A Different Way in the Diagnosis of Meralgia Paresthetica: A Late Response Obtained From Vastus Medialis Muscle

MERALJİA PARESTETİKA TANISINDA FARKLI BİR YÖNTEM: VASTUS MEDİALİS KASINDAN ELDE EDİLEN BİR GEÇ YANIT

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Abstract

Objective: Although nerve conduction studies (NCS) of the lateral femoral cutaneous nerve (LFCN) and somato-sensorial evoked potentials (SEP) generated by the stimulation of LFCN (LFCN SEP) are the main tools in the electro-diagnosis of meralgia paresthetica (MP), these techniques have certain limitations. The analysis of a late response (LR) obtained from the vastus medialis muscle and evoked by the stimulation of LFCN (LFCN-LR) would be useful in the diagnosis of MP. In this preliminary study, we aimed to determine the usefulness of this putative late response.

Material and Methods: Twenty patients with MP (18 unilateral, 2 bilateral), 16 patients with unilateral L3 radiculopathy and 26 controls were included in the study. LFCN-LR, LFCN NCSs and SEP analyses were performed in patients with MP and L3 radiculopathy.

Results: A late response was recorded at 15.7±1.0 ms in control subjects. The onset latency of LFCN-LR was significantly prolonged in patients with MP (20.1±4.1). Asymmetric prolongation of LFCN-LR was the most sensitive parameter in patients with unilateral MP (17/18).

Conclusion: Although the potential effect of indirect excitation of the femoral nerve could not be eliminated, our results suggest that LFCN-LR may be a useful test in the diagnosis of MP.

Key Words: Meralgia paresthetica, lateral femoral cutaneous nerve, somatosensory evoked potentials, late response

Özet

Amaç: Meraljia parestetika (MP) elektrofizyolojik tanısında lateral femoral kutanöz sinir (LFCN) ileti çalışmaları ve somatosensoryel uyarılmış potansiyel (LFCN SEP) çalışmaları başlıca araçlar olmasına rağmen önemli sınırlılıkları vardır. LFCN uyarımı ile elde edilebilecek geç bir yanıt (LFCN-LR) MP tanısında yararlı olabilir. Bu çalışmada varsayılan bu geç yanıtın MP tanısındaki ve ayırıcı tanısındaki katkısının incelenmesi planlanmıştır.

Gereç ve Yöntemler: MP tanısı alan 20 olgu (18 unilateral, 2 bilateral), L3 düzeyinde unilateral radikülopatisi olan 16 olgu ve 26 kontrol olgusu çalışmaya alınmıştır. LFCN sinir ileti çalışmaları, LFCN SEP ve LFCN-LR analizleri yapılmıştır.

Bulgular: LFCN-LR adı verilebilecek bir geç yanıt 15.7±1.0 ms'de ortaya çıkmıştır. LFCN-LR başlangıç latansı MP olgularında belirgin olarak uzun bulunmuştur (20.1±4.1 ms). Unilateral MP olgularında tüm elektrofizyolojik veriler arasında LFCN başlangıç latansının asimetrik uzaması en belirgin anormallik olarak değerlendirilmiştir (17/18).

Sonuç: Bu geç yanıt oluşumunda femoral sinirin indirek uyarımının rolünün ekarte edilememesi ile birlikte bulgularımız LFCN-LR incelemesinin MP tanısında yararlı olabileceğini düşündürmüştür.

Anahtar Kelimeler: Meraljia parestetika, lateral femoral kutanöz sinir, somatosensoryel uyarılmış potansiyeller, geç yanıt

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Meralgia paresthetica (MP) is an entrapment neuropathy of lateral femoral cutaneus nerve (LFCN) in the inguinal region. Electrophysiological diagnosis mainly depends on the demonstration of conduction abnormalities of involved lateral femoral cutaneus nerve.[1,2] Different techniques were defined to evaluate the nerve conduction of LFCN.[1,3,4] The evaluation of conduction abnormalities of LFCN is difficult because of frequently observed anatomical variations of this nerve.[1,5] Additionally, invasive procedures were needed frequently for the analysis of nerve conduction of LFCN.[4] The other method

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described in the diagnosis of MP is evaluation of somatosensory cortical evoked potential (SEP) evoked by LFCN (LFCN SEP).[2,6,7] However, there are contradictory reports about the sensitivity of this technique in the electrodiagnosis of MP.[2,8] Additionally, this technique takes relatively longer time.

