



Interlink of Pain and Motor Controls: An Enigma



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Editorial

It's not certain nor very clear that pain causes changes in motor control or motor changes lead to pain, or both. Where two researches Farfan [1] & Panjabi [2] have presented their models amongst others presenting that deficits in the motor control lead to poor control of joint movement, repeated micro trauma and pain. Later on with this models Janda [3] has put forward his theory and suggested that population who have mild neurological signs are more subjected to have pain. Furthermore slow reactions have been attached to increase risk of musculoskeletal injuries [4]. Therefore however their conversations may be true, and draws a result that Pain leads to motor control changes, numerous studies have been supported this hypothesis and studies by producing of changes in control that have been identified in clinical patients [5-7]. Relevantly there are many studies proposed to clear the theories that are confused to explain the effect of pain on motor controls.

Mainly this change in motor controls due to pain includes changes in excitability at the spinal or cortical level, changes in proprioception or afferent mediated control, or specific cortical effects imparted by aspects of pain, for its demands on the CNS resources, stress, anxiety and fear. Wide spread changes in excitability have been identified at many levels of the motor system during pain. Reflex inhibition of motor neuron excitability has also been suggested to occur in association with the swelling [8] and any injury to joint and its respective structures [9]. It has been often focused in the literature about the interlinks of motor controls and pain, where the both compromise the range of movement, reduction of strength and endurance of muscles and effects the resultant activities of daily, resulting in hypoactive and hyperactive activities. Its aware that pain always compromises our works in meeting the demands on daily.

There are two major theories available on this hypothesis:

- (i) That changes in muscle activity cause spinal pain (muscle-tension or pain-spasm-pain model) or
- (ii) Changes in muscle activity serve to restrict spinal motion (pain adaptation model).

One more agenda or factor to be considered in this hypothesis is that accuracy of movement control is dependent on the sensory element of the motor system. Inaccurate afferent input would affect all aspects of motor control from simple reflex responses especially for instance those arising from mechanoreceptors. In muscles or other elements of spine. Where as many studies are also argued that sensory acuity may reduce after repetitions due to fatigue there by decreased muscle endurance with injury or pain may lead to impaired sensory acuity through increased fatigability. There by this can lead to pain and change in the motor controls finally leading to reducing the accuracy of the daily activities.

It is well aware that from the daily activities muscle pain interacts with the movement performance. This article illustrates how muscle pain affects the muscle control in different ways.

Resting muscle activity and muscle pain

Increased resting muscle activity after saline induced muscle pain is found compared with base line recordings, but not compared with a sham pain condition where patient gets a condition which is very painful without having the actual pain in stimulation [10]. This indicates us to understand that hyperactivity is not present owing to the muscle pain. In another study, a transient increase in the resting electromyography (EMG) activity during infusion of hypertonic saline was recorded in contrast with infusion of isotonic saline [11]. The point here to be learnt was on-going muscle pain did not produce sustained increased EMG activity, and where as experimental muscle pain doesn't cause any changes in resting EMG activity between repeated maximal voluntary contractions [12].

Static muscle activity and muscle pain

The maximal voluntary contraction during saline induced muscle pain is significantly lower than in the control condition [13]. Attenuation of maximal voluntary contraction force during experimental muscle pain was not associated with the changes in contractile properties of muscle fibers, but to a pure central effect; that is for a motor control modulation. A clinical demonstration of the observed decrease in muscle strength during voluntary

isometric contractions of a painful muscle has also been made in musculoskeletal pain patients.

In fibromyalgia patients, the reduction in strength during voluntary isometric contractions of painful muscle has also been made in musculoskeletal pain patients. In fibromyalgia patients, the reduction in strength is suggested to be due to deficient central activation of motor units because supramaximal stimulation of ulnar nerve shows no difference in the strength of the adductor pollicis muscle between patients and a control group [14]. During static contraction, experimental muscle pain causes reduction in endurance time [15]. The different findings between sub maximal and maximal contractions may be explained by changes in the descending neural drive cannot be voluntarily increased during maximal voluntary contractions. An interesting observation is that the muscle pain during static contractions not only decreases the muscle activity of the painful muscle, but also attenuates synergistic muscles [15].

Dynamic muscle activity and muscle pain

Experimental models of muscle pain have also been used in occupational settings (low load with repetitions) where saline-induced neck muscle pain was found to cause changes in motor strategies, for example a decreased working rhythm and a muscle coordination change which may be interpreted as protective. Many studies of the relationship between work-related muscle pain and muscle activity have been carried out an attempt to find a valid predictor for the development of neck-shoulder pain. A decreased frequency of unconscious gaps in the low-level EMG activity was found to predict the patients who developed neck-shoulder pain [16]. A higher EMG level during low load repetitive work was found among the group of workers who developed neck shoulder complaints, highlighting that the level of activity may have some prognostic relevance for the development of chronic neck-shoulder pain [17].

Neurophysiologic mechanisms: Lund [18] suggested the pain-adaptation model to explain the link between activity in nociceptive afferents, a central pattern generator, the motor function, and coordination of muscles. This pain-adaptation model predicts increased muscle activity in antagonistic phases and decreased muscle activity in agonistic phases during muscle pain. Such a co-ordination may produce a decrease in movement amplitude and velocity. The pain-adaptation model includes inhibition and excitation of motor neurons according to functional phases of the painful muscle. Findings indicate coordination and control change for muscle pain is not voluntary, but caused by reflex mechanism. There by the interactions between muscle pain and motor control depends on specific motor related task, muscle pain cause no related changes in EMG activity at rest and reduces maximal voluntary contractions and endurance time during sub maximal contractions. Moreover, muscle pain can also cause loss of coordination in the dynamic exercises .The functional adaptation to muscle pain may also involve increased muscle activity, reflecting changed muscle coordination and strategy.

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