

# SENSORY TRICK IN CERVICAL DYSTONIA: A NEUROPHYSIOLOGICAL STUDY OF SENSORIMOTOR INTEGRATION



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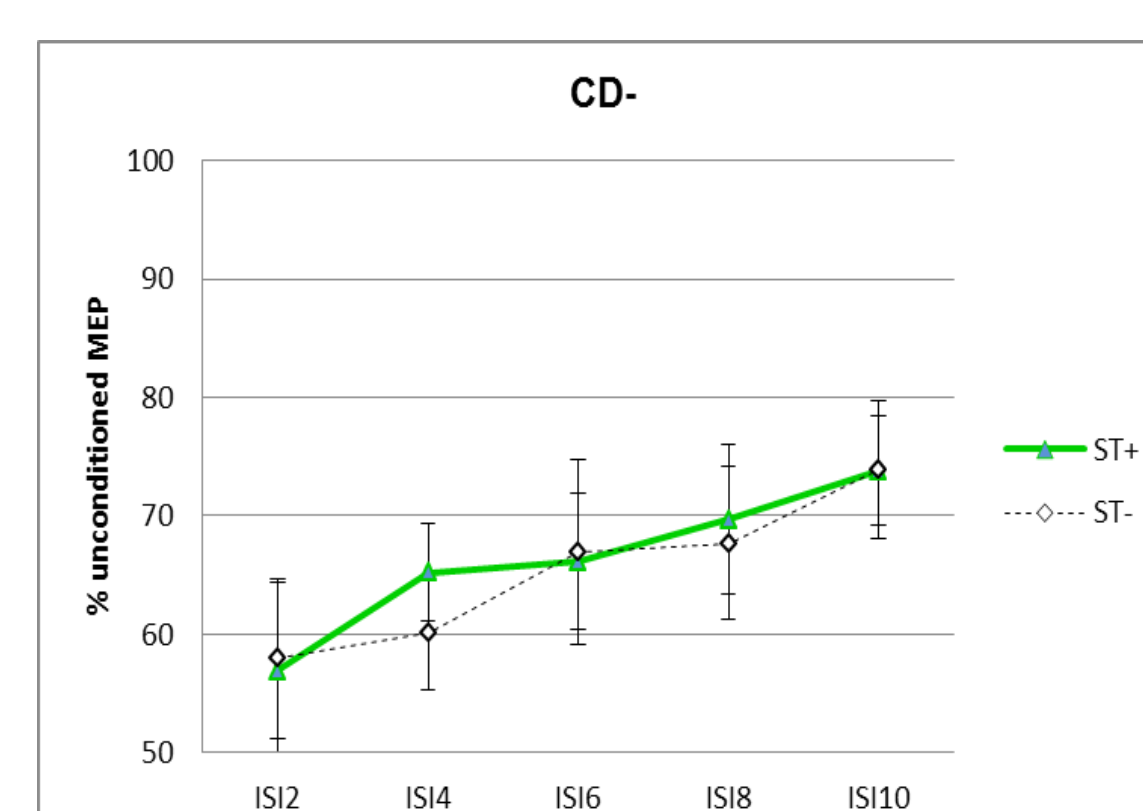
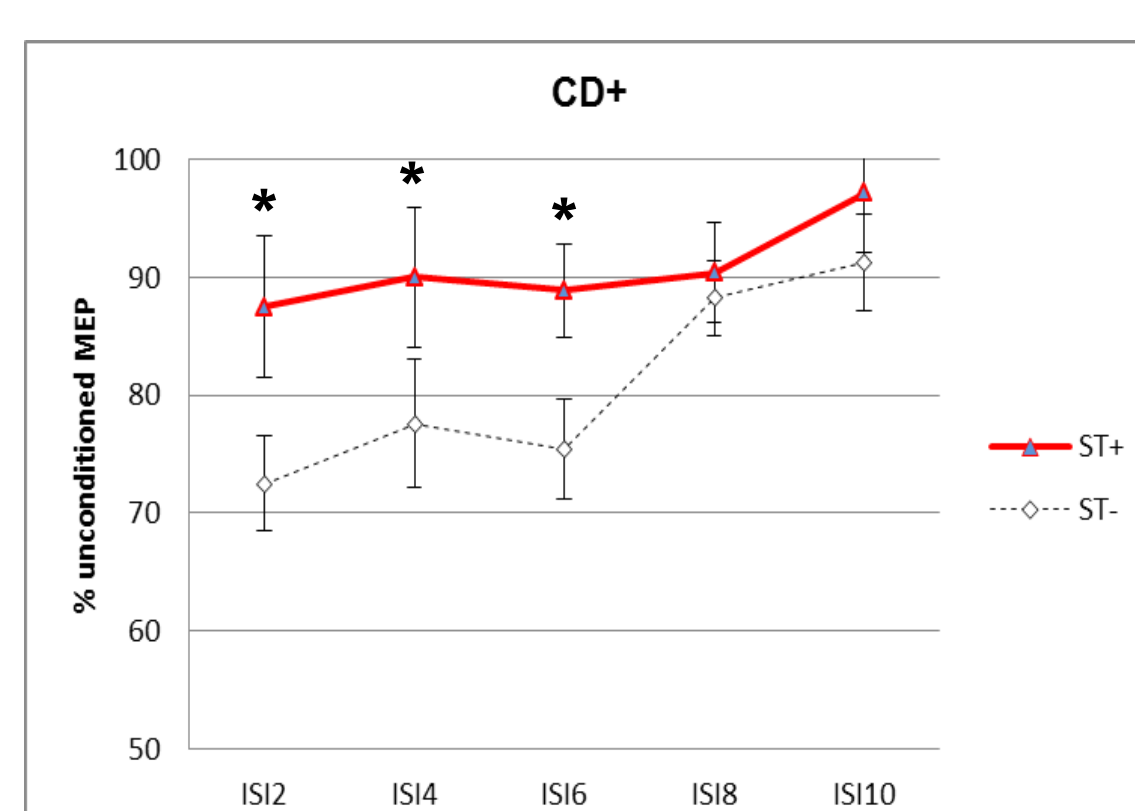
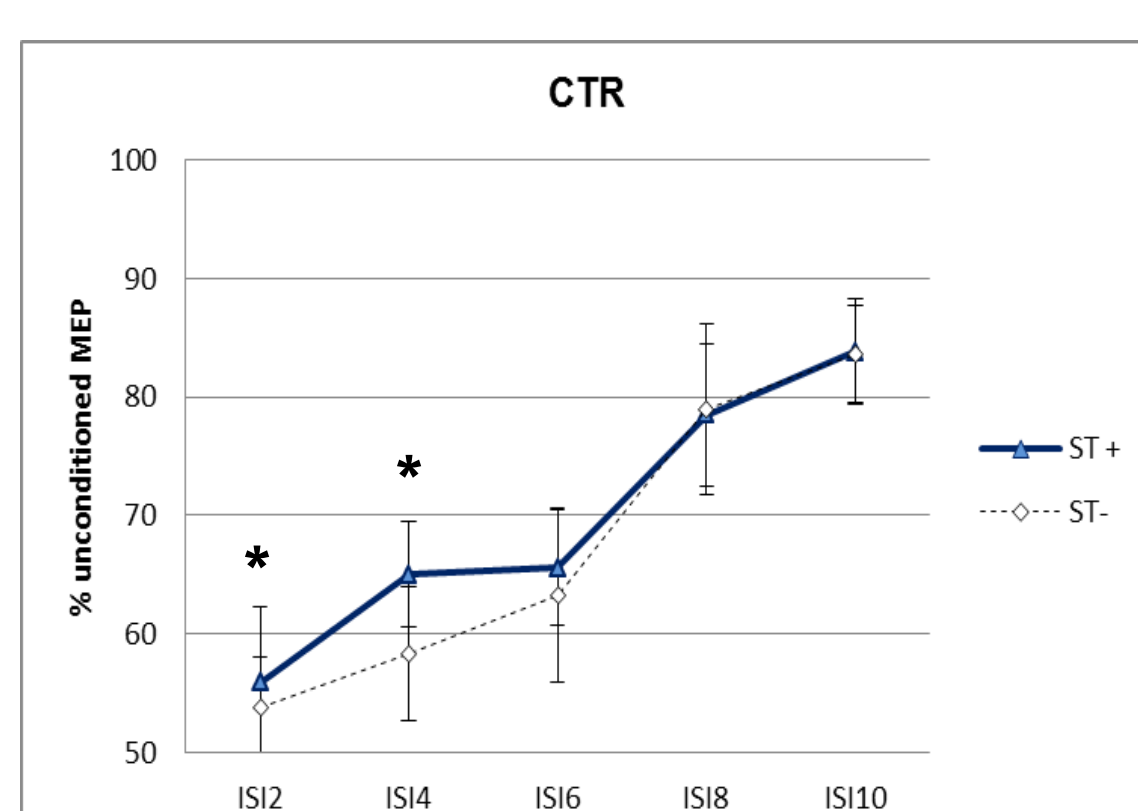
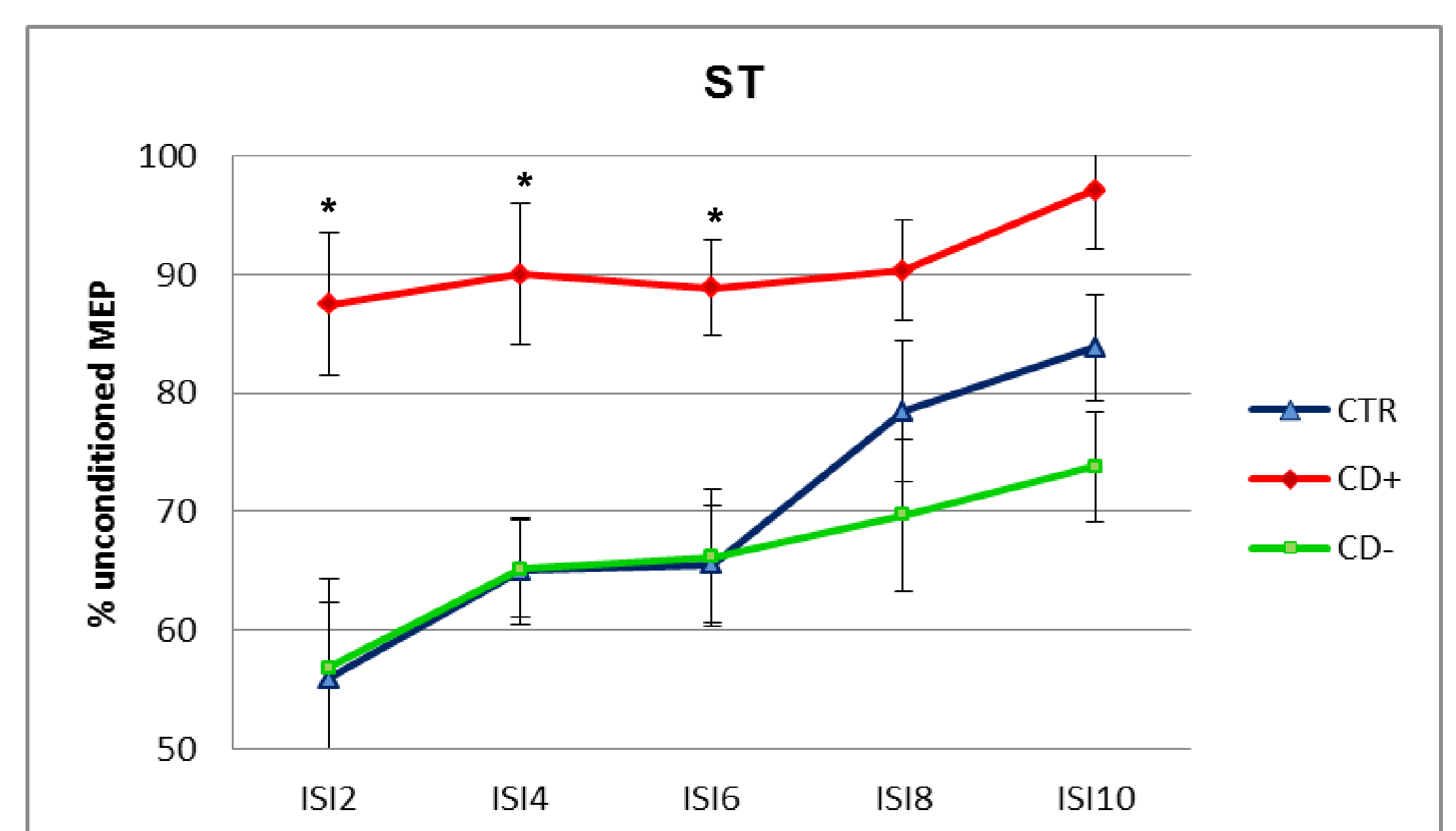
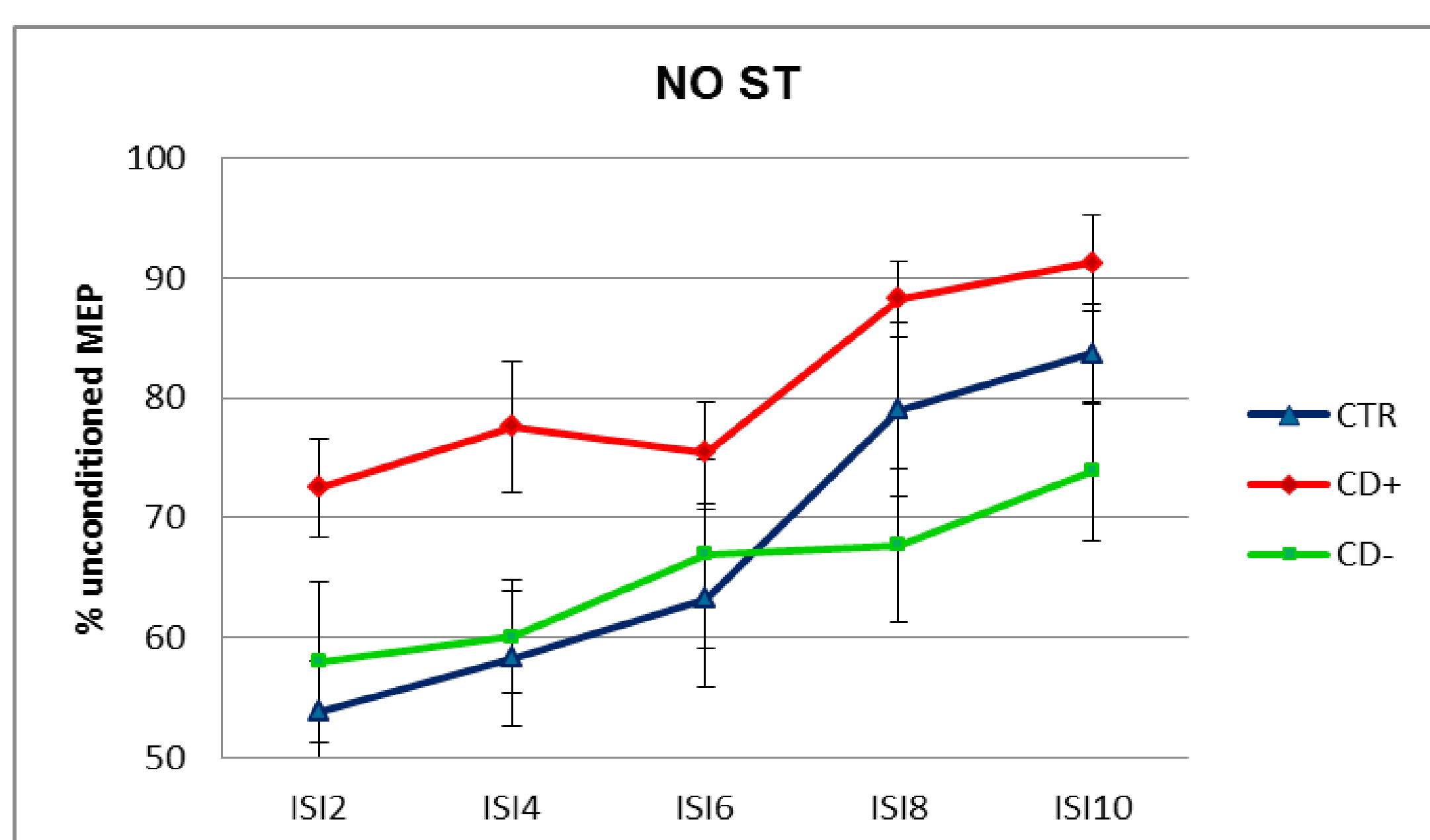
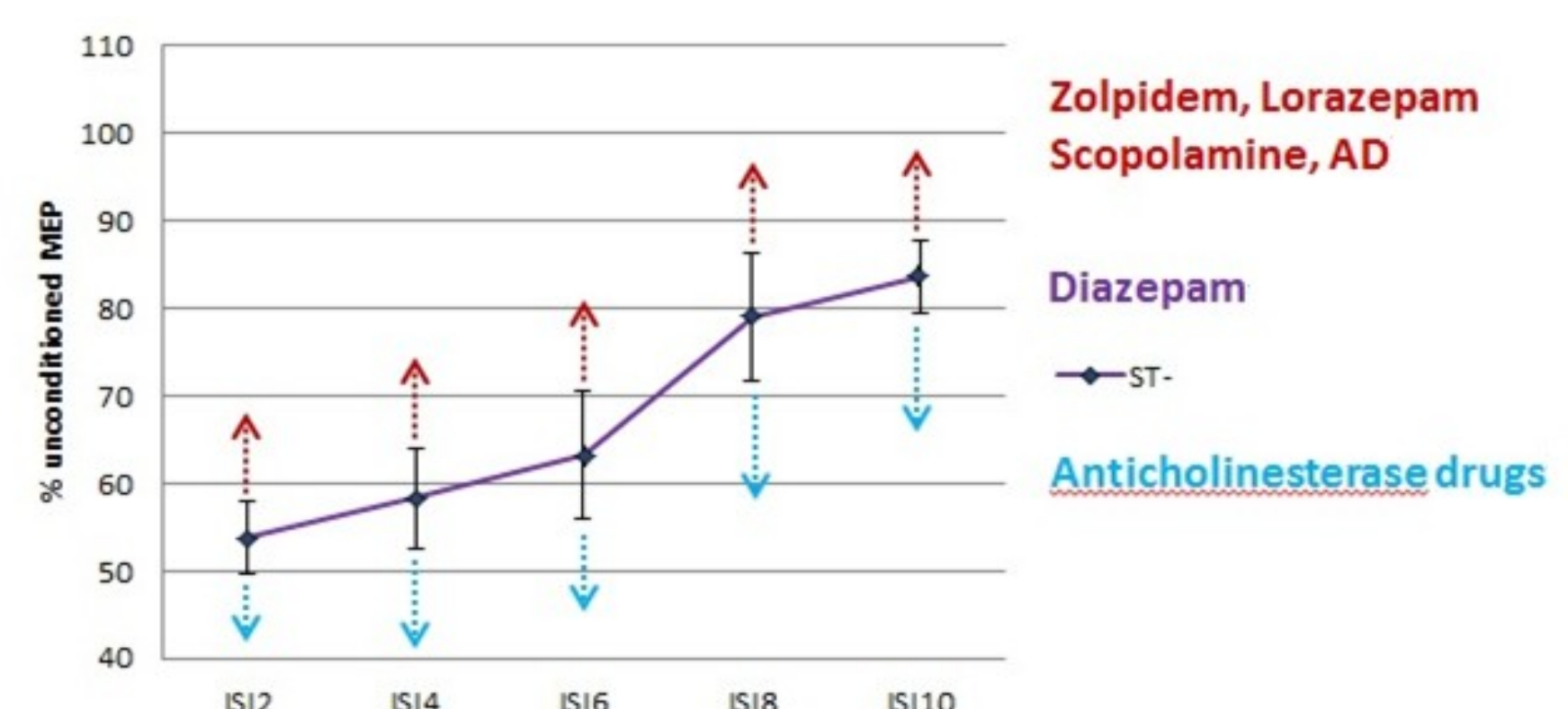
**INTRODUCTION** - Although dystonia is previously regarded as a pure motor disorder, several lines of evidence, including neurophysiological, neuroimaging and experimental findings, suggest that both motor and somatosensory