Journal of Neurology, Neurosurgery, and Psychiatry 1992;55:20–24

Changes in corticospinal facilitation of lower limb spinal motor neurons after spinal cord lesions

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Abstract

The projections from the cortex to the motor neurons of lower limb muscles were examined in 33 normal subjects and 16 patients with incomplete spinal cord lesions. Corticospinal neurons were excited by transcranial magnetic stimulation and the effects on single spinal motor neurons determined from peristimulus time histograms (PSTHs) of single tibialis anterior (TA) and soleus (SOL) motor units. In normal subjects magnetic stimulation produced a short latency facilitation of TA motor units but had little or no effect on SOL motor units. In the patients with spinal cord lesions magnetic stimulation also produced facilitation of TA but not SOL motor units; however, the mean latency of the TA facilitation was significantly longer (by about 14 ms) in the patient group. The F wave latencies were normal in all patients tested, suggesting that central rather than peripheral conduction was slowed. The duration of the period of increased firing probability (in TA motor units) was also significantly longer in the patients with spinal cord lesions. These changes may reflect the slowing of conduction and dispersal of conduction velocities in the corticospinal pathways as a consequence of the spinal cord lesion. No significant correlations were found between the delay of the TA facilitation and the clinical deficits in this group of patients.

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Received 22 January 1991 and in revised form 18 April 1991.
Accepted 13 June 1991

Transcranial stimulation of the human motor cortex by electrical[1] or magnetic[2] stimuli produces contractions of contralateral limb muscles. The latency and brevity of the facilitation of motor neurons[3–5] and the distribution of the effects to various motor neuron pools[6] are thought to indicate that these short latency effects result from the activation of the fast corticospinal pathway.[4,7] The amplitude and latency of the evoked compound muscle action potentials have been shown to be abnormal in a variety of neurological disorders.[8–11] Transcranial stimulation may be used to detect corticospinal involvement in patients with spinal cord lesions,[12,13] and this technique could be used to predict outcome after spinal cord injury.[14]

The purpose of this study was twofold. The first was to examine the pattern and characteristics of the short latency corticospinal projections to lower limb spinal motor neurons in patients with incomplete spinal cord lesions. The second was to determine whether there was a relation between the characteristics of these projections and the patient's clinical profile.

Methods

Patient selection

Subjects with incomplete spinal cord lesions were identified by physicians at Lyndhurst Hospital and screened by one of us (JB). Patients with weakness of the lower limbs who were able to voluntarily contract the tibialis anterior (TA) muscle and showed an increase in resistance to passive stretch and exaggerated deep tendon reflexes were considered for this study. Patients having cardiac pacemakers, intracranial metal implants, or a history of epilepsy were excluded. The experimental procedures were approved by the local ethical review board and informed consent was obtained.

Clinical examination

A clinical neurological examination was carried out on all patients to document the level and duration of the lesion and the degree of motor and sensory impairment. The overall severity of the spinal cord lesion was assessed and a Frankel grade (A to E) assigned. In this grading system grade A corresponds to complete motor and sensory loss; B to complete motor loss, but incomplete sensory loss; C to incomplete motor loss severe enough to impair function; D to incomplete motor loss with some function (in grades C and D sensation may be variably preserved); and E to normal motor and sensory function.[15] Strength was scored from 0 (no movement) to 5 (normal). The ankle jerks were scored from 0 (no response) to 4 (brisk), a score of 2 being normal. The flexion response was tested by pinching the toes and was scored from 0 (no response) to 2 (limb movement). The patient's current medication was noted on the day of testing.

Magnetic stimulation

A Cadwall MES-10 electromagnetic stimulator was used for all studies. When this device is triggered capacitors are discharged, causing a current to flow into a 14 turn circular coil (inside and outside diameter 7·5 cm and 9·0 cm respectively) encased in epoxy resin. This current varies the magnetic field, which in turn induces circular currents in nearby conducting tissues. These currents are proportional to the

rate of change of the magnetic field and the initial stored charge.[16] The charge is preset according to a linear scale ranging from 0% to 100%. The maximum magnetic field generated by this stimulator is 2 Tesla (according to the manufacturer's specifications) and the induced current waveform is an underdamped sinusoid lasting 800 μs with an initial peak at approximately 5 μs and a peak of opposite polarity at 135 μs.

