Dynamic Chiropractic

BACK PAIN

Spinal Motor Implications of Lumbar Radiculopathy

Brad McKechnie, DC, DACAN

Muscular atrophy is the direct result of the loss of stimulus from the anterior horn cells found within lamina IX of the anterior horn to the peripheral musculature. This loss of stimulus may be the result of an intrinsic spinal cord pathology, as in the case of polio, or it may be the result of an intraspinal neoplasm. However, these clinical entities are not as common as are disc herniations, central canal stenosis, and lateral recess stenosis, which affect the existing nerve roots inside the spinal canal and as they exit the intervertebral foramen. This discussion therefore will be directed toward the effects of disc herniation and stenosis.

Clinical examination for motor loss secondary to radiculopathy is targeted toward the motor function of the distal extremity musculature in order to gain information as to the level of pathology and degree or radiculopathy involving the affected spinal nerve. However, the distal extremity muscles are not the only muscles affected by a nerve root lesion to a lumbar nerve root. The erector spinae muscles are innervated by branches of dorsal primary rami which have their origin from spinal nerves. Any pathology which decreases neural transmission (such as a disc protrusion) may result in denervation and atrophy of paraspinal muscles as well as the muscles of the involved distal extremity.

A recent study concluded that mild pathological changes noted in paraspinal muscles of lumbar disc patients may have been the result of denervation, muscle spasm induced ischemia, or by exceptional strains applied to the muscles studied.[1] The muscle atrophy noted was more pronounced in older patients and in patients with long-standing histories of lumbar disc herniation.

A similar study[2] noted connective tissue fibrosis and atrophic changes within the multifidus muscles of lumbar disc patients. The conclusions of this study indicated that denervation secondary to lumbar disc herniation may have induced changes in the mitochondrial metabolism of muscle cells or the changes may have been secondary to inactivity. It was also noted that immobilization produced striking increases in intramuscular connective tissue. Based on these findings, they could only speculate as to whether the increase in muscular connective tissue observed was due to denervation or due to immobilization.

A key issue identified[2] pointed to the strong correlation between the amount of fibrosis and the degree of disability the patient experienced one year postoperatively, indicating that a great deal of intramuscular fibrosis may be a factor impairing the recovery of lumbar spine function. Therefore, any chiropractic treatment regimen for lumbar radiculopathy must address this element of muscular involvement.

A treatment regimen directed solely at pain relief may only be partially successful in returning the patient to full duty and could conceivably result in an increased change of reinjury. During a recent ergonomic analysis for a large retail building supply store it was noted that the cost for a repeat injury to a worker treated by the "standard" pain relief treatment regimen resulted in a $1,465

increase in the amount of expenditures for the second injury over the cost of the initial injury.

In conclusion, an effective conservative treatment regimen for lumbar radiculopathy should include lumbar paraspinal muscle rehabilitation as a key component due to the possibility of lumbar paraspinal muscle atrophy and fibrosis which may be secondary to denervation or immobilization. Clinical suspicion of paraspinal muscular pathology should elevate with the chronicity of the complaint.

References

1. Siong-Zeng Z, et al: Histochemistry and morphology of erector spinae muscle in lumbar disc herniation. Spine, 14(4):1989.

2. Lehto M, et al: Connective tissue changes of the multifidus muscles in patients with lumbar disc herniation: An immunohistologic study of collagen types I and II and fibronectin. Spine, 14(3):1989.

Brad McKechnie, D.C., D.A.C.A.N.
Pasadena, Texas

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