# **Clinical note**

# Neurapraxia of the common peroneal nerve- a rare complication resulting from wearing a KBM prosthesis: a case report

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#### Abstract

This clinical note describes a 47-year-old man who had a traumatic amputation of the left lower leg. Two months after wearing a Kondylen Bettung Münster (KMB) prosthesis, he developed a compression neuropathy of the common peroneal nerve of his right leg after sitting cross-legged. This troublesome complication can be avoided by giving accurate information to the patient.

# **Case report**

# Previous history

A 47-year-old man had an accident at work. A lathe overturned and fell on his left leg. There was a comminuted fracture and extensive injury of soft tissue just above the ankle. A guillotine amputation was performed because there was no chance of recovery. A defect of the skin was covered with a transplant taken from the amputated leg. After two months, when the wound had healed, the patient got a temporary KBM prosthesis. Rehabilitation therapy started.

Four months after the amputation there was a sudden loss of muscle power of the dorsal flexors of the right foot. There was also a numb sensation at the dorsal site of the foot. These changes arose after sitting with legs crossed for about one hour.

Examination showed a nearly fully established paralysis of the dorsal flexors and the peroneal muscles. The patient walked with foot drop. He experienced a loss of sensation in the area of the common peroneal nerve. A nerve conduction study was performed. The peroneal nerve was stimulated and there was no motor conduction response. The sensory conduction response was in the normal range. There was a focal slowing of the maximum nerve condition velocity across the neck of the fibula. Electromyography showed denervation potentials in the muscles innervated by the common peroneal nerve.

A compression neurapraxia of the common peroneal nerve was diagnosed.

The patient was given an orthosis to support the right foot. He used the orthosis for three months. At that time reinnervation had taken place. A new nerve conduction velocity study and electromyography revealed also signs of reinnervation. Muscle force was recovered and sensation was normalised.

Seven months after the amputation a reconstruction of the stump was carried out. The patient received a definitive KBM prosthesis.

#### Follow-up

One year after the injury, the patient started working again. Eighteen months after amputation of the lower left leg, there are no problems with the use of the prosthesis. He does not experience functional restrictions of the right foot. From investigation it appears that muscle force is normal and sensation is undisturbed.

#### Background

Nerve dysfunction which arises by compression, can be caused by a direct as well as an indirect factor. If pressure is acute and focused, the direct mechanical influence is pronounced. If pressure is chronic, ischaemia primarily causes indirect damage (Mumenthaler and Schliack, 1991).

The common peroneal nerve is vulnerable to compression and stretching injury because of its anatomic location. It curves around the neck of

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the fibula and is applied to the pereosteum for about 10 cm. In addition, the nerve is exposed over a bony prominence, covered only by skin (Berry and Richardson, 1976; Stuart *et al.*, 1989).

The patient described in this case report had a drop foot and paraesthesia over the dorsum of the foot. These two symptoms are characteristic of paralysis of the common peroneal nerve (Berry and Richardson, 1976; Muhammed *et al.*, 1995). In this case the actual cause of the peroneal nerve palsy was direct mechanical pressure.

### Discussion

A single episode of nerve compression can be followed by paralysis, caused by local demyclination at the site of the injury. This demyelination follows movement of the nodes of Ranvier, which displace in the same direction in all fibres namely towards uncompressed tissue. The displaced nodes are then no longer in their usual position under the Schwann cell junction. Thus, the paranodal myelin is stretched on one side of the node and invaginated on the other. Degeneration of the damaged myelin follows. It causes a conduction block which may take months to recover (Ochoa *et al.*, 1972).

Ischaemia causes the condition of rapidly reversible physiological block. When a limb goes to sleep, it recovers when posture is altered and blood supply of the nerve is restored. However, nerve fibres can withstand a period of 4 to 6 hours of ischaemia, without the development of structural damage.

It might be possible that ischaemia during a period of compression exaggerates or potentiates the directe effect of the compressive force (Gilliatt, 1980).

Because the common peroneal nerve lies superficial and is relatively fixed, it is frequently injured by compression.

The patient was sitting relaxed in an easy chair for one hour, doing a puzzle. During that time he sat cross-legged, right over left. When he rose and intended to walk, he noticed a slapping gait and a changed scnsation.

Physical examination excluded diabetes, alcoholism, vascular disease and a root syndrome of L5. The sustained compression by the lateral rim of the prosthesis was suspected to be the cause of this complaint. A light orthosis was made to wear inside the shoe. It prevented the drop of the foot and protected against inversion injuries.

In general the prognosis for recovery is good (Stuart *et al.*, 1989; Berry and Richardson, 1976; Mumenthaler and Schliack, 1991). However, it was difficult to convince the patient of this. At the time of this complication he was still undergoing a rehabilitation programme because of the amputation. While wearing an orthosis he was no longer independent. This case demonstrated that the patient wearing a KBM prosthesis, was more disabled by this new impairment than a person without an amputation of the lower leg would have been. It was hard for him to accept that he could not drive his car (not allowed by Dutch law).

In three months the symptoms of the compression neurapraxia spontaneously faded away. One year after the amputation he started working again as a vocational instructor in the metal industry.

## Conclusion

A compression neurapraxia of the common peroneal nerve can occur after sitting crosslegged, especially while wearing a KBM prosthesis. This is an additional complicating factor in physical as well as in psychological rehabilitation.

It is important to warn patients against this troublesome complication, which is easily avoided.

#### REFERENCES

- BERRY H, RICHARDSON PM (1976). Common peroneal nerve palsy: a clinical and electrophysiological review. J Neurol Neurosurg Psychiatry 39, 1162-1171.
- GILLIATT RW (1980). Acute compression block. In: The physiology of peripheral nerve disease./edited by Sumner AJ.-Philadelphia: WB Saunders. p287-315.
- MUMENTHALER M, SCHLIACK H (1991). Peripheral nerve lesions. Diagnoses and Therapy./translation of the 5th German edition.-Stuttgart: Georg Thieme Verlag.
- MUHAMMED N, CAMPBELL P, SMITH IS (1995). Peripheral nerve entrapment syndromes: diagnoses and management. Br J Hosp Med 53, 141-146.
- OCHOA J, FOWLER TJ, GILLIATT RW (1972). Anatomical changes in peripheral nerves compressed by a pneumatic tourniquet. J Anat 113, 422-455.
- STUART JD, MORGAN RF, PERSING JA (1989). Nerve compression syndromes of the lower extremity. Am Fam Physician 40, 101-112.