Review Article

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Efficacy of Neurodynamic Technique on Athletes with Lower Lumbar Radiculopathy: Narrartive Review

Rashmi Gupta*, Saurabh Sharma**

Abstract

Lumbar radiculopathy is associated with nerve root injury. It involves radiating pain below knee with neural deficit in neural distribution pattern. Neurodynamic technique has been advocated as an effective treatment for lumbar radiculopathy. The purpose of this review is to analyse the mechanism and the effectiveness of neurodynamic technique and to examine the effect of neurodynamic technique on motor nerve conduction velocity (mNCV) and surface electromyographic activity (sEMG).

Keywords: Neural Mobilisation; Lumbar Radiculopathy; NCV; sEMG.

Introduction to Lumbar Radiculopathy

Lumbar radiculopathy is defined as the radiating leg pain below knee with neurological deficits in the distribution of the nerves (BW Koes et al; 2007). The major symptoms of lumbar radiculopathy involve pain, numbness, paresthesia and loss of muscle strength. The clinical presentation of lumbar radiculopathy depends on the nerve root impinged. In this study we are including the subjects with lower lumbar radiculopathy (L4-L5 and L5-S1).

Clinical presentation of L4 radiculopathy includes pain on the medial side of the leg, paresthesia over the medial leg along the course of saphenous nerve. There may be weakness of tibialis anterior, quadriceps and hip adductors. Knee jerk may be hypo-reflexive or absent in radiculoapthy (Tarulli & Raynor, 2007).

Clinical presentation of L5 radiculopathy includes pain along the lateral thigh, lower leg and dorsum of foot. Patient may experience paresthesia over the lateral lower leg, dorsum of foot and great toe. There may be weakness of toe extensors and flexors, ankle dorsiflexors, evertors and invertors and hip abductors. Internal hamstring reflex is affected in this radiculopathy. Patient might experience foot drop (Tarulli & Raynor, 2007)

There are several causes of lumbar radiculopathy like intervertebral disc herniation, degenerative lumbar spondylosis and other conditions such as neoplasm, infection, inflammatory. Hemorrhagic areas which are considered red flags.

Diagnosis of Lumbar Radiculopathy

Physical examination: physical examination is performed using a battery of tests based on established criteria (Surie et al 2010). It includes the following test - Provocative test : straight leg raise test, crossed straight leg raise test, Motor testing: heel walk for L4, great toe extension strength for L5, Sensory testing: pin prick test on medial ankle for L4, pin prick on great toe for L5, Reflex testing : patellar reflex for L4.

Subjects with sensory deficits, absence of reflex (or diminution of reflex) and loss of muscle strength represents moderate to severe case of lumbar radiculopathy. However in this study subjects with radiating complaints in the leg below knee, pain on coughing, sneezing of straining, positive straight leg raise test, NPRS more than 4 and symptoms for at least 3 months or more will be included.

When the spinal nerves comes out of the intervertebral foramen, the arachnoid matter and the dura matter forms the inner and outer epineurium which is the outermost covering of the nerve. When the nerve is compressed, nociceptors on the outer epineurium are stimulated which results in pain.

Author Affiliation: *MPT, Jamia Millia Islamia, New Delhi. **Assistant Professor, CPRS, Jamia Millia Islamia, New Delhi 110025.

Reprint Request: Saurabh Sharma, Assistant Professor, Centre for Physiotherapy and Rehab Sciences, Jamia Millia Islamia, New delhi -110025.
E-mail: saurabh14332003@yahoo.com

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Nerve compression leads to blockage of intraneural circulation and axoplasmic flow leading to upregulation of inflammatory mediators which leads to generation of pain (Tapp & Boyd 2006, Kobayashi et al 2004)

Lumbar Radiculopathy, Neural Mobility and Nerve Conduction

Nerve compression leads to microvascular alteration causing inflammatory mediators upregulation. Inflammation leads to adhesion formation between herniated disc and nerve root resulting in decreased nerve gliding (Kobayashi et al,2004). Nerve conduction block, mechanical sensitization, intra-neural edema and increase of sodium channel density have been reported in acute and subacute stages of nerve root compression (Chen et al 2003, Kabayashi et al 2004, Rempel et al 1999).

