Common Peroneal Neuropathy in Patients after First-Time Stroke*

Atzmon Tsur MD

Department of Rehabilitation, Western Galilee Hospital, Nahariya, Israel

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Abstract

Background: Common peroneal neuropathies, usually located at the fibular head, are one of the causes of drop foot, a condition often evaluated in the electromyography laboratory.

Objectives: To study the motor conduction properties of the common peroneal nerve and its branches of distribution in patients with paralyzed drop foot, several weeks after their first stroke, assuming that its inversion position can cause neuropathy around the fibular neck.

Methods: We performed peroneal nerve conduction study on 76 legs of 38 patients, 12–73 days after their first stroke. All the patients had flaccid drop foot on the involved side. The stimulating electrode was placed at the postero-lateral aspect of the fibular neck. Motor nerve conduction latency and compound muscle action potential amplitude were measured along the proximal part of the deep and the superficial peroneal nerve, comparing the paralyzed to the sound leg. Paired sample *t*-test and paired *t*-test were used to compare the nerve conduction properties between the sound and the paralytic leg. The linear liaison between the two legs was determined by Pearson coefficient and the test based on it.

Results: The differences between motor conduction latencies and between CMAP amplitudes, comparing the paralyzed to the sound side, recorded in both the deep peroneal nerve and the superficial peroneal nerve, were statistically significant ($P < 0.05$).

Conclusions: It seems that the permanent equino-varus position of the paralyzed foot might affect common peroneal nerve conduction properties at the level of the fibular neck by demyelination, axonopathy, or both. Possible reasons for these pathological changes are nerve traction or nerve compression, but temperature changes in the paralytic leg should also be considered. Ankle-foot orthoses can be prescribed for prevention or correction of deformities of the foot and ankle and reduction of the weight-bearing forces.

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The common peroneal nerve is a continuation of the sciatic nerve, originating from nerve roots L4, L5, S1 and subdivided under the fibular head as:

- Deep peroneal nerve, which supplies the muscles tibialis anterior, extensor digitorum longus and brevis, extensor hallucis longus (motor) and the first web space of the foot (sensory).
- Superficial peroneal nerve, which supplies the muscles peroneus longus and brevis (motor) and the dorsal part of the foot (sensory).

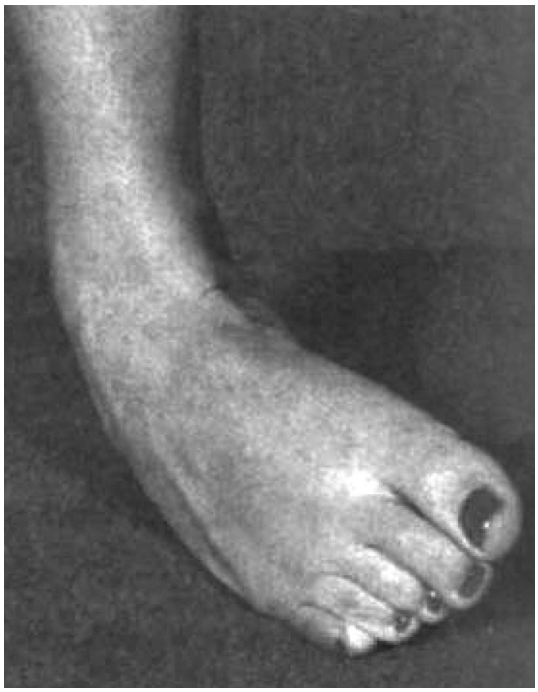

Figure 1. Foot in inversion position

The common peroneal nerve is injured more frequently than either of its branches alone. In fact, common peroneal neuropathy is the most frequent lower extremity mononeuropathy. The CPN can be injured by a stretch injury or by direct compression. Most peroneal neuropathies occur at the knee level around the fibular neck. Compression of the CPN at this level may cause pain and paresthesia in the lateral aspect of the leg and the dorsal aspect of the foot, as well as paresis of the foot extensor and pronator muscles and the toes' extensor muscles, also known as "drop foot" [1,2]. People suffering from severe or even mild ankle sprain, caused by inversion movement of the foot, reveal a "high" lesion of the peroneal nerve around the fibular neck [3-10].

The present study was undertaken to investigate whether a continuous inversion position of the paralyzed foot, seen in hemiplegic patients after acute stroke and imitating the foot position during ankle sprain [Figure 1], causes the same lesion. Our objective was to study the motor conduction properties of the common peroneal nerve and its branches of distribution in

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CMAP = compound muscle action potential

CPN common peroneal nerve

hemiplegic patients with drop foot after their first stroke. The conduction properties of motor nerve fibers in the peroneal nerve are usually calculated from small distal muscles [11], but it may be difficult to measure peroneal nerve velocity if the extensor digitorum brevis is atrophied, as in our patients. In this situation the latency and the amplitude can be calculated by recording the compound muscle action potential in a proximal muscle such as the tibialis anterior or the peroneus longus, by a simple technique that enables the examiner to test more functional muscles than the extensor digitorum brevis.

