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Motor conduction alterations in patients with lumbar spinal stenosis following the onset of neurogenic claudication

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Abstract The pathogenesis of neurogenic claudication is thought to lie in relative ischemia of cauda equina roots during exercise. In this study we will evaluate the effect of the transient ischemia brought on by exercise on motor conduction in patients suffering from lumbar spinal stenosis (LSS). We will also evaluate the sensitivity of motor evoked potentials (MEPs) in detecting motor conduction abnormalities before and after the onset of neurogenic claudication. Thirty patients with LSS and 19 healthy volunteers were enrolled in the study. All LSS patients had a history of neurogenic claudication and the diagnosis was confirmed with a CT myelogram. Both groups underwent a complete electrophysiological evaluation of the lower extremities. The motor evoked potential latency time (MEPLT) and the peripheral motor conduction time (PMCT) were measured. The subjects were asked to walk on a flat surface until their symptoms were reproduced. A new set of electrophysi-

ological tests was then performed. Exercise did not produce claudication in any of the control group subjects. Twenty-seven patients did have claudication. The pre-exercise MEPLT and nerve conduction studies in the control group fell within the normal range. In the patient group, 19 patients had increased baseline values for MEPLT to at least one muscle. There was a significant difference between the MEPLT and the PMCT values measured before and after exercise in the patients with signs of neurological deficit. This difference was not found to be significant in patients without neurological deficits (t -test $P < 0.05$). It may be concluded that exercise increases the sensitivity of MEPs in detecting the roots under functional compression in LSS.

Key words Lumbar spinal stenosis · Motor evoked potentials · Neurogenic claudication · Physical exercise · Ischemia

Introduction

Neurogenic claudication is characterized by intermittent pain and dysfunction brought on by exercise. The location of the pain in the lower extremity has a lumbo-sacral distribution [11]. Neurogenic claudication has been reported in various conditions [2, 5, 7, 21], but it is generally associated with lumbar spinal stenosis (LSS) [25, 34]. The

pathogenesis of neurogenic claudication is thought to be lie in relative ischemia of active cauda equina roots during exercise [3, 8, 12, 23, 32]. This may be at the site of anastomosis of the two vascular systems supplying blood to the roots (the vasa corona of the spinal cord proximally and the intermediate segmental artery distally). The two systems anastomize at about two-thirds length distal to the spinal cord [26]. It is this area that may be hypovascularized and particularly vulnerable to ischemic insults, espe-

cially when there is an adjacent compressive process compromising the blood supply.

In order to evaluate the functional state of the spinal cord and roots, different electrophysiological testing protocols have been put forward and tested for accuracy and reliability. The use of somatosensory evoked potentials (SSEPs) has been rather disappointing in assessing root compression in the lumbar spine. The peripheral nerve stimulated to produce the SSEP often comprises many roots, and thus a monoradiculopathy is easily masked by normal responses mediated by the intact roots [15]. Stimulation of individual dermatomes through dermatomal SSEPs has proven more promising in lumbar spinal compression [10, 24, 28, 30], yet its use is still rather limited [1]. Motor evoked potentials through transcranial magnetic stimulation have been used to evaluate the functional state of the descending motor pathway. Motor conduction has been shown to be altered in clinical conditions as a result of spinal cord and root compression [6, 9, 13, 14, 19, 31, 33].

Few studies have evaluated the effects of the ischemic process on the various electrophysiological parameters. London and England [20] studied the effect of claudication on the F-wave and reported an increase in F-wave latency time in two cases they isolated. Manganotti et al. [22] performed a similar study and reported similar observations in three of five patients tested. Kondo et al. studied the effect of claudication on nerve conduction along the sensory pathway [18]. They performed SSEPs on patients with spinal stenosis and reported altered SSEPs following the onset of claudication.

By using transcranial magnetic MEPs, this study will assess the effect of the transient ischemic process that brings about claudication on motor conduction along the entire motor pathway. Additionally, it will test the hypothesis that the sensitivity of MEPs in detecting motor dysfunction is increased if the test is performed after the occurrence of claudication.