Neurodynamics

Shacklock gave the concept of neurodynamics in 1995. Neurodynamics is composed of mechanical physiological mechanisms. It includes interaction between mechanics and physiology of nervous system which are interdependent on each other. According to the mechanics, body acts as a container of nervous system and the musculoskeletal system is the mechanical interface (MI) of the nervous system which can be divided as Central mechanical interface and Peripheral mechanical interface. Central mechanical interface consists of cranium, spinal and radicular canals housing the neuraxis, cranial nerves, meninges and nerve roots. Peripheral mechanical interface is composed of nerve beds in the limbs and torso where the nerves are presented with bone, fascia and fibrosseous tunnels.

During daily activities, body moves resulting in changing dimensions of MI imposing force on neural structures. In order to protect against such compromise, nervous system undergo distinct mechanical events such as elongation, sliding, cross sectional changes,angulation and compression. When these mechanism fails, symptoms may occur. Joint angulation and destination of the nerves are the two features which combine to cause neuro mechanical responses.

During the early range of a tension test, nerves are wrinkeled sitting loosely in their bed. When movement exerts tension in the nerve, the nerve loose their slack (Sunderland and Bradley, 1961) and begin sliding (Breig, 1978). During the mid range of movement, nerve slides more rapidly because of sufficient tension. Towards end range, neural sliding depletes and there is a more marked increase in neural tension (Charnley, 1951).

Neural sliding does not always occur in one direction. The nerve convergence occurs towards the joint where the nerve elongation is initiated. Sequence of body movement influences the neural sliding as when dorsiflexion is added to the SLR, the nerves slides towards ankle. Neural tissues possess viscoelastic properties. With constant loading neural tissue elongate progressively given that elastic limit is not exceeded and enough time is allowed, they come back to their original length when the load is removed (Kwan et al, 1992). Peripheral nerves and nerve roots possess high risk of plastic deformation with excessive loading (Sunderland and Bradely, 1961)

Physiological responses includes intraneural blood flow, axonal transport, mechanosensivity and sympathetic activation. Excessive tension reduces intraneural microcirculation by stretching and strangulation of vessels. If the vascular capabilities are exceeded by excessive stretching, nerve damage occurs (Lundborg and Rydevik, 1873). Intracellular cytoplasmic movement is sensitive to hypoxia (Ochs and Hollingsworth, 1971; Leone andOchs, 1978). Nerve compression leads to hypoxia (Sunderland, 1976) and reduces axonal transport (Mackinon and Dellon, 1988). Axonal transport is found to be reduced at pressures as low as 30 mm Hg which can be achieved during daily activities like wrist flexion/ extension (Rydevic et al 1981; Gelberman et al 1981)

Mechanosensitivity refers to the impulse activation occuring when a neural structure comes across a mechanical stimuli such as pressure or tension. When a nerve is injured or inflamed, impulses are more readily evoked (Calvin et al 1982; Howe et al 1977). When a nerve is compressed or stretched, action potentials generate in the sympathetic nerve fibres causing increased sweating.

Changes in neural mechanics or physiology can lead to pathodynamics. Pathodynamics is a combination of pathomechanics and pathophysiology.

According to Shacklock, neural tension test should be called as neurodynamic test as they evoke both mechanical and physiological reactions which should be included in clinic thinking. The aim of using these tests in assessment is to mechanically stimulate and move neural tissues in order to determine their mobility and sensivity to mechanical stresses.

Neural Mobilization Techniques

There are two techniques according to butler and

shacklock. Gliding technique – Also known as sliders. These are the manoeuvre causing sliding movements between the non neural and neural structures. These are carried out in non provocative manner (butler 2000, Shacklock 2005). Tensile loading technique – these techniques enable the neural tissue to movements which causes lengthening of nerves. These are not stretches. Tensile loading techniques are performed in an oscillatory manner gently engaging resistance during the movement(butler 2000, Shacklock 2005).

Mechanism by which Neurodynamic Technique Exerts its Effect

There are many mechanism proposed by different authors. According to a study conducted by Brown et al (2011) on the tibial nerves of cadavers, neural mobilisation increases dispersion of fluid, thereby, reducing intraneural oedema. Injured neural tissue or regenerating nerve fibres become hyperexcitable leading to the formation of abnormal impulse generating sites (AIGS) which repeatedly produces impulses (Devor & Seltzer,1999). AIGS are mechanosensitive, chemosensitive and produces spontaneous firing. Neural mobilisation causes inhibition at dorsal horn thereby reducing thermal pain (Beneciuk et al., 2009). Another study conducted by Santos et al, 2012 on animal model says that neural mobilisation leads to decrease in Nerve Growth Factor (NGF) and Glia Fibrillary Acid Protiens (GFAP) in the dorsal root ganglion and spinal cord leading to reduction in hyperalgesia and allodynia.

