

## Nerve Compression Secondary to Weight Loss

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### Abstract

**Background:** The objective of this review was to evaluate the clinical and electrophysiological findings of eight obese patients following a weight loss of more than 25 kg through diet, bariatric surgery or anorexia nervosa, coming out with peripheral nerves compression signs and symptoms.

**Methods:** Eight patients were studied; seven of them had walking difficulty, detecting clinical evidence of common fibular nerve injury, and another one shows unilateral ulnar nerve injury. Neurophysiological study was performed: Electromyogram (EMG). Electroneurogram (ENG): Motor and sensory conduction. Late F responses and H reflex.

**Results:** The common fibular nerve compression (seven patients) and ulnar compression (one patient) was confirmed by appropriate electrophysiological procedures. Widespread pathology is rejected.

**Conclusion:** In the peripheral nerve compression pathology there are multiple factors to consider and many neurophysiological procedures available to diagnose it. Excessive weight loss is an exceptional cause but it is essential to think about it as diagnostic, and the correct treatment will avoid an unnecessary surgical decompression.

**Keywords:** Nerve compression; Weight loss; Drop foot

### Introduction

The Peroneal mononeuropathy is the most common entrapment neuropathy in lower limbs. It is presented as a foot drop. Motor deficit for the extension of foot and toes, eversion deficit, sensory symptoms in autonomous territory and occasionally pain, are common complaints. The most common cause is external compression [1] and direct trauma; others causes are less frequent, such as the existence of tumor mass trapping peroneal tunnel, cysts, vascular etiology, endocrine, nutritional deficit, chronic infection, idiopathic, etc. [2,3].

Although rare, the relationship between peroneal paralysis and excessive weight loss has been well documented over the past two decades [4-10]. Different authors have made reference to a postural mechanism of maintained crossing legs [11-13]. Compressive neuropathy of the Ulnar Nerve in the epitrochlear channel is the second most frequent cause in upper limbs, but, to the best of our knowledge, there is no reference in relation to weight loss. Neurophysiological studies are needed for diagnosis, to identify the affected nerve, assess the severity of the injury and its prognosis [14-19].

### Objective

To characterize clinical and neurophysiological a small series of patients with obesity and weight loss of more than 25 kg secondary to diet, bariatric surgery and anorexia nervosa, who posteriorly develop symptoms and signs corresponding to a peripheral nerve involvement.

### Patients and Methods

Eight patients aged between 23 and 62 years (average 41.3) were studied (five women and three men). Seven had difficulty walking, presenting clinical symptoms (motor and sensory deficits, unilateral and/or bilateral) on the territory of common peroneal nerve. Another patient presented clinical symptoms corresponding to unilateral involvement of the ulnar nerve. Informed consent was obtained in all patients, before neurophysiological exams were performed.

In the Neurophysiological study, electromyogram (EMG) was carried out in dependent muscles of cited nerves. In common peroneal nerve, biceps femoris (caput brevis), tibialis anterior, lateral peroneal

and extensor digitorum brevis were studied. In the case of ulnar nerve, flexor carpi ulnaris, abductor digiti minimi and first dorsal interosseous were analyzed. In all muscles, activity at rest, at maximum effort and analysis of motor unit potentials (MUP) was valued. Electroneurogram (ENG) with segmental motor nerve conduction studies, late F responses and sensitive antidromic studies in the territories clinically involved (both sides) were performed to confirm the diagnosis. Moreover, sural nerves, radial nerves, and both bilateral H reflexes were studied to rule out any generalized polyneuropathy.

### Results

Eight patients were studied: five women and three men aged between 23 and 62 years (mean 41.3 years). Common peroneal nerve involvement in 7 patients (four bilateral, 57%; and three unilateral, 43%). The average weight loss was 40.6 kg and the average time of symptomatic onset was 8.8 months. Causes: 5 patients due to diet, 2 patients due to bariatric surgery, and 1 due to anorexia nervosa (Table 1).

