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Review

A comprehensive review of pain interference on postural control: from experimental to chronic pain

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Abstract: Motor control, movement impairment and postural control recovery targeted in rehabilitation could be affected by pain. The main objective of this comprehensive review is to provide a synthesis of the effect of experimental and chronic pain on postural control throughout the available literature. After presenting the neurophysiological pathways of pain, we demonstrated that pain, preferentially localized at low back or in the leg induced postural control alteration. While proprioceptive and cortical excitability seems modified with pain, spinal modulation assessment might provide new understanding of the pain phenomenon related to postural control. Literature highlight that the motor control of trunk muscles in patient presenting with low back pain could be dichotomized in two populations, where the first one over-activate trunk muscles, the second one under-activate trunk muscles, and both generating increase of tissues loading. Taking account all this findings, will help clinician to provide adapted treatment for managing both pain and postural control.

Keywords: pain; postural control; rehabilitation

1. Introduction

Chronic pain is defined by the International Association for Study of Pain (IASP) as « an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage » [1] lasting more than 3 months [2]. By affecting more than 30% of population worldwide [3], chronic pain leads to economic burden and has dramatic impact on biological, psychological, sociological factors resulting in poor quality of life [4-6]. While medical care focused in pain perception, psychological and functional disability should be considered [6]. It has been clearly demonstrated that pain interferes with sensorimotor control [7-11], and more especially with postural control [12-20].

Postural control, either in static or dynamic conditions, is an essential requirement to perform daily activities [21]. The upright standing human body, classically represented by an inverted pendulum model, is intrinsically unstable as reflected by the movement of the centre of mass (CoM) [22]. To maintain upright standing, the postural system requires efficient functioning of the sensorimotor mechanisms and the ability to detect body sways through reliable sensory systems integrating these sensory cues provided by the visual, vestibular, proprioceptive and exteroceptive systems [23-29]. The integration of sensory information appears to be dynamically regulated related the available sensory information depending on environmental conditions, a process referred to as sensory reweighting [30-33]. When one (or more) of the sensory systems is altered, the Central Nerv-

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ous System regulates balance by attributing a higher weight to the remaining afferent information [30]. Both, sensory integration and reweighting are used by the neural control system to generate a corrective torque at the ankle to resist the deviations of the human body from an upright reference position [34]. Balance control, commonly assessed by measuring center of pressure (COP) displacement, could represent one of the sensorimotor control signatures observed in patients with chronic pain.

The main aim of this comprehensive review is to provide a synthesis of the effect of experimental and chronic pain on postural control by combining knowledge from the literature and identifying potential impact of pain on the sensorimotor mechanisms involved in postural control. This review also summarize evidence supporting the importance of including postural control in the clinical assessment of patient suffering from chronic pain. Improving knowledge of pain interference on postural control could help for developing new and adapted therapeutic approaches for patients presenting chronic pain.

2. From nociception to pain

The process leading to pain starts with stimulation of nociceptors [35]. There are two main classes of nociceptors. The first comprises myelinated afferents of medium diameter (Aδ) which mediate acute and well-localized "first" or rapid pain. The second class of nociceptor consists of small diameter unmyelinated "C" fibers that transmit poorly localized "secondary" or slow pain [36]. Myelinated Aδ nociceptors respond to mechanical and thermal stimuli, while unmyelinated C-fiber polymodal nociceptors generally respond to mechanical, thermal, or chemical stimulation. Specific nociceptors are only excited when stimulus intensities reach the noxious range, suggesting that they possess biophysical and molecular properties allowing them to selectively detect and respond to harmful stimuli [36]. Ion channels on peripheral nociceptors can be activated by direct stimulation or by molecules released at a site of inflammation (bradykinin, prostaglandins, histamine, serotonin, and others), leading to depolarization of small primary afferent of the first order neurons expressing these channels [37]. Action potentials, with a frequency proportional to the intensity of the stimulus, propagate along the axons of myelinated or unmyelinated nociceptive fibers through the dorsal root ganglion (DRG) to the axonal endings of the spinal cord, which are organized into anatomically and electrophysiologically distinct laminae [35].

