

Matters arising

Peroneal neuropathy during weight reduction

Sir: Sotaniemi¹ reported 10 patients who had developed evidence of peripheral nerve lesions during slimming. In nine of them this took the form of unilateral or bilateral peroneal nerve palsy. In the absence of a history of habitual leg crossing or other posture that might have provoked compression of the common peroneal nerve at the head of the fibula, Sotaniemi concluded that his patients must be suffering from a metabolic neuropathy due to slimming. No neuro-physiological data were offered to substantiate this claim however, and the patients' symptomatic lesions were confined to the territories of the common peroneal nerves. It is also difficult to assess the assertion that there was no evidence of a local lesion at the head of the fibula in these cases, since only motor conduction velocities are quoted. A personally studied case suggests slimming increases vulnerability to a local compressive lesion.

A 41-year-old business man was advised to lose weight after sustaining a myocardial infarction. In the course of 12 months he lost 9 stones (57 kg). During the latter part of this year he developed a right-sided foot drop. Examination revealed wasting and weakness in the territory of the right common peroneal nerve with a little sensory loss on the dorsum of the right foot. There was also weakness in the territory of the asymptomatic left common peroneal nerve. There was no weakness or sensory loss outside these territories and all reflexes were obtainable. Nerve conduction studies revealed normal motor conduction velocities in the common peroneal nerves (47 m/s on the left; 46 m/s on the right). There was evidence of local lesions at the head of the fibula on both sides however. On the left the muscle action potential recorded by surface electrodes from extensor digitorum brevis was 3.3 mV during stimulation of the peroneal nerve at the ankle but only 1.0 mV with stimulation above the head of the fibula. On the right the comparable figures were 0.7 mV from stimulation at the ankle, 0.6 mV from the neck of the fibula but less than 0.1 mV from above the head of the fibula. Nerve action potentials were measured orthodromically as described by Gilliatt and Willison,² except that the separate subcutaneous needle electrodes above and below the head of the

fibula were referred to a distant electrode on the front of the knee.³ On the left the nerve action potential recorded at the neck of the fibula during stimulation at the ankle was 7.0 μ V; recorded above the head of the fibula it was 3.5 μ V. On the symptomatic right side a nerve action potential of 3.5 μ V was recorded at the neck of the fibula, but no response could be picked up more proximally.

These results were interpreted as showing some axonal loss in the right peroneal nerve together with evidence of conduction block at the head of the fibula bilaterally. There was no evidence of a diffuse abnormality of nerve conduction.

Six months later some partial recovery had occurred but the patient was obliged to use a foot-drop brace on the right leg. His wife recalled an episode of chest pain and breathlessness in which he had collapsed and been briefly unconscious. This may have represented one incident of trauma to the nerve at the knee, rendered vulnerable by severe weight loss. There seems no need, in this case at least, to postulate a metabolic neuropathy in order to account for the appearance of peroneal nerve palsies.

I am grateful to Professor RW Gilliatt for referring this patient for neurophysiological assessment.

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References

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- 2 Gilliatt RW, Goodman HV, Willison RG. The recording of lateral popliteal nerve action potentials in man. *J Neurol Neurosurg Psychiatry* 1961;24:305–18.
- 3 Gilliatt RW. Peripheral Nerve Compression and Entrapment. In: Lant AF, ed. *Eleventh Symposium on Advanced Medicine* London: Pitman Medical 1975:144–63.

Sotaniemi replies

I thank Dr Harrison for his interest and comments on my paper referred to above. I would like to emphasise that the case history, the mode of the onset of the symp-

toms and the investigatory findings of patient reported by Dr Harrison were completely different from those of the patients described in my article. Particular attention paid to the patients' case histories failed to reveal any kind of trauma or postural, occupational or toxic factors possibly attributable to nerve dysfunction. What was common to all the cases in my report were the rapidity of the weight loss (at least 5 kg per month) and the adherence of a strict diet for 2 to 4 months. The patients underwent careful clinical, laboratory, X-ray and ENMG investigations. No co-existent disease or local or generalised determinant of nerve dysfunction could be established. The ENMG findings were not typical of what is seen in compressive peroneal nerve lesion.

The lack of any verification of local nerve injury in both clinical and ENMG investigation performed, the gradual onset of the symptoms without acute manifestations, and the characteristics of the carefully scrutinised anamnestic information revealing no known factors contributing to nerve damage, suggest that mechanical injury is not necessarily the only determinant of dysfunction to be taken into account. In the case reported above, the compressive aetiology was evident even anamnestically.

The aetiology of peripheral neuropathy associated with weight reduction may be multifactorial, compression and related mechanical factors having an important role, as was already mentioned in my article. However, both obesity itself as well as fasting are known to have their metabolic characteristics which may also be involved in the development of nerve dysfunction. In order to obtain further elucidation on the matter we should be able to investigate future patients soon after the onset of the harmful symptoms. According to my experience, it has been the rule that a person who has developed harmful manifestations during the slimming regimen has immediately normalised his diet. Usually, several weeks elapse before the patient is examined, and the investigatory findings obtained several weeks of "normal" diet may not reliably reflect the conditions during fasting. Examination of the somatosensory evoked potentials might also provide us further information of the eventual complications of fasting.

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