The Hoffman reflex (H-reflex) is electrically evoked reflex responses recorded from calf muscles.[9] This reflex response reflects the continuity of S1 spinal segment, afferent and efferent pathways. This reflex response can also be evoked from quadriceps muscle in healthy adults.[10] It was reported that vastus medialis is most appropriate muscle for recording of this reflex.[11] Vastus medialis H-reflex (vm-HR) has not been widely used clinically. This reflex is evoked by the stimulation of the femoral nerve.[10,11] The amplitude but not onset latency of this potential can be influenced by posture and remote muscle contractions.[11,12] Latency and amplitude alterations of vm-HR in patients with radiculopathy were reported in a previous study.[13] If a reflex response similar to vm-HR, could be recorded by the electrical stimulation of LFCN, this investigation can be extremely useful in the diagnosis of MP. This putative reflex response can not be called as an H reflex because LFCN does not contain muscle spindle afferents.

Our aim was to determine the diagnostic sensitivity of this reflex response (LFCN-LR) evoked by electrical stimulation of LFCN in patients with MP. On the other hand, we planned to compare the results with electrophysiological findings obtained from the patients with L3 radiculopathy.

Material and Methods

Subjects

Twenty consecutive patients diagnosed as MP according to the symptoms and clinical findings were analyzed in this study. The patients were 12 males and 8 females whose ages ranged from 35 to 67 years (mean 52.8±9.6). The disease duration ranged from 3 months to 15 years (mean 6.1±4.1 years). Eight-teen had unilateral, two had bilateral involvement (Twenty-two extremities were affected). All patients had numbness, paresthesias and pain located in the anterolateral thigh. Disputed patients with motor or reflex disturbance in their lower extremities were excluded from the study. No patient with MP showed any other neurological or systemic disorders at the time of the electrophysiological analysis. Sixteen patients diagnosed as L3 radiculopathy according to clinical and radiological findings were also evaluated in this study. The patients with L3 radiculopathy were 10 males and 6 females whose ages ranged from 30 to 71 years (mean 52.0±12.3). All patients had unilateral L3 radicular involvement. The disease duration ranged from 2 months to 7 years. The diagnosis of L3 radiculopathy was confirmed by clinical examination and MRI investigation. The electrophysiological criterion for the diagnosis of L3 radiculopathy was a denervation pattern in at least two muscles innervated by L3 root. The data obtained from patients with MP were compared with the data obtained from patients with L3 radiculopathy and twenty-six control subjects (15 males, 11 females). Controls' ages ranged from 34 to 65 (mean 50.8±9.5). Mean heights of the patient groups and controls were not different ($p>0.05$) (163.1±6.2 for MP, 166.2±6.1 for L3 radiculopathy and 165.2±7.8 for controls). This study was approved by the local ethical committee

Electrophysiological Investigations

For the electrophysiological analysis, subjects were tested in supine position. All subjects were instructed to avoid any movement during the tests. Head and neck posture of all subjects was at the neutral position. Medelec Synergy EMG equipment was used for recordings. During the measurements, skin temperature was between 30-32 °C in all subjects.

The sensory nerve conduction study of LFCN was performed according to the method described by Spevak and Prevec, (1995) previously. LFCN was stimulated with standard surface bipolar electrodes placed 6-10 cm below the anterior superior iliac spine. The stimulus duration was 0.1

ms. Stimulation intensity was between from 60 to 120 V (85.5±19.2V). Sensory nerve action potential was recorded with bar recording electrode (Medelec 16934). Recording electrode was placed 19-24 (21.8±1.1) cm distal to the stimulator electrode on the anterolateral aspect of thigh (on the line between anterior superior iliac spine and lateral side of patella). The ground electrode was placed between stimulating and recording electrodes. Thirty-two responses were recorded and averaged. Every trial was repeated at least twice. The oscilloscope sweep time was 10 ms. Amplifier filters were between 50 Hz and 10 kHz. The onset latency, peak to peak amplitude of sensory nerve action potential (SNAP), conduction velocity of LFCN and interside differences of these parameters were analyzed.

