EVOKED POTENTIALS ELICITED BY STIMULATION OF THE LATERAL AND ANTERIOR FEMORAL CUTANEOUS NERVES IN MERALGIA PARESTHETICA

DENNIS J. CORDATO, PhD, FRACP, CON YIANNIKAS, FRACP, JILL STROUD, JEAN-PIERRE HALPERN, PhD, FRACP, RAYMOND S. SCHWARTZ, FRACP, MEHMET AKBUNAR, and MELISSA COOK

Department of Neurology, Concord Repatriation Hospital, Hospital Rd, Concord, New South Wales 2139, Australia

Accepted 20 August 2003

The lateral femoral cutaneous nerve (LFCN) is a pure sensory nerve that originates from the lumbar plexus. Mononeuropathy of the LFCN was first described by Bernhardt in 1878.2 The term “meralgia paresthetica,” derived from the Greek “meros” (thigh) and “algia” (pain), was coined by Roth in 1895.10 The condition is characterized by pain or dysesthesia in the lateral thigh in the distribution of the LFCN.1,3,5–9,11–16 It is mostly unilateral, but 8–12% of patients experience bilateral symptoms.6

Electrophysiological studies performed to confirm the diagnosis of an LFCN lesion include LFCN conduction studies and somatosensory evoked potentials (SEPs) recorded from the scalp. The most appropriate electrodiagnostic test has been debated, with some authors favoring evoked potentials because of the false-positive results using LFCN conduction studies4 and others favoring LFCN conduction studies because of the false-negative results using evoked potentials.7,12 The aims of this study were to: (1) determine the spectrum of LFCN-derived evoked potential abnormalities in patients with clinical symptoms and signs of meralgia paresthetica, and (2) investigate the usefulness of eliciting SEPs by stimulation of both the lateral femoral cutaneous and anterior femoral cutaneous nerves of the thigh (representing two different skin areas with a common L2 distribution) in distinguishing meralgia paresthetica from a radiculopathy or plexopathy. For the purposes of this study, meralgia paresthetica was defined as a lesion of the LFCN (with the exclusion of proximal lumbar or paravertebral involvement).

SUBJECTS AND METHODS

Eighty consecutive patients referred to the Burwood Neurology Clinic in Sydney, Australia, between 1994 and 1998, for electrodiagnostic evaluation of clinical symptoms and signs consistent with a diagnosis of meralgia paresthetica underwent bilateral studies of SEPs elicited by stimulation of the lateral and anterior femoral cutaneous nerves of the thigh. All patients presented with sensory discomfort in the anterolateral portion of one or both thighs and had clinically altered sensation in the lateral aspect of
thigh in the distribution of the LFCN, with normal muscle strength and retained deep tendon reflexes. Five patients were excluded because of the presence of a proximal lumbar radiculopathy (four patients) and coexisting cervical myelopathy (one patient). Sixty-five patients (37 right- and 28 left-sided) with a clinical diagnosis of meralgia paresthetica presented with unilateral symptoms, whereas 10 patients had bilateral symptoms. All patients underwent lumbar computerized tomography or magnetic resonance imaging that excluded L2 or L3 root pathology. Electromyography of muscles innervated by L2, L3, and L4 (including iliopectos, adductor longus, quadriceps femoris, and tibialis anterior) was similarly normal in the 75 patients (53 men and 22 women) with meralgia paresthetica. Their mean age was 51 years (SD, 13; range, 23–75 years), mean height was 170.5 cm (SD, 9.6; range, 145–186 cm), and mean duration of symptoms was 21 months (SD, 37; 95% confidence intervals or CI 13–30 months; range, 1–276 months).

Five patients presented with clinical symptoms suggestive of meralgia paresthetica but were found to have different pathologies, including two patients with an L2 lumbar radiculopathy due to degenerative disease (in whom lateral and anterior femoral cutaneous SEPs were prolonged on the symptomatic side) and two patients with diabetes mellitus and a proximal radiculopathy (in whom bilateral lateral and anterior femoral cutaneous SEPs were prolonged). Electromyography of muscles innervated by the L2 and L3 segments in the four patients showed no active denervation, but there was evidence of polyphasic units of increased duration and a reduced recruitment pattern of motor units, consistent with a radiculopathy. One patient, in whom SEPs of the bilateral anterior femoral and lateral femoral cutaneous thigh were also prolonged, was found to have coexisting degenerative disease of the cervical spine.

