SURROUND INHIBITION ABNORMALITIES IN FOCAL HAND DYSTONIA MAY NOT DEPEND BY ALTERATIONS IN SOMATOSENSORY INTEGRATION

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BACKGROUND

Several evidence suggest that surround inhibition, suppression of excitability in an area surrounding an activated neural network, is a powerful operational system within sensory-motor cortex operating during the acquisition of new motor tasks in order to select the appropriate muscle sequence to be stored within the final motor engram[1-2]. This mechanism is thought to be lost in dystonia and this should explain the development of redundant motor memories which could culminate in overflow phenomena and overt dystonia[3-4].

OBJECTIVE

To explore if sensory information is abnormally processed and integrated in focal dystonia and if surround inhibition phenomena are operating during sensory-motor plasticity and sensory-motor integration in normal humans and in patients with focal hand dystonia.

PATIENTS & METHODS

✓ 8 patients with focal hand dystonia (6 male, 2 female, mean age 50 years) and 8 age- and sex-matched healthy subjects were recruited.

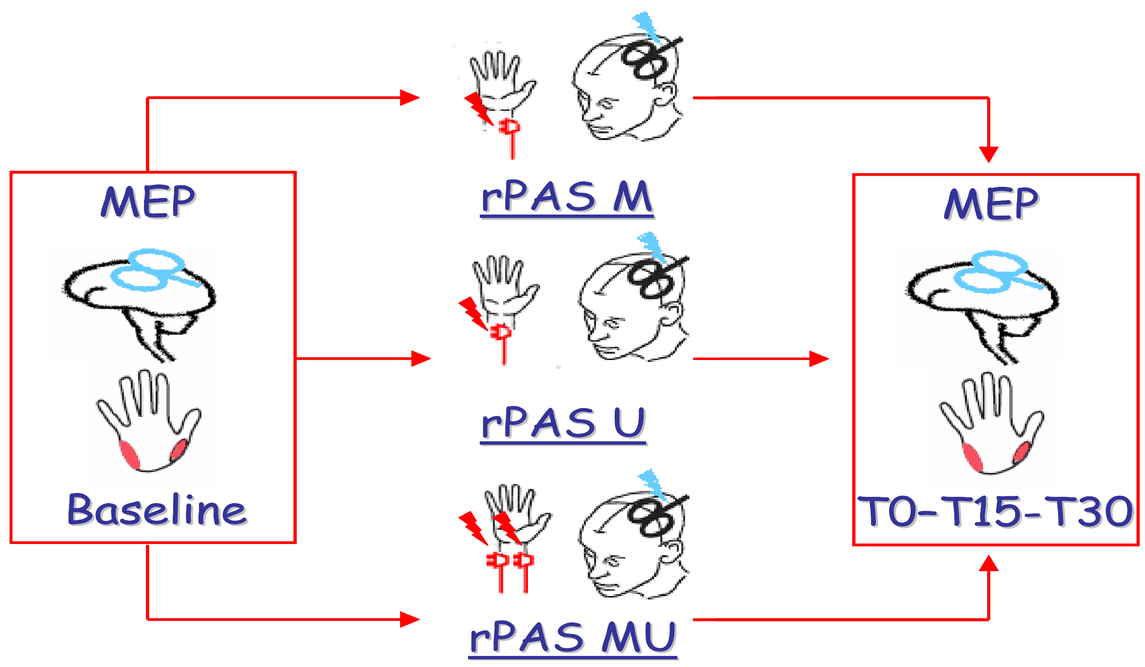
✓ Sensori-motor plasticity was evaluated using the paradigm of repetitive paired associative stimulation (rPAS) at a frequency of 5 Hz[5]. rPAS consisted of 600 pairs of stimuli which were continuously delivered at a rate of 5 Hz for two minutes. Each pair of stimuli consisted of an electrical conditioning stimulus given to the right median nerve (rPAS M) or right ulnar nerve (rPAS U) or right median + ulnar nerve (rPAS MU) followed after 25 msec by a biphasic transcranial magnetic stimulus given to the left motor cortex (M1). The intensity of the electrical stimulus was set at twice the sensory threshold, while the intensity of rTMS was individually adjusted to 90% of active motor threshold (AMT). Patients and healthy subjects underwent the three different sections of rPAS which were given at least 1 week apart.

✓ Cortical excitability of the primary motor hand area was tested with single pulse TMS, by recording motor evoked potentials (MEP) amplitudes, before and after up to 30 minutes after the three different rPAS sections. The MEPs were recorded in the controlateral abductor pollicis brevis (APB) and abductor digiti minimi (ADM).

✓ We also evaluated the ratio MU/(M+U) x 100 to explore surround inhibition mechanism during sensory-motor plasticity.

✓ In 5 patients with focal dystonia, spatial somatosensory integration using the paradigm of Tinazzi[3] and temporal somatosensory integration using the paradigm of Frasson[4] were evaluated.