The SEP evoked by LFCN (LFCN SEP) was recorded by Fz/Cz derivation. Stimulator electrode was placed 12 cm below to the anterior superior iliac spine. Stimulus intensity was three fold of the perception threshold of electrical stimulation for both sides. Five hundred responses were recorded and averaged. Every trial was repeated at least twice. Oscilloscope sweep time was 100 ms. Amplifier filters was between 2 Hz and 2kHz. Repetition rate of electrical stimulation was 3 Hz. The stimulus was square wave current pulse and its' duration was 0.1 ms. Ground electrode was placed at Fpz. The peak latency and peak to peak amplitude of first cortical response (P0) and interside differences of these parameters were analyzed.

For the analysis of LFCN-LR, LFCN was stimulated from same place with analysis of LFCN SNAP (10 cm below to the anterior superior iliac spine). Stimulus intensity was progressively increased to obtain a stable H reflex. It was between 95 and 170 V (140.9±18.6). Strong electrical stimulus was avoided from excitation of femoral nerve by volume conduction. During the recording, subjects were requested to sustain slight activation of quadriceps muscle (about 50% of maximal voluntary contraction). This muscle activity was monitored visually from oscilloscope. Active electrode was placed on the belly of vastus medialis muscle. Reference one was placed on the tendon (just proximal to the patella). Bar electrode was used for recording. It was oriented in a 45° angle with the femoral axis. At least five successive responses (reproducible responses) were recorded for each side. Stimulation rate was 0,1 Hz. The stimulus duration was 1.0 ms. Oscilloscope sweep time was 50 ms. Amplifier filters were between 2Hz-10kHz. The responses obtained by low electrical current and similar waveform to M response were accepted as vm-HR. The onset latency, peak to peak amplitude of LFCN-LR, the amplitude ratio of LFCN-LR and vastus medialis CMAP (LR/M ratio) were analyzed. Additionally, interside differences of the onset latencies and peak to peak amplitudes of HR were analyzed.

Conventional concentric EMG of L3 innervated muscles (vastus lateralis, iliopsoas and adductor longus) were investigated in both patient group and controls.

Statistical Methods

ANOVA and Tukey post-hoc tests were used to compare the electrophysiological data from patient groups and controls. The effect of height on the latencies of LFCN vm-HR and SEP were corrected by analysis of covariance. Height was accepted as covariant in these tests. The maximum Type I error rate level was accepted as %5. Normal limits were defined as mean ± 3SD.

Results

All patients with MP had numbness, paresthesias and pain located in the anterolateral thigh. No motor finding was observed in any patient with MP. Eight patients with L3 radiculopathy had motor involvement (weakness at the hip flexion), 13 patients showed diminished patellar reflex response and sensorial findings (numbness and paresthesias) located at the L3 dermatomal area. Ten had both motor and sensorial symptoms. All patients with radiculopathy showed unilateral bulged or herniated disc at the L2-3 level in the MRI investigation. Twelve had the involvement of lower lumbar segments in their

Table 1. Lateral femoral cutaneous nerve conduction and SEP values obtained from patients with MP and controls. Statistical differences were more profound between affected side of patients and controls. The peak to peak amplitude of P0 did not show significant difference between healthy subjects and affected sides of patients.

