SD 12) years)) or had an incomplete motor SCI (ASIA C (n=2) or D (n=14) (mean 35 (SD 16 years)) (table 1). The level of lesion ranged from C4 to T12 (one patient with incomplete SCI at L3) with about one third at the cervical and two thirds at the thoracic level (see table 1).

All patients were admitted to our centre between 1993 and 1997 for primary rehabilitation. They underwent daily locomotor training on a treadmill (roughly 300 m of walking (about 15 minutes), 1.5 km/h speed, 5 days a week) starting between day 32 to 347 after SCI (mean 96 (SD 64)). The training on average lasted 137 days (SD (SD 72). In all patients with complete SCI and at the beginning of the training in most patients with incomplete SCI (13 of 16), leg movements had to be assisted by physiotherapists and body weight had to be partially unloaded. Unloading was achieved by suspending the patients over the treadmill by a parachute harness connected to counterweights. The amount of unloading was adapted to the patients' capability of performing stepping movements (up to 80% of body weight). After training had finished, 11 patients with incomplete SCI needed neither unloading nor external assistance and were able to perform stepping movements on normal ground conditions; two patients could walk without unloading but needed some assistance. In all patients with complete SCI body weight support could be reduced during the course of the training, although none of these patients could perform stepping movements without assistance after finishing locomotor training.

Recordings at three points were made weekly: EMG activity from the gastrocnemius medialis muscle using surface electrodes; the force exerted on the treadmill using force plates located underneath the treadmill; and from movements of the knee joints using mechanical goniometers fixed at the lateral aspect of the right and left knees. Follow up recordings were also made after finishing the locomotor training as outpatients. The number of outpatient measurements and the intervals between them varied due to the availability of the patients (a mean of five measurements over a mean period of 1.9 years (SD 1.4), range 0.13–5.8 years)).

We also analysed the time course of the amplitude of muscle action potentials (MAPs) of the abductor hallucis muscle evoked by tibial nerve stimulation in four patients with complete SCI, for more than 4 years.

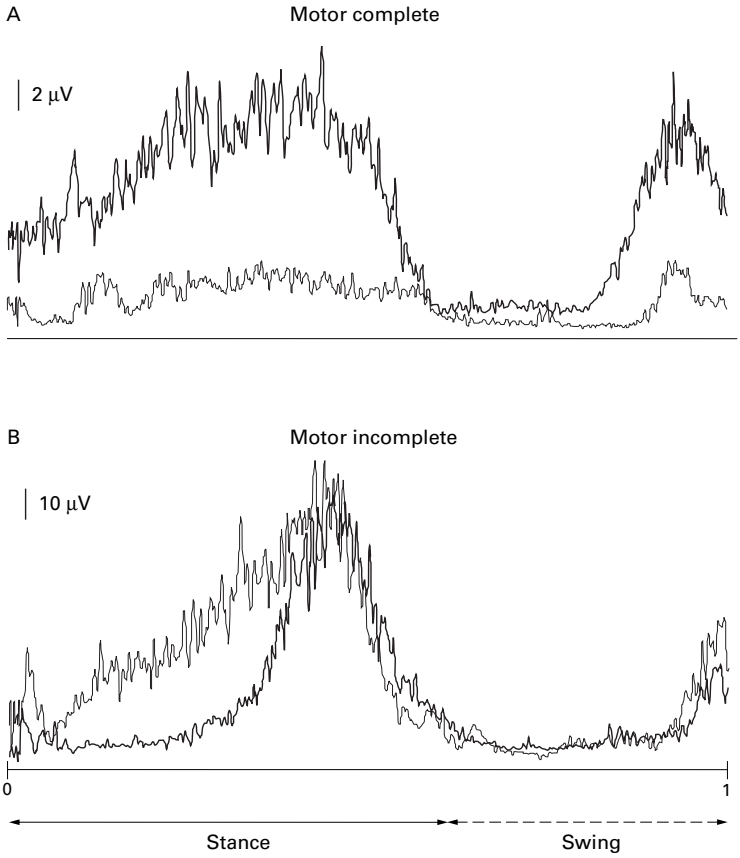

Figure 1 Rectified and averaged (n=20 strides) gastrocnemius EMG during one step cycle of a patient with (A) complete paraplegia T4 and (B) a patient with incomplete tetraplegia C4. The black lines represent the EMG activity at the end of the locomotor training. The grey lines indicate the activity at (A) 335 days and (B) 893 days after finishing locomotor training. Note the different calibrations of the EMG signal in (A) and (B).

