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Characterization of chronic pain and somatosensory function in spinal cord injury subjects

Ruth Defrin[a], Avi Ohry[b], Nava Blumen[b], Gideon Urca[a,*]

[a]*Department of Physiology and Pharmacology, Sackler School of Medicine, Tel-Aviv University, Ramat Aviv, Israel 69978*
[b]*Department of Neurorehabilitation, Sheba Medical Center, Tel-Hashomer, Israel*

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Abstract

The pathophysiology of the chronic pain following spinal cord injury (SCI) is unclear. In order to study it's underlying mechanism we characterized the neurological profile of SCI subjects with (SCIP) and without (SCINP) chronic pain. Characterization comprised of thermal threshold testing for warmth, cold and heat pain and tactile sensibility testing of touch, graphesthesia and identification of speed of movement of touch stimuli on the skin. In addition, spontaneously painful areas were mapped in SCIP and evoked pathological pain – allodynia, hyperpathia and wind-up pain evaluated for both groups. Both SCIP and SCINP showed similar reductions in both thermal and tactile sensations. In both groups thermal sensations were significantly more impaired than tactile sensations. Chronic pain was present only in skin areas below the lesion with impaired or absent temperature and heat-pain sensibilities. Conversely, all the thermally impaired skin areas in SCIP were painful while painfree areas in the same subjects were normal. In contrast, chronic pain could be found in skin areas without any impairment in tactile sensibilities. Allodynia could only be elicited in SCIP and a significantly higher incidence of pathologically evoked pain (i.e. hyperpathia and wind-up pain) was seen in the chronic pain areas compared to SCINP. We conclude that damage to the spinothalamic tract (STT) is a necessary condition for the occurrence of chronic pain following SCI. However, STT lesion is not a sufficient condition since it could also be found in SCINP. The abnormal evoked pain seen in SCIP is probably due to neuronal hyperexcitability in these subjects. The fact that apparently identical sensory impairments manifest as chronic pain and hyperexcitability in one subject but not in another implies that either genetic predisposition or subtle differences in the nature of spinal injury determine the emergence of chronic pain following SCI.

Keywords: Chronic pain; Somatosensory function; Spinal cord injury

1. Introduction

Chronic pain in the paralyzed body regions is one of the most severe outcomes of traumatic spinal cord injury (Nepomuceno et al., 1979; Davidoff et al., 1987; Beric' et al., 1988; Boivie, 1994). This type of chronic pain, often referred to as central pain, occurs in 11–94% of spinal cord injury patients, with percentages varying greatly between reports (Melzack and Loeser, 1978; Nepomuceno et al., 1979; Davidoff et al., 1987; Mariano, 1992; Fenollosa et al., 1993). In some cases pain is so severe as to produce a drastic impairment in everyday function, even to a greater extent than the motor impairment (Nepomuceno et al., 1979).

The pathophysiology of the chronic pain following spinal cord injury is still unclear. However the characteristics of the sensory loss which always accompanies this type of chronic pain led to several hypotheses. An early case reported by Browder and Gallagher (1948) suggested that chronic pain might develop following lesion of the Dorsal columns (DC). This hypothesis did not get additional support since subsequent observations showed that discrete lesions of the lemniscal system give rise only to parasthesias and not to chronic pain (Cassinari and Pagni, 1969; Pagni, 1977).

In a later study Beric' et al. (1988) examined 13 spinal cord injury (SCI) subjects with chronic pain. These subjects exhibited an almost complete loss of temperature and thermal pain sensations below the level of lesion, whereas vibration and touch sensations were significantly less impaired (Beric' et al., 1988). The authors concluded that the imbalance between the spinothalamic and DC systems is the main underlying cause of this central pain. In a recent study conducted by Eide et al. (1996), painful and painfree skin areas below the level of lesion, were examined in 16 SCI patients with chronic pain. Both areas showed reduction in thermal (temperature and thermal pain) and tactile sensibilities compared to normal areas above the lesion, but the

* Corresponding author. Tel.: +972-3-6408754.
E-mail address: gurca@post.tau.ac.il (G. Urca).

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reduction in thermal sensibilities in pain free areas was much smaller in magnitude. There was no significant difference in thermal and tactile sensibilities between painful and pain-free areas (Eide et al., 1996). Based on these findings the authors concluded that central pain is not dependent on the damage to either the spinothalamic tracts (STT) or DC, but more probably on hyperexcitability of nociceptive neurons.