Effectiveness of Neurodynamic Technique

In 2008, Ellis and Hing conducted a systemic review of RCTs to analyse the therapeutic efficacy of neural mobilisation. 10 RCTs (11 articles) were included that discussed therapeutic effect of neural mobilisation. 8 of the 11 studies concluded a positive benefit from using neural mobilisation in the treatment of altered meurodynamics or neurodynamic dysfunction. However, in consideration of methodological quality, there is limited evidence to supports use of neural mobilisation. Further studies are needed in this direction.

Schaffer et al conducted a study in 2011 in which he classified patients with low back and leg pain in to four groups: Neuropathic Sensitization , Denervation, Peripheral nerve sensitization and musculoskeletal. Neural mobilisation was given for 7 sessions twice a week in all of the groups. Number of responders to neural mobilisation were greater in peripheral nerve sensitization group (55.6%) compared to other groups. Thus, patients with peripheral nerve sensitization were the appropriate candidates for neural mobilisation.

In another study conducted by Cleland and Hunt et al in 2004, effectiveness of neural mobilisation was demonstrated in a patient with lower extremity neurogenic pain. 14 physiotherapy sessions over a period of 48 days was conducted out of which 8 sessions included neural mobilisation. Visual as well as statistically significant improvement was observed following implementation of neural mobilisation technique.

Nerve Conduction Velocity (NCV)

Nerve conduction velocity is an important aspect of nerve conduction studies. It is the speed at which an electrochemical impulse propagates down a neural pathway. (National Institutes of Health, 31 October 2013).In this study, we are concerned with motor nerve conduction velocity.

Motor nerve conduction velocity can be obtained by electrically stimulating the nerve and then recording the CMAP (Compound Muscle Action Potential) from the surface electrodes placed over the muscles innervated by that nerve. Recording electrodes are placed over the target muscles using adhesive conductive pads. Reference electrode is placed over a neutral (inactive) area while the active electrode is placed above the belly of muscle. CMAP is recorded. Latency is the time from the stimulus artefact to the onset of response. To record motor nerve conduction velocity, nerve is stimulated at 2 sites- proximal point and a distal point. Proximal and distal latencies are recorded (A Mallik et al, 2016). The motor nerve conduction velocity is recorded in the following way:

mNCV= distance between distal and proximal point divided by difference between distal latency and proximal latency.

Effect of Neurodynamic Technique on MNCV

Some authors examined the effect of neural mobilisation on mNCV. According to them, improvement was observed in motor nerve conduction velocity following application of neurodynamic technique. Dongwook Han et al (2013) conducted a study in which he examined the effect of median nerve mobilisation in open as well as closed kinetic chain conditions on median motor nerve conduction velocity. 20 healthy female college students were recruited and divided in to 2 groups

in this study. One group performed self CKC median nerve mobilisation while the other performed the same in OKC. A statistically significant improvement was observed in CKC group as compared to OKC group in mNCV in the wrist elbow section.

Misook HA et (2012) conducted a similar study on the similar population which compared the effect of median nerve mobilisation performed by the therapist and the effect of self median nerve mobilisation on the median motor nerve conduction velocity. It was concluded that physical therapist's application of median nerve mobilisation was more effective than self median nerve mobilisation.

Effect of Neurodynamic Technique on Surface Electromyography (SEMG)

EMG is a technique which involves the development, recording and analysis of myoelectric signals. In lumbar radiculopathy, neuromuscular imbalance is found in the lumbar region. Tobias Renkawitz et al conducted a study in 2006 analysing the association of low back pain with neuromuscular imbalance, and trunk extension strength in athletes before and after the implementation of back exercise program. They found that significant association exist between neuromuscular imbalance and LBA.

According to a study conducted by Giselle Horment Lara et al., there was a reduction in sEMG amplitude of ipsilateral and contralateral erector spinae after application of self neurodynamic sliding technique.

Conclusion and Clinical Implications

The literature search examined the effect of neural mobilisation on mNCV. According to them, improvement was observed in motor nerve conduction velocity following application of neurodynamic technique. A statistically significant improvement is observed in studies having CKC group as compared to OKC group in mNCV .

EMG is a technique which involves the development, recording and analysis of myoelectric signals. In lumbar radiculopathy, neuromuscular imbalance is found in the lumbar region. It is concluded from literature search that significant association exist between neuromuscular imbalance and LBA. The practical and clinical application of this review that since erector spinae EMG activity decreases with neural mobilisation so it gives a chance for the inner core to initiate muscular activity which was inhibited due to pain and inactivity.

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