RESEARCH PAPER

Electrodermal Activity Dysregulation and Compression Therapy in Patients with Lumbosacral Radiculopathy: A Narrative Review

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ABSTRACT

Background: lumbosacral radiculopathy is a painful disorder that starts in the buttocks and travels down the leg due to irritation or compression of the sciatic nerve. Condition is usually harmless and goes away on its own. Pain in the lower back that travels down one or both legs is a hallmark of this condition, as are neurological symptoms such as paresis, impaired sensation, or a lack of reflexes, all of which point to irritation of nerve roots or a malfunction in the nervous system. Humans exhibit Electrodermal activity (EDA), a trait that results in constant change in the skin's electrical properties. One another name for EDA is skin conductance (SC). According to EDA, the condition of the skin's sweat glands determines the skin's resistance and skin conductance condition changes.

Purpose: This review aimed to explore the EDA dysregulation in patients with lumbosacral Radiculopathy and also to which extent the compression therapy in previous literature may have effect on it.

Methods: By searching for papers published between 2000 and 2024 that were relevant, a thorough literature search was performed using PubMed, Cochrane Library, Web of Science, Scopus, and Google Scholar. Electrodermal activity (EDA), lumbar radiculopathy, and compression treatment(CT) were the search phrases employed. We only considered observational studies, systematic reviews, meta-analyses, and randomized controlled trials (RCTs) that were published in English.

Results: Studies have linked compression treatment interventions to changes in autonomic nervous system activation, changes is the EDA activity which reflects better on the patients functional activity, and allow better patient engagement in rehabilitation programs.

Conclusion: By addressing the neurological aspects of rehabilitation, compression treatment shows promise as an intervention for lumbosacral radiculopathy. Adding CT to interdisciplinary rehabilitation programs has the potential to boost patient engagement and quality of life improvement that enhance functional rehabilitation for those patients as supported by the previous data.

Keywords: Electrodermal activity, Compression therapy, Lumbosacral Radiculopathy

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INTRODUCTION

lumbosacral radiculopathy is a painful disorder that starts in the buttocks and travels down the leg due to irritation or compression of the sciatic nerve. Disc herniation or rupture pressing on the roots of the L5 or S1 nerve accounts for the vast majority of cases. Some common reasons include spinal stenosis, spondylolisthesis, and degenerative disc disease. Additionally, sciatica may be caused by a number of less common non-spinal conditions. There are several examples, including piriformis syndrome, trauma, surgical complications, gynecologic illnesses, and herpes zoster. If it persists for more than a year or causes episodes to happen again and again, it is considered chronic (Christopher et al., 2018; Davis et al., 2021).

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The condition known as lumbosacral radiculopathy is usually harmless and goes away on its own. Pain in the lower back that travels down one or both legs is a hallmark of this condition, as are neurological symptoms such as paresis, impaired sensation, or a lack of reflexes, all of which point to irritation of nerve roots or a malfunction in the nervous system. The herniation of a disc can compress a nerve, which can cause a variety of symptoms including impaired motor skills, altered postural stability, pain that radiates to the lower extremities, and problems with balance when standing. Disabilities, worse health state, and less capacity to work results from it, (Frost and Brown.2016, Alexander et al., 2024).

There were no documented variations between males and females when it came to the epidemiologic aspects of lumbosacral radiculopathy. Patients in their 40s and 50s also have the greatest rate of occurrence. It is believed that the lifetime incidence falls somewhere in the range of 10% to 40%. Very seldom occurs in those less than 20 years old, with a yearly incidence rate of 1% to 5%. A hereditary predisposition has been suggested by some research. (Davis et al., 2021).

If you've previously had sciatica symptoms, exercising increases your chance of getting them again, but if you've never had them, it decreases your risk. According to Davis et al. (2020), occupations that demand individuals to be in physically demanding positions, such as machine operators, truck drivers, and others, have been associated with occupational tendency.

When the nucleus pulposus moves out of its normal location in the intervertebral space, the result is a spinal herniated disc. Back discomfort is often caused by it. Many people get back discomfort due to this. Many people who have herniated disc discomfort can recall the exact moment when they felt the pain first. Herniated disc pain, in contrast to mechanical back pain, may spread into the lower extremities and feels like a burning or stinging sensation. Additionally, changes in feeling or weakness may accompany more severe instances. Myelopathy, or discomfort indicative of nerve compression or spinal cord dysfunction, may occur in rare cases due to herniated disc injuries (Kobayashi et al., 2004; Dydyk et al., 2020).