functions may be defective (Abbruzzese and Berardelli, 2003<sup>1</sup>; Hallett, 1995<sup>2</sup>). Consequently, abnormal processing of the somatosensory input in the central nervous system may lead to inefficient sensorimotor integration, thus contributing to the generation of dystonic movements. The broad spectrum of sensory tricks (ST) used in patients with dystonia, and especially of adult-onset primary cervical dystonia (CD), is a direct indicator of the sensory influence on motor output. The physiological mechanisms underlying sensory trick are still unknown, and only a few studies investigated this phenomenon.

**OBJECTIVE** - The aim of this study was to evaluate sensorimotor integration in CD patients examining, by means of SAI paradigm, the inhibitory effect of the somatosensory afferent volley on the motor cortex output and to assess if the sensory trick may change SAI profile. In addition we studied short-latency and long-latency components of somatosensory evoked potentials (SEPs) in order to assess the integrity of the somatosensory pathway.

**PATIENTS AND METHODS** - SAI was performed in 25 patients with primary CD, 15 with effective sensory tricks (CDST+) and 10 without sensory tricks (CDST-). SEPs were recorded in 14 patients with primary CD, 7 CDST+ and 7 CDST-. 10 and 14 healthy, age-matched, subjects were recruited as controls, respectively for SAI and SEPs experiments. SAI was studied using the technique described by Tokimura et al. (2000)<sup>3</sup>. SEPs were recorded using standard parameters and peak-to-peak amplitude and latency of short latency (N20, P25, N30, P45, N60) and long-latency components (P100, N140, P200) were measured. Tests were performed at rest and during ST

**RESULTS** - Clinical data are reported in the Table below. At rest, there were no differences on SAI between CTR and CD patients. During ST, SAI was