The neurophysiological study (ENG) showed a compound muscle action potential (CMAP) with dispersed morphology and decreased amplitude, mainly in proximal segments; distal average latency was normal, and there was a slowing in motor conduction velocity at the level of fibular head (FH), in all of the seven patients, with varying degrees of intensity (from 36% to 64%) (Figure 1). In one patient, there was a slowing in motor conduction velocity at the level of the epitrochlear channel (ulnar nerve). There was also alteration on antidromic sensory conduction of Superficial Peroneal and Ulnar Nerves. On late F responses, there was absence and/or decreased of

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Nº	Age (years)	Gender	Motor and sensitive nerve. Laterality.	Weight	Time of weight loss	CAUSE
1	34	Female	Common Peroneal <b>Bilateral</b>	30 Kg	3 months	Diet
2	42	Male	Common Peroneal Unilateral	30 Kg	12 months	Diet
3	23	Female	Common Peroneal Unilateral	45 Kg	8 months	Diet
4	54	Female	Common Peroneal <b>Bilateral</b>	85 Kg	6 months	Bariatric surgery
5	32	Male	Common Peroneal Unilateral	30 Kg	12 months	Diet
6	33	Female	Common Peroneal <b>Bilateral</b> (clinical unilateral affection)	30 Kg	6 months	Anorexia
7	51	Male	Common Peroneal <b>Bilateral</b> (clinical unilateral affection)	25 Kg	12 months	Diet
8	62	Female	Ulnar Nerve. Unilateral	50 Kg	12 months	Bariatric surgery
<b>MEAN, % n</b>	<b>41.37</b>	<b>Female: 62.5% (n=5)</b> <b>Male: 37.5% (n=3)</b>	<b>Bilateral Affection: 57% (n=4)</b> <b>Unilateral Affection 43% (n=3).</b> <b>Peroneal nerve: 87.5% (n=7)</b> <b>Ulnar nerve 12.5% (n=1)</b>	<b>40.62 kg</b>	<b>8.87 months</b>	<b>Diet: 62.5% (n=5)</b> <b>Bariatric Surgery: 25% (n=2)</b> <b>Anorexia: 12.5% (n=1)</b>

Table 1: Demographic and clinical characteristics of the series analysed.