	Patients with MP (n:20)				
	Affected side	Normal side	Controls (n:23)	p¹	p²
Onset Latency (ms)	4.0±0.5	3.6±0.5	3.4±0.3	0.005	0.5
Peak to peak Amplitude (µV)	1.9±0.9	4.1±2.1	4.7±1.1	0.0001	0.003
NCV (m/s)	55.6±7.3	63.9±6.9	64.4±3.8	0.0001	0.002
Peak latency of P0* (ms)	32.6±3.0	28.4±3.4	30.4±2.2	0.01	0.002
Peak to peak amplitude of P0	0.9±0.4	1.2±0.6	1.7±0.4	0.0001	0.5

*First cortical response of LFCN SEP.
p¹ The comparison of affected side of patients with MP and controls.
p² The comparison of affected and healthy sides of patients with MP.

Table 2. Lateral femoral cutaneous nerve conduction and SEP values obtained from patients with L3 radiculopathy and controls.

	Patients with radiculopathy (n16)				
	Affected side	Normal side	Controls (n:23)	p¹	p²
Onset Latency (ms)	3.4±0.5	3.7±0.5	3.4±0.3	0.2	0.7
Peak to peak Amplitude (µV)	4.7±1.2	4.8±0.9	4.7±1.1	0.9	0.7
NCV (m/s)	63.8±4.4	63.9±4.1	64.4±3.8	0.9	0.9
Peak latency of P0 (ms)	30.9±2.2	31.0±1.8	30.4±2.2	0.9	0.07
Peak to peak amplitude of P0	1.1±0.6	1.2±0.7	1.7±0.4	0.001	0.9

p¹ The comparison of affected side of patients with L3 radiculopathy and controls.
p² The comparison of affected and healthy sides of patients with L3 radiculopathy.

MRI investigation. Mean values of LFCN sensory nerve conduction velocity (LFCN SNCV) and peak to peak amplitude of LFCN SNAP obtained from control group was 64.4±3.8 m/s and 4.7±1.1 µV respectively. LFCN SNAP was obtained in 13/22 extremities with MP (59.1%). This potential could be recorded in all patients with L3 radiculopathy and control subjects. The peak to peak amplitude of LFCN SNAP was significantly diminished in patients with MP compared to controls and patients with L3 radiculopathy (p: 0.0001 and 0.0001 respectively). LFCN SNCV was significantly decreased in patients with MP compared to controls and patients with L3 radiculopathy (p: 0.0001 and 0.001 respectively) (Tables 1 and 2). Interside differences in the peak to peak amplitudes of LFCN SNAP exceeding 2.8 µV and in SNCV exceeding 8.2 m/s were considered as abnormality. These parameters did not show significant difference in patients with L3 radiculopathies. Nerve conduction abnormalities of LFCN were observed in 20/22 extremities (90.1%) with MP (specificity 96.2%). 18/22 extremities had absent or reduced LFCN SNAP amplitude. Increased interside difference in the nerve conduction velocities was observed in 7/22 extremities. Decreased conduction velocity was observed in only 4/22 extremities.

The latency of P0 component of LFCN SEP was 30.4±2.2 ms. The peak latency of first cortical response of LFCN SEP (P0) was prolonged in patients with MP compared to the patients with L3 radiculopathy and controls (p: 0.001 and 0.0001 respectively). The amplitude of P0 was decreased in patients with MP compared to controls and to the patients with L3 radiculopathy (p: 0.0001 and 0.01). These parameters were also not changed between patients with L3 radiculopathy and controls (p: 0.1). Interside difference exceeding 2.8 ms in the peak latencies of P0 was considered abnormal. 16 patients (17/22 extremities) with MP (sensitivity: 77.3%, specificity: 100%) showed

SEP abnormalities. Main abnormality was increased interside difference in the latency of P0 component (14/22 extremities).