The stimulating coil was positioned flat on the scalp overlying the motor cortex with the centre aligned midway between the vertex and either C3 or C4 (international 10–20 system of electrode placement).[17] Single stimuli were delivered about every 3 s at an intensity just below that which produced a contraction of the weakly contracted TA muscle. In patients with spinal cord lesions in whom a threshold intensity for lower limb activation could not be established in this way, the intensity was set 10% higher than that at which a visible contraction was first evoked in any relaxed upper limb muscle.

Motor unit recordings

Single motor unit recordings were obtained in turn from the TA and SOL muscles contralateral to the site of stimulation by using a concentric needle electrode with a recording surface area of 0·07 mm^2 (Dantec 13L49). The needle was positioned near a motor unit that was activated by gentle voluntary contraction or was discharging spontaneously. The motor unit action potentials were amplified (bandpass 10 Hz to 10 kHz), extracted with a window discriminator, and displayed on a storage oscilloscope using a delay line. The pulse from the discriminator was led to a loudspeaker and tachometer. Subjects were provided with visual and auditory feedback of the unit's discharges and instantaneous firing rate and were instructed to keep the unit discharging steadily while a minimum of 100 magnetic stimuli were delivered to the contralateral cortex.

Peristimulus time histograms (PSTHs) of each individual motor unit's discharge times were generated to record changes in firing probability produced by the magnetic stimulus. The mean background firing probability of a given unit was calculated from the 100 ms prestimulus portion of each PSTH. In the post stimulus portion a coherent cluster of at least two bins (bin width 1 ms) with amplitudes greater than the background mean plus two standard deviations was defined as a period of increased firing probability.[18] The area of the peak of increased firing probability above the mean background level (expressed as extra counts per 1000 stimuli) provided an estimate of the magnitude of the underlying composite excitatory postsynaptic potential (EPSP) produced by the descending volley, and the duration of the PSTH peak provided an estimate of the EPSP rise time.[19–22] The latencies of the peaks were corrected for the unit's rise time to estimate the conduction time from the brain to the motor unit in the muscle of interest.

The patient data were compared (by *t* tests) with normal values previously obtained in this laboratory and reported elsewhere.[23]

F waves and central conduction time

The shortest latency F wave[24] from 10 trials was measured from TA in response to supramaximal electrical stimulation of the common peroneal nerve at the head of the fibula. Compound muscle action potentials were recorded from TA with the active electrode placed at the approximate location of the needle electrode insertion.

The central conduction time was estimated using the following formula:

$$\text{Central conduction time} = \text{TA latency}_{mag} - ((\text{F wave} + \text{M wave})/2 + 1)$$

where TA latency$_{mag}$ refers to the latency of the facilitation of the TA motor unit in that subject after magnetic stimulation. All measures refer to their respective latencies (in ms), and 1 ms is allowed for the synaptic delay between the corticospinal axons and the alpha motor neuron.[6]

Table 1 Summary of clinical findings in 16 patients with spinal cord lesions

Case No	Age (years)	Sex	Aetiology	Lesion	Time after lesion (years)	Frankel grade (A–E)	TA strength (0–5)	Ankle jerk (0–4)	Flexion response (0–2)	Latency of TA facilitation (ms)*	Estimated CCT (ms)
1	26	M	Trauma	C_4	0·5	D	3	4	1	35	15·9
2	45	M	Trauma	C_4	0·2	D	3	4	0	36	17·5
3	32	M	Trauma	C_5	2·0	C	5	3	2	42	ND
4	36	M	Trauma	C_5	0·6	D	1	3	1	37	18·0
5	17	M	Trauma	C_6	0·6	C	1	4	1	42	ND
6	37	M	Trauma	C_6	0·7	D	1	4	1	39	19·0
7	27	F	Trauma	C_6	0·8	D	2	2	1	56	36·6
8	23	M	Trauma	C_7	3·0	C	2	4	2	31	10·9
9	56	M	Trauma	T_4	5·5	D	1	3	1	38	ND
10	24	M	Vascular	T_6	0·1	D	2	4	1	42	ND
11	67	M	TSP	T_8	17·0	D	2	3	1	41	23·9
12	53	M	TSP	T_8	5·0	C	2	3	2	41	15·4
13	53	M	Ep	T_9	2·0	C	4	2	2	39	18·0
14	28	M	Trauma	T_{10}	1·0	D	5	4	0	60	ND
15	32	M	Trauma	T_{12}	0·5	D	4	4	1	Absent	—
16	31	M	Trauma	T_{12}	0·2	D	3	3	0	38	16·4

*Corrected for the risetime of the motor unit action potential; see text for details on scoring.
TSP = Tropical spastic paresis.
Ep = Ependymoma.
CCT = Central conduction time.
ND = Not done.