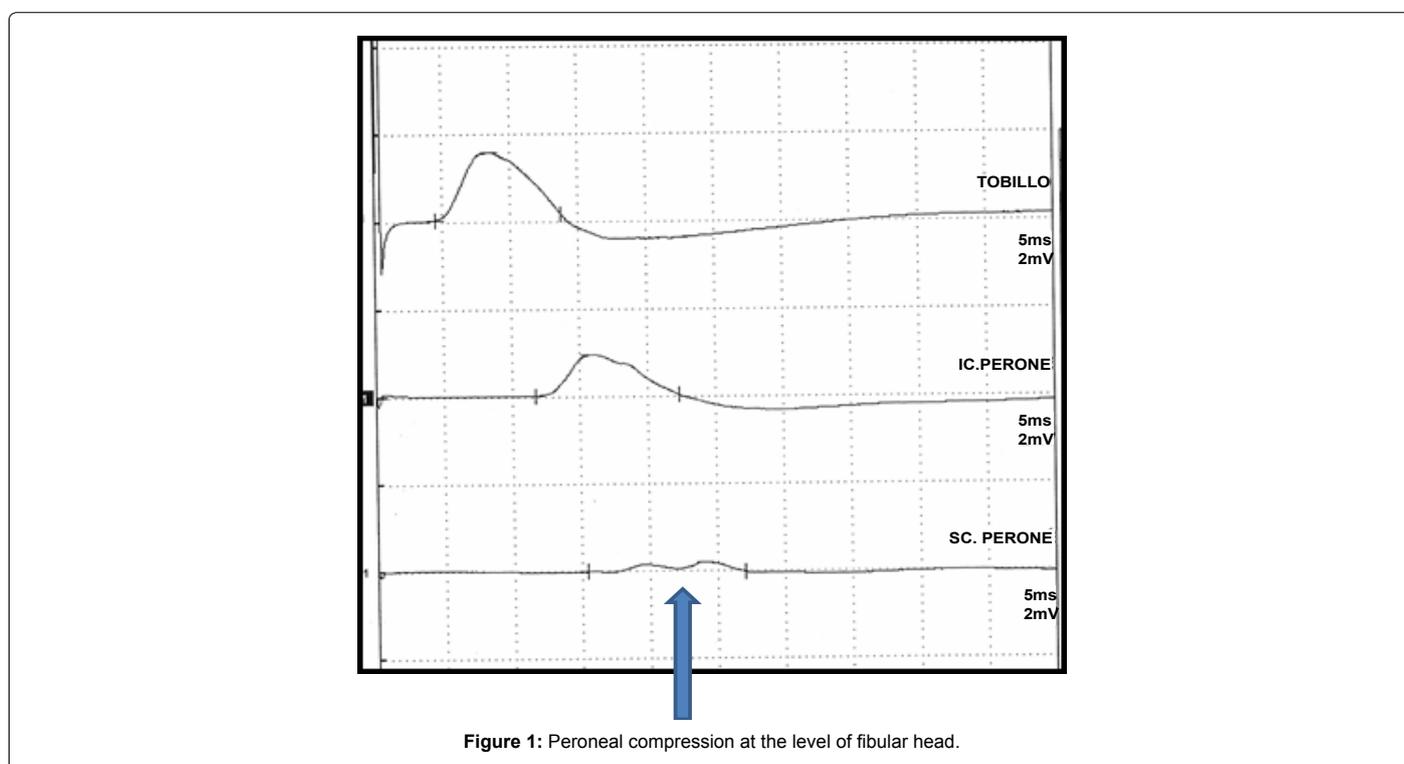


Figure 1: Peroneal compression at the level of fibular head.

responses with prolonged persistence of minimum latency (Figure 2). On EM, active denervation signs were observed, such as fibrillations and positive waves at rest (Figure 3) and a partial loss of motor units on muscles dependent from peroneal nerve, from tibialis anterior to distal muscles. The same pattern was observed in the case of ulnar neuropathy. MAP analysis showed characteristics of reinnervation in different chronological phases and to a high percentage (Figure 4). All patients were normal in sensitive conduction velocities both on sural and Radial nerves and in bilateral H reflexes [20] (Figure 5).

