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RESEARCH ARTICLE

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Inhibition of pain and pain-related brain activity by heterotopic noxious counter-stimulation and selective attention in chronic non-specific low back pain

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Abstract—The aim of the present study was to assess inhibition of pain and somatosensory-evoked potentials (SEPs) by heterotopic noxious counter-stimulation (HNCS) and by selective attention in patients with chronic non-specific LBP. Seventeen patients and age/sex-matched controls were recruited (10 men, 7 women; mean a ge ± SD: 43.3 ± 10.4 and 42.7 ± 11.1, respectively). On average, patients with LBP reported pain duration of 7. 6 ± 6.5 years, light to moderate disability (19.3 ± 5.7/100) and low clinical pain intensity (21.8 ± 1.5/100), while pain catastrophizing, state and trait anxiety and depressive symptoms were not significantly different between groups (all *p*'s >0.05). HNCS and selective attention had differential inhibitory effects on pain and SEP, but no difference was observed between groups. Across both groups, HNCS decreased pain ($p = 0.06$) as well as the N100 and the N150 components of SEP (*p*'s <0.001), while selective attention only decreased pain ($p < 0.01$) and the N100 ($p < 0.001$). In contrast, the P260 was decreased by HNCS only when attention was directed toward the HNCS stimulus ($p < 0.01$). This indicates that patients with the characteristics described above do not show altered pain inhibitory mechanisms involved in HNCS and selective attention. Importantly, this experiment was carefully designed to control for non-specific effects associated with the repetition of the test stimulus and the effect of an innocuous counter-stimulation. It remains to be determined if these results hold for patients with severe LBP and psychological symptoms or whether symptom severity may be associated with pain inhibition deficits.

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Key words: CPM, DNIC, SEP, HNCS, nociception, selective attention.

INTRODUCTION

Acute low back pain (LBP) is a costly health condition affecting up to 80% of individuals at least once over their lifetime (Manchikanti et al., 2009). In 5–10% of cases, acute pain persists and becomes chronic (Manchikanti et al., 2009). In these individuals, who represent 85% of patients with chronic LBP, the clinical condition is defined as chronic non-specific LBP (Deyo and Phillips, 1976).

Although multiple factors are thought to be involved in chronic non-specific LBP, its pathophysiology remains unclear. Like other chronic pain conditions, one of the mechanisms that may contribute to LBP is the alteration of pain inhibition processes, including those involved in hypoalgesia induced by heterotopic noxious counter-stimulation (HNCS) (Kosek and Hansson, 1997; Lautenbacher and Rollman, 1997; Coffin et al., 2004; Pielsticker et al., 2005; Piche et al., 2010). HNCS hypoalgesia, also called conditioned pain modulation (CPM), has shown clinical relevance considering that the function of its underlying mechanisms has a predictive value for the development of postoperative chronic pain (Yarnitsky et al., 2008) and the efficacy of pain medication for the treatment of neuropathic pain (Yarnitsky et al.,

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Abbreviations: CPM, conditioned pain modulation; DNICs, diffuse noxious inhibitory controls; EMG, electromyography; HICS, heterotopic innocuous counter-stimulation; HNCS, heterotopic noxious counter-stimulation; PAG, periacqueductal gray matter; RIII-reflex, nociceptive flexion reflex; SEPs, somatosensory-evoked potentials; WDR, wide-dynamic-range.

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2012). Experimentally, the integrity of pain mechanisms underlying HNCS hypoalgesia can be assessed by the application of tonic pain (HNCS/conditioning stimulus) to a distant body site during a series of painful phasic stimuli (test stimulus).

