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Motor Conduction Time Along the *Cauda Equina* in Patients With Lumbar Spinal Stenosis

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Study Design. Magnetic lumbar stimulation was used to detect spinal nerve degeneration in patients with lumbar spinal stenosis (LSS).

Objective. To evaluate delays in the motor conduction time in the *cauda equina* of patients with LSS.

Summary of Background Data. Previous studies suggested a bilateral slowing of motor conduction in the cauda equina in LSS. Among several methods, only magnetic stimulation is sufficiently sensitive for detecting potential degeneration in LSS. A recent study demonstrated the direct calculation of the *cauda equina* motor conduction time using magnetic stimulation at proximal and distal sites of the *cauda equina*. We used this technique to determine potential degeneration in patients with LSS.

Methods. Twenty adult subjects and 15 patients with LSS were investigated. Lumbosacral roots were stimulated at intervertebral levels L1–L2 and L5–S1 by magnetic coil stimulation. The muscle responses to stimulation were recorded from the gastrocnemius-soleus, and anterior tibialis muscles on both sides with bipolar surface electrodes. The response latency from stimulations at the L5 spine level were subtracted from those at the L1 level on the same side. This value represented the conduction time from the proximal to distal ends of the cauda equina.

Results. The mean conduction time along the cauda equina was significantly prolonged in patients with LSS compared with controls. The mean *cauda equina* motor conduction time was 1.97 ± 0.67 milliseconds in controls and 3.57 ± 2.22 milliseconds in patients with LSS ($P = 0.00$).

Conclusion. Determining the motor conduction time along the cauda equina using L1 and L5 magnetic stimulation provides an effective alternative method for evaluating the lumbar motor roots in patients with LSS.

Key words: lumbar spinal stenosis, magnetic coil stimulation, motor roots, cauda equina. **Spine 2009;34: 1410–1414**

Lumbar spinal stenosis (LSS) is a degenerative disease of the spine. Patients with LSS experience pain in the buttocks and/or legs that can be aggravated by walking and relieved after rest in the absence of vascular changes in the legs (*i.e.*, neurogenic claudication).

Magnetic resonance imaging is the method of choice for evaluating patients with LSS. However, asymptomatic LSS is not an uncommon finding in a healthy population.[1,2] In addition, systematic studies have shown that magnetic resonance imaging findings and interpretations are not significantly related to the clinical diagnosis of LSS.[3–5]

Electrodiagnosis is a well-known method for investigating LSS. Among several electrophysiological methods, a needle electromyographic examination (NEE) in an appropriate myotomal distribution has been accepted as the most useful test because it can detect spontaneous denervation activity and motor unit abnormalities.[6–8] However, routine NEE at the myotomal levels of both legs and the paraspinal muscles only show the end results of compression and injury to the lumbar nerve roots. To detect potential denervation in related myotomal muscles, there must be degeneration of the root motor nerve axons.[9–11] Therefore, NEE may be more sensitive in patients with severe or advanced LSS.

Axonal degeneration or loss is not always observed in patients with lumbar root compression. In these cases, a demyelination process and slowing of focal conduction is usually inferred.[12–14] Several studies have shown the slowing or blocking of root nerve conduction were associated with neurogenic claudication by assessments of F-wave latencies,[15] the H reflex recruitment curve,[16] somatosensory-evoked potentials (SSEP),[17–19] and the time for motor conduction to reach leg muscles after transcranial magnetic stimulation.[19,20] A SSEP technique that employed multiple peripheral and spinal recordings allowed the detection of spinal cord/*cauda* lesions at and above the conus medullaris that involved sensory pathways.[20–24] Similarly, a lumbar magnetic stimulation (LMS) technique applied to only one site in the lumbar region has also suggested that there may be bilateral slowing of motor conduction at the *cauda equina* level in LSS.[19,20,25] Furthermore, Maccabee *et al* have shown that the *cauda equina* motor conduction time can be directly calculated by stimulating at the proximal and distal sites of the *cauda equina*.[26] This method has also been used in patients with LSS; but in that study the bilateral abductor hallucis muscles were recorded.[27]

In this study, we recorded representative myotomal muscles of the gastrocnemius-soleus (mainly S1) and tibialis anterior (mainly L5) bilaterally in patients with LSS.

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We hypothesized that the motor conduction time in the *cauda equina* from spinal levels L1–L5 should be prolonged in patients with LSS compared with controls.