ESPERIMENTAL DESIGN



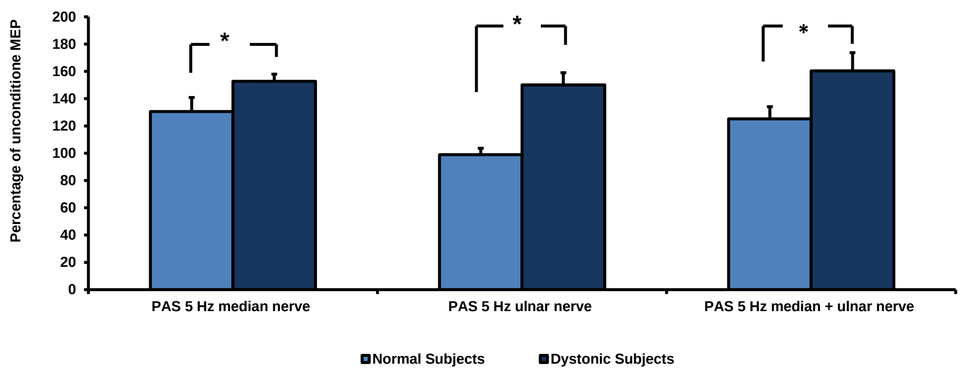
5Hz rPAS of left M1

Continuous train of 600 paired stimuli using ISI of 25 ms between the peripheral and transcranial stimulus

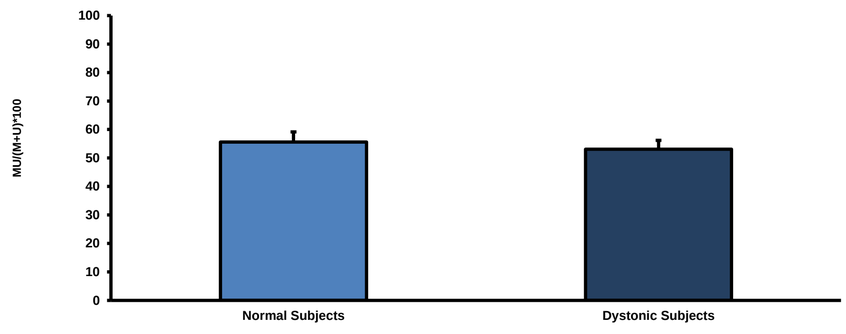
RESULTS

Our data demonstrated that this novel and rapid PAS paradigm pointed out the same set of abnormalities already found with classical PAS in patients with dystonia: the amount of facilitation was larger compared to normal subjects and the spatial specificity was lost (Fig. 1). A three-factorial ANOVA demonstrated a significant time x group x conditioning interaction (F=7.9; p=0.005). Moreover, MU/(M+U) x 100 ratio after PAS was similar (about 50%) in healthy subjects and dystonic patients (Fig. 2). Finally Spatial (Fig. 3) and Temporal Somatosensory integration (Fig. 4) were normal in focal dystonia.

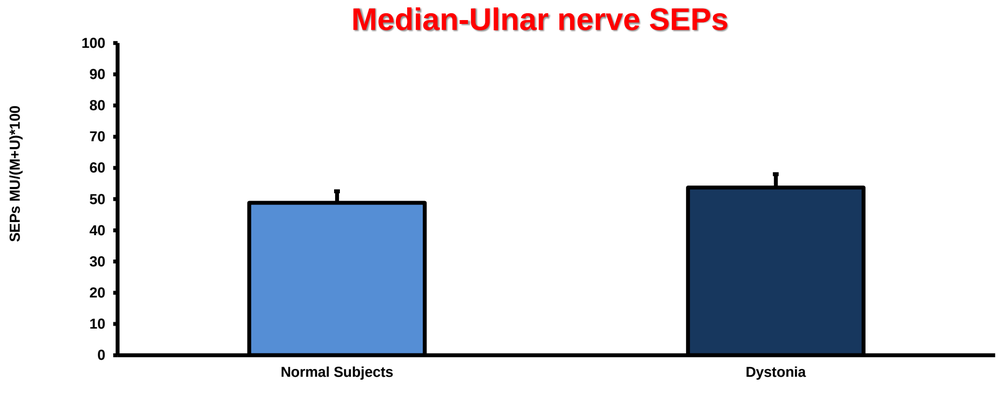
Abnormal Plasticity (Fig. 1)



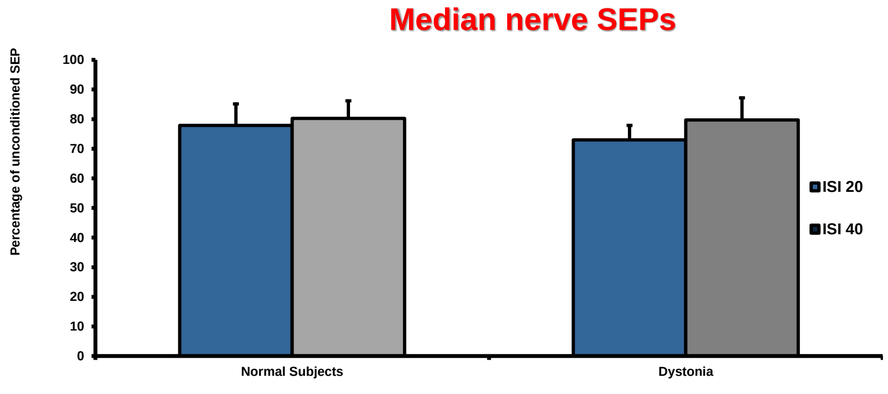
MU/ (M+U) x 100 ratio during PAS protocol (Fig. 2)



Spatial Somatosensory Integration (Fig. 3)



Temporal Somatosensory Integration (Fig. 4)



CONCLUSIONS

These data suggest that surround inhibition phenomena during sensory-motor plasticity are normal in focal dystonia. We could speculate that, the greater is the spreading of dystonia in the body parts, the lesser is the ability to integrate and discriminate afferent sensory inputs coming simultaneously from adjacent body parts. Our findings may set the stage for future research in generalized dystonia, to better understand if abnormalities in somatosensory integration could contribute to the formation of abnormal and redundant motor memories.

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