Somatosensory changes in orthodontics—findings from quantitative sensory testing (QST) studies

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Several orthodontic procedures induce pain at some degree during the treatment. Even though orthodontic pain has been extensively studied from the clinical perspective, the spreading of standardized somatosensory examination of the oro-facial structures opens a window on the knowledge of the neurophysiological mechanisms underlying such pain. This article provides an overview on the chairside and comprehensive intraoral somatosensory testing, and summarizes the current findings on the somatosensory changes in orthodontic and orthognathic surgery patients. Signs of sensitization have been reported both during orthodontic treatment and after orthodontic-surgical treatment; therefore, clinicians should be aware of the available psychophysical methods to adequately assess the oro-facial somatosensory functions. (Semin Orthod 2018; ■:1–8) ***© 2018 Elsevier Inc. All rights reserved.***

Introduction

The somatosensory system is a complex system of sensory neurons and pathways that responds to different stimuli. This system conveys the perception of touch, pressure, pain, temperature, position, movement, and vibration, which arise from surface tissues or deep organs. Anatomically, it is a 3-neurons system that relays sensations detected in the periphery and transfers them through the spinal cord, the brainstem, and the thalamic nuclei to the somatosensory cortex in the brain. Alterations in the integrity of this neural axis might result into disturbances of the somatosensory functions, which are common signs of neurological illnesses and peripheral nerve diseases.[1]

Notwithstanding, pain is not simply a reflex of peripheral afferents, and nociceptive pathways are subject to dynamic (excitatory and inhibitory) modulations.[2] After intense, repeated and sustained nociceptive stimuli, temporary or permanent changes in neuronal activity can occur. As a matter of fact, neurons are able to change in form and function in response to alterations in their environment, and this ability goes under the name of "neural plasticity".[3] Following an injury, when nociceptors produce lots of neuropeptides, two major effects can occur: sensitization of the existing receptors, bringing the membrane potential closer to the depolarization threshold, and recruitment of new receptors, stimulating the production of new nociceptive terminals. This phenomenon is called "peripheral sensitization", which clinically results in an increased sensitivity to painful (hyperalgesia) and non-painful (allodynia) stimuli.[4] Central sensitization occurs when sustained neuronal changes arise in the dorsal horn of the spinal cord, and the central nervous system turns into a persistent state of high reactivity. Also, repression of the descending pain inhibiting pathways plays

Abbreviation: ALL, Dynamic Mechanical Allodynia; CDT, Cool Detection Threshold; CPT, Cold Pain Threshold; HPT, Heat Pain Threshold; MDT, Mechanical Detection Threshold; MPS, Mechanical Pain Sensitivity; MPT, Mechanical Pain Threshold; PHS, Paradoxical Heat Sensation; PPT, Pressure Pain Threshold; QST, Quantitative Sensory Testing; QualST, Qualitative Sensory Testing; TSL, Thermal Sensory Limen; VDT, Vibration Detection Threshold; WDT, Warm Detection Threshold; WUR, Wind-up Ratio

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1073-8746/12/1801-$30.00/0

https://doi.org/10.1053/j.sodo.2018.04.004

a role in the development of the central sensitization phenomenon. Therefore, when central sensitization occurs, pain is maintained even after the initial injury might have healed.[2]

For long time, the integrity of the nervous conduction system has been addressed by means of electrophysiological tests. However, the propriety of the nerves to conduct electricity reflects only the capacity of large fibers, and is poorly related to the transmission of the sensory function.[5] Being able to objectively measure the somatosensory function, or dysfunction, allows a deeper understanding of the mechanism underlying the sensory experience and the pathophysiology of pain.[6] Clinical neurophysiology has offered several tools for the detailed investigation of the somatosensory functions. In particular, the orofacial area and the trigeminal nerve system have been studied for years by eliciting brainstem reflexes such as blink reflex, jaw jerk reflex, and masseter inhibitory reflex.[7] These tests can measure a wide and complex range of nerve transmissions, but still do not provide information on specific sensory afferents. Moreover, even when similar stimuli are used, lack of standardization in measurement techniques makes it difficult to combine results from different studies.[8]

Quantitative sensory testing (QST)

Quantitative Sensory Testing (QST) are psychophysical methods that have been firstly introduced in a clinical paper more than 40 years ago,[9] and since then numerous publications and research advances have been published. In the last decades, QST methods gained increasing interest in the pain research community since they are non-invasive, comprehensive and easy to perform clinical methods to assess the sensory nerve function, able to acquire objective and mechanistic information on the somatosensory system. A comprehensive battery of QST allows the standardized activation of specific nociceptive and non-nociceptive nervous transmitters (Aβ-, Aδ-, and C-fibers), the control of the stimulus application in terms of intensity, duration, and modality, the quantitative assessment of the evoked response, and the detection of both somatosensory loss and gain of function (negative and positive signs).[10] Sensory experiences can be quantified through several parameters: sensory threshold (the minimal intensity required at which the stimulus is firstly perceived), pain threshold (the intensity at which the stimulus is felt as painful), and tolerance levels (used mainly for studying pain).[5]

QST techniques have found large application in the evaluation of the sensory abnormalities of peripheral nerve diseases, such as diabetic neuropathy, carpal tunnel syndrome, multiple sclerosis, and many others (for review see Zaslansky and Yarnitsky).[11] In regards to the craniofacial district these tests, especially when used in combination, resulted able to evaluate the whole trigeminal and facial system, covering not only the brainstem complex, but a whole sensory axis, from the peripheral skin or mucosa receptors to the brain.[7]

In 2006, a comprehensive protocol of standardized QST has been developed by the German Research Network on Neuropathic Pain (DFNS). This protocol has been validated for the somatosensory evaluation of neuropathic pain patients in different region of the body, including the face.[12,13] Based on the DFNS protocol, the Special Interest Group of Oro-facial Pain (SIGOFP), during an international taskforce on the somatosensory testing, has proposed a comprehensive battery of QST methods for both screening and examination procedures specifically related to the oro-facial region.[14] This battery of QST demonstrated feasibility of adaptation both to the extraoral and intraoral regions,[8,15] and also showed good intra- and inter-examiner reproducibility in different patient settings.[8,16,17] It has to be underlined that the assessment of the intraoral sensory profiles required some changes due to the limited space available for the testing in the oral cavity.[8]

The comprehensive QST battery comprises 7 tests measuring 13 parameters, that can be grouped as following: 1) *thermal testing* - cool and warm detection threshold (CDT, WDT); thermal sensory limen (TSL); paradoxical heat sensations (PHS); cold and heat pain threshold (CPT, HPT); 2) *mechanical testing* - mechanical detection thresholds (MDT); mechanical pain threshold (MPT); mechanical pain sensitivity (MPS); dynamic mechanical allodynia (ALL); wind-up ratio (WUR); vibration detection thresholds (VDT); pressure pain threshold (PPT). With this protocol, the large Aβ-fibers are tested with the detection of mechanical touch and vibration sensation, while the small Aδ- and C-fibers are studied by measuring thermal detection, thermal