Twenty control subjects (10 men and 10 women) with no prior history of a neurological condition and a normal neurological examination underwent lateral and anterior femoral cutaneous SEP studies of both thighs for the purpose of providing normative data. Their mean age was 48 years (SD, 17; range, 23–73 years) and mean height was 166.5 cm (SD, 9.9; range, 145–182 cm). All control subjects provided informed consent to participate.

Recordings for both control subjects and patients were performed in a quiet room with subjects supine in a semi-reclining chair. Skin temperature was > 30°C in all subjects. Two gold electroencephalographic surface electrodes (10-mm cup, 1.5-meter cable; Nicolet, Madison, Wisconsin) were placed with the cathode 3 cm proximal to the anode over the center of the skin area innervated by the LFCN (at a point halfway between the iliac crest and the superior aspect of the patella) and on the medial aspect of the inner thigh (at a parallel position in the skin area innervated by the medial division of the anterior femoral cutaneous nerve of the thigh). The skin at the recording and stimulating sites was prepared with an abrasive skin prepping gel (Omni-Prep, Weaver & Co., Aurora, Colorado) using cotton-tip applicators and Eleflex paste (Nihon Kohden, Tokyo, Japan) was applied to the cups of both stimulating and recording electrodes. Stimuli were delivered sequentially to the lateral and anterior femoral cutaneous regions of both thighs at 2.5 times the sensory threshold at a rate of 2.41 Hz. Two-channel SEPs were recorded from the scalp also using gold electroencephalographic surface electrodes (Nicolet), with the active electrode for channel 1 placed 2 cm posterior to Cz and for channel 2 placed 1 cm ipsilateral to Cz (to the right for right thigh stimulation and to the left for left thigh stimulation), with Fz as reference point. The latency to the first positive peak (P37) and amplitude from P37 to second negative peak (N45) were measured. Mean values, standard deviations, 95% confidence intervals, and ranges of SEP latencies and amplitudes elicited by stimulation of skin areas innervated by the right and left LFCNs anterior femoral cutaneous nerves of the thigh, as well as side-to-side comparisons, are summarized in Table 1.

RESULTS

Control Subjects. In all control subjects, absolute latencies of SEPs to LFCN stimulation were ≤ 40 ms and amplitudes > 50% of contralaterally elicited SEPs. Amplitudes of lateral femoral cutaneous SEPs were also > 50% of SEPs elicited by ipsilateral anterior femoral cutaneous nerve stimulation in all 20 control subjects. The absolute latency difference between right and left femoral cutaneous SEPs was ≤ 5 ms in all subjects. The latency difference between ipsilateral and anterior femoral cutaneous SEPs was ≤ 5 ms in 19 of the 20 control subjects. The upper limits of the range of values of lateral femoral cutaneous SEPs in the controls were used for the evaluation of patient results. A normal result satisfied the following three criteria: (1) absolute latency of lateral femoral cutaneous SEP ≤ 40 ms, (2) absolute latency difference between symptomatic and asymptomatic lateral femoral cutaneous SEPs ≤ 5 ms, and (3) amplitude of symptomatic
lateral femoral cutaneous SEP > 50% of contralateral response. Hence, abnormalities of SEPs derived from the LFCN were divided into the following categories: (1) absent SEP; (2) absolute latency > 40 ms; (3) SEP latency ≤ 40 ms but > 5 ms interside latency difference; and (4) SEP latency ≤ 40 ms and ≤ 5 ms interside latency difference but amplitude ≤ 50% of contralateral response.

**Patients with Meralgia Paresthetica.** Thirty-five patients (47%) with meralgia paresthetica had an absolute SEP latency to LFCN stimulation of >40 ms. The SEP amplitude was <50% of the contralateral response in 15 of the 35 patients. Fourteen patients (19%) had an absent response; 8 patients (11%) had an SEP latency ≤ 40 ms and ≤5 ms interside latency difference but amplitude ≤ 50% of contralateral response; and five patients (7%) had an SEP latency ≤ 40 ms but >5 ms interside latency difference. The false-negative rate for lateral femoral cutaneous SEPs in patients with a clinical diagnosis of meralgia paresthetica was 17% (13 patients). The inclusion of SEP latencies and amplitudes following stimulation of the anterior femoral cutaneous nerve of thigh (lateral femoral cutaneous SEP latency > 5 ms difference to ipsilateral anterior femoral cutaneous nerve) identified an additional two patients whose results would otherwise have been deemed normal. Of the 10 patients with bilateral symptoms, nine had lateral femoral cutaneous SEPs that were abnormal bilaterally, with the response either absent or >40 ms in latency. In one patient with bilateral symptoms, the lateral femoral cutaneous SEP latency on the less symptomatic side was <40 ms but latency and amplitude were >5 ms and <50%, respectively, when compared with the ipsilateral anterior femoral cutaneous SEP. Figure 1 shows the lateral femoral and anterior femoral cutaneous SEP responses of a patient with unilateral meralgia paresthetica.

