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Diagnostic value of cauda equina motor conduction time in lumbar spinal stenosis

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HIGHLIGHTS

- The prolongation of cauda equina motor conduction time was statistically significant in the lumbar spinal stenosis (LSS) group.
- Lumbar laminar electrical stimulation is an easy and reliable method to demonstrate motor conduction delay in cauda equina.
- In LSS, chronic compression may occur earlier in the cauda equina root fibers within the spinal canal.

ABSTRACT

Objective: Lumbar spinal stenosis (LSS) is a chronic degenerative disease with pain in the back, buttocks and legs aggrevated by walking and relieved after rest without associated vascular disease of lower extremities observed in patients between 50 and 60 years. Several studies, using different methods indicated an association between slowing or blocking of root-nerve conduction and LSS. None of the previous research had applied the more conceivable methods such as recording the cauda equina potentials from the lumbar level or stimulating the spinal roots within the canal using either leg nerves or muscles. In this study, electrical lumbar laminar stimulation was used to demonstrate prolongation of cauda equina motor conduction time in lumbar spinal stenosis.
Methods: Twenty-one LSS patients and age matched 15 normal control subjects were included in the study. Lumbar laminar electrical stimulation from L1 and L5 vertebra levels were applied by needle electrodes. Compound muscle action potential (CMAP) from gastrocnemius muscles were recorded bilaterally. Latency difference of CMAPs obtained from L1 and L5 spine levels were accepted as the cauda equina motor conduction time (CEMCT).
Results: CEMCT was significantly longer in patient group when compared to normal controls. Mean latency difference was 3.59 ± 1.07 msec on the right side, 3.49 ± 1.07 msec on the left side in LSS group, it was 1.45 ± 0.65 msec on the right side, 1.35 ± 0.68 msec on the left side on normal control group ($p < 0.0001$).
Conclusions: The prolongation of CEMCT was statistically and individually significant in patient group. This may indicate that lower lumbosacral motor roots were locally and chronically compressed due to lumbar spinal stenosis. Lumbar spinal stenosis may have induced local demyelination at the cauda equina level.
Significance: Since the prolongation of CEMCT was found only in patients with LSS, the method of laminar stimulation can be chosen for patients with uncertain diagnosis of LSS.

1. Introduction

The most frequent cause of lumbar spinal stenosis (LSS) is a degenerative disease of the spines encountered in patients who are 50 to 60 years old (Chad, 2007; Hall et al., 1985; Arbit and Pannullo, 2001). Cardinal symptom is "neurogenic claudication" (NC) that is defined as pain in the buttocks and legs aggrevated by walking and relieved after rest in the absence of vascular changes in the legs (Chad, 2007; Arbit and Pannullo, 2001; Adamova et al., 2005).

Radiological methods including magnetic resonance imaging (MRI) are important in the diagnosis of LSS (Haig et al., 2005). However, radiologically proven LSS can be found in many people

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without any complaints and findings (Jensen et al., 1994; Boden et al., 1990). Despite very mild radiological findings, some patients may have severe clinical symptoms of LSS (Haig et al., 2006a,b, 2007).

A chronic neural compression in LSS can be detected by using electrodiagnostic methods. Among the electrodiagnostic methods the myotomal needle EMG was found to be the most beneficial one (Plastaras, 2003; Wilbourn and Aminoff, 1988; Fisher, 2002; Zileli et al., 2002; Ertekin et al., 1994; Haig et al., 2005; Chiodo et al., 2007) but this EMG can be more useful only in patients with advanced LSS.

Assessment of F-wave latencies (Plastaras, 2003; London and England, 1991; Adamova et al., 2005; Tang et al., 1988; Tsao, 2007; Egli et al., 2007; Chad, 2007), the H-reflex and its recruitment curve (Tsao, 2007; Pastor and Valls-Sole, 1998), somatosensory evoked potential (Tsao, 2007; Egli et al., 2007; Snowden et al., 1998; Leinonen et al., 2003; Kondo et al., 1989; Storm and Kraft, 2004; Saadeh and Illis, 1994; Aminoff and Eisen, 2005), transcranial magnetic stimulation and leg muscle motor evoked potentials (Saadeh and Illis, 1994; Lang et al., 2002; Baramki et al., 1999) were studied. But this kind of studies have two shortcomings. First of all their stimulation points are far from the target organ. Secondly, LSS diagnosis confirmation with root pathology could be difficult in each case and these procedures did not help to diagnose properly (Lee et al., 1978; Hall et al., 1985; Petropoulos, 1989; Martens and Hoogmartens, 1984; Bartolozzi et al., 1992; Leyshon et al., 1981).

It is more conceivable to record the cauda equina potentials from the lumbar level or to stimulate the spinal roots within the canal using either leg nerves or muscles.

