

INTERSEGMENTAL MODULATION BY PERIPHERAL AFFERENTS IS MODIFIED IN HEMIPARESIS FOLLOWING STROKE

Dyer JO^{1,2,3,*}, Bourbonnais D^{1,2,3}, Fleury J and Forget R^{1,2,3}

¹Centre de recherche interdisciplinaire en réadaptation, Université de Montréal, Canada

²Institut de réadaptation de Montréal, Université de Montréal, Montréal, Québec, Canada

³École de réadaptation, Université de Montréal, Montréal, Québec, Canada

* Presenting author: Dyer JO. Email address: jomerdy@gmail.com

Abstract

Intersegmental pathways linking quadriceps (Quads) to soleus (Sol) were explored in 11 stroke subjects and compared to those of 14 healthy controls. Relationships between changes in these circuits and motor impairment were also assessed.

Subjects were seated and the effects of the conditioning stimulation of femoral nerve (FN) over the voluntary isometric contraction of Sol at 20% and 40% of maximal EMG were measured. FN stimulation produced a short-lasting (26 ± 1 ms) and brief (8 ± 2 ms) facilitation ($25 \pm 44\%$) of Sol EMG, in 40% of controls. In stroke subjects, this facilitation had a similar latency and duration but was observed in a greater proportion of patients (64%) and with an increased amplitude ($111 \pm 99\%$). This abnormal facilitation was correlated to spasticity ($r=0.88$; $p<0.001$). In all control subjects, the facilitation was followed (34 ± 4 ms after FN stimulation) by an inhibition (decrease of $48 \pm 22\%$) of Sol EMG, for a duration of 59 ± 34 ms. In contrast, hemiparetic subjects did not show on average the FN-induced inhibition of Sol EMG.

It is argued that the malfunction of intersegmental circuits could contribute to the motor impairment observed at the paretic lower limb.

1. INTRODUCTION

Spinal pathways are thought to assist muscular coordination at the lower limb, in human bipedal stance and gait (Meunier et al., 1994). Several basic sensorimotor mechanisms are involved in the regulation of motoneurons excitability at one medullar segment. The malfunction of these segmental mechanisms is well documented in hemiparesis following a stroke. In fact, reciprocal inhibition (Yanagisawa et al., 1976), homosynaptic Ib inhibition (Delwaide and Oliver, 1988) and recurrent inhibition (Katz and Pierrot-

Deseilligny, 1982) were all found to be impaired at the paretic lower limb.

Although the impairment of segmental pathways is well established in hemiparesis, few studies have investigated the possible malfunction of intersegmental pathways following stroke. In human, intersegmental projections of group Ia proprioceptive afferents (Meunier et coll., 1993) and recurrent inhibition via Renshaw cell interneurons (Meunier et coll., 1990) could affect the activity of motoneurons located at different levels within the lumbar spinal cord. Thus, there are intersegmental pathways linking quadriceps (Quads) to soleus (Sol) in human. These circuits can be explored by measuring the modulation of Sol reflex (Meunier et al., 1990) and voluntary activity (Forget et coll., 1998) after the conditioning stimulation of the femoral nerve (FN). This heteronymous modulation consists in a short-latency (22 ms) and short-lasting (8 ms) facilitation of Sol activity. The characteristics of this facilitation are suggestive of a monosynaptic facilitation via group Ia afferents from Quads to Sol motoneurons (MNs) (Meunier et al., 1993). Moreover, this facilitation is immediately followed by a long-lasting (50 ms) inhibition (decrease of 40%) of Sol activity (Meunier et al., 1990). It has been suggested that this inhibition is due to the activation of Quads Renshaw cells (Meunier et al., 1990; 1996). Heteronymous spinal connections between Quads and Sol in human may contribute to the coordination of the reciprocal activity of these muscles during gait. The malfunction of these pathways could be responsible for the abnormal coactivation between Quads and Sol observed in hemiparesis following a stroke.