**DISCUSSION**

The utility of lateral femoral cutaneous SEPs in the diagnosis of meralgia paresthetica has been subject to debate, with some authors reporting a relatively low incidence of abnormal findings and others re-

### Table 1. The SEPs elicited by stimulation of the lateral femoral and anterior femoral cutaneous nerves of the thigh: latencies and amplitudes in 20 control subjects.

<table>
<thead>
<tr>
<th></th>
<th>Lateral femoral cutaneous SEP</th>
<th></th>
<th>Anterior femoral cutaneous SEP</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Latency (ms)</td>
<td>Interside latency difference (ms)</td>
<td>Amplitude (μV)</td>
</tr>
<tr>
<td>Mean</td>
<td>35.0</td>
<td>1.84</td>
<td>1.47</td>
</tr>
<tr>
<td>SD</td>
<td>2.84</td>
<td>1.45</td>
<td>0.95</td>
</tr>
<tr>
<td>95% CI</td>
<td>34.1–35.9</td>
<td>1.1–2.5</td>
<td>1.2–1.8</td>
</tr>
<tr>
<td>Range</td>
<td>30.6–40</td>
<td>0.1–4.7</td>
<td>0.3–5</td>
</tr>
</tbody>
</table>

Latency and amplitude mean values, SD, and 95% CI include right and left sides (n = 40).

*Latency difference between lateral femoral and anterior femoral cutaneous SEPs.

![Figure 1](image-url)
porting a high incidence of abnormalities in clinically symptomatic patients. For example, Wiezer et al.\textsuperscript{15} found unilaterally absent (10 patients) or delayed responses (8 patients) among 24 patients with unilateral symptoms of meralgia paresthetica. Three patients (12.5\%) with clinically symptomatic meralgia paresthetica had normal findings, which is comparable to the false-negative rate in our study. In contrast, Seror\textsuperscript{12} reported a low incidence of abnormality (8 of 30 patients) using lateral femoral cutaneous SEPs, whereas the same 30 patients were all found to have abnormal sensory nerve conduction study findings. Lagueny et al.\textsuperscript{7} similarly found a low incidence of SEP abnormalities (abnormal interside latency SEP differences in only 4 of 19 patients); if the authors had included a >50\% amplitude reduction compared with the contralateral side as an electrodiagnostic criterion, an additional six patients would have been identified as having abnormal results.

An LFCN conduction study is also used as an electrodiagnostic test in meralgia paresthetica. However, its diagnostic utility is limited by the presence of false-positive findings. For example, in the study by Lagueny et al.,\textsuperscript{7} three control subjects (15\%) were excluded because of the absence of sensory nerve action potentials. Low-amplitude sensory responses were also found in some of the retained controls. Furthermore, the methodology required relocation of the stimulating cathode six or seven times in some patients (possibly due to anatomical variations of the stimulating cathode six or seven times in some patients) and a change in the type of recording needle in others, especially in obese patients, to obtain the most reliable result. We acknowledge that anatomical variations in superficial sensory nerve and dermatomal distributions also may have occurred in our patients and controls.

Anterior femoral cutaneous thigh SEPs marginally improved the diagnostic utility of evoked potential studies in our study by identifying a significant latency difference between SEPs elicited by lateral femoral and anterior femoral cutaneous stimulations in two patients whose results were deemed to be otherwise normal. Anterior femoral cutaneous thigh SEPs were also of value in confirming the diagnosis of meralgia paresthetica in patients with bilateral symptoms and helped exclude the diagnosis of meralgia paresthetica in four patients with proximal L2 pathology. Electromyography was also useful in confirming proximal L2 pathology in this setting and hence should be performed.

In conclusion, lateral femoral cutaneous SEPs are a useful diagnostic tool in meralgia paresthetica. A comparison of anterior femoral and lateral cutaneous thigh SEPs is of value in making a diagnosis of meralgia paresthetica when the disorder is bilateral as well as in distinguishing meralgia paresthetica from a proximal lumbar radiculopathy or plexopathy.

REFERENCES