MOTOR NEURON INVOLVEMENT IN EXPERIMENTAL LUMBAR NERVE ROOT COMPRESSION. A LIGHT AND ELECTRON MICROSCOPIC STUDY
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INTRODUCTION. It is generally considered that the genesis of radiculopathy associated with the degenerative conditions of the spine may result from both mechanical compression and circulatory disturbance. However, few studies have looked at changes of neurons within the lumbar cord caused by disturbance of axonal flow and the axon reaction as a result of mechanical compression of the ventral root. The lumbar cord should not be overlooked when considering the mechanism of motor weakness in the legs so it is important to understand the morphologic and functional changes that occur in lumbar motor neurons as a result of nerve root compression. In this study, we employed morphological methods to examine the changes of motor neurons using the nerve root compression model.

MATERIALS AND METHODS. In mongrel dogs, the sixth and seventh lumbar laminae were removed, and the seventh lumbar nerve root was exposed widely on one side under general anesthesia. The nerve root was clamped with a clip for microvascular suturing at the midpoint between the dural sac and dorsal root ganglion. The 7th nerve root was exposed to compression at 7.5 gram force (gf) clamping power. In the present study, the strength of the spring clips used for nerve root compression was determined with an Instron-type tensile tester. After awakening from the anesthetic, the animals were maintained for 1 week, or 3 weeks and then sacrificed. The animals were fixed by intracardiac perfusion with 4% paraformaldehyde and 1% glutardehyde. After the compression was determined with an Instron.

RESULTS. After 1 and 3 weeks postlesion time point were performed using a nonparametric test (Wilcoxon rank-sum test) with SPSS statistical software, version 11.0J (SPSS Inc, Chicago, IL). The other sections were postfixed in 2% OsO4, impregnated with 2% uranyl acetate, dehydrated in graded ethanol, and embedded in epoxy resin. For light microscopy, -3μm thick toluidin blue stained sections were used. For electron microscopy, ultrathin sections contrasted with uranyl acetate and lead citrate were examined under an electron microscope.

DISCUSSION. Disturbance of axonal flow therefore threatens the survival of neurons and appears to be one cause of neurological dysfunction. In this study, compression of the peripheral branches of motor neurons in the nerve root led to impairment of axonal flow and central chromatolysis in the neurons of the ventral horn, where the peripheral branches of these neurons originated. Chromatolysis is a reactive change of neuronal perikarya to axonal injury. This reaction reflects an alteration in the arrangement and concentration of RNA-containing material in the cell, leading to changes in protein synthesis of importance for axonal regeneration. It seems likely that sustained mechanical compression of the nerve root could result in irreversible damage to the motor neurons. The morphologic changes that we observed in lumbar motor neurons after mechanical compression of the nerve root therefore reflect the metabolic response to axonal degeneration and regeneration. If the disturbance of axonal flow caused by compression and the resulting central chromatolysis are mild, the neuron can recover fully after compression is relieved. However, it seems likely that sustained mechanical compression of the nerve root could result in irreversible damage to the neurons of the ventral horn, such as apoptosis. Chromatolysis does not necessarily foreshadow neuronal cell death after axotomy, although specific signals during chromatolysis may be required to initiate apoptosis after axotomy.

CONCLUSION. As clinicians, we often come across patients with cauda equina and nerve root compression due to lumbar disc herniation or lumbar canal stenosis who continue to experience muscle atrophy and weakness long after surgical decompression of the nerve root, particularly patients with a long history of weakness in the lower extremities before surgery.

REFERENCES