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STUDY OF SUPRASEGMENTAL AND SEGMENTAL CONTROL OF MUSCLE INNERVATION IN AMBULATORY SPINAL CORD INJURY PATIENTS

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ABSTRACT

Spinal and supraspinal generators of gait are not completely understood in healthy man, and even less in motor disabled patients. In order to distinguish between segmental and suprasegmental interaction in the neurocontrol of ankle flexors and extensors during gait, ambulatory spinal cord injury (ASCI) patients with intact segmental mechanisms as well as suprasegmental mechanisms above and impaired descending suprasegmental influence below the level of injury were used as a model. The purpose of this study was to describe the impaired suprasegmental control. Measurements in a sitting position examined monosynaptic stretch reflex (H-reflex) with superimposed volitional activity or vibratory tonic reflex, to independently assess the reciprocal relation between antagonists and presynaptic inhibition. EMG activity of tibialis anterior and triceps surae (soleus) muscles was recorded in 6 ASCI males with lesions from C3 to T10 (neural levels C5 to T11), 20 to 299 months post-onset, and compared to the recordings in 4 neurologically healthy adults. Both the reciprocity and presynaptic inhibition were impaired in all the patients. Measurements during gait, including EMG activity, joint angles, and foot-floor contacts, revealed severe kinesiological disabilities as well as altered motor unit activity in the ASCI patients, when compared to the control group. Central pre-programmed activity, recorded at the tibialis anterior in the control group, was diminished in the ASCI patients and this lack generated either tonic hyperactivity with coactivation of both ankle antagonists or hypotonia with motor deficits and an exaggerated segmental phasic stretch reflex. Additionally, there was also no evidence of suprasegmental functional stretch reflex, which was recorded in the control group. In spite of the insufficient descending control, the released segmental activity did not prevail.

INTRODUCTION

Studies using simultaneous recordings of motor unit activity, changes in leg joint angles and sequences of foot-floor contacts, have made it possible to demonstrate the presence of two distinct control mechanisms of the central nervous system activating the leg muscles during gait. The first one is a preprogrammed control of muscles and is related to the phase of gait but is independent of foot contact with the floor (1,2). The second is a central nervous system mechanism which is dependent upon and triggered by the stretching and loading of the muscle group at the foot-floor contact(3). It consists of the so-called short latency segmental stretch reflex and longer latency segmental and suprasegmental functional stretch reflex (4,5).

In incomplete spinal cord injury patients who recover the ability to ambulate with or without support, their gait is controlled by segmental and impaired suprasegmental mechanisms. Thus, the question is what kind of neuro-control is present during the gait of such patients. Theoretically, there is a possibility that an ambulatory spinal cord injury patient uses released segmental stretch reflex activity with minimal pre-programmed central descending neuro-control. On the other hand, released segmental stretch reflexes can generate exaggerated phasic activity which is an obstacle for the functional utilization of residual suprasegmental neuromuscular control.

To assess the degree of functional integrity between the upper motor neurone and segmental mechanisms, the influence of volitional suprasegmental control on the facilitation and suppression of the segmental stretch reflex (H-reflex) of the soleus muscle was tested first in this study. During the test, volitional motor activity was induced respectively in the soleus and tibialis anterior muscles which were later studied during gait. Also, the degree of impairment of presynaptic inhibition of the soleus was studied by testing the effect of vibration on the amplitude of the H-reflex response. After these two tests, the pattern of EMG activity of the soleus and the tibialis anterior was analyzed as related to foot-floor contact in order to differentiate between EMG activity dependent on floor contact and EMG activity dependent only on central pre-programming in the absence of immediate contact with the environment. It became important, therefore, to study residual central neurocontrol and its interference with segmental reflex mechanisms in addition to the quantification of gait.

MATERIAL

These studies were carried out on 6 ambulatory spinal cord injury (ASCI) patients, and on 4 control subjects. The patients were 28 to 59 years old, with motor and sensory incomplete lesions at bony levels C3 to T10 (neural levels C5 to T11) and were 20 to 299 months post onset (Table 1).