LFCN-LR is a negative onset biphasic potential. Its' shape was similar to M response of this muscle (Figure 2). The control value of onset latency of LFCN-LR was 15.6±0.9 ms. Normal values of the amplitude and LR/M amplitude ratio were 1.6±0.9 and 0.41±0.2, respectively. Onset latency of LFCN-LR was prolonged in patients with MP in compared to the patients with L3 radiculopathy and controls (p: 0.0001 and 0.0001) (Figure 1). Peak to peak amplitude of LFCN-LR was not changed in patients with MP and L3 radiculopathy. There was also not any difference in LR/M amplitude ratio between patient groups and controls (p>0.05) (Table 3). The onset latencies of LFCN-LR exceeding 18.3 ms or interside differences of this parameter exceeding 1.5 ms were considered abnormality (Figure 3). Nineteen patients with MP (20/22 extremities), showed either prolonged onset latency of LFCN-LR or increased interside difference in onset latency of LFCN-LR (sensitivity 90.1%, specificity 100%) (Table 4). Most prominent abnormality was the increased interside difference in the latency of LFCN-LR (17/18 extremities). No patients with radiculopathy showed any abnormality in the latency or amplitudes of LFCN-LR (Table 4).

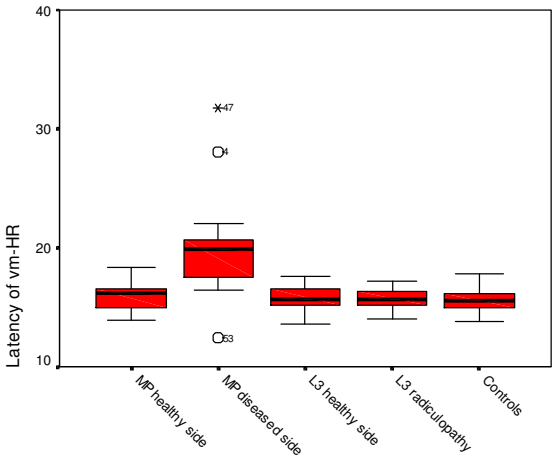

Figure 1. Onset latencies of vm-HR in patient groups and controls.

Concentric EMG investigations disclosed the neurogenic pattern observed in the L3 innervated muscles in all patients with L3 radiculopathy. Only one patient showed on-going dennervation pattern (the existence of fibrillation potentials and polyphasic motor unit potentials) in vastus lateralis. Others had chronic denervation (enlarged motor unit potentials, decreased recruitment) pattern in L3 innervated muscles.

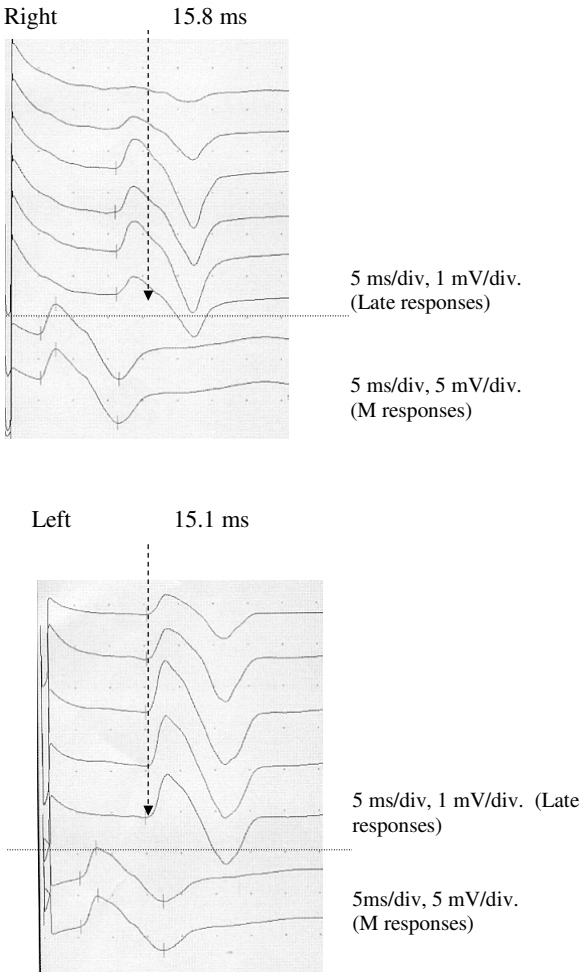

Figure 2. LFCN vm-HR recordings from a control subject. Arrows indicate vm-HR onset latencies.