Lumbosacral Radiculopathy and neuropathic pain

Lumbar region pain is common among patients with radiculopathy, and the pain is almost always experienced by one side of the body. Paresthesia, along with soreness or a burning feeling deep in the buttocks, is a common symptom that patients report. Those who have ipsilateral limb weakness, a less common consequence, may report a heavy sensation in the affected leg (Bogduk, 2009; Colloca et al., 2017).

Radiculopathy and neuropathic pain are linked through nerve root injury and inflammation. Mechanical compression, for example, can cause demyelination and ischemia. Inflammatory mediators, such as cytokines like TNF-α, can make nociceptors more sensitive. Ectopic discharges, which originate from damaged nerve fibers, can cause pain on their own. Central sensitization can also develop and increase the perception of pain. Pain that is allodynia, hyperalgesia, radiating pain along a dermatomal distribution, electric shock-like pain, and burning are all symptoms of neuropathic pain, which is caused by these processes (Colloca et al., 2017; Karcz and Gharibo, 2024).

The fundamental processes of chronic radiculopathy go beyond basic mechanical compression including Central sensitization that occurs when the central nervous system becomes more excitable due to repeated nociceptive input, which in turn increases the perception of pain. This can happen as a result of nerve damage, fibrosis, long-term compression, demyelination, or axonal injury. According to Treede et al. 2015, chronic pain may be caused by neuroplastic alterations that alter the brain's structure and function.

Electrodermal Activity Sources and Mechanisms:

A branch of the neurological system, the autonomic nervous system (ANS) controls the heart, lungs, smooth muscles, and glands. It was formerly known as the vegetative nervous system. A number of unconscious processes are regulated by the autonomic nervous system (ANS), which includes the heart rate, contraction force, digestion, respiration rate, pupillary response, urine, and sexual desire. Mainly the mechanism that controls the fight-or-flight response included is this system (Jänig and Wilfrid 2008).

The conventional division of the autonomic nervous system into the parasympathetic and sympathetic branches (i.e., motor only) remains in place. The sympathetic branch originates in the lower back and thoracic region of the spinal cord and ends at the level of L2-3. The parasympathetic division's neurons start from the sacral (S2-S4) spinal cord and cranial (oculomotor, facial, glossopharyngeal, and vagus) nerves, a phenomenon known as craniosacral "outflow" (Jänig and Wilfrid 2008).

The human body's electrodermal activity is what causes the skin's electrical properties to vary continuously. Skin conductance level (SCL), skin conductance response (SCR), sympathetic skin response (SSR), electrodermal response (EDR), skin conductance, and psychogalvanic reflex (PGR) are some of the previous names for electrodermal activity (EDA).

The electrodermal activity theory postulates that perspiration gland health determines skin resistance. The sympathetic nervous system regulates perspiration, and changes in skin conductance reflect emotional or physical excitement. Sweating and an increase in skin conductivity are both effects of a highly aroused sympathetic branch of the autonomic nervous system. So, skin conductivity may be used to judge how someone is feeling emotionally and sympathetically. According to Boucsein and Wolfram (2012), EDA is a continuous measurement (signal curve with time) and it is expressed in microsiemens (μS).

It is not possible to consciously regulate skin conductance. Sympathetic activity, which controls human behavior, thoughts, and emotions subtly, instead modulates it independently. Hence, skin conductance provides direct

insights into self-regulation of emotions, (Grimnes et al., 2011).

Fingers, palms, and foot soles all exhibit unique bio-electrical phenomena. A device that shows the change in electrical conductance between two places over time, known as an EDA . The two current path are along the surface of the skin and through the body . A little quantity of electricity is passed through the body during active measurement, (Grimnes et al., 2011)

A frequent measure of autonomic nervous system activity, electrodermal activity has a long history of use in psychological research. EDA integrates changes in autonomic sympathetic arousal with emotional and cognitive states; it is a sensitive psychophysiological indicator. In order to aid users in managing their anxiety, some biofeedback treatment devices employ EDA as a measure of the user's stress reaction. If you want to know how your nervous system is doing without having to endure the pain and expense of conventional EEG-based monitoring, EDA is the way to go. Another possible application is as a stand-in for psychological stress. According to Yang and McCoy (2021).

Affection of Electrodermal activity in lumbosacral radiculopathy patients:

Nonmyelinated axons are four times more concentrated than myelinated axons in most somatic nerves, and SSR affects nonmyelinated C fibers in somatic nerves, suggesting that axon and preganglionic fiber loss may be the cause of sympathetic system dysfunction in radiculopathy with longer SSR latencies. Allodynia and hyperalgesia may be brought on by excitatory neurotransmitters such as substance P, calcitonin, gene-related peptide, vasoactive intestinal peptide, and neuropeptide Y, which may be induced by the chronic compression or constriction of spinal neurons during surgery, (Son et al., 2007, Amin and Faghih.2022).