The nociceptive Aδ and C fibers surround the outermost layer of the dorsal horn. They enter the dorsal horn and end in the superficial layers (called Rexed Laminae I and II) or extend into the deep layers (Lamina V) probably via interneurons [36]. The lamina II plays a key role in the modulation of pain in the spinal cord [38, 39]. The lamina II, also known as the substantia gelatinosa system, acts as an inhibitory mechanism on central transmission cell (T)-cells. Stimulation of nociceptive Aδ and C fibers inhibits the substantia gelatinosa cells, reducing the output and their inhibitory action on the (T)-cells, leading to an increase in their activity. The reduction in the ability of (T)-cells to receive or respond to the stimuli, is the hallmark of the gate control theory at the spinal level [40]. As a reminder, the (T)-cells are located in the dorsal horn of the spinal cord. They receive a balanced input of large of Aβ and small Aδ and C fibers activity in the peripheral nerves. Inhibitory interneurons, located in the substantia gelatinosa, can be activated by large afferents and can modulate the transmission of pain by projection to small fibers and central transmission cells [40].

Because pain is a complex multifactorial subjective experience, a large brain network is engaged during nociceptive processing. Numerous central nervous system structures (e.g., the anterior cingulate cortex, thalamus, and insula) consistently respond to transient nociceptive stimuli causing pain. Activation of this pain matrix or pain signature has been related to perceived pain intensity, both within and between individuals [2013]. Following integration into the dorsal horn, nociceptive information is conducted via two phylogenetically distinct systems, the medial and the lateral systems, to the higher centers of

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brainstem and brain. The medial system is involved in the affective and cognitive dimension of pain, pain memory, and autonomic responses [42, 43]. This medial pathway projects directly to the higher brain structures and mainly includes the spinoreticular tract, the spinomesencephalic tract, the spinoparabrachial tracts, the spinohypothalamic tract and the spinothalamic tract fibers. A component of the spinoreticular tract projects to the lateral reticular formation involved in motor control. The other component projects to the medial, pontomedullary reticular formation and, from there, to the thalamocortical circuits. A major target of the spinomesencephalic tract is the parabrachial nucleus of the pons, a region involves in the integration of the cardiovascular, autonomic and motivational response to pain. Other collaterals of the spinohypothalamic pathway project at the thalamus and also innervate the medulla and pons of the brainstem, sites of origin of the descending modulatory pathways [for review, please see 39]. The lateral system provides information on the location and duration of pain and plays an important role in the sensory-discriminating component of pain. This lateral system is formed by the spinocervical pathway, which projects to the lateral cervical nucleus at the C1-C3 level, and the nuclei of the dorsal column, which project to the cuneate and gracile nuclei of the dorsal column of the spinal cord. From the lateral cervical nucleus, information travels by the cervicothalamic tract to several thalamic nuclei, including the ventroposterior and posterior nucleus groups, and by a cervicomesencephalic pathway to the midbrain, including the periaqueducal grey and superior colliculus. With regard to the nuclei of the dorsal column, the output neurons project by the medial lemniscus to the ventroposterior and posterior groups of thalamic nuclei and to the superior colliculus [for review, see Millan, 39].

3. Interaction between pain and postural control

Experimental pain has been used to determine the potential impact of pain on balance control. By inducing heat pain on the lower leg muscle (45°C), Blouin et al. [15] showed a significant increase in COP velocity in comparison with the non-pain condition (i.e., heat stimulation at 40°C). Similarly, other studies have reported that unilateral hypertonic saline injection at infrapatellar fat pad [18], thigh [17] or leg muscles [16] led to significant increase in body sways and muscle activities. In addition, it has been reported that pain induced by electrical stimulation on the dorsum of the feet caused larger COP displacement [14]. By inducing different level of pain (weak, moderate, extreme), the authors observed that the COP displacement scaled with the level of pain. Furthermore, they reported that pain induced at the hand did not change COP displacement, showing specificity of the pain location related to postural control interference. The authors concluded the painful stimulation affects postural control via the sensorimotor mechanisms rather than cognitive processes related to perception of pain.