The recording of the cauda equina root action potentials have been tested by some leg nerves but they are scarce in LSS (Osawa et al., 2003). However the evaluation of the cauda equina motor conduction time has been investigated by using lumbar percutaneous magnetic coil stimulation at the dorso-lumbar spine levels (Maccabee et al., 1996). The motor conduction time along the cauda equina using L1 and L5 magnetic stimulation have provided an effective alternative method of the lumbar motor roots evaluation in patients with LSS (Şenocak et al., 2009). Since position of the cauda equina limits application of the magnetic stimulation to that point and sometimes leads to suboptimal results, better results may be obtained by performing recording close to the cauda equina. Epidural stimulation technique from uppermost lumbar level to lumbosacral level may give more precise results about CEMCT in normal control and patients with LSS. Stimulation of the cauda equina and motor roots at the lumbar laminar/spine level were previously reported in normal subjects and in patients with LSS (Zileli et al., 2002; Ertekin et al., 1994). However, one of the previous studies evaluated the CEMCT because they stimulated roots from one segmental level.

We can proposed that the determination of the CEMCT is important for the nerve-root pathology, because the chronic root compression could produce not only the axonal degeneration, but also probable local demyelination. Besides this, the CEMCT is diagnostically important in a selected patients with LSS.

2. Material and methods

Twenty-one prediagnosed LSS patients (14 women, 7 men) (age 42–78, mean 65.2 ± 9.1) who had radiological lumbar imaging (computed tomography and/or magnetic resonance imaging) without any known systemic disease (such as diabetes mellitus and malignancy), polyneuropathy, spinal cord disease or any spinal cord operation previously were chosen for the study. Fifteen normal control subjects (12 women, 3 men) nearly at the same age group (age 36–77, mean 58.6 ± 14.1) were selected among persons referred to EMG laboratory with suspicion of radiculopathy whose EMG results were all found to be normal. Control subjects had either normal MRI and/or CT or their radiological appearence of lumbar spines were not indicative of LSS. Their clinical examination have no objective neurological signs. History of patients put forward 90.47% intermittent neurogenic claudication (19 patients), 4.8% back ache in rest (1 patients), 4.8% (1 patient) right leg and backache in rest. In neurological examination, bilateral achilles reflex loss was determined in 4 patients (19%), unilateral loss was in 3 patients (14.3%). No other neurological symptoms were recorded. Patients did not have any complaint of urinary or bowel dysfunction. They did not have any other objective neurological signs other than complaints just mentioned above during rest or during walking/standing with lumbar extension. On the other hand, backache and/or pain at the buttocks and legs aggrevated by walking and/or long standing position with lumbar extension were told by patients and they relieved just after the rest.

The institutional review board approved the study protocol and informed concent was obtained from all patients.

A two channeled Medtronic-Key point EMG (Denmark-Skovlunde) was used for all examinations. After neurological and physical examination, routine electrophysiological tests were applied to all patients (nerve conduction studies and others: median and ulnar motor and sensory examination, bilateral sural sensory and peroneal, tibial motor examinations, bilateral tibial and peroneal F-responses, bilateral H reflex, muscle EMG L3-5, S1 myotomes: bilateral tibialis anterior, gastrocnemius, rectus femoris and tensor facia lata muscles). H-reflex examination was applied to the patient in prone position. At the same prone position, one Ag–AgCl surface electrode was fixed to belly of soleus muscle, reference Ag–AgCl surface electrode was placed just above the Achilles tendon. Mean distance between active and reference electrode was 17.3 cm. Two consecutive peripheral motor responses were recorded from both soleus muscles with stimulation from each popliteal region. Latencies and peak to peak amplitudes of peripheral motor responses were calculated and recorded.

Lumbar laminar stimulation was performed with subject lying down in prone position as described before (Ertekin et al., 1994).

Teflon coated monopolar needle electrodes were used (50 mm with diameter of 26 G, 38 mm with diameter of 26 G) for laminar stimulation. Active electrode was placed on L1 inter disc space after finding T12 vertebra by palpating 12th rib in the course of chest wall. Long needle (50 mm) was used as a cathode introduced between the spinal processes of L1 and L2 at the midline with a right angle. When examiner had felt the long needle touch bony place, insertion was stopped. The tip of the needle electrode (cathode) was placed at the dorsal part of laminae of the lumbar spine (Fig. 1A and B). Short needle (38 mm) was inserted in the midline subcutaneously, 2 or 3 levels above the cathode. Afterwards rectangular electrical pulses of 1.0 msec duration with increasing intensity were delivered at the laminar level, motor threshold level was obtained. Stimulus intensity was set to two and half times of threshold level. Four consecutive responses were recorded from both soleus muscles by bipolar Ag–AgCl surface electrodes. L1 latency was calculated from the proximal site of stimulation. The same procedure was done for L5 spine position, needle was placed between L5 and S1 space by palpating upper edge of sacrum. L5 latency was obtained on both sides and L1–L5 latency difference called as cauda equina motor conduction time (CEMCT) was calculated for right and left sides. This procedure was performed on both normal controls and patients. CEMCT values from the patients with lumbar stenosis were compared with those obtained from the control group.

All statistical analysis were performed with SPSS 18 for Windows. Descriptive analysis for all parameters were done,