

Medial calcaneal neuropathy: a missed etiology of chronic plantar heel pain

Emmanuel Kamal Aziz Saba, Sarah Sayed El-Tawab,
Hussein Al-Moghazy Sultan

Department of Physical Medicine,
Rheumatology and Rehabilitation, Faculty of
Medicine, Alexandria University, Alexandria,
Egypt

Correspondence to Emmanuel Kamal Aziz
Saba, MD, Department of Physical Medicine,
Rheumatology and Rehabilitation, Faculty of
Medicine, Alexandria University, Alexandria,
21131, Egypt. Tel: +20 354 454 23;
e-mail: emadaziz55@yahoo.com

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Introduction

Medial calcaneal neuropathy had been implicated as a source of chronic plantar heel pain.

Aim

The aim was to determine the presence of medial calcaneal neuropathy as a cause of chronic plantar heel pain.

Patients and methods

The present study included 43 heels obtained from 38 patients with chronic plantar heel pain and 30 apparently healthy volunteers as a control group. Clinical examination was done. Sensory nerve conduction study of the medial calcaneal nerve was performed. This was a single-center, public hospital-based study. It was designed as a cross-sectional examination of consecutive patients with chronic plantar heel pain.

Results

There were 27 (62.79%) heels, from 23 (60.52%) patients, who had medial calcaneal neuropathy. From them, unobtainable medial calcaneal nerve response was present in 10 (37.03%) heels of nine (39.13%) patients. Medial calcaneal neuropathy was the solitary cause of chronic plantar heel pain in 10 (37.03%) heels from 10 (43.48%) patients. However, it was associated with other local heel pathologies in the remaining patients. The majority of them were having plantar fasciitis in nine (33.34%) heels from five (21.73%) patients.

Conclusion

Medial calcaneal neuropathy is present in a considerable number of patients with chronic plantar heel pain. It should be taken into consideration during the assessment of any patient with chronic plantar heel pain.

Keywords:

chronic heel pain, medial calcaneal nerve, medial calcaneal neuropathy, plantar heel pain, tarsal tunnel syndrome

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Introduction

Plantar heel pain is a commonly encountered problem that causes significant discomfort. Several causes have been implicated to cause chronic plantar heel pain [1,2]. The neurological etiologies of it include entrapment neuropathy of medial plantar nerve, lateral plantar nerve, or medial calcaneal nerve (MCN) branches of the posterior tibial nerve [1]. Medial calcaneal neuropathy is considered to be a form of tarsal tunnel syndrome (TTS), when it occurs solitary and is not associated with medial or lateral plantar neuropathies [3–5].

Medial calcaneal nerve is a sensory nerve. It is the first branch that arises from the posterior tibial nerve at the level of the ankle [6–9]. The MCN provides sensory innervation to the inferior, medial, and posterior aspects of the heel and the calcaneus [8].

Medial calcaneal neuropathy is considered to be the second most common reported neuropathy that causes

plantar heel pain of neural origin [10–13]. It is usually missed in the differential diagnosis of plantar heel pain, especially the chronic ones [3,13–16]. The diagnosis of it could be difficult because its symptoms are often subjective and are mimicking those associated with other heel and foot pathological conditions [5,13]. There are scanty studies that assessed this issue.

The aim of this study was to determine the presence of medial calcaneal neuropathy as a cause of chronic plantar heel pain.

Patients and methods

The present study included 43 heels obtained from 38 patients with chronic plantar heel pain from those

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attending the outpatient clinic of Physical Medicine, Rheumatology and Rehabilitation Department, Main University Hospital, Alexandria Faculty of Medicine, and 30 apparently healthy volunteers (30 heels) as a control group. The volunteers included medical staff, their relatives and patients' relatives. Plantar heel pain of at least 3 months duration was considered to be chronic [17]. Exclusion criteria included diabetes mellitus, endocrine disorders, metabolic disorders, and neurological disorders including lumbosacral radiculopathy, peripheral neuropathy, and TTS affecting the medial and/or lateral plantar nerves. The study was explained to the participants and an informed consent was given by each. The study had been approved by the ethics committee of the Faculty of Medicine, Alexandria University, Egypt.