The purpose of this study was to attempt and resolve the contradictory conclusions of these two studies (Beric' et al., 1988; Eide et al., 1996) by reexamining the sensory profile of SCI patients with chronic pain. To gain further information on the conditions necessary for the emergence of chronic pain following spinal cord injury, the sensory profile of SCIP patients was compared with that of pain free SCI patients.

2. Materials and methods

2.1. Subjects

Forty male and female patients participated in this study. These included fifteen subjects with incomplete spinal injury and chronic pain (average age 38.9 ± 9.2 years), seven subjects with incomplete spinal cord injury without chronic pain (average age 37.6 ± 9.1) and 18 healthy volunteers without neurological damage or chronic pain (average age 35.6 ± 7.4 = SD). SCI subjects included in this study were recruited from the outpatient population of the rehabilitation center on a voluntary basis. They were all paraplegics with spinal cord injury restricted to lumbar and thoracic segments (L3–T4) so that the hands were considered intact areas above lesion. All patients suffered from traumatic spinal injuries (Mainly due to car accidents). The chronic pain, in spinal cord injured patients, was always referred to regions below the spinal injury and had all the characteristics of central pain (Davidoff et al., 1987; Beric' et al., 1988; Boivie, 1994). The average duration of chronic pain in these patients was 14.9 years (range 2–35 years). Since eight (53%) SCI subjects of the chronic pain group regularly used analgesic medications, they were asked if possible, to take the medication following the testing session and not prior to, only in those days of their arrival.

Experiments were approved both by the Tel Aviv University and The Sheba Medical Center human rights committees. All subjects participated in this study on a voluntary basis. Informed consent was obtained from all subjects after receiving a full explanation of the goals and protocols of the study.

Testing took place in a quiet room. Temperature in the room was maintained at 22 ± 2°C. The subjects were seated in their wheelchairs or a comfortable armchair with the tested limb resting on a supporting structure. Each subject underwent three sessions, comprising of all tests, at a minimal interval of 1 week between sessions. Sensory testing was conducted below the level of injury. Since all subjects suffering from chronic pain complained of pain in the legs, we divided the testing regions into three areas. One in midthigh, one in the middle of the lateral shin, and one in the center of the dorsal aspect of the foot. SCI subjects with and without chronic pain were all tested in these areas (standardized areas), except for cases in which patients refused to allow further testing in very painful and/or sensitive areas. In addition, since chronic pain patients complained of pain sensations originating in areas not designated as testing areas (e.g. upper thigh, medial shin), testing was also conducted in all areas from which chronic pain was reported to originate. Since spinal injury level was not identical for all patients, testing was not conducted on all areas in all patients. Thus, patients injured at the T12 level were tested in all three areas, whereas those suffering from an L2 lesion were tested in the shin and foot only. A total of 68 areas were tested in chronic pain patients and 38 areas in spinal cord injury patients without chronic pain. Both normal and abnormal sensations were tested. All the following tests were first conducted on the dorsal surface of the hands (intact skin areas above lesion) so that SCI subjects could experience normal sensation before being tested below the lesion.

2.2. Thermal testing of spinothalamic function

Sensations of warmth, cold and heat and cold pain were tested to evaluate spinothalamic function (Willis, 1985; Nathan et al., 1986). The threshold of each thermal sensation was measured by means of a computerized thermal stimulator (TSA 2001, Medoc Inc., Ramat Ishai, Israel). The principles of the Peltier stimulator were described elsewhere (Fruhstorfer et al., 1976; Wilcox and Giesler, 1984; Verdugo and Ochoa, 1992). Briefly, passage of current through the Peltier element produces temperature changes at rates determined by an active feedback system (1–10°C/s). As soon as the target temperature was attained, probe temperature actively reverted to a preset adaptation temperature by passage of an inverse current.

Sensory thresholds: warmth, cold and heat-pain threshold was measured using the method of limits (Gescheider, 1985). For warmth and cold threshold determination, subjects received four successive, increasing or decreasing, stimuli starting from an adaptation temperature of 32°C, at a rate of 2°C/s with inter-stimulus intervals of at 15 s. For heat-pain threshold determination subjects received four successive stimuli starting from an adaptation temperature of 35°C at a rate of 2°C/s. A minimal interval of 30 s was kept between successive stimuli in the same session, in order to avoid possible sensitization or suppression of cutaneous receptors (Yarnitsky and Ochoa, 1990; Verdugo and Ochoa, 1992). The subject was asked to press a switch at the first pain sensation perceived. Warm, cold and heat-pain thresholds were the average reading of four successive stimuli in each session. In those areas with complete sensory loss, a cut-off temperature was assigned for each modality.