DATA ANALYSIS

The EMG recordings were amplified (μV amplifier; bandpass filter, 30–300 Hz) and, together with the biomechanical signals, were converted (a/d) into a digital signal and transferred to a PC system. Signals were sampled at 500 Hz. The EMG signals were rectified and averaged over 20 step cycles. The force signal indicating the heel strike and beginning of the stance phase served as a trigger to average the EMG signal and to normalise the recordings to one stride cycle (for further details see Dietz *et al*[1 6]

To investigate changes in amplitude of EMG activity in the gastrocnemius medialis muscle as a function of time, the signal energy (root mean square (RMS)) was determined for the stance (5%–25% before the end of the stance phase) and for the swing phase (5%–25% after the end of stance). The knee joint signal was used to detect the end of the stance phase. During the stance phase, the main activation and during swing no activation of the gastrocnemius medialis muscle was expected to occur.[6] The RMS during swing was subtracted from the RMS during stance to eliminate noise and tonic muscle activation. To allow intersubject comparison within the two groups of patients with complete and incomplete SCI, measurements were normalised to the mean RMS amplitude of the three final recordings before finishing locomotor training. These values have been correlated with the time elapsing after discharge. Because the number of the recordings differed between patients, Pearson's weighted correlation coefficient was taken for calculation.

A Dantec Keypoint 1000 EMG machine was used to record M waves. The maximal amplitude of muscle action potentials was obtained by a supramaximal stimulation (single rectangular wave stimuli of 0.5 ms duration, ≤100 mA) of the tibial nerve at the level of the ankle. The M waves (MAPs) were recorded in four patients over the abductor hallucis muscle using surface electrodes. The maximal MAP amplitude (baseline to peak) was determined. The registration was done before, during, and after the locomotor training in patients with complete SCI to assess whether muscle atrophy due to disuse had occurred. The MAP amplitude of the four patients has been correlated with the time elapsing after discharge (Pearson's correlation coefficient).

Results

Figure 1 shows two examples of gastrocnemius muscle EMG during one step cycle of (A) a patient with complete paraplegia T4 and (B) a patient with incomplete tetraplegia C4 (central cord syndrome). The EMG signals were rectified, averaged (n=20 step cycles), and smoothed. The black lines represent EMG activity of the gastrocnemius medialis muscle at the end of locomotor training. The grey lines indicate the EMG activity at (A) 335 days and (B) 893 days after finishing locomotor training. In the patient with incomplete SCI the amplitude of the EMG signal was somewhat larger during early stance during the late recording, but otherwise similar at the two time points. In the patient with complete SCI the EMG signal was considerably reduced in amplitude during the late recording.

Figure 2 shows the course of locomotor activity in patients (A) with complete and, (B) incomplete SCI over time after locomotor training had finished. In both groups of patients there was a significant (p>0.001) increase of EMG activity of the gastrocnemius muscle during the period of locomotor training (left side of time=0). This increase seemed to be greater in the group of patients with incomplete SCI. In patients with complete SCI the absolute level of RMS amplitude at this time was about one third of the corresponding activity level seen in incomplete paraplegic patients.

After finishing locomotor training all patients with incomplete SCI used their locomotor capability every day on normal ground conditions (see methods). In these patients EMG activity of the gastrocnemius medialis muscle changed marginally over time after the end of training (r=−0.01; p>0.05, see fig 2 A. No patient showed a significant decline). By contrast, all patients with complete SCI never performed stepping movements after locomotor training stopped. These patients developed a significant decrease of gastrocnemius medialis muscle EMG activity over time (r=−0.29; p<0.01, see fig 2 B). The regressions between patients with complete and those with incomplete SCI were significantly different (p<0.01).