Demographic data collection and history taking were done stressing on the presence of subjective burning pain and/or paresthesia or numbness over the territory of MCN. Clinical examination was done stressing on the musculoskeletal and neurological examination of the lower limb. Assessment of sensation over the territory of the MCN was done to detect the presence of objective sensory loss in the form of hypoesthesia or anesthesia [3,4,18].

Electrophysiological studies were conducted on a Nihon Kohden Neuropack MEB-7102 mobile unit with a two-channel evoked potential/electromyography measuring system (Nihon Kohden Corporation, Tokyo, Japan). Skin temperature at the site of the recording electrodes was maintained around 32–34°C by means of an infrared lamp. The skin of the stimulation and recording sites were abraded with a commercial cleansing stone especially the heel and cleansed with alcohol. The ground electrode was placed between the recording electrodes distally and the stimulation site proximally. Conduction distances were measured by a measuring tape with a precision of 1 mm [19].

Sensory nerve conduction study of the MCN was done by using an antidromic technique. It was done for patients and for the apparently healthy volunteers (control group). The following was applied: the sweep speed was 2 ms/division and the sensitivity was 5–10 μ V/division. The filter bandwidth was 20 Hz–2 kHz. The bipolar stimulator had a production current ability of 50 mA. The pulse duration was 0.2 ms. Signal averaging was applied. Responses were superimposed to ensure reproducibility. Supramaximal stimulation was ensured. Measurements of the sensory nerve action potential (SNAP) included onset latency (in milliseconds), peak latency (PL)

(in milliseconds), amplitude (in microvolts), and conduction velocity (CV) (meter per second) using onset latency [19].

The antidromic technique for doing the sensory nerve conduction study of the MCN was as follows: the active recording surface disc electrode was placed in a point at one-third the distance between the apex of the heel to a point midway between the navicular tuberosity and the prominence of the medial malleolus. The reference surface disc electrode was placed on the apex of the heel. Electrical stimulation was done at about 2 cm posterior to the medial edge of the tibia, at about 10 cm proximal to the active recording electrode [7]. Figure 1 is an illustration of the technique of MCN sensory nerve conduction study.

The diagnosis of medial calcaneal neuropathy depended on the presence of subjective burning and tingling sensation or objective sensory loss in the territory of the MCN with or without the presence of positive Tinel's sign in an area inferior and posterior to the medial malleolus. This was confirmed with the results of the sensory conduction study of the MCN [3,4]. Patients with medial calcaneal neuropathy were considered group I, whereas group II included patients without medial calcaneal neuropathy.

Statistical analysis of data was done using the statistical package for the social sciences (SPSS version 17) software [20]. Descriptive measures [count, frequency, minimum, maximum, mean, and standard deviation (SD)] as well as analytic measures (Mann–Whitney test, Kruskal–Wallis test, and Pearson's Chi-square-test) were used. Correlation study was done using

Figure 1



This is an illustration for the stimulation technique and recording electrodes position for the medial calcaneal nerve sensory conduction study.

Spearman's correlation test. Statistical significance was assigned to any *P* value at less than 0.05. The reference cutoff values of the electrophysiological studies were calculated by rounding the mean plus or minus two SD to measure the upper limit of normal or the lower limit of normal, respectively.

Results

The present study included 43 heels obtained from 38 patients with chronic plantar heel pain [26 (68.42%) women]. Their mean age was 40.67±9.91 years (range: 21–62 years). The control group consisted of 30 asymptomatic heels that were obtained from 30 apparently healthy individuals [20 (66.67%) women]. Their mean age was 39.00±11.58 years (range: 19–60 years). There were no statistically significant differences between patients and control group as regards sex ($\chi^2=0.024$, $P=0.878$) and age ($Z=-0.404$, $P=0.686$). The clinical characteristics of the patients and controls are summarized in Table 1.

Reference values for different parameters of the MCN SNAP from the controls are shown in Table 2.

The patients were subdivided into two groups according to the presence of medial calcaneal neuropathy. Group I included patients with medial calcaneal neuropathy, whereas group II included patients without medial calcaneal neuropathy. There were 27 (62.79%) heels obtained from 23 (60.52%) patients who had medial calcaneal neuropathy (group I) proved clinically and electrophysiologically. From them, unobtainable medial calcaneal nerve response was present in 10 (37.03%) heels of nine (39.13%) patients. The comparison between the two patient groups and the control group as regards the results of the nerve conduction study of the MCN are shown in Table 3.