■ Materials and Methods

This investigation was approved by the local ethics committee at Dokuz Eylül University and each participant provided informed consent.

Fifteen patients with LSS (10 women, 5 men) were recruited from the outpatient departments of Physical Medicine and Rehabilitation at Dokuz Eylül University Medical School. The mean age was 52.86 ± 9.8 years old (range: 35–72). Inclusion criteria were: pain in the buttocks or legs induced or aggravated by walking, a maximum walking distance of less than 500 m due to pain or accompanying neurologic deficits, relief or improvement of pain after about 5 minutes of standing or sitting, healthy foot pulses, and signs of a spinal stenosis in either computer tomography or magnetic resonance images as circumferential narrowing of the central canal due to diffuse disc bulging, facet hypertrophy, ligamentous thickening, and central canal area less than 1.5 cm^2 or an anteroposterior diameter distance of less than 11.5 mm.[28] All patients received a neurologic examination and a routine electrophysiological test to exclude other neurologic problems. Twenty adult subjects (14 women and 6 men) without back pain or leg pain and without any neurologic symptoms or findings were enrolled for control group. This group was recruited from patients' healthy relatives. The mean age of the control group was 46.65 ± 11.45 years old (range: 19–62). There were no significant differences for age between the control and the patient groups.

The lumbosacral roots were stimulated with a 90-mm circular coil (Novametrix Magstim 200, 2 Tesla version, Whitland, Dyfed, Wales, UK) at the L1–L2 and L5–S1 intervertebral levels. Apart from using the circular coil, we used the method described by Maccabee *et al*[26] for stimulation; particularly the method for adapting the position of the coil to the vertebral column. The maximal responses from leg muscles that had a low threshold were determined by orienting the magnetic coil vertically to a nearly parallel position along the long axis of the vertebral column at the L1 spine level. The stalk of the magnetic coil electrode was oriented cranially. The maximal responses from the leg muscles could be systematically measured every 10° from the vertical line until the stalk of the coil was at 60° from the vertical line. In this cranially-oriented position, the magnetic coil stimulation at the L1 invariably elicited motor responses from the tibialis anterior and gastrocnemius-soleus muscles. The coil orientation was adjusted until the low threshold motor response exhibited a stable onset latency and a rather constant shape. However, the amplitude of the response was much more variable than the shape and the onset latency at the proximal spine (L1–L2) coil position. The second stimulation site was at the L5–S1 spine level. Here, the stalk was oriented caudally, and the magnetic coil was positioned either at the midline or 30° to 80° from the vertical axis of the spine. Several lateral approaches (resulting in the latter positions) were tested to explore whether a maximal response could occur with shorter onset latency than that observed with the proximal electrode.

Thus the aim of this method was to obtain 2 different motor responses from each muscle investigated, the proximal (L1) and the distal (L5). In the proximal stimulation (L1), the maximal output of the stimulator sometimes elicited excitation points that spread out from the lumbosacral roots and the response latency was similar to that obtained from the distal stimulation (L5). When this occurred, the positions of the proximal electrodes were changed and/or checked again.

The muscle response to each stimulation was recorded at the gastrocnemius-soleus and tibialis anterior muscles on both sides with bipolar surface electrodes (Medelec 40-mm bar electrode). The surface recording electrodes were placed on the belly of the muscles investigated for L1 and L5 stimulations. Four to 16 responses were recorded consecutively. The muscle response latencies were measured from the same side of the body that was stimulated. The L1 (proximal) latency was longer than that from the L5, thus motor conduction times through the *cauda equina* were calculated by subtracting the L5 time from the L1 time.[26] L1 and L5 measurements were obtained from 2 muscles (the gastrocnemius-soleus and tibialis anterior) bilaterally. The *cauda equina* motor conduction time values from the patients with lumbar stenosis were compared with those obtained from the control group.

All statistical analyses were performed with SPSS 11.0 for Windows. A probability value of $P < 0.05$ was considered significant. Normal distribution of all measurements was checked by using Kolmogorov Smirnov test. All of the variables found to be normally distributed. Independent samples *t* test was performed to evaluate the differences in motor response latencies between the patients with LSS and controls.