Materials and methods

In a cross-sectional study, 30 patients with lumbar spinal stenosis and 19 healthy volunteers were enrolled in the study, which was approved by the local ethics committee. All LSS patients had a history of neurogenic claudication and the diagnosis was confirmed with a CT myelogram. Both groups underwent the same electrophysiological evaluation, which consisted of nerve conduction studies and MEPs to the lower extremities. Exclusion criteria for

the study included prior spinal or brain surgery and known or suspected peripheral vascular disease. The study hypothesis and protocol were explained to each participant by a clinical coordinator. All patients and controls gave a complete history and underwent a thorough physical examination.

Disposable silver-silver chloride pellet electrodes (Graphics Controls, Gananoque, Ontario) were placed in a tendon-belly montage over the following muscles bilaterally: vastus lateralis (VL); extensor digitorum brevis (EDB); abductor hallucis (AH). MEPs were produced using a Magstim 200 magnetic stimulator (Nova-matrix, Whitland, Wales), with a figure-of-eight cone coil producing 2.1 T. The stimulator intensity used was at 10% above threshold. The shortest latency time of five successive stimuli was used as the motor evoked potential latency time (MEPLT). The motor response was recorded using standard electromyographic (EMG) equipment. EMG activity was filtered using a bandpass of 16 Hz to 16 kHz, which was digitized at a sampling rate of 10 kHz/channel and stored. Nerve conduction studies were performed to the EDB and the AH muscles using a TD20 EMG/EP machine (TECA, Pleasantville, NY) and M- and F-waves were recorded. The peripheral motor conduction times (PMCT) of the deep peroneal nerve (EDB) and the tibial nerve (AH) were calculated according to the following formula [16]:

$$\text{PMCT} = (\text{M} + \text{F} - 1)/2$$

The PMCT was used to calculate the central motor conduction time (CMCT) to L5 and S1 in the following manner:

$$\text{CMCT} = \text{MEPLT} - \text{PMCT}$$

After performing this initial electrophysiological evaluation and recording the various resting latency times, the subject was asked to walk on a flat surface until his/her symptoms were reproduced or for 15 min, whichever came first. Following the physical exercise, a new set of electrophysiological tests (MEP, nerve conduction) was performed and the post-exercise MEPLT, PMCT, and CMCT were obtained.

MEP latency times were compared with previously published results [4, 6]. The influence of side (left, right) and exercise on the MEPLT and PMCT was evaluated (ANOVA). In addition, a paired *t*-test was used to compare the pre-exercise with the post-exercise MEPLT and PMCT in both study groups. A correlation test was used to compare the MEPLT and the PMCT values obtained both before and after the stress test.

Results

The LSS group consisted of 19 men and 11 women and had a mean age of 66.6 years (range: 38.3–89.3 years). The control group (healthy volunteers) consisted of 10 men and 9 women and had an average age of 62.18 years (range: 41.1–85.9 years). The clinical examination did not reveal any neurological abnormalities in any of the controls. The patient group was divided into two sub-groups according to the presence (sub-group P, *n* = 10) or absence

Table 1 Mean pre-exercise and post-exercise motor evoked potential latency time (MEPLT), in milliseconds with the minimum and maximum values in brackets, measured in the control group

	VL pre-exercise	VL post-exercise	EDB pre-exercise	EDB post-exercise	AH pre-exercise	AH post-exercise
Left	25.5 (23.0–29.0)	25.6 (22.8–29.7)	40.5 (35.0–44.5)	40.3 (35.0–45.5)	41.5 (37.5–43.5)	41.4 (37.0–44.2)
Right	25.6 (22.0–29.5)	25.8 (21.1–30.1)	40.5 (36.3–45.3)	40.4 (36.9–46.8)	41.2 (37.3–44.0)	41.6 (36.0–45.2)

for the vastus lateralis (VL), the extensor digitorum brevis (EDB), and the abductor hallucis (AH) muscles bilaterally

(sub-group A, $n = 20$) of clinical signs of neurological deficit. In sub-group P, six patients had a predominantly motor deficit. Their clinical signs included weak or absent deep tendon reflexes (patellar $n = 3$, Achilles $n = 4$) and distal muscular weakness (L4: $n = 2$, L5: $n = 5$, S1: $n = 5$). The remaining four patients in the P sub-group had a loss of light-touch sensation along the L4 dermatome ($n = 1$), along the L5 dermatome ($n = 3$) and along the S1 dermatome ($n = 4$).