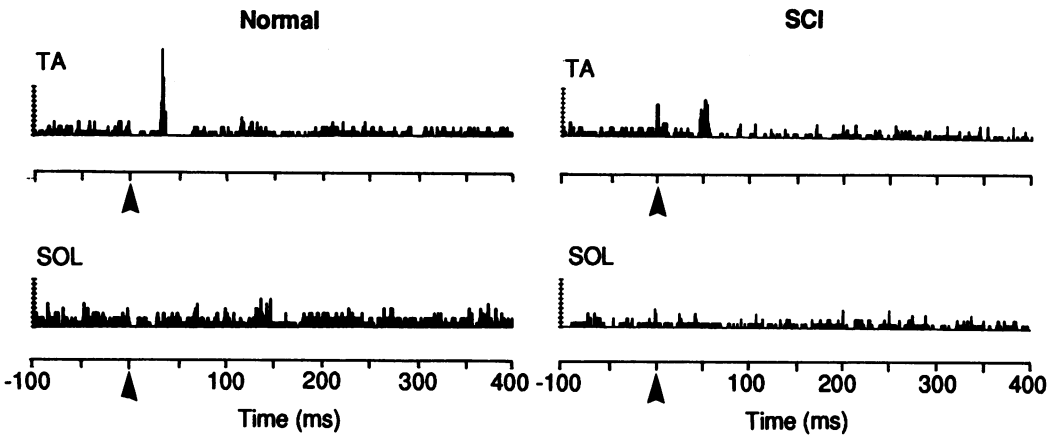

Figure 1 Representative PSTHs from a normal subject (left) and patient (case 12) (right) showing changes in the firing probability of single TA and SOL motor neurons in response to magnetic stimulation applied over the contralateral motor cortex at time zero (arrowhead). There is a strong facilitation of TA motor units (top) but not of SOL motor units (bottom). The apparent facilitation at time zero of the PSTHs from the patient is due to stimulus artefact.

Results

Clinical findings

Studies were carried out on 33 normal subjects (mean age 25·0 (SD 6·7)) and on 16 patients with incomplete spinal cord lesions (mean age 36·7 (14·1)) due to trauma (12 patients), tropical spastic paraparesis (two), ependymoma (one), or vascular lesion (one). All but one patient (case 8) were able to take a step with aids and one patient (case 7) walked regularly with canes. All patients had Frankel grades of C or D and all had some sensory loss. A summary of the clinical findings is presented in table 1.

Motor unit recordings

Magnetic stimuli applied to the contralateral hemisphere resulted in strong facilitation of TA motor units in all normal subjects and in 15 out of 16 patients. In the one patient in whom a response was not observed the stimulus may have been insufficient, although the intensity was chosen according to the criteria described in the methods. Only three of the normal subjects and three of the patients showed short latency facilitation of SOL motor units and in all cases the facilitation was much less than that of TA units. Typical recordings taken from a normal subject and a patient with a spinal cord lesion are shown in fig 1. The means of the PSTH peak areas (normalised to 1000 stimuli) for both groups are shown in fig 2. The relative magnitudes of the facilitations of TA and SOL motor units are similar in normal subjects and patients with spinal cord lesions.

The characteristics of the shortest latency PSTH peaks of TA motor units are summarised in table 2. (The general absence of responses in SOL precluded analysis of the short latency effects on these motor units.) The mean latency of the facilitation of TA units was significantly longer in patients with spinal cord lesions (41·1 ms) than in normal subjects (27·1 ms), $t_{(df = 47)} = 9·06$, $p < 0·001$. This is illustrated in fig 3, which shows PSTHs obtained from TA motor units in a patient and a normal subject of identical height. In only one patient (case 8) did the latency of the facilitation of TA fall within two standard deviations of the mean calculated for normal subjects.

The latency of TA facilitation will depend on the subject's height and on the conduction velocity of the alpha motor neuron axon. The mean heights for the two groups were 167·9 (7·5) cm (normal subjects) and 174·8 (6·1) cm (patients with spinal cord lesions), $t_{(df = 47)} = 2·71$, $p < 0·01$. There was no evidence that the conduction velocity in the alpha motor neuron axon was slow in the patient group. F waves were measured for 10 patients, nine of whom showed delayed facilitation of TA motor units after magnetic stimulation. In all cases the F wave latencies were normal.[13]