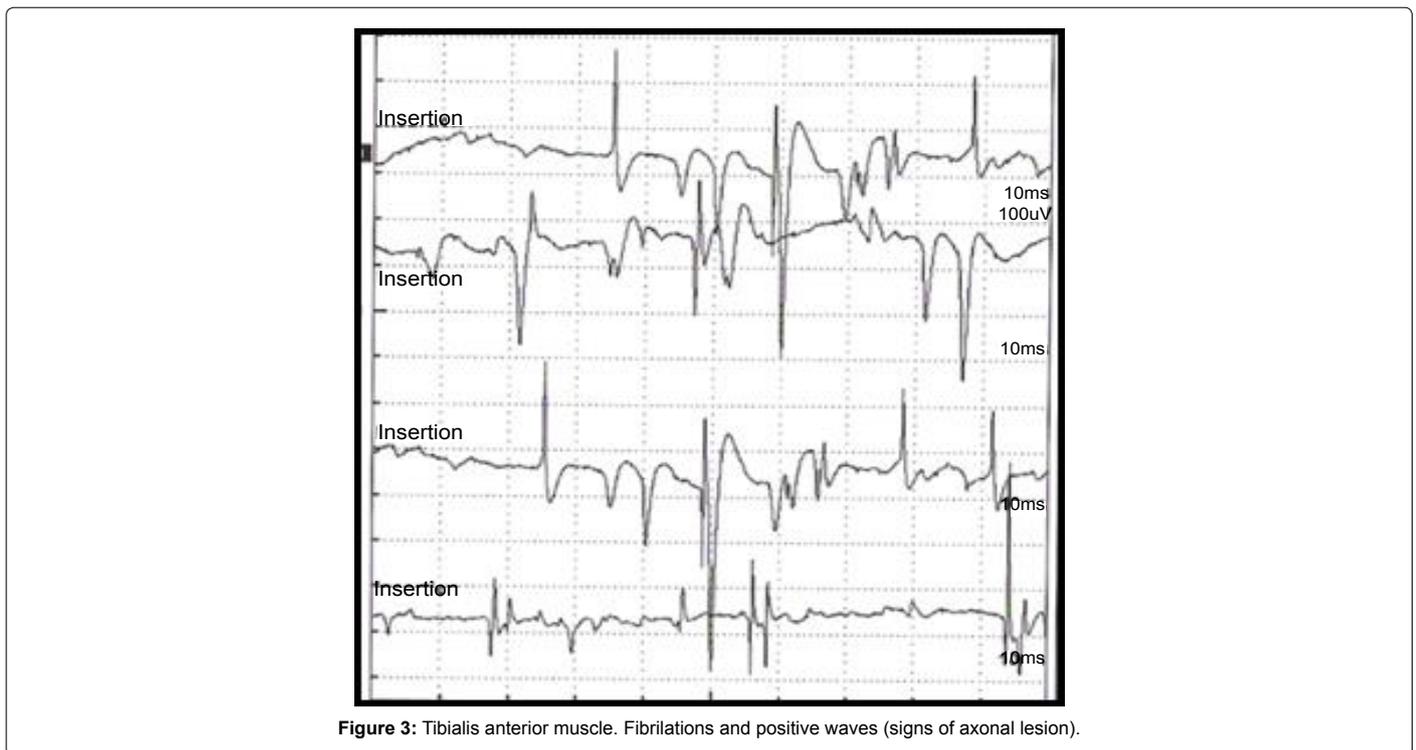
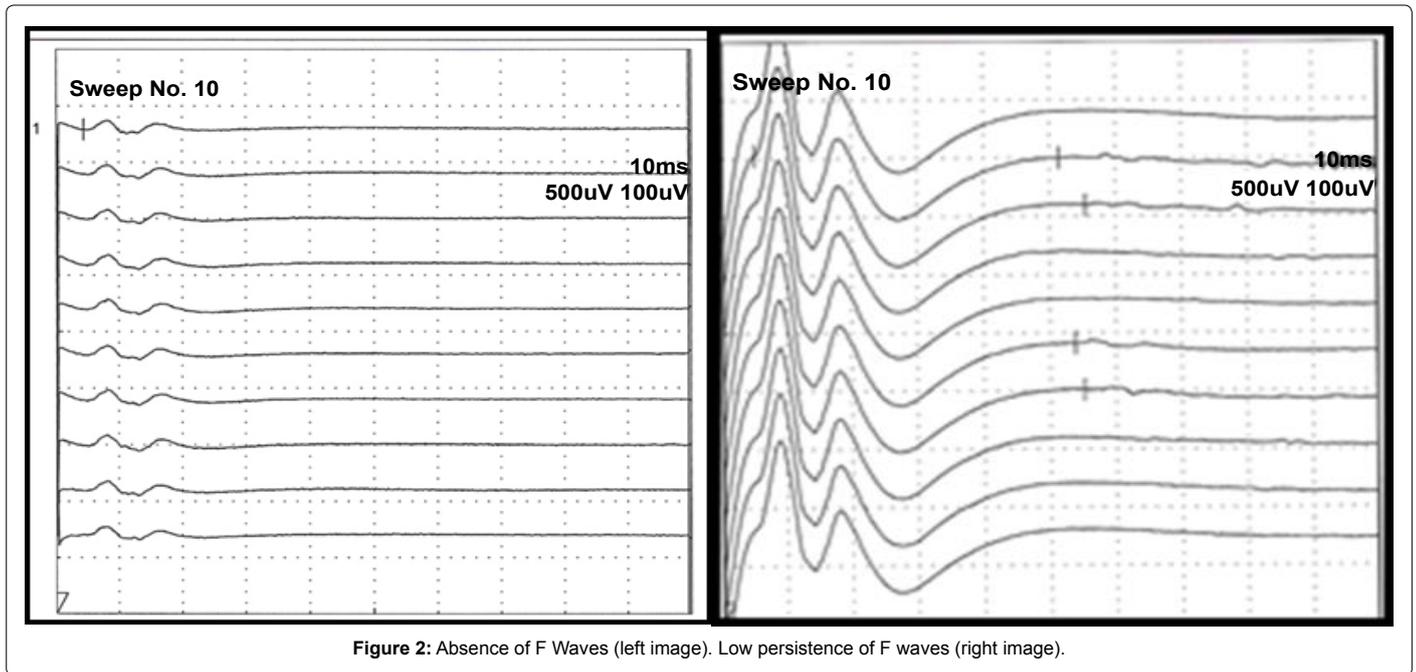
## Discussion