Since incoordination of extensor muscles results in abnormal synergies at the paretic lower limb, the questions then arise whether these spinal mechanisms of regulation of soleus excitability by FN afferent 1) are modified in hemiparetic subjects as compared

to those of healthy subjects and if so, 2) to what extent these malfunctions are related to leg motor impairment?

In this study, the integrity of the intersegmental pathways linking quadriceps (Quads) to soleus (Sol) were explored in hemiparetic subjects and compared to that of healthy control subjects. Moreover the possible relationship between motor dysfunction at the paretic lower limb and changes in the heteronymous spinal modulation were assessed.

2. METHODS

The FN was stimulated (pulse duration = 0.5 ms & intensity = H max/2 of rectus femoris) on the right side of 14 control subjects (41 ± 14 y. old) without orthopaedic or neurological deficit and on the affected side of 11 hemiparetic subjects (45 ± 14 y. old). The subjects were seated and instructed to produce a voluntary isometric contraction of Sol at two levels of activation (20% and 40% of maximal EMG). The EMG signal was first amplified (5000 x), filtered (30 Hz to 1 kHz) and digitized at a sampling rate of 3000 Hz. The effect of FN stimulation on Sol integrated EMG activity was assessed from 22 to 99 ms after FN stimulation within 6 consecutive time windows of 12 ms duration. Surfaces of Sol integrated EMG (iEMG) recorded during these time frames were compared to the baseline EMG activity before FN stimulation, at the two levels of Sol activation and between the hemiparetic and the control subjects. The Chedoke-McMaster Stroke Assessment (CMSA) was used to assess motor performance of the hemiparetic subjects at the leg and the foot. A Composite Spasticity Index (CSI) evaluated levels of spasticity and the walking time at comfortable and maximal speed over a 5-meter distance was used to measure gait velocity. Spearman rank-order statistics were used to correlate motor impairment measures and EMG modulation of Sol induced by FN stimulation.

3. RESULTS

Within both experimental groups, the contraction level of voluntary EMG activity (i.e.: 20% or 40% of maximal EMG at Sol) did not affect the pattern of modulation of Sol EMG activity by FN stimulation. However, at a comparable level of baseline voluntary EMG activity, patterns of Sol modulations were different between the two groups.

In 40% of the control subjects, a short-latency (26 ± 1ms) and short-duration (8 ± 2ms)

facilitation (25 ± 44% increase of baseline values) was observed after FN stimulation. In all control subjects, the FN stimulation produced a marked inhibition (48 ± 22% decrease of baseline values) of Sol voluntary EMG activity at a mean latency of 34 ± 4 ms and duration of 59 ± 34 ms.

A higher percentage of the hemiparetic subjects (64%) showed the FN-induced facilitation. This facilitation was also of greater amplitude (111 ± 99 %) compared to control values (p = 0.016) but the latency (26 ± 3ms) and duration (13±7 ms) characteristics were not significantly different between the two groups. In contrast to the control subjects, the majority of hemiparetic subjects did not show the FN-induced inhibition at latencies corresponding to the pattern observed in healthy subjects (*cf. Figure 1*). At both voluntary contraction levels, baseline EMG activity of hemiparetic subjects were correlated with the spasticity index (CSI) scores (r = -0.760; p = 0.007 at 20% of Sol max EMG level and r = -0.836; p < 0.005 at 40% level) and also with the motor performance (CMSA) scores at the leg (r = 0.81; p = 0.003 at 20% level and r = 0.81; p = 0.009 at 40% level). Facilitation of Sol at short latencies (within 22 and 34 ms post-stimulation) was correlated with CSI scores at 20% (r = 0.88; p<0.001) and 40% (r = 0.66, p=0.038) contraction levels. Facilitation was also correlated to CMSA scores at the foot (r = -0.651; p = 0.030) at 20% of max EMG at Sol.