Exercise did not produce claudication in any of the control group subjects. The pre-exercise and the post-exercise MEPLT and PMCT fell within the normal range

(Table 1). There was no significant difference between the pre- and post-exercise values of MEPLT and PMCT (t -test, $P = 0.7483$ and 0.9734 respectively).

Nineteen patients had increased baseline values for MEPLT and PMCT to at least one muscle (7/10 in the P sub-group, 12/20 in the A sub-group). Exercise produced claudication in 27 patients (17 in the A sub-group, 10 in the P sub-group). The MEPLT as well as the PMCT and the calculated CMCT of the two patient sub-groups is presented in Table 2. The number of roots displaying increased MEPLT are listed in Table 3. The percentage of change in the post-exercise MEPLT for the two patient sub-

Table 2 The mean and range (in parentheses) motor conduction latency times (in milliseconds) measured in the two patient sub-groups^a. The latency times are presented for the left and right VL, EDB, and AH muscles

Muscle	Sub-group	MEPLT (Motor Evoked Potential Latency Time)			PMCT (Peripheral Motor Conduction Time)			CMCT (Central Motor Conduction Time)		
		Pre-Exercise	Post-Exercise	Difference	Pre-Exercise	Post-Exercise	Difference	Pre-Exercise	Post-Exercise	Difference
Lt. VL	P sub-group	26.3 (23.0–30.5)	27.3 (24.1–32.6)	1.0	NA	NA	NA	NA	NA	NA
	A sub-group	24.7 (20.0–27.2)	25.0 (20.3–29.0)	0.3	NA	NA	NA	NA	NA	NA
Rt. VL	P sub-group	27.0 (23.3–33.0)	28.4 (24.5–35.7)	1.4	NA	NA	NA	NA	NA	NA
	A sub-group	25.7 (22.0–29.5)	25.8 (21.3–31.0)	0.1	NA	NA	NA	NA	NA	NA
Lt. EDB	P sub-group	44.3 (37.0–57.2)	47.8 (40.2–61.5)	3.5	30.5 (23.2–38.4)	33.7 (26.2–42.0)	3.2	13.8 (10.3–20.0)	14.0 (10.7–20.6)	0.2
	A sub-group	40.1 (34.5–48.0)	40.4 (34.4–50.3)	0.3	24.2 (23.5–31.9)	24.2 (23.5–31.9)	0.0	15.9 (8.3–44.5)	16.2 (10.3–46.8)	0.3
Rt. EDB	P sub-group	44.4 (39.0–60.0)	47.2 (40.4–61.5)	2.8	29.7 (23.3–38.1)	31.2 (23.4–38.8)	1.5	15.8 (12.8–22.0)	16.0 (13.3–22.7)	0.2
	A sub-group	40.4 (34.2–49.5)	40.8 (34.4–50.0)	0.4	26.3 (22.6–31.8)	26.5 (22.6–31.8)	0.2	14.1 (10.2–17.8)	14.3 (10.4–18.3)	0.2
Lt. AH	P sub-group	44.9 (37.8–56.5)	47.1 (39.9–58.5)	2.2	30.3 (25.3–39.8)	32.2 (26.3–41.3)	1.9	14.6 (12.3–19.8)	14.9 (12.7–20.4)	0.3
	A sub-group	41.1 (36.0–49.5)	41.3 (36.4–50.0)	0.2	27.9 (23.7–35.5)	28.0 (23.7–35.5)	0.1	13.2 (9.6–15.7)	13.4 (10.3–16.4)	0.2
Rt. AH	P sub-group	46.1 (39.0–60.3)	48.6 (41.0–63.3)	2.5	30.4 (25.5–42.3)	32.6 (26.7–44.8)	2.2	15.8 (13.1–18.8)	16.0 (13.6–19.4)	0.2
	A sub-group	41.2 (35.1–49.0)	41.6 (35.3–49.5)	0.4	27.9 (23.7–32.8)	28.1 (23.7–32.8)	0.2	13.3 (9.6–16.3)	13.6 (9.8–17.4)	0.3