Localized neuropathy of common peroneal nerve at the level of the fibular head is the most common of all entrapment neuropathies in lower limbs. At ulnar nerve, the most common site of entrapment is the cubital tunnel, being the second in frequency on upper limb, after

carpal tunnel syndrome. The most common cause of neuropathy on both nerves is external compression and direct trauma; other causes may be the existence of mass, diabetes, vascular etiologies, idiopathic and so on. Currently, it must also be considered as possible causes of nerve compression weight loss in a short period of time by Diet, Weight Loss Surgery [21-23], nutritional deficiency and cancer [24-26], and anorexia nervosa [27-32].

In our series of cases, all patients referred progressive onset of symptoms. In seven patients (cases 1-7, Table 1) crossing legs as a habit was very common. In one patient (case 1, Table 1) intense physical exercise (dancing all night long) was followed by clinical bilateral drop foot afterwards. In case 8 (Table 1), maintained elbow bent over a hard table was a common posture and the probable cause of compression.

Regarding the etiology, as the literature states (including ours),



weight loss secondary to diet (5 cases) was the most frequent; followed by weight loss secondary to bariatric surgery (2 cases); and weight loss secondary to anorexia nervosa (one case).

The peroneal compression usually presents clinically as unilateral involvement, but the neurophysiological study confirms that in some patients it is bilateral; that is the reason why the study must be performed on both sides (two of our cases) in order to prevent clinical manifestation.

The mechanism of compression on these nerves is provided in both territories by the loss of muscle mass and the changes in the protective subcutaneous tissue, favored by persistence of forces over a common site of susceptible compression on related nerves (head of fibula for peroneal nerve and epitrochlear channel for ulnar nerve) enhanced by sustained postures. In the case of common peroneal nerve (located at the level of fibular head), the compression is enhanced by crossing legs [2,11-13,33-35]. In the case of the ulnar nerve, by localized pressure on the Epitrochlear channel (direct compression over the elbow on flexed position).

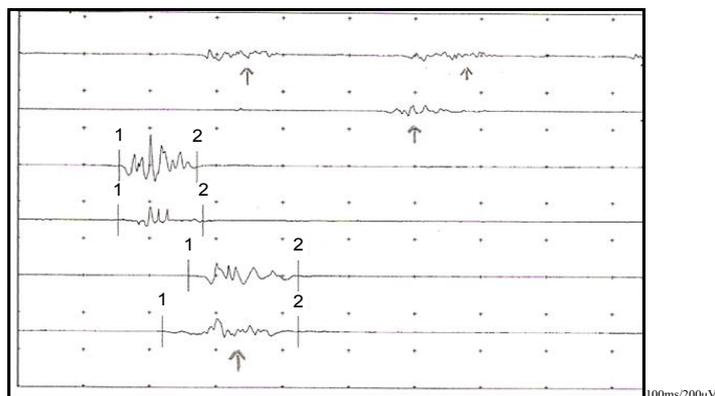


Figure 4: MAP (motor action potential) analysis shows characteristics of re-innervation in different chronological phases and to a high percentage.