Patients and Methods

The study was a retrospective analysis of data on patients hospitalized in our rehabilitation department between the years 1998 and 2004. We routinely perform nerve conduction tests on all patients who have a drop foot after stroke. Motor conduction studies for both deep and superficial peroneal motor nerve were tested in 76 legs of 38 adult patients after their first stroke who had hypotonic drop foot on the involved side. Their mean age was 65 ± 9 years (range 43–80 years). The test was done 12–73 days after their first-time stroke (mean 38 ± 19). All patients had complete paralysis of the foot and toes' extensor muscles at the time of the examination, when they were asked to contract them in a lying and sitting position. Exclusion criteria were previous stroke, neuromuscular disease, radiculopathy or peripheral nerve injury in a lower limb, polyneuropathy, and diabetic neuropathy.

The plantar flexion and inversion position of the paralyzed foot was not corrected by an elastic band at least until our nerve conduction test. Some of the patients were wheelchair-dependent. When the other patients exercised in the physiotherapy institute, the involved foot was attached to the ankle by a strap in a neutral position in order to facilitate walking and even heel strike at the start of the stance phase.

The study was performed in a warm room and the temperature was maintained at about 23°C. Skin temperature was not measured due to technical reasons. Patients were either lying in bed or seated on a wheelchair with their legs propped on a regular chair. The tests were done following the method described first by Devi et al. [12]. For the proximal deep and superficial peroneal motor nerve conduction, the stimulating cathode was placed at the posterior-lateral aspect of the fibular head. The point for the recording surface electrodes was placed 8 cm from the cathode over the tibialis anterior in the first test and over the peroneus longus in the second test. The reference electrodes were secured respectively over the tendons of the tibialis anterior and peroneus longus at the ankle [Figure 1]. All patients were studied on a Nicolet Viking NT electromyography machine. The electrical nerve stimulation level was 175 volt, well tolerated by all the patients, and the stimulator pulse duration of square wave was 0.1 msec. The latency was measured from the stimulus artifact to the onset point, and the amplitude was determined from baseline to the highest negative peak [12-15].

Results of the paralyzed leg were compared to those obtained in the sound leg. Means and standard deviations of the latencies and amplitudes were calculated. This method is particularly helpful in determining the conduction properties of the peroneal nerve so that the diagnosis of paralysis can be made in the presence of atrophy of the extensor digitorum brevis.

Statistical analysis

Paired sample *t*-test and paired *t*-test were used to compare the nerve conduction properties, i.e., motor latency and CMAP amplitude, between the sound and the paralytic leg. The linear correlation, comparing the different parameters between the two legs, was determined by Pearson coefficient and the test based on it.

Results

Motor nerve conduction latencies in the deep peroneal nerve, registered in the tibialis anterior muscle, were longer in the paralytic side than in the sound side (paired sample *t*-test, $P < 0.001$). Motor nerve conduction latencies in the superficial peroneal nerve, registered in the peroneus longus muscle, were also longer in the paralytic than in the sound side (paired *t*-test, $P < 0.001$) [Table 1]. The CMAP amplitudes were higher in the sound than in the paralytic in both the tibialis anterior muscle (paired *t*-test, $P = 0.031$) and the peroneus longus muscle (paired *t*-test, $P = 0.0105$) [Table 2].

We found a low intensity linear liaison ($r = 0.266$) with regard to the difference between latencies of the two legs in the tibialis anterior muscle and the difference between latencies of the two legs in the peroneus longus muscle. The liaison was determined by Pearson coefficient and the test based on it ($P = 0.053$). The significance is that as the difference between the latencies in the tibialis anterior muscle increase, the same difference in the peroneus longus muscle increases concomitantly. Low to moderate intensity linear liaison ($r = 0.434$) was also found regarding the difference between the two legs' CMAP amplitudes in the tibialis anterior muscle compared to the same difference in the peroneus longus muscle. The liaison was determined by Pearson coefficient and the test based on it ($P = 0.003$). No linear liaison was found between the time passed since onset of the stroke and the difference between the two legs with regard to motor latencies and CMAP amplitudes. No differences were found in motor nerve conduction latencies and CMAP amplitudes, between people examined less than 32.5 days or more than 32.5 days after occurrence of the stroke (median 32.5 days).