Even though some studies did not observed improvement in balance control in individuals with LBP compared healthy counterparts [45, 46], a systematic review, including 16 studies, reported that low back pain results in COP parameters alteration (e.i., increase of COP velocity and sway in anteroposterior direction) [19, 20]. Pain influences sensorimotor response in individuals with LBP, delaying and reducing the COP displacement on unstable surfaces [47], as well as increasing postural sway in the antero-posterior and medio-lateral direction in open eyes [21, 48] , closed eyes [49] conditions, and in a single leg support [50]. Considering all of these result, pain may alter the sensorimotor components of the postural system controlling balance [51-55]. Pain and impaired postural control often imply reduced muscle strength [56], physical inactivity [57] and depression [58]. Musculoskeletal pain is also associated with an increased the risk of falling [12, 13, 59]. Results from various studies also highlighted reduced trunk movements [60] and trunk stiffness [61]. These alterations likely cause postural instability [50] and may an indicator of dysfunctional postural control strategies [61, 62].

Some studies also reported a decreased in proprioceptive acuity, that is, patients with back or neck pain have less accurate positions sense [63, 64] suggesting impairment in body sway perception. More specifically, Popa et al. [65] suggested that the deterioration

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of proprioceptive information of the lower limbs and the trunk determines a reduced accuracy in the sensory integration process and thus a more imprecise internal estimate of the center of mass (CoM) position in individuals with chronic low back pain (CLBP). Consequently, the motor controller needs to increase the safety margin of the CoP shifts with respect to the predicted oscillation of the CoM, reflected by a greater sway. Individuals with CLBP might set ankle stiffness at a higher level in order to compensate for sensory deterioration [66], as already demonstrated by reducing plantar sole sensitivity [24, 25]. Reweighting of proprioceptive input by increasing the gain at the ankle joints (increasing loading of ankle extensors by leaning more forward) may enhance sensory discrimination and help maintaining a critical level of sensory information to adequately cope with postural perturbations [65]. Overall, these sensorimotor changes may alter postural control [67]. Balance disorders may be associated with specific clinical findings, such as reduced muscle strength, impaired cognition, sensory or motor deficits, lower-extremity myofascial trigger points [68] or change in flexibility and coordination [69]. Patients with chronic pain syndrome, such as fibromyalgia, reported larger body sway than healthy controls [70], and balance impairment represents one of the top 10 most debilitating symptoms [71]. It was proposed that fibromyalgia likely affects dynamic balance control because of altered somatosensory inputs to central nervous system, including abnormal perception of pain with light somatosensory stimulation [68].

Persistent pain also alters cognitive processes. As cognition contributes to balance control [72-74], it is crucial to assess the relationship between pain intensity, cognition and balance control. Individuals with severe pain showed less effective executive functioning [75]. Such cognitive deficits are associated with impaired physical functioning including gait speed, balance performance, sit-to-stand and trunk rotation [76]. Because pain alters the sensorimotor mechanisms involve in balance control [77, 78], clinical evaluation should assess balance control.

4. Mechanisms of action of pain and potential mechanisms involved in postural control alteration in pain condition

Pain is intimately linked to the activation of a complex cerebral network as mentioned above, and involves cortical reorganization. Results from studies inducing pain confirmed a causal relationship between pain and cortical changes [10, 79-82]. Experimental pain studies showed increase of the primary motor cortex (M1) activity [83-85]. Using electroencephalography (EEG), Stancák et al. [86] reported that short-lasting painful heat stimuli on the hand decreased beta (β: 15-30 Hz) activity within the sensorimotor cortex. Given the inhibitory role that β oscillations has on the motor cortex [87], the decrease in primary motor cortex (M1) activity suggests that a brief nociceptive stimulus could alter (reduction of the inhibition) the motor region, possibly to facilitate withdrawal responses [88]. In a recent systematic review and meta-analysis, Rohel et al. [10] confirm the inhibitory effect of pain on corticospinal excitability. More specifically, Billot et al. [9] reported that heat pain applied at the tibialis anterior muscle significantly reduced corticospinal excitability either during active muscle contraction or at rest. These results provide evidence that nociceptive sensory input can impact corticospinal excitability at the lower limb. Incoming research, using transcranial magnetic stimulation of the lumbar erector spinae muscles [89], will help to delineate corticospinal excitability modulation with pain.