Table 1

Patient Characteristics:

#	Age	Bony Level of SCI	Onset (months)	Neurological examination	Ambulation
1.	50	C3-4	112	C5 M+S incomplete	Walker, slow, stiff, rt. worse
2.	28	T10	20	T10-11 M+S incomp.	Walker, slow, stiff, all jnts.
3.	49	C5-7	79	C6 M+S incomplete	2 canes, slow, hip/knee flexed
4.	52	C6,7	34	C5,6 Brown-Sequard	no support, left side worse
5.	56	T8	83	T10 Brown-Sequard	no support, right side worse
6.	59	T7-9	299	T7 M+S incomplete	2 canes, slow, left worse

Neurologically healthy adults were used as controls. There were 2 females (28 and 32) and 2 males (36 and 41 years old) for the H-reflex evaluation, and 1 female (28) and 3 males (23 to 41) for the gait study control group.

METHODOLOGY

H-Reflex Evaluation

The H-reflex evaluation was carried out in a sitting position by comparing the amplitude of responses while the subject was relaxed to the amplitude obtained during volitional contraction of the extensor (triceps surae) and flexor (tibialis anterior) muscle groups respectively. The values obtained while relaxed also served as a control for comparison with the amplitude obtained while vibrating the quadriceps femoris muscle and the Achilles tendon.

The H reflex was elicited by electrical stimulation of the tibial nerve at the popliteal fossa. Monophasic, constant current pulses with amplitudes from 10 to 20 milliamperes, a duration of 1 millisecond, and a frequency of 0.2 Hz were applied through a surface, monopolar electrode strapped tightly to the nerve by a frame, while a 5 by 10 cm conductive elastomere/karaya anode was placed above the knee. The position of the stimulating electrode was chosen where the optimal H-reflex was elicited in the soleus muscle. The stimulus amplitude was adjusted to the threshold of the M-wave, which was monitored for the consistency of stimulation. The stimulating pulses were provided by a Disa Neuromatic 2000 EMG analyzer, which was also used for measuring and averaging of the single H reflex responses.

Bipolar recording electrodes of silver-silver chloride (In Vivo Metric Systems) were placed 3 centimeters apart along the belly of the soleus muscle, between the lateral and medial heads of the gastrocnemius muscle. A strap ground was placed around the leg between the stimulating and recording electrodes. Simultaneously, EMG activity of the tibialis anterior muscle was monitored by another, equal surface EMG electrode pair over its belly.The electrodes were connected to Gould 13-4615-58 Universal Amplifiers over Gould 11-5407-58 Preamplifiers. Movements in the ankle joint were monitored by a planar, parallelogram ankle electrogoniometer (Ranchos Los Amigos Hospital, Downey CA) connected directly to a Gould 13-4615-58 Universal Amplifier. Ankle movements as well as EMG activity during volitional plantar and dorsal flexion and H-reflexes were monitored on a Gould 2800S stripchart recorder.

Averaged 20 H-reflex responses during volitional ankle plantar and dorsal flexion, vibration of the quadriceps muscle and the Achilles tendon respectively, were normalized by comparison to those obtained in the relaxed state. A pneumatic vibrator with an approximate frequency of 100 Hz and a displacement of 1 to 2 millimeters was used as the vibratory stimulus.

Activity during gait

The motor activity during different sequences of gait was studied together with its segmental and suprasegmental neurocontrol by simultaneous recording of EMG activity of the ankle extensor (triceps surae) and flexor

(tibialis anterior) muscles, of joint angles, and of the foot-floor contacts.

