

ABSTRACT: We present three patients with signs and symptoms of meralgia paresthetica (MP) after long-distance walking and cycling. No other possible causes of MP, such as trauma or exogenous compression, were present. A neuropathy of the lateral femoral cutaneous nerve was confirmed in all patients with somatosensory evoked potentials. We propose that conduction block due to local ischemia during repetitive muscle stretching was the probable cause for the neuropathy.

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MERALGIA PARESTHETICA AFTER STRENUOUS EXERCISE

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Meralgia paresthetica (MP) is a sensory mononeuropathy of the lateral femoral cutaneous nerve (LFCN) that is characterized by abnormal sensation over the lateral thigh.⁸ It is often idiopathic. Among the known causes are those associated with weight changes or trauma in the pelvic or lower spine area, and iatrogenic complications of obstetric or orthopedic surgery.^{3,7} Herein we report three healthy, nonobese patients with MP after strenuous exercise.

CASE REPORTS

Patient 1 (male, 25 years old, body mass index of 19.6) walked the annual Nijmegen Four Days Marches, which consist of 4 consecutive days of a 50-km walk (a total of 200 km). He did not train for this event, and wore loose sports clothing. Symptoms started with unilateral numbness in the right anterolateral aspect of the thigh after the third day of marches. His symptoms continued for about a week, and were followed by gradual sensory recovery accompanied by paresthesias in the same area. There were no motor symptoms. Clinical examination at the time of the neurological deficit revealed loss of sensation on the anterolateral side of the right thigh,

consistent with the sensory area supplied by LFCN. No other signs were found. On previous participations in the walking event, the patient had developed similar complaints of anesthesia/paresthesia but did not seek medical attention. Somatosensory evoked potentials (SSEPs) were recorded using an established protocol.¹¹ The skin areas of the LFCN and the ilioinguinal nerve, as well as the dermatomal areas of L-3 and L-4, were stimulated on both sides, using surface electrodes placed in the center of each specific region. The SSEP of the ipsilateral LFCN showed a prolonged latency and a reduced amplitude as compared with the contralateral side. The ilioinguinal SSEPs were of normal latencies and amplitudes (Fig. 1A and B).

Patient 2 (male, 52 years old, body mass index of 24.2) also participated in the Four Days Marches. This patient had no relevant medical history and trained for the walking event, which he undertook for the first time. He developed symptoms of numbness and paresthesias on the lateral side of his right leg during the event. There were no motor symptoms. He consulted us a few weeks later. The diagnosis of MP was considered, with a sensory deficit being found in the LFCN area, without further abnormalities on neurological examination. No SSEPs could be recorded after stimulation of the right LFCN, whereas normal responses were found on the left side. Recovery was gradual but complete within a few months.

Patient 3 (male, 49 years old, body mass index of 23.2) had climbed several mountain passes in the Pyrenees on a bicycle. He had trained for bicycling

Abbreviations: LFCN, lateral femoral cutaneous nerve; MP, meralgia paresthetica; SSEP, somatosensory evoked potential

Key words: lateral femoral cutaneous nerve; marching; meralgia paresthetica; sensory evoked potentials

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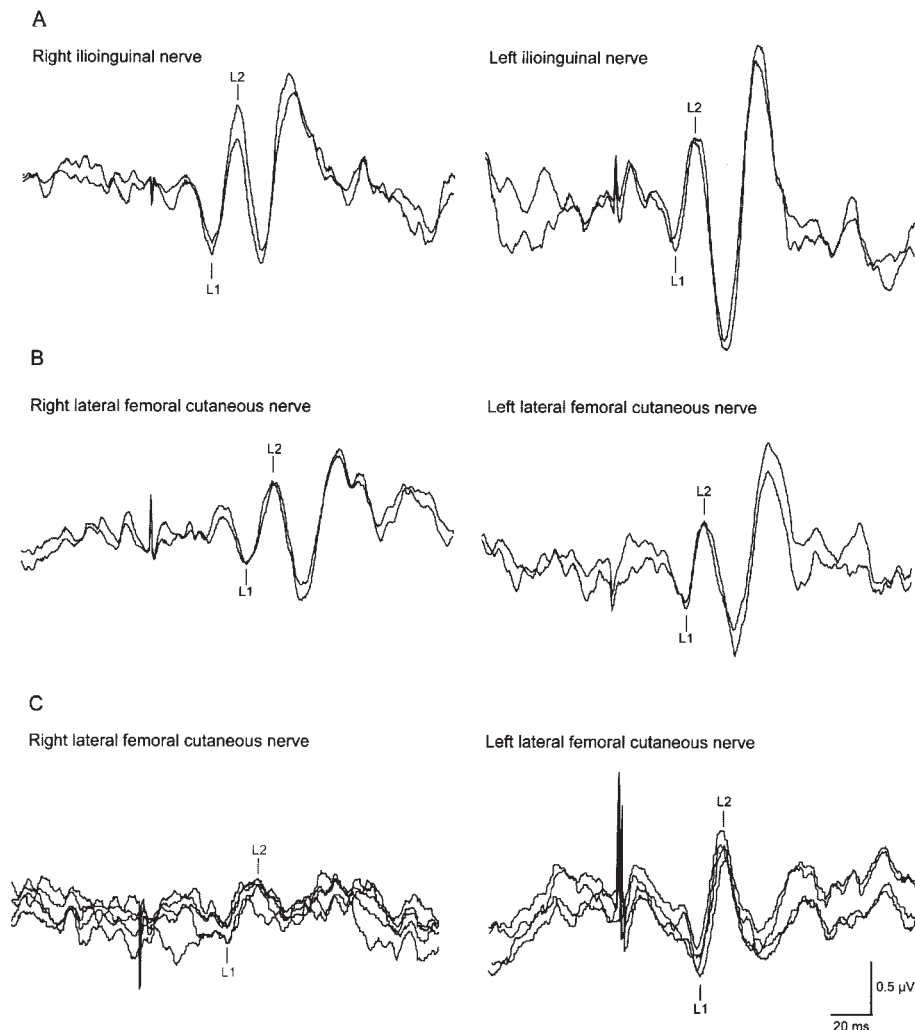


