Introduction: Neurogenic intermittent claudication is the most common presenting symptom in lumbar spinal canal stenosis. It is generally considered that the genesis of neurogenic intermittent claudication may result from nerve root ischemia caused by mechanical stress. However, the exact pathogenesis of neurogenic intermittent claudication still remains uncertain. We investigated the clinical relationship between the local pressure of the intervertebral foramen and the clinical findings in lumbar spinal canal stenosis, and report our findings regarding the pathogenesis of neurogenic intermittent claudication.

Materials and Methods: A total of 20 lumbar spinal canal stenosis patients with L5/S1 vertebral foramens were studied. All patients showed L5 radicular symptom with neurogenic intermittent claudication induced by lumbar spinal canal stenosis.

We classified the patients into two groups based on their walking ability, as the group which was able to walk 100m without rest and the group which was unable to walk 100m without rest.

All patients were evaluated by myelography preoperatively. We assessed all patients as either one-level spinal canal stenosis at L4/5 level or as two-level spinal canal stenosis at L3/4 and L4/5 levels.

The local pressure of the intervertebral foramen was measured in all patients, for all of the L5/S1 vertebral foramens. A micro-tip catheter transducer was used as a pressure transducer. Intraoperatively, after exposing the L4/5 interlaminar space, the catheter transducer was inserted from the L4/5 interlaminar space, and then the tip of the catheter transducer was placed just below the pedicle in the L5/S1 vertebral foramen. The local pressure was continuously measured while the lumbar spine postures were changed under passive movement (flexion, neutral and extension). We defined the rising ratio of the intervertebral foraminal pressure between the lumbar spine flexion and extension as % pressure = (local pressure in lumbar spine extension - local pressure in lumbar spine flexion) / local pressure in lumbar spine flexion.

Results: The local pressure of the group which was able to walk 100m was 19.00±10.32 mmHg for the lumbar spine flexion posture, 23.61±8.97 mmHg for the lumbar spine neutral posture, and 32.11±14.10 mmHg for the lumbar spine extension posture. The % pressure was 98.81±119.65. In contrast, the local pressure of the group which was unable to walk 100m was 14.19±8.16 mmHg for the lumbar spine flexion posture, 28.76±13.64 mmHg for the lumbar spine neutral posture, and 44.83±19.83 mmHg for the lumbar spine extension posture. The % pressure was 276.23±190.84. The local pressure of the intervertebral foramen increased significantly during lumbar spine extension in both groups (p=0.0001, p<0.0051). The patients who had two-level spinal canal stenosis showed a significantly higher local pressure than one-level spinal canal stenosis patients in the lumbar spine flexion posture (p=0.0459); however, no significant differences were seen in the lumbar spine neutral and the extension postures. In addition, there was a significant difference in the % pressure between the two groups (p=0.0102), with the patients with one-level spinal canal stenosis having a higher pressure.

Discussion: Many authors have reported the clinical importance of two-level stenosis. Olmarker et al demonstrated that the compression of the cauda equina at two levels induces ischemia, not only at the location of compression, but also in the nerve root segments between the two compressed sites, and that the functional changes of the spinal nerve roots were more pronounced with two-level compression than with one-level compression at corresponding pressure levels.

We previously demonstrated that the electrophysiological values of the spinal nerve roots, as evaluated by the CMAPs from TA muscle after L5 nerve root stimulation, were found to deteriorate in line with increasing local pressure of the intervertebral foramen. This result suggested that the dynamic and intermittent compression of the spinal nerve root seem to occur in both the intracanal and the intervertebral foramen with lumbar spine extension. In the present study, no significant differences between the preoperative walking ability and the local pressure of the intervertebral foramen for each posture were observed; however, the patients who demonstrated large changes in the local pressure between the lumbar spine flexion and the extension postures tended to show a significantly poor walking ability. These results suggest that the walking ability in lumbar spinal canal stenosis is related to the intermittent and dynamic mechanical stress on the spinal nerve roots with lumbar spine motion, rather than the static stress on the spinal nerve roots with each posture. Moreover, the patients who had two-level spinal canal stenosis had a significantly higher local pressure than that of one-level spinal canal stenosis with the lumbar spine flexion posture. In addition, the patients who had two-level spinal canal stenosis showed a significantly smaller change in the local pressure between the lumbar spine flexion and extension postures than one-level spinal canal stenosis patients. These results suggested that two-level spinal canal stenosis patients demonstrated radicular symptoms with relatively less external stress on their spinal nerve roots in the vertebral foramen than that observed in one-level spinal canal stenosis patients.

We hypothesize that the genesis of neurogenic intermittent claudication in lumbar spinal canal stenosis may be closely related to the intermittent and dynamic mechanical stress on the spinal nerve roots with lumbar spine motion, rather than the static stress with each posture, and that neurogenic intermittent claudication is more likely to occur with multiple-level compression rather than single-level compression.