Beckman surface Ag-AgCl electrodes were placed over bellies of the tibialis anterior and triceps surae muscles. EMG activity during gait was recorded with Elema Shoenander Mingograph EEG16 ink jet recorder (frequency range from 3 to 700 Hz), and by Honeywell Model 96 analog magnetic tape recorder. Angles at the six leg joints were measured in the sagittal plane using MERU electrogoniometers and Clevite Brush Mark 200 8-channel stripchart recorder. Foil tape on the heel and mid-foot was employed to mark the phases of gait through contacts with an 8 meter metal surfaced walkway. The two foot contact patterns were recorded on the Elema stripchart recorder along with the EMG data, on the Brush recorder along with the goniograms, and on the magnetic tape recorder as well. The EMG data were subsequently processed from the analog storage using Preston high speed ADC in Hewlett-Packard 1000 computer system, in order to apply non-recursive filtering and averaging tools to the gait signals.

RESULTS

The reciprocal relationship between the ankle plantar and dorsal flexors was assessed by comparison of the magnitude of the H-reflex during volitional plantar and dorsal flexion respectively, with the one during relaxation. Average values of 20 H-reflex responses during the plantar flexion (empty bars) and dorsal flexion (dotted bars) for 4 neurologically healthy subjects and 6 patients are displayed in Fig. 1. The values were normalized by the averaged 20 relaxed responses. There was no significant difference in facilitation of H-reflex between the healthy subjects (6% in average) and patients (11%in average) during the plantar flexion. On the contrary, the average response was suppressed during the volitional dorsal flexion to 41% of the relaxed one in the healthy subjects and to 60% in the patients. Compared to the control group, the average ratio: facilitation/suppression was 1.40 times smaller in the patients. According to Fig. 1, the reciprocal relationship between ankle extensors and flexors was found to be impaired in the ASCI patients.

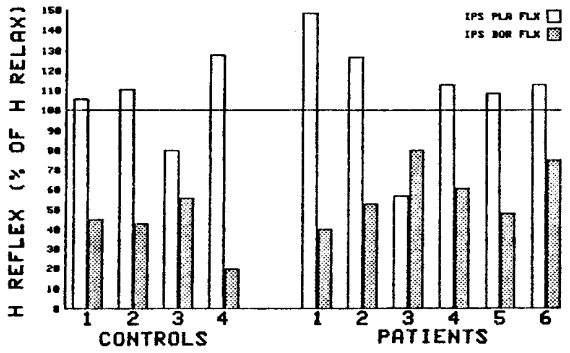

Fig. 1: H-reflex in soleus muscle during volitional extension and flexion of the ankle joint respectively.

When testing the presynaptic inhibition, the H-reflex responses during vibration of quadriceps muscle and Achilles tendon were compared with the ones during the relaxation. In Fig. 2, the averaged 20 responses, normalized by the 20 relaxed ones, are given for the healthy subjects and the patients during the vibration of quadriceps muscle (empty bars) and Achilles tendon (dotted bars). The average response was suppressed to 66% at quadriceps and to 15% at Achilles in the healthy subjects, while it showed 77% at quadriceps and 38% at Achilles in the ASCI patients, when compared to the relaxed response. Fig. 2 additionally displays, that the vibration of Achilles tendon suppressed the soleus H-reflex both in the healthy subjects and ASCI patients considerably more than the vibration of quadriceps muscle - healthy: 2.5 times, patients: 2.7 times. When compared to the control group, the average suppression of H-reflex during vibration of quadriceps muscle and Achilles tendon was smaller in the patients - quadriceps: 1.48 times, Achilles: 1.37 times, displaying thus a diminished presynaptic inhibition.

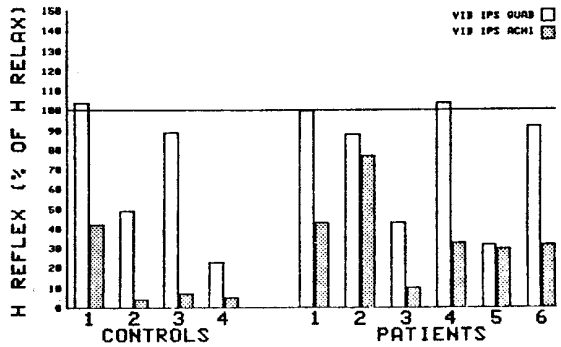

Fig. 2: H-reflex in soleus muscle during vibration of quadriceps muscle and Achilles tendon respectively.