significantly reduced in CDST+ compared to other groups. When CTR mimic the trick, SAI was significantly reduced compared to the rest at ISI2 (p=0.037) and ISI4 (p=0.044). When CDST+ patients performed their ST, SAI was significantly reduced compared to rest condition at ISI2 (p=0.038), ISI4 (p=0.031) and ISI6 (p=0.002). No significant changes were observed in CDST- patients. There were no differences in SEPs between CD patients and CTR and SEPs were not modified by ST.

Clinical characteristics and TMS data					
	Disease duration (y)	TWSTERS severity score	N20 Latency (ms)	RTM (%)	MEP <sub>amp</sub> (μV)
CDST+	10,27 ± 7,91	14,36 ± 5,61	17,72 ± 4,85	48,13 ± 8,25	5,74 ± 3,95
CDST-	13,20 ± 8,72	14,60 ± 3,63	19,04 ± 1,22	53,50 ± 5,30	5,39 ± 2,56
CTR	-	-	19,41 ± 0,95	50,70 ± 8,53	4,52 ± 2,42



**CONCLUSIONS** - Our study confirmed that sensory trick-mediated improvement of CD is related to changes of cortical excitability, changes that are not shown in CD patients without the sensory trick and in healthy controls. Even if the pathophysiological mechanism underlying sensory trick is still unknown, an impairment of sensorimotor integration rather than a simple alteration of the afferent input or motor output is the most probable hypothesis.