There were statistically significant differences between parameters of MCN SNAP among the two patient groups and control group as regards the PL, CV, and

Table 1 Clinical characteristics of patients and controls

Clinical characteristics	Patients (<i>n</i> =43 heels obtained from 38 patients)	Controls (<i>n</i> =30 heels obtained from 30 healthy participants)	Test of significance	<i>P</i> value
Heel side (right/left) [<i>n</i> (%)]	25 (58.14)/18(41.86)	17 (56.67)/13 (43.33)	$\chi^2=0.016$	0.900
Duration of complain (mean±SD) (years)	1.88±1.52	NA	NA	NA
Objective sensory loss at the territory of the MCN [<i>n</i> (%)]	27 (62.79)	NA	NA	NA

MCN, medial calcaneal nerve; *n*, number of heels; χ^2 , value of chi-square test; NA, not applicable. $P<0.05$, significant.

Table 2 The determined reference cutoff values of medial calcaneal nerve sensory conduction study parameters obtained from the control group

MCN SNAP parameters	Mean±SD	Range	NL	Rounded NL
Peak latency (ms)	2.31±0.23	1.72–2.50	2.77	2.8
CV (m/s)	57.36±12.31	42.30–83.30	32.74	32.7
SNAP amplitude (μV)	6.39±2.16	4.00–11.00	2.07	2.1

CV, conduction velocity; MCN, medial calcaneal nerve; NL, upper (latency) or lower (conduction velocity and amplitude) limit of normal; SD, standard deviation; SNAP, sensory nerve action potential.

Table 3 Comparison of the clinical characteristics and different medial calcaneal nerve sensory conduction study parameters between the two patient groups and the control group

Clinical characteristics and MCN sensory conduction study parameters	Group I (<i>n</i> =27 heel from 23 patients)	Group II (<i>n</i> =16 heel from 15 patients)	Control (<i>n</i> =30 heels obtained from 30 healthy participants)	Test of significance	<i>P</i>
Women [<i>n</i> (%)]	15 (65.22)	11 (73.33)	20 (66.67)	$\chi^2=0.297$	0.862
Age (mean±SD) (years)	42.29±9.54	37.93±10.22	39.00±11.58	$K=2.180$	0.336
Side (right/left) [<i>n</i> (%)]	13 (48.15)/14 (51.85)	12 (75.00)/4 (25.00)	17 (56.67)/13 (43.33)	$\chi^2=2.981$	0.225
Duration of complain (mean±SD) (years)	2.31±1.76	1.17±0.65	NA	$Z=-2.193$	0.028*
MCN PL (ms)	4.65±1.08 ^{†‡}	2.25±0.34	2.31±0.23	$K=37.776$	<0.0001*
MCN CV (m/s)	25.69±6.75 ^{†‡}	59.66±11.25	57.36±12.31	$K=37.839$	<0.0001*
MCN SNAP amplitude (μV)	3.32±1.92 ^{†‡}	6.35±2.68	6.39±2.16	$K=18.745$	<0.0001*

CV, conduction velocity; Group I, patients with medial calcaneal neuropathy; group II, patients without medial calcaneal neuropathy; *K*, value of Kruskal–Wallis test; MCN, medial calcaneal nerve; *n*, number of heels; PL, peak latency; SD, standard deviation; SNAP, sensory nerve action potential; χ^2 , value of chi-square test; *Z*, value of Mann–Whitney test. [†]Significant difference (Mann–Whitney test) between group I and control group regarding MCN PL, MCN CV, and MCN SNAP amplitude ($P<0.05$). [‡]Significant difference (Mann–Whitney test) between group I and group II regarding MCN PL, MCN CV, and MCN SNAP amplitude ($P<0.05$). * $P<0.05$, significant.

amplitude of MCN SNAP, as well as, among the two patient groups, and among group I (patients with medial calcaneal neuropathy) and control group. Regarding the disease duration of chronic plantar heel pain, it was significantly longer among group I patients when compared with group II patients (Table 3).

There was a statistical significant negative correlation between the MCN SNAP amplitude and duration of complaints ($r=-0.435$, $P=0.013$). Otherwise, there was no statistically significant correlation between the MCN SNAP parameters and age ($P>0.05$).