General characteristics of gait of the ASCI patients were described by goniograms of the joint angles in sagittal plane, as well as by average stride length and gait velocity. The results in Fig. 3 clearly demonstrated severe impairment in all kinesiological parameters, when compared to the control group.

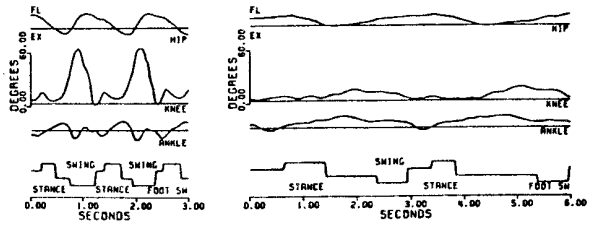

Fig. 3: Joint angles in healthy adult (left) and patient (right) during gait.

The goniograms of a neurologically healthy subject on the left side of Fig. 3 are given in the same amplitude and time scale as the ones of an ASCI patient on the right side. There, severely restricted ranges of motion and altered patterns of activity can be noticed besides 2.5 times slower gait. Similar goniograms were recorded in 3 ASCI patients, while in the other 3 the patterns approached those in the control group, although the excursions in all joints were limited.

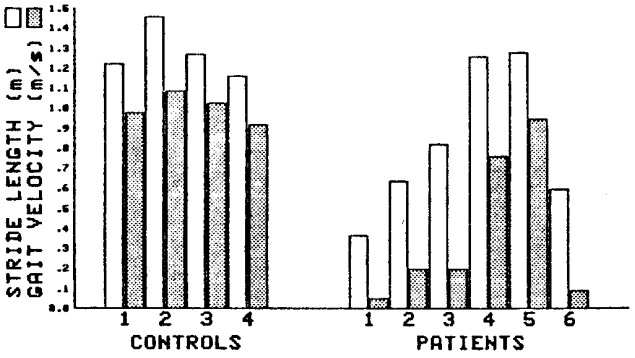

Fig. 4: Average stride length and average velocity of gait.

In Fig. 4, average stride length in meters (empty bars) and average gait velocity in meters per second (dotted bars) are given for the healthy subjects and ASCI patients. The foot contact data revealed in average 2.7 times slower gait with 1.5 times shorter stride length in the ASCI patients, when compared to the control group.

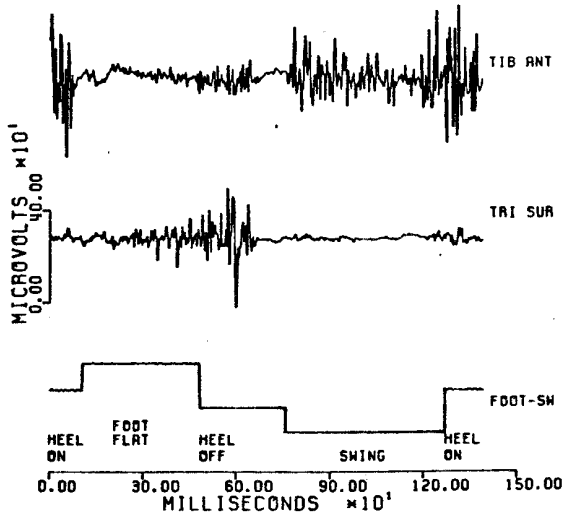

Fig. 5:

EMG activity of tibialis anterior and triceps surae (soleus) during stride in neurologically healthy adult person.

As an extensor muscle, the soleus is active only during the stance phase of gait. In electromyographic studies of four neurologically healthy subjects walking at normal speeds, an increasing EMG activity of the soleus was recorded in the stance phase while it was absent in the swing phase, as shown in Fig. 5. It is of interest to notice that there was no activation of the EMG at the heel contact or at the onset of flat foot. Thus, there was no evidence for the short segmental stretch reflex responses, while the delay of 100 or more milliseconds from the onset of flat foot to the onset of characteristic increasing soleus EMG activity strongly suggested, that this activity depended on the load applied to the soleus and was a result of the long-loop functional stretch reflex response.