Neurographic recordings were performed over time in four patients with complete SCI to assess eventual signal changes in EMG activity due to muscle atrophy. The MAP amplitude (abductor hallucis muscle) evoked by tibial nerve stimulation did not change over time (r=0.05; p>0.05).

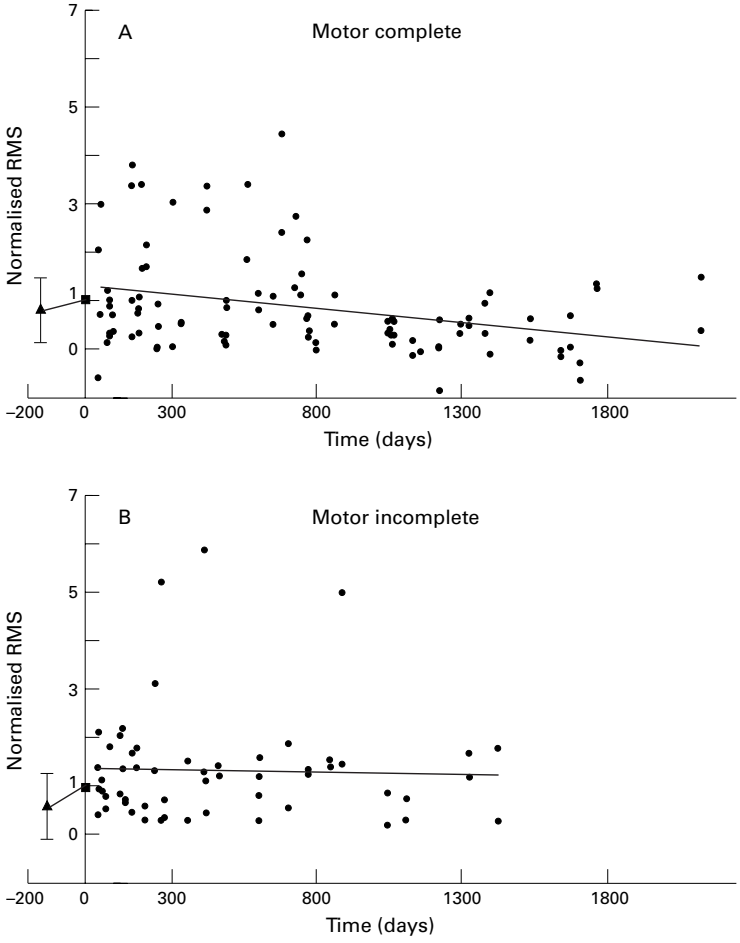

Figure 2 Course of gastrocnemius EMG activity after finishing locomotor training: (A) at t=0, 16 patients with complete motor paraplegia (ASIA A or B; n=99 recordings) and (B) 16 patients with incomplete paraplegia (ASIA C or D; n=55 recordings). The RMS values were normalised to the mean of the final three EMG recordings before finishing locomotor training (filled squares). Each point represents one recording (average 5/patient). The filled triangle reflects the normalised mean RMS amplitude of the first three recordings of all patients at the beginning of locomotor training. In patients with complete paraplegia the EMG amplitude declines over the time course (y=−0.0006x+1.3039; r=−0.29; p<0.01) whereas in incomplete paraplegic patients the level of EMG activity remains about constant (y=0.0000424x+1.3527; r=−0.008; p>0.05).

Discussion

Several reports indicate that patients with incomplete SCI profit from locomotor training.[2 6] The basis for this improvement in locomotor activity seems to be mainly due to an adaption of spinal neuronal networks to a physiological proprioceptive input after SCI. In patients with incomplete SCI a strengthening of cortical input[12] might also contribute to the functional improvement.