■ Results

Figure 1 shows the motor responses recorded from the tibialis anterior muscle after magnetic stimulation at the L1 and L5 spine levels in a control patient. We found that the magnetic stimulation with the coil oriented in vertical or near vertical positions produced a maximum of about 1.97 milliseconds (mean value for control group) longer motor latencies from L1 than those obtained from the L5 level (Table 1). There were no significant differences in latencies measured from the muscle groups on different sides of the body in the control group.

On the other hand, the mean conduction time along the *cauda equina* was significantly prolonged in patients with LSS (Table 1; Figure 2) on both sides. We found that the absolute latency values were significantly prolonged from the L1 level to both the tibialis anterior and the gastrocnemius-soleus muscles, and from the L5 to the tibialis anterior muscle. However, the latency values from the L5 level to the gastrocnemius-soleus muscle were not significantly different from controls. This suggests that in patients with LSS, alterations in the motor conduction time may begin at the proximal end of the *cauda equina*.

Of 20 control subjects, the highest *cauda equina* motor conduction time was about 3.5 milliseconds (range: 0.6–3.5 milliseconds). However, of 15 LSS patients, the *cauda equina* conduction time was greater than 3.5 milliseconds for 7 patients in the left soleus, 5 in the left tibialis anterior, 7 in the right soleus, and 7 in the right tibialis anterior.

In all, except 5 patients with LSS there were no NEE abnormalities. In 2 patients with LSS, we found abnormal spontaneous activity and in 5 patients we observed

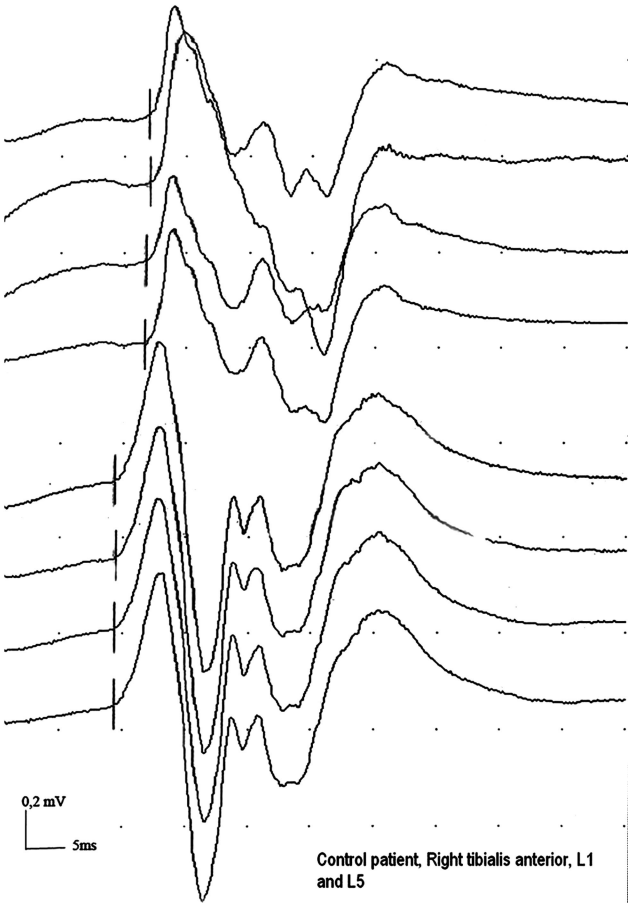

Figure 1. Electrophysiological responses to magnetic stimulations at the L1 (upper) and L5 (lower) spinal levels were recorded in the tibialis anterior muscle in a control subject. Four stimulations were given at each site (L1 and L5). The small vertical lines indicate the different latencies for the L1 and L5 conduction times.

some neurogenic motor unit abnormalities in tibialis anterior, extensor *digitorum brevis*, gastrocnemius, and vastus medialis muscles varying in patients. The NEE did not cover the paravertebral muscle electromyographic study.