^aSubgroup P were those who showed clinical signs of neurological deficit, and sub-group A were those who did not

Table 3 The number of affected roots in the two patient sub-groups before and after exercise

Patient sub-group	Increased MEPLT to Rt. L4	Increased MEPLT to Lt. L4	Increased MEPLT to Rt. L5	Increased MEPLT to Lt. L5	Increased MEPLT to Rt. S1	Increased MEPLT to Lt. S1
P-sub-group						
Pre-exercise	$n = 2$	$n = 3$	$n = 3$	$n = 3$	$n = 6$	$n = 5$
Post-exercise	$n = 3$	$n = 4$	$n = 4$	$n = 6$	$n = 8$	$n = 6$
A-sub-group						
Pre-exercise	$n = 5$	$n = 4$	$n = 6$	$n = 6$	$n = 7$	$n = 5$
Post-exercise	$n = 7$	$n = 6$	$n = 7$	$n = 7$	$n = 9$	$n = 9$

Table 4 Distribution of patients according to the percentage change in their post-exercise MEPLT

Muscle	Patient sub-group	Total number	Percentage change in post-exercise MEPLT			
			-3-0%	0-5%	5-8%	> 8%
Left VL	P sub-group	<i>n</i> = 10	<i>n</i> = 2	<i>n</i> = 0	<i>n</i> = 5	<i>n</i> = 3
Right VL		<i>n</i> = 10	<i>n</i> = 2	<i>n</i> = 1	<i>n</i> = 2	<i>n</i> = 5
Left EDB		<i>n</i> = 10	<i>n</i> = 0	<i>n</i> = 2	<i>n</i> = 2	<i>n</i> = 6
Right EDB		<i>n</i> = 10	<i>n</i> = 0	<i>n</i> = 5	<i>n</i> = 2	<i>n</i> = 3
Left AH		<i>n</i> = 10	<i>n</i> = 0	<i>n</i> = 0	<i>n</i> = 9	<i>n</i> = 1
Right AH	A sub-group	<i>n</i> = 10	<i>n</i> = 0	<i>n</i> = 0	<i>n</i> = 7	<i>n</i> = 3
Left VL		<i>n</i> = 20	<i>n</i> = 7	<i>n</i> = 10	<i>n</i> = 1	<i>n</i> = 2
Right VL		<i>n</i> = 20	<i>n</i> = 5	<i>n</i> = 14	<i>n</i> = 1	<i>n</i> = 0
Left EDB		<i>n</i> = 20	<i>n</i> = 10	<i>n</i> = 4	<i>n</i> = 5	<i>n</i> = 1
Right EDB		<i>n</i> = 20	<i>n</i> = 3	<i>n</i> = 14	<i>n</i> = 3	<i>n</i> = 0
Left AH		<i>n</i> = 20	<i>n</i> = 7	<i>n</i> = 12	<i>n</i> = 0	<i>n</i> = 1
Right AH		<i>n</i> = 20	<i>n</i> = 6	<i>n</i> = 9	<i>n</i> = 5	<i>n</i> = 0

groups is presented in Table 4. The nerve conduction (M-wave) to the EDB and the AH did not reveal any abnormal latency times. The increase in MEPLT following exercise was found to be significant in the P sub-group (*t*-test, $P < 0.0001$). This difference was not found to be significant in patients with no clinical signs of a neurological deficit (*t*-test $P > 0.05$). The MEPLT to the VL muscle showed the same trend as did the MEPLT to the EDB and the AH muscles. It was increased in five patients at rest and it increased in two new roots following exercise in the P sub-group. In the A sub-group, there were 9 affected L4 roots prior to exercise and 13 after exercise. There was excellent correlation between the MEPLT and the PMCT values obtained both before and after exercise ($r > 0.973$).