In contrast to the soleus, the tibialis anterior, as an antagonistic flexor muscle, was active during the initial swing phase of gait and through the terminal swing to the first part of the loading phase. The activity of tibialis anterior appeared when the foot was not in contact with the floor or started before the heel contact and ended before flat foot event. Therefore, it had to depend mainly on the pre-programmed suprasegmental motor control. Fig. 5 illustrates the characteristic EMG patterns of both tibialis anterior and soleus muscles in a neurologically healthy subject.

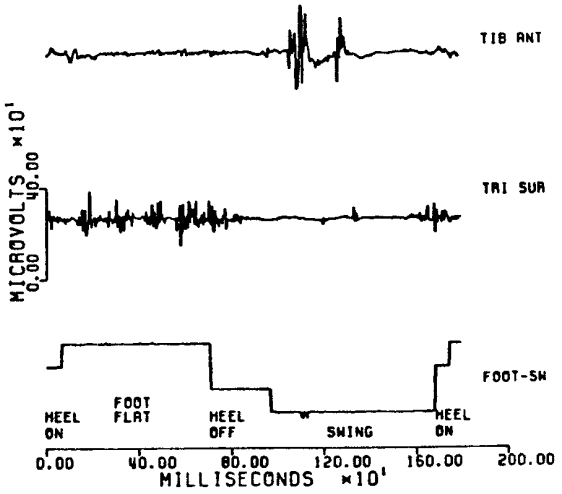

Fig. 6:

EMG activity of tibialis anterior and triceps surae (soleus) during stride in ASCI patient: hypotonic pattern with clonogenic extensor activity.

A similar study of the pattern of EMG activity during gait in the 6 ambulatory spinal cord injury patients revealed that the activity of the soleus was profoundly altered. The characteristic, steadily increasing EMG activity during the flat foot phase was replaced by repetitious clonogenic bursts, as in Fig.6 or by an increased simultaneous coactivation of both tibialis anterior and soleus, shifted towards the onset of the swing phase, as shown in Fig. 7. The activity of tibialis anterior was either drastically diminished with a consequent drop-foot, or there was a low tonic activity with sporadic EMG bursts of larger amplitude and short duration (Fig. 6 and 7).

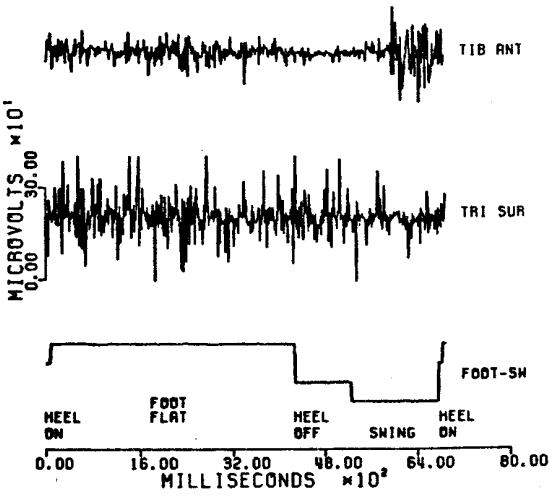

Fig. 7:

EMG activity in tibialis anterior and triceps surae (soleus) during stride in ASCI patient: hypertonic pattern with coactivation of antagonists.

During this study, the presence of continuous tonic soleus activity with the coactivation of tibialis anterior, as shown in Fig. 7, was found in two patients, suggesting that impaired pre-programmed mechanisms facilitated an excessive tonic activity. At the same time, the contact of foot with the floor did not modulate the centrally induced ongoing activity. In the other four patients, the soleus activity was present before the foot contact with the floor, later evidencing short, segmental stretch reflex and loss of characteristic EMG pattern, as in Fig. 6.