Table 4 illustrates the different final diagnosis of all patients in the study. Among the patient group with medial calcaneal neuropathy (group I), medial calcaneal neuropathy was the solitary cause of chronic plantar heel pain in 10 (37.03%) heels obtained from 10 (43.48%) patients. However, it was associated with other local heel pathologies in the remaining patients. The majority of them were plantar fasciitis in nine (33.34%) heels from five (21.73%) patients.

Discussion

A potential etiology for chronic plantar heel pain is entrapment of MCN under the flexor retinaculum or compressed behind the medial malleolus [3,21,22]. However, it is a frequently under-recognized cause of heel pain. The medial calcaneal neuropathy produces symptoms that are frequently subjective because it is a pure sensory nerve. Subsequently, it is usually missed. It was postulated that the entrapment of the MCN is a rare condition [8].

In the current study, medial calcaneal neuropathy was detected in 27 (62.79%) heels obtained from 23

(60.52%) patients. It was the solitary diagnosis for chronic plantar heel pain in 10 (37.03%) patients. Its high prevalence is an indicator of a considerable health problem. Unfortunately, it is usually missed in patients with chronic plantar heel pain. This condition is usually overlooked because the more obvious local musculoskeletal disorders and injuries draw the physician's attention. This could be due to the similarity of its symptoms with other causes of plantar heel pain. Its clinical signs are usually missed and unreliable. In addition, the electrophysiological assessment of MCN is not used routinely [13].

It was reported that MCN is affected in about two-thirds of TTS patients. This is because it arises proximal to the tarsal tunnel and descends superficial to the flexor retinaculum [3]. However, it was postulated that isolated medial calcaneal neuropathy is rare [3]. A study reported 11 heels from 10 patients with chronic heel pain secondary to entrapment of the MCN. Surgical decompression was done with improvement in 10 (90.9%) heels [23]. Another study reported one (4%) patient with solitary medial calcaneal neuropathy among 25 patients with TTS [4]. The present study is not in agreement with this study due to differences in the studied patients. Patients with TTS affecting the medial and/or lateral plantar nerves were excluded in the current study. Seo *et al.* [24] reported four cases with only medial calcaneal neuropathy as an illustration for the efficacy of an electrophysiological technique for assessment of MCN. Cheong *et al.* [25] presented a single case report of medial calcaneal neuropathy as a cause of intractable heel pain.

In the current study, the duration of chronic plantar heel pain was significantly longer among the patient group with medial calcaneal neuropathy. This could be due to the delay in the diagnosis of medial calcaneal

Table 4 Diagnosis of patients with chronic plantar heel pain in the two patient groups

Etiologies of chronic plantar heel pain	Group I (n=27 heel from 23 patients) [n(%)]	Group II (n=16 heel from 15 patients) [n(%)]
Plantar fasciitis	9 (33.34)*	12 (75.00)
Heel pad syndrome (atrophy of heel fat pad)	5 (18.52)	1 (6.25)
Post plaster stiffness and edema after healing of calcaneal fracture	3 (11.11)	1 (6.25)
Plantar enthesopathy due to seronegative spondyloarthropathy	0 (0)	2 (12.5)†
Medial calcaneal neuropathy as the solitary diagnosis	10 (37.03)	NA
Etiology of medial calcaneal neuropathy		
Idiopathic	8 (29.63)	NA
Secondary to lower limb varicosity	1 (3.70)	NA
Secondary to a lipoma	1 (3.70)	NA

Group I, patients with medial calcaneal neuropathy; group II, patients without medial calcaneal neuropathy; NA, not applicable. *Nine (33.34%) heels with plantar fasciitis were present in five (21.73%) patients from which four patients had bilateral affection. †Two (12.5%) heels with plantar enthesopathy due to seronegative spondyloarthropathy were present in one (6.67%) patient.

neuropathy. Making the patient receive inappropriate medical therapy which is not effective or has only a placebo effect with slight improvement of the condition then recurrence after stopping the therapy [13].