Although a consistent and reproducible deficit in HNCS hypoalgesia was reported in some chronic pain conditions, including fibromyalgia (Lautenbacher and Rollman, 1997; Staud et al., 2003; Julien et al., 2005; Vierck, 2006; Johannesson et al., 2007; de Souza et al., 2009; Goffaux et al., 2009; Staud, 2009; Normand et al., 2010) and irritable bowel syndrome (Coffin et al., 2004; Wilder-Smith et al., 2004; King et al., 2009; Heymen et al., 2010; Piche et al., 2010, 2011, 2013; Bouhassira et al., 2013), assessment of HNCS hypoalgesia in patients with LBP led to mixed results. In one study, HNCS hypoalgesia was decreased in females but not in males with chronic LBP compared with controls (Correa et al., 2015). In another study, HNCS produced hyperalgesia instead of hypoalgesia in some participants and the proportion of such participants was greater in the group of patients with chronic LBP compared with controls (Rabey et al., 2015). In contrast, other studies showed that patients with chronic LBP have similar HNCS hypoalgesia compared with controls (O'Neill et al., 2014; Owens et al., 2015; Dubois et al., 2016). Comparable pain inhibition was also reported in patients with LBP and controls in a study using a spatial summation protocol that triggers pain inhibition processes (Julien et al., 2005). Such divergent findings between studies may be explained, in part, by attentional processes, including selective attention. Accordingly, we reported that the net hypoalgesic effect of HNCS is smaller when participants are instructed to direct their attention toward the test stimulus as opposed to the conditioning stimulus (HNCS) (Ladouceur et al., 2012). Therefore, the inconsistent reduction in HNCS hypoalgesia reported in some chronic pain conditions, including LBP, may partly reflect a difference in the allocation of attention.

Accordingly, patients with chronic pain tend to present an attentional bias toward pain-related information compared with healthy controls (Pearce and Morley, 1989; Snider et al., 2000; Beck et al., 2001; Pincus and Morley, 2001; Roelofs et al., 2002; Haggman et al., 2010; Schoth et al., 2012; Crombez et al., 2013), especially those with greater fear of pain (Crombez et al., 1999a; Eccleston and Crombez, 1999; Roelofs et al., 2002). This bias leads to increased selective attention toward pain (Asmundson et al., 1999; Crombez et al., 1999b; Peters et al., 2002), which in turn may lead to altered pain inhibition and play a role in chronic LBP (Roelofs et al., 2005; Haggman et al., 2010).

By its unpleasant and alarming features, pain captures attention. Disengaging selective attention from a painful stimulus in patients with a bias for pain information may then be affected because of the priority attributed to the painful stimulus relative to other stimuli in the environment. On one hand, a bias of selective attention toward pain sources in acute pain states allows a prompt detection of stimuli that may produce further damage (Roelofs et al., 2002). However, this adaptive behavior becomes maladaptive in a chronic pain state by maintaining or exacerbating pain (Crombez et al., 1999a; Eccleston and Crombez, 1999; Pincus and Morley, 2001).

The aim of the present study was to assess inhibition of pain and pain-related brain activity (somatosensory-evoked potentials: SEPs) by HNCS and selective attention in patients with chronic non-specific LBP. Based on the literature mentioned above, we expected a decrease in the inhibition of pain and pain-related brain activity by selective attention in patients with chronic LBP compared with controls. For HNCS hypoalgesia, conflicting results reported earlier prevent stating a strong hypothesis. However, we hypothesized that the deficit in HNCS hypoalgesia may be more readily observed in patients with LBP compared to healthy controls when attention is directed explicitly toward the test stimulus or the conditioning stimulus as instructed by the task, in all participants.

EXPERIMENTAL PROCEDURES

Ethical approval

All experimental procedures conformed to the standards set by the latest revision of the Declaration of Helsinki and were approved by the Research Ethics Board of "Université du Québec à Trois-Rivières". All participants gave written informed consent, acknowledging their right to withdraw from the experiment without prejudice and received compensation of $ 50 for their travel expenses, time and commitment. The study consisted in three sessions of 120 min during which participants filled questionnaires and completed the experimental protocol. Participants from both groups were recruited by advertisement in the local newspaper and on the campus of "Université du Québec à Trois-Rivières".

Study participants

Participants with chronic non-specific low back pain. Seventeen patients with chronic non-specific LBP were recruited (10 men and 7 women; range 24–55 years; mean ± SD: 43.3 ± 10.4). Patients with chronic LBP were examined by a chiropractor and were included if they were between 18 and 60 years old and suffered from non-specific low back pain for at least one year. They were excluded if they had any neurological disorder, a history of disc herniation, neurological symptoms associated with their back pain or the presence of a major scoliosis (> 20°). They were also excluded if they were diagnosed with clinical depression, anxiety or other psychiatric disorders.

Control participants. Seventeen age- and sex-matched control participants (10 men and 7 women; range 24–55 years; mean ± SD: 42.7 ± 11.1) were included in the study. They were excluded if they had taken any analgesic medication within 2 weeks prior to the experiment and if they had a history of acute or

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