■ Discussion

In previous studies, single-site magnetic stimulations required cortical stimulation and/or single paravertebral lumbar stimulation for direct detection of motor conduction abnormalities within the *cauda equina*. The conventional central motor conduction time is the time it takes a signal to traverse the distance from the motor cortex, over the distal *cauda equina* (at the L5), to the site of peripheral stimulation in the leg muscles. The central motor conduction time reflects the sum of conduction times from the motor cortex to the anterior horn, and then to the distal *cauda equina*; this long distance makes it difficult to extract the conduction time over the *cauda equina*.[26,29–32]

In LSS, when most or even a few lumbosacral roots are involved, an abnormal motor root conduction time should be evident in multiple lower limb muscles. However, these cases have only been studied with the single LMS and the F-wave methods; although these are helpful for the clinician, they do not demonstrate the conduction time along the *cauda equina* motor fibers. In contrast, the method described here allowed the direct measurement of motor (L4, L5, S1) conduction in a segment of the *cauda equina* that may be entirely intrathecal.[26] The most important difference in our method from that of Maccabee *et al*, was the magnetic circular coil that we used instead of a twin coil. In humans, sacral roots (S1) were optimally excited by a vertical or near vertical magnetic coil junction.[26] However, it seems likely that the sites of excitation for lumbar and sacral roots in the distal *cauda equina* were identical for the twin coil and the circular coil methods used in this study and others.[33] Experiments comparing round coil and twin coil excitation in the same subject reveal virtually identical compound muscle action potential onset latencies.[26]

Maccabee *et al* found that mean *cauda equina* conduction times of motor fibers innervating lower limb muscles were 2.3 milliseconds in the tibialis anterior and 3.1 millisecond in the soleus muscles.[26] These values were comparable with those detected in our control subjects. Han *et al* recorded only the abductor hallucis muscle and found a slightly longer conduction time (4.02 ± 0.91 milliseconds) in control subjects.[27] Ertekin *et al* calculated that the conduction difference between the L1 and S1 spine levels was about 2.8 milliseconds in healthy

Table 1. The Conduction Times Along the *Cauda Equina* in Controls and Patients With LSS

	Control Group Mean ± SD (Min-Max) n = 20	Patient Group Mean ± SD (Min-Max) n = 15	*P*
Left L5 to soleus	11.95 ± 1.61 (8.2–14.5)	12.76 ± 1.01 (10–14.4)	0.97
Left L1 to soleus	13.94 ± 1.41 (10.6–16.3)	16.28 ± 2.42 (13.5–21.4)*	0.00
Left L1–L5 to soleus	1.97 ± 0.67 (0.7–3.5)	3.57 ± 2.22 (1.2–8)	0.00
Left L5 to tibialis ant	10.88 ± 1.40 (7.9–13.8)	11.96 ± 1.05 (10–14)	0.01
Left L1 to tibialis ant	12.67 ± 1.42 (9.9–14.9)	15.30 ± 2.92 (11–22)	0.00
Left L1–L5 to tibialis ant	1.79 ± 0.67 (0.6–3.5)	3.33 ± 2.27 (1–8)	0.00
Right L5 to soleus	11.87 ± 1.54 (8.7–14.8)	12.55 ± 1.40 (11–15)	0.18
Right L1 to soleus	13.79 ± 1.72 (10.3–16.2)	15.89 ± 2.10 (13.7–19.5)†	0.00
Right L1–L5 to soleus	1.92 ± 0.56 (1–2.8)	3.44 ± 1.28 (1.4–5.8)	0.00
Right L5 to tibialis ant	10.75 ± 1.61 (7.6–14)	11.90 ± 1.03 (9.5–13.7)	0.02
Right L1 to tibialis ant	12.59 ± 1.39 (10.2–15.3)	15.39 ± 1.47 (13–18)†	0.00
Right L1–L5 to tibialis ant	1.96 ± 0.80 (1.1–4)	3.46 ± 1.59 (1.2–6)	0.00

*There was no motor response in 1 patient.
†There was no motor response in 2 patients.