Using neuroimaging and neurostimulation, numerous studies showed that patients with chronic pain, such as complex regional pain syndrome (CRPS) [90] or phantom limb pain [91], presented cortical reorganization at the M1 level, with a smaller corticomotor representation of the affected limb, compared to pain-free participants. A normalization of the cortical changes was observed in CRPS patients after treatment over 1 to 6 months consisting of graded sensorimotor retuning [92], once pain subsided [92, 93], underlying the fact that cortical reorganization may play a major role in the physiopathology of chronic pain [92, 93]. These results support a causal relationship between pain and cortical

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changes. The cerebrum works as an integrated system of circuits and certain brain areas, other than those classically involved in pain perception and modulation, can be affected by nociceptive stimulations [88].

At a lower anatomical level, spinal control may likewise be affected by pain conditions. To date, experimental pain studies failed to provide strong evidence in a potential inhibitory or facilitatory effect at the spinal level. Farina et al. [94] did not support any modification of H-reflex of the flexor carpi radialis muscle while inducing tonic pain by applied capsaicin cream. Investigating the influence of pain (hypertonic saline into biceps brachii) at cortical and spinal levels, Martin et al. [95] showed cervicomedullary motor evoked potentials increase at rest for both biceps and triceps brachii, and for agonist muscle during constant EMG elbow flexion (biceps) and extension (triceps). In another hand, Le Pera et al. [54] reported H-reflex amplitude reduction in the recovery period after related-pain induced by hypertonic saline injection in the flexor carpi radialis. The authors interpreted this delayed H-reflex depression by inhibition of the spinal motoneurones excitability that overlap the cortical inhibition observed by motor evoked potential amplitude (corticospinal excitability) decrease. In addition to spinal excitability, pain induces steady variations in spinal transmission that could alter motor strategies [52]. For instance, prolonged exposure to nociceptive stimulations from the skin or sore muscles induced large errors in a torque-matching task [96]. The authors reported that participants overestimated the torque level generated by a limb affected by pain. In addition, pain could induce a distortion of the body image, leading to a biased estimation of the body position in space [55].

Assessment of motor control in patients presenting with CLBP considers three main classes of motor tasks, evaluating control of the trunk in steady-state condition (posture and movement) or challenging by predictable or unpredictable perturbations [97]. Regarding the first condition, literature provided inconsistent lumbar extensor muscle activity through 30 studies by reporting higher, no difference, or lower muscle activity [7]. The results may differs depending on anatomical specificity of the muscle, with deeper muscles were more systematically inhibited and superficial muscles activity were preferentially augmented [7]. Likewise, by investigating anticipatory activation of the trunk muscles occurred after expected or unexpected perturbations in CLBP patients, studies reported late activation of the transversus abdominis and multifidus muscles [98-103], no modification [104], or earlier activation [105, 106] In line with these results, the trunk movement alteration observed in CLBP patients may result from proprioception deficiency [107, 108]. Far from placing all these results in opposition, van Dieen et al. [97] propose to dichotomize patients profil/phenotype, where one phenotype includes patients with tight trunk control associated with over-activation of trunk muscle due to excitability increase and causing tissue loading increase, and the second phenotype includes patients with lose control associated with excessive spinal movements due to excitability decrease and causing tissue loading increase. Thereby, in a nutshell, patients suffering from CLBP presents abnormal loading of the tissues in the low back originated from different mechanisms.

5. Conclusions

There is no doubt that pain modify movement and motor control, illustrated by postural control alteration. This review showed that both experimental and chronic pain lead to postural control impairments. While the cortical modification has been largely investigated with pain localized at the upper limb, cortical and spinal modulation focusing on spine and lower limb muscles have to be determined. Finally, different phenotype of motor control by tight or loose trunk control should be considered to provide adapted treatment for managing both pain and postural control in patients presenting with chronic low back pain.

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