FIGURE 1. (A) Normal SSEPs of the left and right ilioinguinal nerve of patient 1. (B) SSEPs of the left and right lateral femoral cutaneous nerve of patient 1, showing a decreased amplitude (right: $0.44 \mu\text{V}$; left: $0.88 \mu\text{V}$) and a prolonged latency (right: 54.4 ms ; left: 34.4 ms) on the right side. (C) SSEPs of the left and right lateral cutaneous nerve of patient 3, showing an almost absent response on the right side.

in the mountains and had undertaken this activity several times previously. He had developed similar and reversible complaints of numbness on a previous occasion. He had not sought medical attention at the time, and reported complete recovery after several weeks. The present symptoms started after he had climbed two mountains, when he developed a painful sensation on the lateral side of the right thigh, lasting for about 1 week, followed by hypesthesia of the same region. On clinical examination, there was a sensory deficit in the LFCN area. No motor signs were present. An extensive electrodiagnostic study was performed, including nerve conduction studies of the femoral, peroneal, posterior tibial, and sural nerves, and needle electromyography of the muscles of the third, fourth, and fifth lumbar

root. These studies were all normal. The ipsilateral SSEP of the LFCN was abnormal, showing a markedly reduced amplitude with normal latency (Fig. 1C).

DISCUSSION

We report three cases of electrophysiologically confirmed neuropathy of the LFCN associated with strenuous exercise. Next to idiopathic causes of MP, known associations are with trauma (iatrogenic as well as non-iatrogenic), external compression (tight clothing,⁷ seatbelt), and weight change (pregnancy, possibly obesity). Patients with anatomical variations of the LFCN may be predisposed to develop this disorder.² Nerve entrapment syndromes associated

with sports are usually reported for the nerves of the arm, whereas case reports of involvement of leg nerves are sporadic.⁶ Among morbidity associated with long-distance road marching (e.g., in the army) or cycling are pain caused by abrasions and blisters on the feet and muscle soreness.^{4,5}

To our knowledge, there are no reports of MP resulting from strenuous walking or cycling. This may imply that it is a rare disorder. It may also be underreported, as patients may choose not to consult a physician because the symptoms generate a low level of distress and are reversible. Diagnosis also depends on knowledge of this entity.¹⁰

The pathophysiological mechanism of the damage to the nerve is probably multifactorial. Repetitive or continuous stretch of muscle may compress surrounding structures.¹ The iliopsoas muscle and the tensor fascia lata are heavily involved in both walking and cycling movements. This may result in compression of the LFCN in the inguinal canal, causing secondary local ischemia. Direct mechanical forces due to the compression may further contribute to the damage. Decreased blood flow to the nerve due to hypertrophic muscle may be another factor. The recurrence of the symptoms on the ipsilateral side in subjects 1 and 3 after the same kind of exercise suggests an anatomical predisposition.

The extent and the time course of recovery of our patients is highly suggestive of neurapraxia of the LFCN.⁹ A disruption of axonal continuity (axonotmesis or neurotmesis) would require more time for recovery. The SSEP findings of prolonged latency and reduced amplitude are concordant with a demyelinating lesion. They suggest a partial (patients 1 and 3) or complete (patient 2) conduction block of the LFCN. However, we have not performed measurements above and below the lesion to confirm a

true block. This is technically very difficult, either with evoked potentials or nerve conduction studies.

In conclusion, we have presented three cases of reversible but recurrent MP occurring after strenuous walking or cycling. Electrophysiological examination revealed LFCN neuropathy with probable conduction block, possibly due to local compression or ischemia.

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