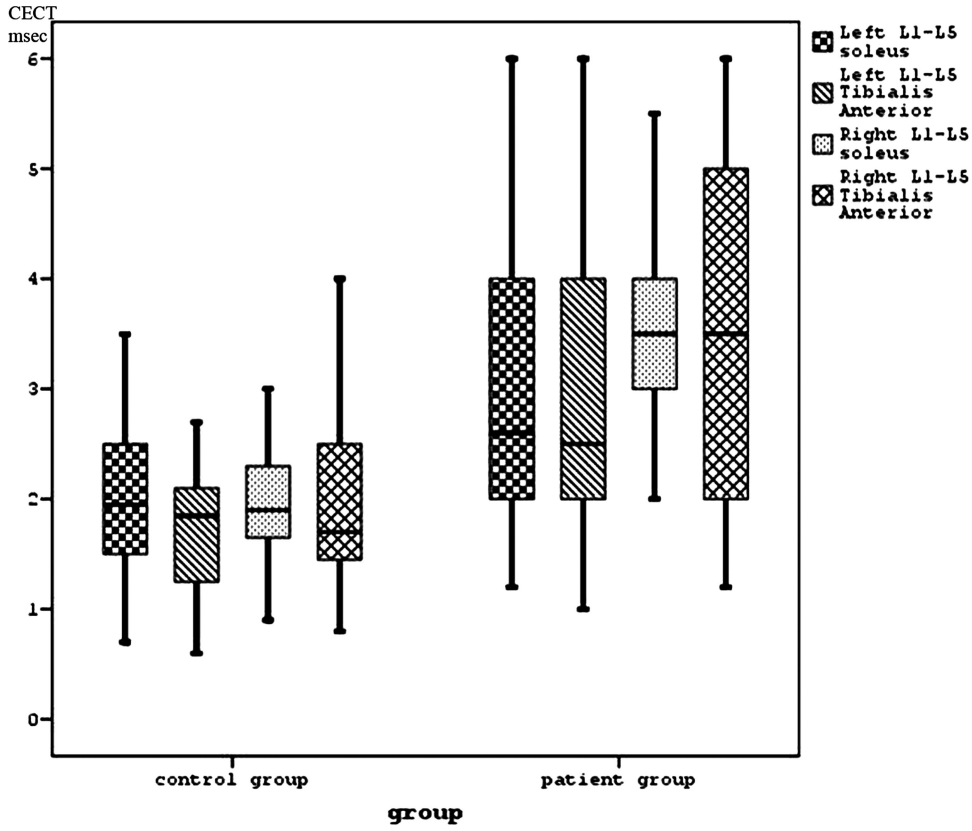

Figure 2. The *cauda equina* conduction times in controls and patients with LSS. The horizontal lines indicate the median conduction times measured in the soleus or tibialis muscles on each side of the body for controls (n = 20) and patients with LSS (n = 15).

human subjects using the epidural recording technique (Figure 2 in their article).[34] This value is within the range of our observations of L1–L5 *cauda equina* motor conduction times. Similarly, Ogura *et al* found a motor conduction latency difference of 2.2 milliseconds from L1–L5 lumbar spine levels to the tibialis anterior muscle by using transcutaneous electrical stimulation applied to lumbar spines (derived from Table 3 in their article).[35]

The mean age of the control group was 6 years younger than the patient group, but there was no statistically significant difference between the groups. As, there are about 0.1 millisecond increase for each decade on the peripheral motor nerves latencies,[36] this 6-year-age difference can not explain this significant latency differences between the groups.

It was suggested that a double compression of the nerve roots may occur in lumbar stenosis; one is within the lumbar spinal canal and the other is around the vertebral foramens.[37] Our results from patients with LSS suggest that the compression may occur earlier in the *cauda equina* root fibers within the spinal canal, and indeed multiple root involvement was detected bilaterally. The *cauda equina* conduction time calculated from L1–L5 reflected the properties of the most affected nerve fiber in LSS, and we found that conduction was not altered before the L5 root compression. For example, the conduction to the gastrocnemius-soleus muscle was not significantly prolonged in patients with LSS compared with controls (Table 1). Moreover, some patients exhibited no NEE abnormalities, including fibrillation; however, the paravertebral muscles were not investigated. Nevertheless, the prolongation in motor conduction along the *cauda equina* was significant. This suggests that the motor root was locally compressed and this may have induced local demyelinization at the level of the *cauda equina*. Other electrodiagnostic methods, including SSEP, F wave latencies, and H-reflex recruitment have yielded delayed responses that might also be explained by focal demyelination of multiple lumbar roots.[15,23] Certainly, in advanced cases of LSS, axonal degeneration may be prominent and some groups have reported NEE abnormalities in paravertebral and lower limb muscles.[3,4]

In conclusion, LMS is correlated with the presences of LSS. Abnormalities in motor conduction in the proximal region of the *cauda equina* may be an early sign of neural degeneration in LSS demonstrated electrophysiologically in this study and also in some researches.[9,14,38] The clinical usefulness of this method must await further studies.

■ Key Points

- *Cauda equina* conduction is significantly prolonged in LSS.
- Lumbar magnetic stimulation is an effective method for diagnosing LSS.
- Degeneration may begin in the proximal *cauda equina* in LSS.

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