Table 1. CMAP latency recorded in the tibialis anterior and the peroneus longus muscles

	Sound foot	Paralyzed foot	*P*
Tibialis anterior	3.25 ± 0.35	3.6 ± 0.48	< 0.001
Peroneus longus	3.57 ± 0.45	4.11 ± 0.53	< 0.001

Table 2. CMAP amplitude recorded in the tibialis anterior and the peroneus longus muscles

	Sound foot	Paralyzed foot	*P*
Tibialis anterior	5.05 ± 1.94	4.4 ± 1.89	< 0.031
Peroneus longus	4.96 ± 1.84	4.30 ± 2.12	< 0.0105

Discussion

Injury to the CPN around the head and neck of the fibula is usually due to compression, traction, or laceration [6-10,16], and has been described mostly in sportsmen after severe ankle sprain [2,5-7,18]. A lesion in the CPN in stroke patients with drop foot and uncontrolled foot inversion after paralysis has not yet been described.

The study on cadavers by Noble [17] showed that the CPN and its branches of distribution are attached to the fibular neck along with the peroneus longus muscle producing a T-form osteo-muscular tunnel. The extensibility of the common peroneal nerve in the popliteal cave is limited to 10–25 mm only. Severe inversion movement may lead to significant displacement of the osteo-muscular tunnel, thereby causing anterior nerve traction (the mechanical explanation) [7,17,18]. The peroneal nerve is supplied by two to three vasa nervorum, attached to the nerve by the endonerve and the perinerve. Severe traction may cause either hematoma or ischemia inside the nerve (the vascular explanation) [4,17,18]. Nitz and co-workers [5] found that 17% of patients with grade II ankle sprains and 86% with grade III sprains had fibrillation potentials in their peroneal innervated leg muscles, despite the lack of clinical signs and symptoms of nerve lesion, suggesting a mild axonal injury. Baccari and team [18] reported six cases of peroneal nerve paralysis following ankle sprain, 0–3 days after the accident. These theories on peroneal nerve vulnerability were evidenced after ankle sprain, during which the foot takes an equino-varus position, but not after stroke in which there is no history of local trauma.

Takebe et al. [19] noted significant slowing in peroneal nerve conduction velocities in the affected extremities of 27 hemiplegic patients and, at the same time, a significant difference in skin temperature between the extremities of the two sides. They supposed that a decreased diameter of the nerve fiber as a result or cause of muscle atrophy could lead to decreased nerve conduction velocity.

Chokroverty and Medina [20] measured bilaterally the motor nerve conduction velocities of the CPN in 44 hemiplegic patients and found a statistically significant difference between the two limbs. In 63% of the patients skin temperature was reduced in the hemiplegic limbs.

We shall adopt the hypothesis that continuous inversion position, as in hypotonic drop foot, may affect the electro-physiological properties of the nerve. It most probably results from nerve traction and compression at the level of the peroneal neck, causing demyelinization and even axonopathy. Myelin loss results in slowing of nerve conduction through the area involved. When compression is severe, ischemic changes occur and cause secondary axonal damage, expressed by reduction of CMAP amplitude. Nonetheless, we must take into consideration that the slowing of conduction velocities of the CPN in the hemiplegic limbs may be related also to the lowering of the skin temperature in the same limbs.

The values of latencies in deep and superficial peroneal motor nerves to the tibialis anterior and peroneus longus, respectively, with a fixed distance of 8 cm, were recorded. This approach may be easily accessible for electrodiagnostic evaluations of peroneal neuropathy, although the nerve conduction velocity is the value required for better clinical diagnosis.

Ankle-foot orthoses are most commonly prescribed for muscle weakness affecting the ankle and the subtalar joints, such as weakness of dorsiflexors and evertors. These orthoses can be prescribed for prevention or correction of deformities of the foot and ankle and reduction of the weight-bearing forces [21,22] in standing and even in sitting positions. As members of the rehabilitation team, the physician, the physiotherapist, the nurse and the orthotist all play a role in educating the patient in the decision-making process involving orthotic prescription, in order to prevent a continuous drop foot and, consequently, peroneal nerve traction.

Conclusions

Severe inversion of the ankle, as in skin temperature reduction in the paralyzed leg of stroke patients with drop foot, can influence the common peroneal nerve conduction properties. We presume that continuous stretch or nerve traction, due to the foot position, is the main reason for this phenomenon. Functional impairment in these patients arises primarily from the brain lesion that causes a loss of foot dorsiflexion and eversion, but the resulting peripheral neuropathy can aggravate the dysfunction.

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Correspondence: Dr. A. Tsur, Dept. of Rehabilitation, Western Galilee Hospital, P.O. Box 21, Nahariya 22100, Israel.
Telefax: (972-4) 910-7726
email: atzmon.tsur@naharia.health.gov.il