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CRITICAL REVIEW

Effectiveness of neural mobilization in patients with spinal radiculopathy: A critical review

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Summary Spinal radiculopathy (SR) is a multifactorial nerve root injury that can result in significant pain, psychological stress and disability. It can occur at any level of the spinal column with the highest percentage in the lumbar spine. Amongst the various interventions that have been suggested, neural mobilization (NM) has been advocated as an effective treatment option. The purpose of this review is to (1) examine pathophysiological aspects of spinal roots and peripheral nerves, (2) analyze the proposed mechanisms of NM as treatment of injured nerve tissues and (3) critically review the existing research evidence for the efficacy of NM in patients with lumbar or cervical radiculopathy.
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Introduction

Nerve roots are susceptible to injury at any level of the spinal column, with a high percentage of these injuries occurring at the lumbar and cervical spine (Konstantinou and Dunn, 2008; Abbed and Coumans, 2007). Spinal radiculopathy (SR) is defined as a disorder of the spinal nerve root(s) most commonly caused by a disc herniation, or a space-occupying lesion that can result in nerve root inflammation, impingement, or both (Wainner et al., 2003). Also, malignant and infectious causes of SR have been reported and hence should always be suspected, as these patients would require medical referral and not any type of physiotherapy intervention (Stafford et al., 2007).

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Depending on the spinal level of nerve root irritation, SR can be further categorized as cervical (CR), thoracic (TR) and lumbar radiculopathy (LR). Epidemiological data for CR has shown an annual incident of 0.1% in males and 0.06% in females in the general population with an increased prevalence occurring in the fifth decade of life (Radhakrishnan et al., 1994). In the lumbar spine, the frequency of LR is highly variable, depending largely on the characteristics of the population studied, with annual values ranging from 2.2% in the general population to 34% in specific working populations (Konstantinou and Dunn, 2008). Men are more likely to have LR in their 4th decade of life, while women have higher rates in their 5th and 6th decade of life (Tarulli and Raynor, 2007). Thoracic disc herniation and diabetes mellitus are two of the most common etiologies for the development of TR. There is no available epidemiological data on TR, however certain data on thoracic disc herniations indicate that they occur in only 0.15—4% of all symptomatic disc herniations of the spine, and they represent less than 2% of all spinal disc surgeries (O'Connor et al., 2002). Since thoracic disc herniations are the less common across the whole spine and since disc herniation is the most common cause of SR (Radhakrishnan et al., 1994), TR should also be less common than CR and LR.

The pattern and location of the patient's symptoms may vary significantly, depending on the level of the affected nerve root (Cleland et al., 2005). The two most commonly affected levels are L4-5 or L5-S1 (90%) among all LRs (Murphy et al., 2009), and C7 (31%—81%), C6 (19%—25%) and C5 (2%—14%) among all CRs (Greathouse and Joshi, 2010). For TR, T11-T12 interspace is affected in 26%—50% of all cases (O'Connor et al., 2002). Common symptoms include weakness, numbness, paresthesia or a combination of all these symptoms (Young et al., 2009), which often cause disability and functional limitations (Cleland et al., 2005). SRs are often accompanied by (radicular) pain, but they are not defined by pain, as they can often occur in the absence of it (Bogduk, 2009).

Pathophysiology of injured nerves

In order to understand the mechanism through which any type of technique can have an effect on neural tissues, it is essential to understand the cascade of events that occur once a nerve has been affected by a mechanical or chemical stimulus that exceeds its threshold of tolerance.

Nerves have the ability to adjust to different types of mechanical stress imposed on them due to normal every day limb movements (Topp and Boyd, 2006). It is important for the integrity of the nerve that the duration and/or degree of the stress never exceeds the nerve's ability to withstand it. Ischemia and impaired function seem to be the first results when intraneural circulation and axoplasmic flow are blocked by compressive, tensile or shear forces (Topp and Boyd, 2006). Animal studies have demonstrated that nerves show time-dependent viscoelastic behavior (Topp and Boyd, 2006). Driscoll et al. (2002) investigated the effect of 16.1% strain on the sciatic nerve of 10 rabbits. They found that 16.1% of strain resulted in nerve blood flow reduction of 78% and that this reduction failed to recover after 30 min of rest. Jou et al. (2000) also found that 24% and 32% lengthening of the sciatic nerve of rats produced 50% drop in nerve blood flow measured with laser Doppler flowmetry. The effects of nerve compression, have also been extensively explored in animal models using various methods (miniature inflatable cuffs or silicon tubes around the nerve) to induce acute or chronic compression (Dahlin and Kanje, 1992; Dyck et al., 1990). Extraneural pressures have been found to inhibit intraneural microvascular blood flow, axonal transport and nerve function with increases of intrafascicular pressure in a dose—response manner (Rempel et al., 1999).

The main sources of compressive stress that will impede blood flow of the nerve root are disc herniations, osteophytes of the facet or uncovertebral joints and stenosis of the spinal canal (Kobayashi et al., 2003). With the contrary to dorsal root ganglion (Bogduk, 2009), root compressions can cause sensory and motor dysfunction but usually not pain (Mulleman et al., 2006). Pain, is typically generated when microvascular alterations as a result of compression lead to upregulation of inflammatory mediators (Kobayashi et al., 2004). Inflammation can ultimately lead to adhesions between the herniated disc and the nerve root that will impair gliding of the nerve root. In the acute and sub-acute stages of nerve root compression, neural conduction block, intraneural edema, mechanical sensitization and increase of sodium channel density have been reported (Chen et al., 2003; Kobayashi et al., 2004; Rempel et al., 1999). Dysfunction can also extend to primary sensory neurons within the dorsal root ganglion (Kobayashi et al., 2004). The result of these changes manifests itself as increased mechanosensitivity. It is worth noting that the critical threshold for duration and magnitude of compression has not been fully determined yet (Rempel and Diao, 2004).

Furthermore, substances contained in the herniated material can cause inflammation and radicular pain without evidence of true mechanical compression (Videman and Nurminen, 2004). This is because the nucleus pulposus is a very powerful inflammatory stimulus (Takahashi et al., 2003; Mulleman et al., 2006) possibly due to its high proteoglycan content (Urban and Roberts, 2003). Takebayashi et al. (2001), found mechanical hypersensitivity in the dorsal root ganglion of 14 rats after implanting nucleus pulposus at the L5 nerve root. In another animal study, induced neuritis in the sciatic nerve of rats produced axonal inflammation characterized by recruitment of macrophages and lymphocytes (Bove et al., 2003). This led to an increase of the pro-inflammatory cytokine, tumor necrosis factor alpha (TNFa), which in turn created spontaneous activity in nociceptors via an increase in sodium channel conductance. Elevated levels of neurotrophines such as nerve growth factor can sensitize C fibers of the nervi nervorum resulting in the release of prostaglandins and bradykinin (Onda et al., 2005; Greening, 2004). Other inflammatory mediators such as serotonin have also been involved (Kato et al., 2008). Interestingly, these inflammatory responses can cause nerve mechanosensitivity without evidence of major axonal degeneration and damage (Bove, 2008). Dilley et al. (2005) found that induced local neuritis in the nerve trunks of adult rats caused small numbers of structurally intact myelinated and unmyelinated afferent fibers to develop increased sensitivity to stretch and pressure. Patients presenting with radicular-like pain without radiculopathy (sensory and motor disturbance) are sometimes provided

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