By subtracting the peripheral conduction time (estimated from the F wave) from the total latency it is possible to estimate the central conduction time. The average estimated central conduction time in the patients with spinal cord lesions was 19·2 (6·9) ms with a range from 10·9 ms to 36·6 ms. Mean central conduction times for normal subjects are between 9·5 ms[13] and 14·8 ms.[25]

The mean duration of the peak of increased firing probability in TA motor neurons was significantly longer in patients than in normal subjects ($t_{(df = 43)} = 2·61$, $p < 0·02$). The width (duration) of a peak is positively related to its

Table 2 Characteristics of short latency facilitations in TA motor neurons in patients with spinal cord lesions and normal subjects. Values are means (SD)

	Recording type	n	Extra counts/ 1000 stimuli	Mean onset latency (ms)	Mean duration (ms)
Patients	SU	12	199·2 (39·9)	40·1 (1·7)	7·4 (1·2)
	SU + MU	16	200·8 (32·8)	41·1 (1·9)	7·5 (1·0)
Normal	SU	33	237·4 (23·3)	27·1 (0·6)	5·1 (0·3)

SU = Single unit.
MU = Multi-unit.

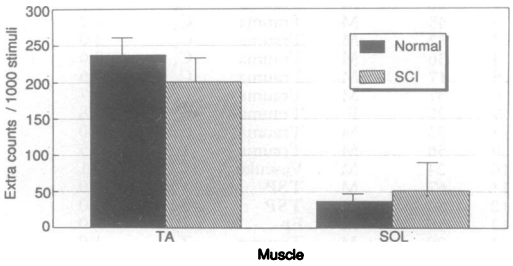

Figure 2 Mean amplitudes (SE) of the periods of increased firing probability (expressed as extra counts per 1000 stimuli) in TA and SOL motor units from 33 normal subjects and 12 patients with spinal cord lesions (single unit studies only). The pattern of activation is the same for both groups.

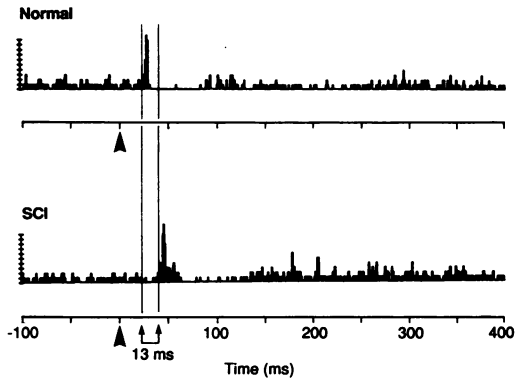

Figure 3 PSTH of a TA motor unit from a normal subject (top) and patient (case 13) (bottom) in response to 100 subthreshold magnetic stimuli applied over the contralateral motor cortex at time zero (arrowhead). The heights of these two individuals are identical (168 cm). The onset of the TA facilitation in the patient is 13 ms later than that of the normal subject (after correction for the rise time of the motor unit action potentials).

magnitude,[26] but this cannot account for our findings. There was no difference between the magnitudes of the peaks in patients and normal subjects ($t_{(df = 43)} = 0{\cdot}84$, $p > 0{\cdot}40$), and first order regression analysis of the relation between peak area and duration (fig 4) shows similar slopes but different y intercepts for the two populations ($p < 0{\cdot}01$).

Some patients had difficulty maintaining a steady contraction of the TA muscle and it was not possible to obtain recordings without occasional contamination by other units. As these patients generally had the greatest motor deficit and might have shown an alteration in projections not seen in other patients, their "multi-unit" recordings were analysed. As shown in table 2, the addition of these data made little difference to the results.

There was no apparent relation between the patient's clinical profile and the magnitude, latency, or duration of the facilitation observed in TA. Correlation coefficients between TA strength and PSTH peak amplitude or latency and between the time post-lesion and PSTH amplitude or latency were in all cases less than 0·20.

Discussion

Magnetic stimulation over the motor cortex in normal subjects produces strong short latency facilitation of TA motor neurons but not of SOL motor neurons. This has been interpreted as reflecting the relative strength of the projections of the fast conducting corticospinal pathway to these motor neurons.[23 27] This pattern seems to be the same in patients with spinal cord lesions, suggesting that the projection pattern of the corticospinal tract is not changed as a result of adult onset spinal cord lesions, even when these are long standing (up to 17 years). In contrast, the pattern of corticospinal projections is altered after lesions of the developing brain.[23 27]

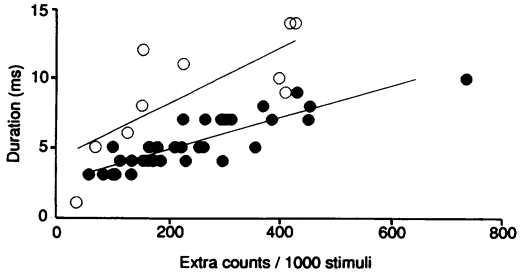

Figure 4 Relation between the magnitude and duration of the PSTH peaks obtained from TA motor units in normal subjects (●) and patients with spinal cord lesions (○).