DISCUSSION

Ambulatory SCI patients were chosen, together with neurologically healthy adult volunteers, to study the influence of segmental and suprasegmental mechanisms in neurocontrol of the ankle flexors and extensors during gait. In these patients, both kinds of mechanisms are preserved, only their interaction is impaired due to the partial lesion of descending pathways. Measurements in a sitting position were used to assess the impairment of upper motor neurone at the spinal cord. Recordings were then made during walking to quantify the gait and to study the segmental and suprasegmental interaction in its neurocontrol.

An active disynaptic Ia reciprocal inhibition, accompanying volitional contractions of ankle extensor and flexor muscles, was clearly demonstrated in healthy subjects (6). By using the same method of triceps surae H-reflex and the conditioning stimulation of peroneal nerve in different pathologies with motor disorders, a reduced reciprocal inhibition, as well as facilitation of the extensor H-reflex in some cases, were disclosed in spinal cord lesions (7). The method was defined further, excluding a role of degree of the volitional contraction, torque, Ib Golgi afferents, group II skin and joint

afferents, and revealing an insufficiency of Ia spindle afferents to produce the intire recorded reciprocal inhibition, which was atrributed also to group II muscle afferents and its suprasegmental regulation was further indicated (8). In this study, the relationship between ankle antagonists was examined by the monosynaptic stretch reflex (H-reflex) with the superimposed volitional activity. The reciprocal inhibition was at least partly present in all the patients, showing the existence of central control.

An inhibition of the monosynaptic H-reflex during conditioning by a polysynaptic tonic vibratory reflex expresses a degree of presynaptic inhibition on the Ia afférents (9). By comparing the soleus H-reflex during soleus contractions while walking and volitional soleus contractions while standing, a lower facilitation during gait was ascribed to a lower suprasegmentally controlled presynaptic inhibition on the Ia fibers (10). Similarly to the reciprocal inhibition, the interaction between the H-reflex and the vibratory tonic reflex indicated diminished, but present presynaptic inhibition at the ASCI patients in this study.

Overall, it was possible to demonstrate partly preserved patterns of healthy adult subjects in the impaired motor organization of ASCI patients included in the study. The recordings of joint angles and foot contacts displayed various degrees of severely impaired gait in the group of patients when compared to the control group. Yet, they were all able to ambulate by themselves, at least on short distance and with an aid of assistive devices.

It was shown in animal studies,that at least a part of ventral sector of the spinal cord had to be preserved for stepping in monkeys, pointing out a supraspinal dependency of previously postulated spinal step generator (11). Comparing muscular responses in monkeys and healthy man, the activity preceding a direct foot contact with the floor during low drops or postural perturbances, depended on vestibular and visual interaction, while after landing, it was modulated by suprasegmental and segmental reflexes (1). The preprogrammed suprasegmental control of gait and its resulting kinesiological parameters showed similar dependency in patients with symmetric patophysiological disabilities (2). In this study, the EMG activity of ankle antagonists showed a diminished interaction of central preprogrammed neurocontrol in the ASCI patients. It reflected either in tonic hyperactivity with the coactivation of ankle plantar and dorsal flexors or in hypotonic activity with the motor deficit predominantly in the dorsal flexors.

A role of the preprogrammed and foot contact dependent activities studied during runing in healthy subjects, showed that a sudden activation was superimposed on a slowly increasing activity after the ground contact, developing additional force in the triceps surae even within the shortest stance measured in sprint (3). Observing force, position and muscular activity of the ankle during a functional stepdown in different conditions, spinal and supraspinal control was required to minimize the error between the actual and the intended movement (5). Contribution of the stretch reflexes integrated in the central preprogrammed innervation was studied further, showing an optimisation of pushing-off, which could not be accomplished by the central preprogramed innervation and long loop reflexes during runing under different conditions (4). In the ASCI patients from this study, the segmental stretch reflex responses appeared in the extensors when the background EMG activity was decreased and the flexors showed absence of motor unit activity, while the

suprasegmental functional stretch reflex innervation was suppressed in all the cases. All this three features can probably be attributed to the impaired preprogrammed descending control.