Chronic pathological pain may be a symptom of trauma to the soft tissues, bones, or nerves. Efferent sympathetic activity and circulating catecholamines sustain the pain and hyperalgesia (sympathetic maintained pain SMP) . Many pain models show that as the disease progresses, either the abnormal sympathetic sprouting into the dorsal root ganglion (DRG) decreases or the sympathetic impact on sensory neurons changes to become less excitatory. Similar to phantom pain, neuralgias, herpes zoster, and fibromyalgia (FM), sympathetic maintained pain (SMP) is a clinical component of several neuropathic pain syndromes. Research has shown that people with FM exhibit symptoms of sympathetic hyperactivity, and that the pain is alleviated by blocking the sympathetic nervous system and then being re-activated by injecting norepinephrine (NE). This supports the idea of FM is considered as sympathetic maintained pain (SMP) disorder . Even in non-neuropathic pain diseases including arthritis, temporomandibular dysfunction (TMD), and back pain, the sympathetic nervous system plays a role. (Chen and Zhang. 2015).

How inflammation at the DRG level stimulates sympathetic fiber sprouting... Another possible positive feedback loop is that the sympathetic fibers that grow into the DRG can further enhance inflammatory activities there. As an example, sympathetic ganglia (SG) activation may be enhanced by ATP release because of the excitatory receptors present on these cells. In addition to increasing the production of proinflammatory cytokines from neurons, sympathetic stimulation of sensory neuron activity may also enhance glial activation. There are some interesting similarities to that showed in the results of the complete sciatic nerve transection (CSNT) paradigm that followed by sympathetic sprouting inhibitory effects later on.(Chen and Zhang, 2015).

The role of sympathetic nerves in producing radicular pain was investigated using a model that had lumbar sympathectomy after root constriction. Sympathectomy alleviated the mechanical hypersensitivity shown in our behavioral evaluation of the lumbar root constriction model. The clinical efficacy of sympathetic nerve block might be explained by this, which implies that the sympathetic nervous system could be associated with a source of radicular discomfort, (Mizano et al.,2007).

The lumbar sympathetic trunk innervates the lower lumbar disc, and neurophysiological and neural tracer experiments have shown that afferents from the disc go to the deep root ganglia of level 2. But sympathetic efferents aren't the only ones involved in back pain. Research using neuroanatomy and immunohistochemistry has shown that the intervertebral disc and its surrounding tissues contain sympathetic neurons. Activation of A-b afferents, which stimulate large dynamic range neurons in spinal cord pain pathways, is the mechanism by which the sympathetic nervous system produces pain, according to neurophysiological investigations. There seems to be an upregulation of alpha-adrenergic receptors in injured peripheral sensory nerves. Changes in nociceptors' phenotype and, eventually, central sensitization, may result from norepinephrine levels that are too high. Clinical symptoms of varying intensity, such as neuropathic pain , refractory condition or radicular pain both with a reversible symptom may result from various sprouting patterns of attachment to the DRG. (, Mizuno et al., 2007).

Compression therapy mechanism of action on neuropathic pain

A neurophysiological approach, it entails repeatedly moving the patient in order to stimulate their cutaneous and proprioceptive receptors. It connects inflated splints to intermittent sensory input, which is enhanced touch, movement, and pressure , the three main inputs that impact developing sensation. The affected limb is encased in an inflatable jacket, which is linked to the air pump via pressure lines. The pump forces blood and lymph out of the compressed region by pressurizing the limb tissues when turned on. This is achieved by filling the air chambers of the jacket, (Sharma et al., 2018).

Among the several vascular treatments available, intermittent pneumatic compression (IPC) stands out.

Also, it's very clear that IPC's mechanical action is tropic for receptors; therefore, increasing blood circulation stimulates the transit of regulatory and immunological chemicals, which impacts the autonomic nervous system's functional state. Ischemia, poisoning, chronic inflammation, and regulation abnormalities may all cause secondary nervous system injury; the IPC can treat those secondary vascular manifestation of nerve illnesses as well as non-vascular nerve pathology with local hyperfunction, (Zaitsev .2022).

CONCLUSION

In individuals suffering from lumbosacral radiculopathy, applying compression therapy helps alleviate neuropathic pain and restores their functional status simultaneously.

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Conflict of interest

All authors have disclosed that they have no competing interests.

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