In all healthy subjects tested, stimulation of FN during the sustained isometric contraction of Sol induced a clear depression of Sol voluntary EMG activity either at 20% or 40% of maximal voluntary EMG activity levels. Mean results for healthy subjects revealed a long-duration (59 ± 34 ms) and a marked decrease (from 40% to 60%) of voluntary EMG activity at Sol at a latency of 34 ± 4 ms after FN stimulation (*cf. Figure 1*). Hemiparetic subjects showed on average no heteronymous inhibition at latencies (from 34 to 90 ms after FN-stimulation) where the phenomenon was observed in control subjects at both level of contraction tested. Moreover, at 40% max EMG at Sol, the modulation of Sol EMG activity from 48 ms to 61 ms after FN stimulation, which corresponds to an inhibitory modulation period in control subjects, was correlated to the degree of spasticity (CSI_{Ankle}).

4. DISCUSSION AND CONCLUSIONS

The short-latency modulation of Sol EMG activity by FN stimulation reveals strong

intersegmental influences of FN onto Sol motoneuronal pool (Meunier et al., 1990). The consistent finding is a marked inhibition of Sol voluntary EMG activity observed in all healthy subjects tested. A short-duration (8 ms) facilitation may precede the heteronymous inhibition in 40% of the cases. However, hemiparetic subjects show, on average, non-significant inhibition after FN stimulation. The results suggest an alteration of this heteronymous inhibition. On the contrary, facilitation was the preponderant influence of FN afferents onto Sol voluntary EMG activity at short-terms latencies.

Previous studies have suggested that the short-latency facilitation of Sol is an heteronymous monosynaptic excitation by group Ia afferents from FN whereas the consecutive long-lasting inhibition results from recurrent inhibition projecting from FN to Sol (Meunier et al., 1990; 1996). Our results suggest an overfacilitation of the monosynaptic heteronymous excitation by Ia afferents pathways and a dysfunction of heteronymous recurrent inhibition pathways from Quads to Sol in hemiparesis. The dysfunction of both spinal mechanisms could have a significant impact in the regulation of Quads and Sol activation and coordination.

In fact, dysfunction of some spinal mechanisms is well documented in spastic hemiparesis. Our results confirm that these dysfunctions usually result in a global facilitation of spinal pathways (Artieda et al., 1991). The functional significance of these spinal mechanisms is still to be established. The correlations analysis revealed that, in the hemiparetic subjects, the amount of EMG activity that can be generated (i.e. baseline levels) and the modulation capability by sensory afferents are related to the level of spasticity and motor impairment.

Intersegmental inhibition of Sol EMG triggered by FN stimulation is modified in hemiparesis following stroke. The modulation observed, which is largely facilitatory, is correlated with the clinical measures of motor impairments.

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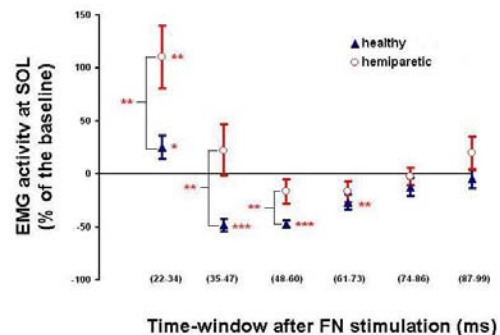


Figure 1 Mean modulation of Sol voluntary EMG activity induced by FN conditioning stimulation for control (filled triangles) and hemiparetic subjects (open circles) at comparable baseline values (ie, 20% and 40% of max EMG at Sol for control and hemiparetic subjects respectively). The modulation of Sol integrated EMG activity was measured from 22 to 100 ms after FN stimulation within 6 consecutive time windows (of 13 ms duration) and was expressed as a % of the baseline EMG activity. Each symbol represents the mean modulation of Sol EMG activity for each group within the time-window of analysis (15 trials averaging per subject). Vertical bars represent 1 SEM. Asterisks indicate statistically significant differences from the baseline EMG activity (p < 0.05 *, p < 0.01 **, p < 0.001 ***).