Discussion

In the present paper, clinical usefulness of a putative late response evoked by the stimulation of LFCN (LFCN-LR) in the electrophysiological

Table 3. The analysis of the vastus medialis H-reflex (vm-HR) obtained from patients with meralgia paresthetica (MP) and L3 radiculopathy and controls. The patients with MP showed prolonged vm-HR onset latency whereas this difference was not observed in patients with L3 radiculopathy.

	Patients with MP		Patients with radiculopathy				
	Affected side	**Normal side**	**Affected side**	**Normal side**	**Controls**	**p[1]**	**p[2]**
Latency of LFCN-LR (ms)	20.1±4.1	16.1±1.4	15.7±0.8	15.7±1.1	15.7±1.0	0.0001	0.9
p[3] (Latency of vm-HR)	0.0001		0.9				
Amplitude of LFCN-LR (mV)	1.5±0.9	2.3±1.5	1.8±1.2	2.2±1.2	1.7±1.0	0.9	0.9
p[3] (Amplitude of vm-HR)	0.1		0.9				
Amplitude ratio	0.32±0.2	0.40±0.2	0.47±0.3	0.62±0.3	0.40±0.2	0.8	0.9
p[3] (HR/M)	0.7		0.5				

p[1]: The comparison of affected side of patients with MP and controls.
p[2]: The comparison of affected side of patients with radiculopathy and controls.
p[3]: The comparison of affected and healthy sides of patients with L3 radiculopathy and MP.

Table 4. Detailed presentation of the results of electrophysiological investigations obtained from patients with MP

Patients	Age	Decreased SNCV	Decreased amplitude of SNAP	IID* of SNCV	Increased latency of P0	IID of P0 latency	Increased latency of LFCN-LR	IID latency of LFCN-LR
YP	65	NR**	NR	NR	+	+	+	+
RA	53	NR	NR	NR	-	+	+	+
MM	56	+	-	+	-	+	+	+
SA	45	NR	NR	NR	NR	NR	+	+
FG	58	NR	NR	NR	-	+	+	+
RA	49	-	+	-	+	+	+	+
CÇ	48	-	+	-	-	+	+	+
MT	47	NR	NR	NR	NR	NR	NR	NR
KE	35	+	+	+	-	+	+	+
UU	56	+	-	+	-	+	+	+
KP	45	-	-	-	-	+	+	+
KA	65	-	+	+	+	+	+	+
FA	52	NR	NR	NR	-	+	+	+
KU	50	-	+	+	-	-	-	+
EC	67	NR	NR	NR	-	+	+	+
IS[1]	60	NR	NR	NR	+	+	+	-
IS[1]	60	-	+	-	+	-	+	-
MS	35	-	+	+	-	-	+	+
HS[1]	61	+	+	-	-	-	-	-
HS[1]	61	NR	NR	NR	-	-	-	-
MK	42	-	+	+	+	+	+	+
EP	67	-	-	-	-	-	+	+

* IID: Increased interside difference **NR: No response [1] Bilateral involvement

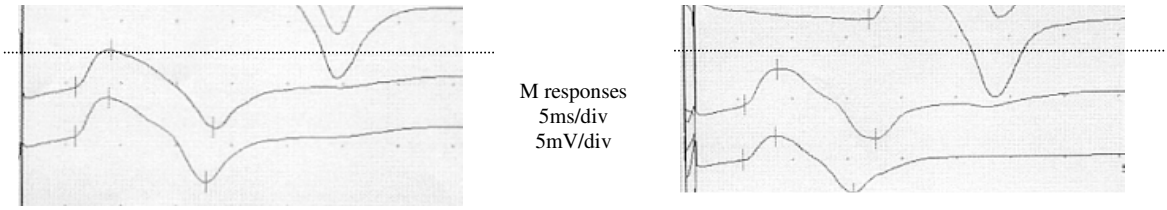

Figure 3. LFCN nerve conduction studies (NCS) (A) and vm-HR recordings (B) from a patient with unilateral MP. An amplitude reduction in sensory nerve potential without latency prolongation (A) and prolonged latency in vm-HR onset latency (B) were observed. Arrows indicate the onset latencies of vm-HR.

damage of LFCN. In mild MP, no difference in NCS may be observed by this method.[1] Decreased conduction velocity of LFCN in some patients (4/22) was explained by the vulnerability of rapid conducting, myelinated nerve fibers at the entrapment site in MP. Increased interside difference in the NCV of LFCN was more frequently observed (7/22). Most common NCS abnormality was diminished LFCN SNAP amplitude (9/22) probably due to degeneration of some fibers in the entrapment site.