Compared with the healthy subjects, intermittent reciprocal activations neccessary for the optimal movements during gait, were disturbed in the ASCI patients by the ongoing activity of both antagonists weighted towards the extensors. However, the presumed released segmental phasic activity did not prevail in motor disabilities of these patients. In spite of the insufficient suprasegmental control, they walked with difficulties, using the residual central influence on segmental control. In other words, they did not walk by the disinhibited spinal cord but by the control of brain.

Is it possible to influence impaired functions of upper motor neurone in such ASCI patients by an external control? Yes. However, in the past we learned, that externally induced neuromuscular responses can be so widespread, that are functionally limited. In order to obtain the optimal functional response, it will be neccessary to integrate the externally induced muscular activity with the residual suprasegmental and segmental neuromuscular control.

REFERENCES

1. Lacour, M., Vidal, P. P., Xerri, C., Dynamic characteristics of vestibular and visual control in rapid postular adjustments, In Advances in Neurology 39, Motor Control Mechanisms in Health and Disease, J. E. Desmedt (Ed.), 589-605, Raven Press, New York 1983.
2. Conrad, B., Benecke, R., Carnehl, J., Hoehne, J., Meinck, H. M., Patophysiological aspects of human locomotion, In Advances in Neurology 39, Motor Control Mechanisms in Health and Disease, J. E. Desmedt (Ed.), 717-726, Raven Press, New York 1983.
3. Dietz, V., Schmidtbleicher, D., Noth, J., Neuronal mechanisms in human locomotion, J.Neurophysiol. 42, 1212-1222, 1979.
4. Dietz, V., Noth, J., Significance of spinal stretch reflexes in human locomotion, In Advances in Physiological Sciences 1, Regulatory Functions of the CNS, Principles of Motion and Organization, J. Szentagothai, M. Palkovits, J. Hamori (Eds.), 169-177, Akademiai Kiado, Budapest, Pergamon Press, New York, 1980.
5. Freedman, W., Minassian, S., Herman, R., Functional stretch reflex (FSR) - a cortical reflex?, In Progress in Brain Research 44, Understanding the Stretch Reflex, S. Homma (Ed.), 485-490, Elsevier, Amsterdam 1976.
6. Tanaka, R., Inhibitory mechanism in reciprocal innervation in voluntary movements, In Progress in Clinical Neurophysiology 8, Spinal and Supraspinal Mechanisms of Voluntary Motor Control and Locomotion, J. E. Desmedt (Ed.), 117-128, Karger, Basel 1980.
7. Yanagisawa, N., Reciprocal reflex connections in motor disorders in man, In Progress in Clinical Neurophysiology 8, Spinal and Supraspinal Mechanisms of Voluntary Motor Control and Locomotion, J. E. Desmedt (Ed.), 129-141, Karger, Basel 1980.
8. El-Tohamy, A., Sedgwick, E.M., Spinal inhibition in man: depression of the soleus H reflex by stimulation of the nerve to the antagonist muscle, J. Physiol. 337, 497-508, 1983.

9. Delwaide, P. J., Martinelli, P., Crenna, P., Clinical neurophysiological measurement of spinal reflex activity, In Spasticity: Disordered Motor Control, R. G. Feldman, R. R. Young, W. P. Koella (Eds.), 345-371, Symposia Specialists Inc., Miami 1980.
10. Morin, C., Katz, R., Mazieres, L., Pierrot-Deseilligny, E., Comparison of soleus H reflex facilitation at the onset of soleus contractions produced voluntarily and during the stance phase of human gait, Neuroscience Letters 33/1, 47-53, 1982.
11. Eidelberg, E., Walden, J. G., Nguyen, L. H., Locomotor control in macaque monkeys, Brain 104, 647-663, 1981.