The magnitude of the facilitation of TA motor units in the patients with spinal cord lesions was about the same as in normal subjects. This may be related to the selection of patients (who had to be able to contract TA) and to the way the intensity of the stimulus was chosen. The responses were, however, significantly delayed. The delay was probably not due to an increased peripheral conduction time as the F wave latencies were normal. F waves represent activity in large diameter alpha motor neuron axons whereas the motor units first recruited by voluntary contraction are probably of small diameter. If the delays in the PSTH peaks were due to an increased peripheral conduction time in the patients with spinal cord lesions there could have been selective slowing of conduction in small diameter alpha motor neuron axons. The delay is more likely to be occurring in the central nervous system. The patient group was on average slightly taller (by 7 cm) than the normal subjects, but this could not account for the longer latencies since this difference in height would be expected to produce an increase in latency of about 0·5 ms.[28] The possibility that we mistook a late response to magnetic stimulation for the short latency facilitation is unlikely as these responses rarely occur in the absence of short latency facilitation.[29] Furthermore, the late responses recorded from TA occur at latencies in excess of 100 ms.[29] The delayed facilitation of TA units in the patients with spinal cord lesions is therefore attributed to pathological changes in the corticospinal tract.

If so, there are several possible explanations. It could be that the fastest conducting corticospinal axons have been selectively lost and that the prolonged central conduction time reflects conduction in the smaller (<4 μm), slower conducting axons which constitute about 90% of the corticospinal tract.[30] If this were the case the central conduction times, assuming a conduction velocity of 20 m/s over a distance of 560 mm,[31] would be expected to be more than 28 ms. Our data do not support this.

An alternative explanation is that the descending volley is transmitted via a pathway other than the direct (monosynaptic) corticospinal tract. For example, a facilitatory pathway may exist whose effects are normally hidden in the period of inhibition which follows alpha motor neuron activation by the corticospinal volley.[32] Only the rubrospinal tract is known to have a similar pattern of projections onto spinal motor neurons, but it is believed to be vestigial in humans.[33]

A third possibility is that the conduction in the zone of the lesion has been slowed owing to

focal demyelination. Assuming normal central conduction to be about 65 m/s[34] and conduction in demyelinated fibres to be 2 m/s,[35] then demyelination over a 15–58 mm (mean 22 mm) segment would be required to explain total central conduction times of 16–37 ms (mean 19 ms).

Studies describing the pathology after spinal cord injury in humans report macroscopic cord lesions extending longitudinally 1–5 cm[36] and microscopic lesions including haemorrhages and disruption of myelin sheaths throughout 4–5 segments adjacent to the lesion.[37] This being the case, a demyelinated segment extending from 15–58 mm in length would be quite possible. Local demyelination would also explain the wider PSTH peaks in the patient group as the duration of the PSTH peak (which is related to the rise time of the underlying composite EPSP) would be expected to be longer because of the temporal dispersion from the non-uniformity of conduction velocities in the demyelinated region. In normal subjects the facilitation of TA is remarkably brief, suggesting that the corticospinal axons activated by cortical stimulation have very similar conduction velocities. Magnetic resonance imaging would have been helpful in determining the presence and extent of demyelination in patients with spinal cord injury[38] but was not available.

Similar delays in cortically evoked muscle activation have been observed in cases of cervical spondylosis, trauma,[12] and stenosis.[13] Like Masur *et al*[13] we were unable to show a significant correlation between the magnitude of the delay and the patient's functional abilities. Delayed central conduction may be related more strongly to disability in degenerative central nervous system diseases such as multiple sclerosis.[9]

In conclusion, this study provides evidence that the pattern of corticospinal projections to lower limb spinal motor neurons is not altered after incomplete spinal cord injury. The TA facilitation in response to cortical magnetic stimulation is significantly delayed in affected patients, probably reflecting slowing of conduction in the corticospinal pathway. The degree of slowing was not shown to be related to the clinical deficit in this group of patients.

This research has been funded by the Ontario Easter Seal Research Institute and the Canadian Medical Research Council. We thank the patients and staff of Lyndhurst Hospital for their support.

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