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**Abstract**

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# The effect of paresthesia free spinal cord stimulation tech-niques on A delta fibers: a preliminary study

Mariateresa Giglio <sup>1</sup>, Alberto Corriero <sup>2</sup>, Emmanuella Ladisa <sup>3</sup>, Chiara Abbatantuono <sup>3</sup>, Marina De Tommaso <sup>3</sup>, Filomena Puntillo <sup>4</sup>

1. Department of interdisciplinary Medicine, Università degli studi di Bari, Bari, ITA 2. Department of Interdisciplinary Medicine (DIM), University of Bari Aldo Moro, Bari, ITA 3. Department of Translational Biomedicine and Neuroscience, University of Bari Aldo Moro, Bari, ITA 4. Department of Intedisciplinary Medicine, University of Bari Aldo Moro, Bari, ITA

**Corresponding author:** Mariateresa Giglio, mariateresa.giglio@uniba.it

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## Abstract

### Introduction

Tonic or paresthesia-spinal cord stimulation (P-SCS) is an efficacious treatment for several chronic refractory pain conditions, such as persistent spinal pain syndrome 2 (PSPS 2) (1). A recent study (2) demonstrated that P-SCS inhibits the somatosensory potentials, acting on large-diameter peripheral fibers, i.e., A beta fibers. High frequency SCS (HF-SCS) is a spinal cord stimulation in which kilohertz frequency pulses are delivered, while burst SCS (B-SCS) mimicks the activation of endogenous neuronal bursts. Both these techniques do not cause an activation of the A beta fibres, while the effects on A delta and C fibers are unknown (3). According to the latest guidelines (4), the most reliable technique to evaluate the nociceptive pathway are laser evoked potentials (LEPs). The aim of our study will be to investigate the action HF-SCS and B-SCS on the modulation of A delta fibres, using laser evoked potentials.

### Materials and methods

Adult patients treated with HF-SCS or B-SCS for PSPS 2 were enrolled. These patients were subjected to stimulation with laser evoked potentials in 2 times, with the stimulator off and on. The LEPs were evoked by stimulating the knee bilaterally, the dorsum of the feet and in the dorsal bilateral region (T12-L2) with a Nd:YAP stimulator; the right hand was used as control. Recording electrodes were 62. The exclusion criteria were poor cooperation and cognitive disorders. Paired t test and Wilcoxon test were used to compare LEP latencies and amplitude. A p value <0.05 was considered statistically significant.

### Results.

Eight patients were included, 2 M, 6 F., the median age was 67 + 7. The analysis of LEP showed a significant statistical reduction of P2 wave in the knee when the PF-SCS was turned on, while other waves showed no significant reduction.

### Conclusion

These preliminary results suggested that PF-SCS is able to modulate the activation of A delta fibers, even if only a significant result was retrieved in the reduction of the P2 component.

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