We used Fz/Cz derivation for LFCN SEP recording. The SEP abnormalities were higher when Fz/Cz derivation was used in a previous study.[2] Contradictory studies concerning the sensitivity of LFCN SEP in the diagnosis of MP have been reported.[2,7,14] SEP analysis in the diagnosis of MP was first performed by Eisen and Elleker in 1980.[15] In 1983, Synek and Cowan evaluated the value of SEP in the diagnosis of MP. They studied LFCN SEP in two patients with MP. Their results supported the diagnosis of MP in one patient. Increased interside differences of P0 component peak latency was a useful parameter in the evaluation of LFCN SEP in the diagnosis of MP.[6] Po and Mei demonstrated that LFCN SEP abnormality was found in all of their 22 patients with MP.[7] Their normality ranges (mean ± 2SD) were different from present study (mean ± 3 SD) and this difference in the methodology of both studies may cause this discrepancy between results. On the other hand, the sensitivity of LFCN SEP in the diagnosis of MP was very low in another study.[2] Our results also disclosed that LFCN SEP had lower sensitivity than other methods in the diagnosis of MP. Another disadvantage of SEP analysis is the requirement of relatively long time interval for EMG laboratory. It was concluded that SEP should not be recommended as a routine procedure in the MP diagnosis.

Main topic of the present study was to evaluate the clinical usefulness of the LFCN-LR in the diagnosis of MP. We stimulate the LFCN at the 10 cm distal to the anterior superior iliac spine because LFCN has a superficial course as a single trunk at this site. On the other hand, this stimulation site is not affected from the variations of this nerve and allows the reliable analysis of entrapment site.[1]

In the present study, we aimed to avoid from indirect excitation of the femoral nerve by the volume conduction. We think that observing no vastus medialis M response during the LFCN evoked late response recording even by high stimulus intensities was a convincing finding for reliability of this technique. Nevertheless, there are some doubts about this late response. LFCN does not contain rapid conductive muscle spindle afferents (1a fibers) which are mainly responsible in the generation of H reflex. Therefore, it can not be expected somewhat early response for LFCN.

Patients with L3 radiculopathy were participated in this study because clinical similarities between in some patients with MP and lumbar radiculopathy have been reported previously.[16] Our aim was to determine the usefulness of LFCN-LR in the differential diagnosis of lumbar radiculopathy and MP. L3 radiculopathy may affect this reflex arch at the radicular level. Sabbahi and Khalil demonstrated that the amplitude of vastus medialis H reflex was diminished and the latency of this response was prolonged in patients with L4 radiculopathy.[13] We could not demonstrate any difference in electrophysiological parameters in present study. There was an amplitude asymmetry between diseased and healthy sides of patients with L3-4 radiculopathy in our study but this difference did not reach statistical significance. Different stimulation procedures can cause this discrepancy between the results of these studies. Some fibers can escape from injury and may provide normal conduction along the involved root because partial involvement of a single root is not an uncommon pattern in radiculopathies.[9] Additionally, LFCN is originated from L2 and L3 spinal nerves and one of two roots may not be influenced from disc disease.[17] It seems that a compression neuropathy affecting the afferent arch of nerve trunk results more profound alterations in this reflex arch.

In this preliminary study, our findings suggest that LFCN-LR can be an electrophysiological tool

in the diagnosis of MP. It seems that lumbar radiculopathies as clinical condition mimicking MP do not cause any significant alterations in this late response. Nevertheless, the excitation of femoral nerve by volume conduction is still a problem. The application of more localized stimulation procedures of LFCN such as near nerve stimulation can be helpful in explanation of the nature of this late response.

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