There are many etiologies that lead to medial calcaneal neuropathy. These include compression of the nerve by hypertrophic flexor retinaculum, tendonopathy, any space occupying lesions as ganglion, lipoma, neuroma, and lower limb varicosities [5,26]. Moreover, these include foot deformity as hind foot valgus deformity, prolonged weight bearing periods as walking and standing, lower limb edema of any cause, obesity, post surgical scarring and inflammatory arthropathies as rheumatoid arthritis [5,26]. It may be caused by chronic microtrauma associated with repetitive biomechanical overload. The heel fat pad acts as a natural shock absorber for the body. This is deficient in case of heel pad syndrome due to atrophy of heel fat pad. Because the branches of the MCN usually lie superficial to the intrinsic foot muscles and plantar fascia, subsequently, they are likely to be traumatized and irritated following heel fat pad atrophy [13,27]. Repetitive trauma to the heel can lead to extraneural, as well as, intraneural fibrosis involving the MCN [28]. It could be affected by direct trauma to the heel which lead to contusion and the bleeding is healed by adhesions and perineural fibrosis [5].

In the present study, medial calcaneal neuropathy was associated with plantar fasciitis in nine (33.34%) heels. The current study is in agreement with Rose *et al.* [12] and Chang *et al.* [29]. Rose *et al.* [12] reported that solitary medial calcaneal neuropathy was associated with plantar fasciitis in 22.68% of their patients, whereas 49.48% of their patients had medial calcaneal neuropathy in association with medial plantar neuropathy [12]. Chang *et al.* [29] reported that medial calcaneal neuropathy was associated with plantar fasciitis in six (23.1%) patients with unilateral plantar fasciitis. This could be because the etiology of both conditions; i.e. medial calcaneal neuropathy and plantar fasciitis; could be the same which is excessive load that could be the result of prolonged standing and walking with excessive activity [10].

The current study is in agreement with Park and Toro [7], who assessed sensory nerve conduction study of the MCN by using an antidromic technique, regarding PL and amplitude of the MCN SNAP. However, it does not agree with them regarding the SNAP CV which was much slower in the current study. This could be

due to differences in the study population. Park and Toro [7] studied participants who were younger than the control group of the current study (mean age: 30 years; range: 22–45 years).

Chang *et al.* [29] estimated the lowest normal MCN CV to be 34.3 m/s and the lowest normal SNAP amplitude to be 6.1 μ V. The current study is in agreement with Chang *et al.* [29] regarding the lowest normal MCN CV. However, it is not in agreement with them regarding the SNAP amplitude. This could be due to difference in the study population who were older than those of the current study (mean age: 50.3 \pm 5.3 years), as well as, the difference in the techniques of MCN recording between the two studies [29].

The current study had few limitations. First, the relatively larger number of female patients included in the current study. This could be because men seek medical advice only in late stages of their illness; they are usually seeking medical advice in the health insurance services related to their employment; and also they are working in the morning and tend to seek medical advice in the private clinics in the evening and at night. Second, the current study was conducted in the Physical Medicine, Rheumatology and Rehabilitation outpatient clinic and did not include other clinics where patients with chronic plantar heel pain could attend as Orthopedic Surgery outpatient clinic and Internal Medicine (Rheumatology subunit) outpatient clinic.

In conclusion, medial calcaneal neuropathy is present in a considerable number of patients with chronic plantar heel pain. It should be taken into consideration during the assessment of any patient with chronic plantar heel pain.

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Conflicts of interest

There are no conflicts of interest.

References

- 1 Scher DL, Belmont PJ, Bear R, Mountcastle SB, Orr JD, Owens BD. The incidence of plantar fasciitis in the United States military. *J Bone Joint Surg* 2009; 91:2867–2872.
- 2 Oztuna V, Ozge A, Eskandari MM, Colak M, Golpınar A, Kuyurtar F. Nerve entrapment in painful heel syndrome. *Foot Ankle Int* 2002; 23:208–211.
- 3 Oh J. Neuropathies of the foot. *Clin Neurophysiol* 2007; 118:954–980.
- 4 Oh SJ, Kim HS, Ahmad BK. The near-nerve sensory nerve conduction in tarsal tunnel syndrome. *J Neurol Neurosurg Psychiatry* 1985; 48: 999–1003.