The aim of this study was to evaluate the long term effects of such training on the leg extensor EMG activity in patients with incomplete and complete SCI. We concentrated on the EMG activity of the gastrocnemius medialis muscle, as this muscle was shown earlier[1 6] to be most sensitive to locomotor training (by contrast with the tibialis anterior muscle) and as a consequence was overriding antigravitational function during locomotion. The results indicate that a regular locomotor training is effective in improving locomotor (gastrocnemius medialis EMG) activity in both patients with complete and those with incomplete SCI. Different long term effects of this training on locomotor activity between the two groups of patients was found.

In patients with incomplete SCI who regained ambulatory capacity, either functional or therapeutic,[9] leg extensor EMG activity did not change over time after finishing locomotor training. All these patients used their "learned" locomotor activity daily to walk at least short distances. Obviously this practice is sufficient to maintain the level of locomotor activity achieved during training. However, there is evidently no futher improvement in the more practical use of the locomotor pattern.

Patients with complete SCI could not use their acquired locomotor activity after finishing locomotor training as they remained unable to induce stepping movements. These patients lost the "learned" capacity of spinal neuronal networks to produce reasonable leg extensor EMG activity during assisted walking. The preserved MAP amplitude over time indicates that the decline in EMG activity is not due to muscle atrophy because of disuse, or peripheral nerve degeneration.

It is recommended that locomotor training is started in patients with incomplete SCI as early as possible so as to use a long training period and to establish optimal conditions for the time when some supraspinal control is regained. By such an approach a high level of locomotor function can be achieved. For patients with complete SCI the results may be of importance for future interventional therapies promoting some regeneration. In such a situation a maintained level of trained locomotor activity may be of benefit to regain locomotion.

We thank Monica Stüssi and Evelyn Windisch for their technical assistance and Thomas Erni for statistical support. This work was supported by the Swiss National Foundation (No 31–53526.98).

1 Dietz V, Colombo G, Jensen L. Locomotor activity in spinal man. *Lancet* 1994;**344**:1260–3.
2 Wernig A, Müller S. Laufband locomotion with body weight support improved walking in persons with severe spinal cord injuries. *Paraplegia* 1992;**30**:229–38.
3 Barbeau H, McCrea DA, O'Donovan MJ, *et al*. Tapping into spinal circuits to restore motor function. *Brain Res Rev* 1999;**30**:27–51.
4 Forssberg H, Grillner S, Halbertsma J, *et al*. The locomotion of the low spinal cat II. Interlimb coordination. *Acta Physiol Scand* 1980;**108**:283–95.
5 Forssberg HA. Developmental model of human locomotion. In: Grillner S, Stein PSG, Stuart DG, *et al*, eds. *Neurobiology of vertebrate locomotion. Wenner-Gren international symposium series.* Vol 45. London: Macmillan, 1986:458–501.
6 Dietz V, Colombo G, Jensen L, *et al*. Locomotor capacity of spinal cord in paraplegic patients. *Ann Neurol* 1995;**37**: 574–82.
7 Wernig A, Nanassy A, Muller S. Laufband (treadmill) therapy in incomplete paraplegia and tetraplegia. *J Neurotrauma* 1999;**16**:719–26.
8 Harkema SJ, Hurley SL, Patel UK, *et al*. Human lumbosacral spinal cord interprets loading during stepping. *J Neurophysiol* 1997;**77**:797–811.
9 Curt A, Dietz V. Ambulatory capacity in spinal cord injury: significance of somatosensory evoked potentials and ASIA protocol in predicting outcome. *Arch Phys Rehabil* 1997;**78**: 39–43.
10 Metz GAS, Curt A, van de Meent H, *et al*. Validation of the weight-drop contusion model in rats: a comparative study of human spinal cord injury. *J Neurotrauma* 2000;**17**:1–17.
11 Maynard FM, Bracken MB, Creasy G, *et al*. International standards for neurological and functional classification of spinal cord injury. *Spinal Cord* 1997;**35**:266–74.
12 Davey NJ, Smith HC, Wells E, *et al*. Responses of thenar muscles to transcranial magnetic stimulation of the motor cortex in patients with incomplete spinal cord injury. *J Neurol Neurosurg Psychiatry* 1998;**65**:80–7.