Discussion

The symptoms of LSS are often spectacular, yet physical examination usually renders few findings. The paucity of clinical signs seen in this cohort is consistent with what has been reported by others [11, 29]. The physical examination was positive in only 30% of the patients in the present cohort (P sub-group). The symptoms were present in all the patients and the diagnosis was confirmed with a CT myelogram. As for the MEPs, they were positive in 63% of the patients (19/30). This is consistent with what has been reported by others regarding LSS [6]. In an effort to increase the sensitivity of the MEP test, the patients were asked to walk until they produced their symptoms. The stress test increased the number of patients with abnormal MEPLTs to 23 (76%). When we separated the patients into the two clinical sub-groups, we found that in the P sub-group, the number of patients with abnormal MEPLT increased from 7 (70%) to 10 (100%), while in the A sub-group it went from 12 (60%) to 13 (65%). Additionally, the difference in pre-and post-exercise MEPLT in the P sub-group was clearly higher than in the A sub-group (Table 2).

Exercise and the occurrence of claudication increased the number of roots with abnormal MEPLTs from 17 to 23 in the P sub-group (Table 3) and from 33 to 45 in the A sub-group. Although the change in the average MEPLT in the A sub-group was not significant, it is the increase in the number of roots that is significant. Generally, change in the MEPLT was found to be 5% or more of the baseline value in the P sub-group, while it was 5% or less in the A sub-group. All the patients in this study have a confirmed stenotic process affecting their spinal roots, yet 60% of those patients have no clinical signs of compression (sub-group A). This finding has been reported by others and may be due to a slowly progressive compressive process, allowing hemodynamic compensatory mechanisms to take place. When these mechanisms finally fail (as most likely happened to the patients in the P sub-group), the post-exercise changes in the MEPLT are found to be more significant (Table 4). As for the A sub-group, where compensatory mechanisms are not yet saturated, there is little destruction of the neural tissue and the overall function is still preserved at rest, but becomes deficient following exercise. This may also explain why there was an increase in the number of roots with abnormal MEPLT following exercise.

In order to exclude peripheral neuropathy, we measured nerve conduction velocities in all patients and we calculated the PMCT. We found that there was an excellent correlation between PMCT and MEPLT both before and after exercise ($r > 0.973$). This finding is of certain clinical interest, because it gives additional confidence when evaluating higher segmental levels using MEPs (i.e., the L4 level).

After separating the patients into two sub-groups, we found that three patients in the P sub-group had clinical signs of compression but normal MEPLT at rest, while 12 patients in the A sub-group had increased MEPLT but no clinical findings. Since nerve root ischemia is assumed to be the cause of neurogenic claudication [3, 8, 12, 23, 32], then it is probable that exercise produced a subclini-

cal ischemic condition in the root that affected neural conduction temporarily. In the present study we found that the onset of claudication increased the MEPLT in the patients who had signs of neural deficit. This group of patients had a documented neural compression that produced an increase in their MEPLT before the onset of claudication. When these patients were asked to reproduce their symptoms, the MEPLT was found to increase even further, indicating the occurrence of an acute process that overlays a chronic one. We believe that the mechanical compression is responsible for the chronic increase in latency time, but it is the ischemic process that further decreases nerve conduction in an acute fashion. This is in agreement with the results of studies concerning how ischemia affects neural conduction conducted by Kobrine et al. [17] on the spinal cord of the monkey and by Rydevik et al. [27] on the pig cauda equina. As to why only 73% of patients with symptoms of claudication had abnormal MEPLTs, we believe that in the 27% who had normal MEPLTs, the chronic mechanical compression might have been below threshold and so the ischemic process was not important enough to produce prolonged latency times.

Conclusion

The appearance of neurogenic claudication in LSS is a result of an acute ischemic process affecting the spinal root. This process produces changes in motor conduction that can be measured using MEPs. Clinical examination is positive in about 30% of patients with LSS, while MEPs are prolonged in 66% of patients. This percentage was increased to 76% following exercise. Exercise increases the sensitivity of MEPs in detecting the roots under functional compression in LSS. The MEPLT of the VL muscle may be helpful in evaluating motor conduction along the L4 pathway, provided that peripheral neuropathy is ruled out.

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