- 5 Ahmad M, Tsang K, Mackenney PJ, Adedapo AO. Tarsal tunnel syndrome: a literature review. *Foot Ankle Surg* 2012; 18:149–152.
- 6 De Prado M, Cuervas-Mons M, Golanó P, Rabat E, Vaquero J. The tarsal tunnel syndrome. *Fuß and Sprunggelenk* 2015; 13:227–236.
- 7 Park T, Toro D. The medial calcaneal nerve: anatomy and nerve conduction technique. *Muscle Nerve* 1995; 18:32–38.
- 8 Louisia S, Masquelet AC. The medial and inferior calcaneal nerves: an anatomic study. *Surg Radiol Anat* 1999; 21:169–173.
- 9 Govsa F, Bilge O, Ozer MA. Variations in the origin of the medial and inferior calcaneal nerves. *Arch Orthop Trauma Surg* 2006; 126:6–14.
- 10 Rose B, Singh D. Inferior heel pain. *Orthop Trauma* 2016; 30:18–23.
- 11 Johnston MR. Nerve entrapment causing heel pain. *Clin Podiatr Surg* 1994; 11:617–624.
- 12 Rose JD, Malay DS, Sorrento DL. Neurosensory testing of the medial calcaneal and medial plantar nerves in patients with plantar heel pain. *J Foot Ankle Surg* 2003; 42:173–177.
- 13 Alshami A, Souvlis T, Coppieters M. A review of planter heel pain of neural origin: differential diagnosis and management. *Man Ther* 2008; 13: 103–111.
- 14 Hossain M, Makwana N. 'Not plantar fasciitis': the differential diagnosis and management of heel pain syndrome. *Orthop Trauma* 2011; 25:198–206.
- 15 Juliano PJ, Harris TG. Plantar fasciitis, entrapment neuropathies, and tarsal tunnel syndrome: current up to date treatment. *Curr Opin Orthop* 2004; 15:49–54.
- 16 Stroud CC. Heel pain, plantar fasciitis, and tarsal tunnel syndrome. *Curr Opin Orthop* 2002; 13:89–92.
- 17 Saba EKA, El-Sherif SM. Ultrasound-guided versus palpation-guided local corticosteroid injection therapy for treatment of plantar fasciitis. *The Egyptian Rheumatologist* 2016; 38:123–131.
- 18 Tu P, Bytowski JR. Diagnosis of heel pain. *Am Fam Physician* 2011; 84:909–916.
- 19 Preston DC, Shapiro BE, editors. *Electromyography and neuromuscular disorders: clinical-electrophysiologic correlations*. 3rd ed. London: Elsevier; 2013.
- 20 Statistical package of social science. *Statistical package of social science Version 17, Documentation*. London: University of Cambridge computing service; 2007.
- 21 Beskin JL. Nerve entrapment syndromes of the foot and ankle. *J Am Acad Orthop Surg* 1997; 5:261–269.
- 22 Llanos LF, Vila J, Nunez-Samper M. Clinical symptoms and treatment of the foot and ankle nerve entrapment syndromes. *Foot Ankle Surg* 1999; 5:211–218.
- 23 Henricson AS, Westlin NE. Chronic calcaneal pain in athletes: entrapment of the calcaneal nerve?. *Am J Sports Med* 1984; 12:152–154.
- 24 Seo JH, Oh SJ. Near-nerve needle sensory conduction study of the medial calcaneal nerve: new method and report of four cases of medial calcaneal neuropathy. *Muscle Nerve* 2002; 26:654–658.
- 25 Cheong IY, Kim KH, Park BK, Kim DH. Medial calcaneal neuropathy as a cause of intractable heel pain. *Am J Phys Med Rehabil* 2016; 95:e62.
- 26 McSweeney SC, Cichero M. Tarsal tunnel syndrome: a narrative literature review. *Foot* 2015; 25:244–250.
- 27 Davidson MR, Copoloff JA. Neuromas of the heel. *Clin Pod Med Surg* 1990; 7:271–288.
- 28 Plotkin D, Patel D, Roberto T. Heel neuroma: a case study. *J Foot Ankle Surg* 2009; 48:376–379.
- 29 Chang CW, Wang YC, Hou WH, Lee XX, Chang KF. Medial calcaneal neuropathy is associated with plantar fasciitis. *Clin Neurophysiol* 2007; 118: 119–123.