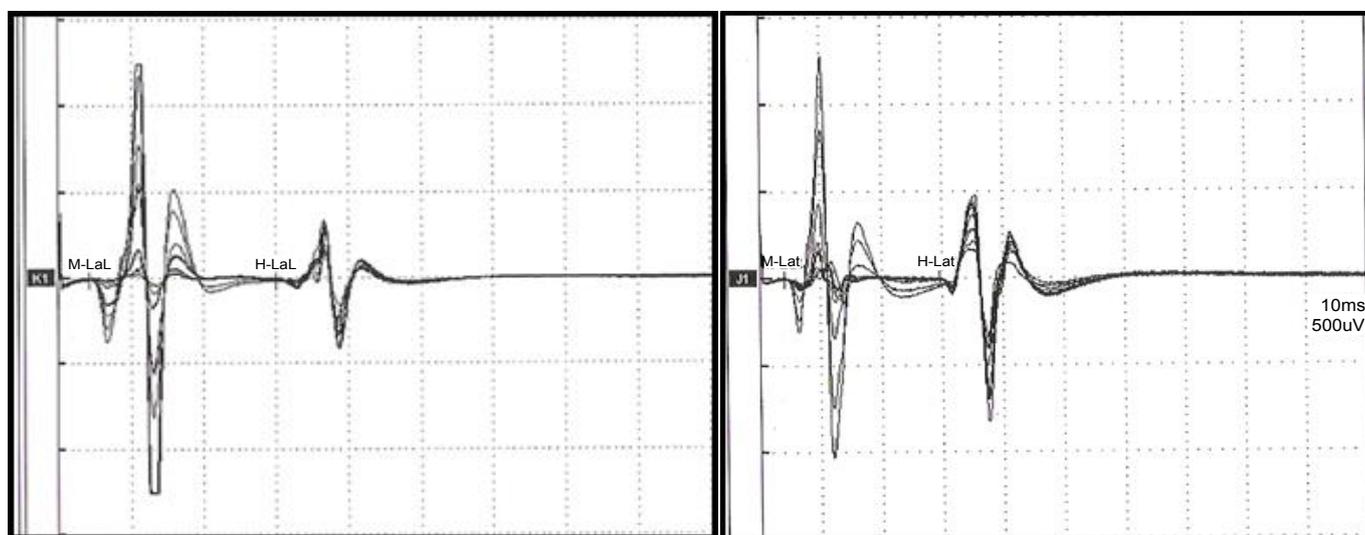


Figure 5: Tibial nerve-soleus: Left H Reflex shows 29.7 ms latency and right H reflex shows 30.0 ms latency, both values are normal. Polyneuropathy is ruled out.

In cases secondary to bariatric surgery, the existence of polyneuropathy by nutritional deficit has been described [24,36,37]. In our series of cases, polyneuropathy has been ruled out by the neurophysiological study over different nerves, and the presence of located mononeuropathy has been confirmed.

In cases of Anorexia Nervosa, compression is favored by secondary weight loss due to malnutrition and facilitated by the postural mechanism of maintained crossing legs, as it was in our patient.

The neurophysiological study confirmed in all cases a compressive neuropathy localized, with demyelinating character, decreased motor conduction velocity and decreased antidromic sensory velocity, at the compression site. Besides, there was axonal compromise, objectified in the analysis of the PUMS, showing characteristics of reinnervation (long duration potentials and polyphases) at different time phases and a high percentage. The latter indicates a subacute or chronic evolution and a good prognosis for functional recovery, suggesting the desirability to start conservative treatment.

In our series of cases, the treatment was basically rehabilitation, to prevent complications, to preserve the range of motion by mobilizations, to maintain muscle trophism with electro stimulation and foot orthotics

to prevent drop foot (Adapted to each patient based on convenience). Gait training in case of peroneal compression was performed. In all patients, a good performance for functional recovery was achieved, without the need of surgical decompression, as other authors refer [21]. In the case of ulnar neuropathy it was decided to make a surgical relocation of ulnar nerve and the patient improved.

## Conclusion

For peripheral nerve compression, there are multiple risk factors to consider and neurophysiological diagnosis means to use. Excessive and rapid weight loss is a rare cause, but it is important to be considered nowadays, in order to prevent and to diagnose earlier, and to perform the appropriate treatment for a better recovery.

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