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Latent tarsal tunnel syndrome with the provocation of flexor spasms in a paraplegic person. Case Report

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A 71-year-old paraplegic woman presented with a complaint of insomnia caused by severe flexor spasms of the lower limbs at night, aggravated for a few hours by taking hot baths. Lidocaine injection of the tarsal tunnel diminished the flexor-withdrawal reflex, which was easily elicited by light pinching or sustained compression over the tarsal tunnel, and strongly suggested the existence of the tarsal tunnel syndrome. Surgical decompression of the tarsal tunnel significantly reduced the flexor spasms both in terms of frequency and of duration.

Keywords: paraplegia; flexor-withdrawal reflex; syringomyelia; tarsal tunnel syndrome

Introduction

There has been a considerable amount written on the tarsal tunnel syndrome. However, no instances of this disorder provoking a paraplegic person to develop severe flexor leg spasms have hitherto been described.

Case report

A 71-year-old woman who was paraplegic presented with a 2 month history of insomnia caused by severe flexor spasms in her legs. In 1987, she was first admitted with complaints of progressive weakness of her legs, and she was not ambulatory because of her spastic paraparesis. On radiological examination, the thoracolumbar spine was seen to be severely scoliotic with holocord syringomyelia being demonstrated in the magnetic resonance images. Shrinkage of the syrinx was obtained by means of a syringo-subarachnoid shunt operation and when she was discharged she was able to walk. However, the neurological condition of her legs progressively deteriorated, and she was not ambulatory for the ensuing 3 years during the last two of which she had flexor spasms of her legs, aggravated at night-time. Leg casts were required to prevent knee flexion during sleep, and during the 2 months before readmission to hospital, flexor spasms of her legs occurred, especially for a few hours after taking a hot bath. She had become exhausted because of insomnia.

On her readmission in 1994, she was paraplegic, the deep tendon reflexes were exaggerated in both legs and diminished in the upper extremities except for the right triceps, the right plantar reflex was extensor, and all sensory modalities were diminished below the seventh thoracic segment with apallaesthesia in the legs and loss of position sense. Her blood-picture and urine analysis data were normal. On radiological examination, the syrinx was still found to be shrunk. Electrophysiological examination did not reveal any obvious abnormalities. Not only painful stimulation to the soles of the feet but also sustained compression over the posterior tibial nerve at its course posterior to the internal malleolus, or even a light pinch of the big toe, would quickly elicit a flexor-withdrawal reflex (Figure 1a), which was a one phase reaction of the limb not extending to the original position thereafter in either leg. Flexor-withdrawal reflexes markedly receded for a few hours after the injection of 2 ml 2% lidocaine into the tarsal tunnel (Figure 1b). Although not endorsed, a diagnosis of tarsal tunnel syndrome was strongly suggested.

She was operated on for decompression of the tarsal tunnel in both legs under general anaesthesia. The operations did not reveal any obvious changes in relation to the posterior tibial nerves in their tunnel. After the operation she stated that her feet felt warm and comfortable, and although the flexor-withdrawal reflex could be easily elicited by scratching the sole (Figure 1c), elicitation by pinching the big toe or sustained compression over the posterior tibial nerve posterior to the internal malleolus were markedly reduced soon after the operation (Figure 1d). Also the nocturnal flexor spasms were reduced in frequency and severity and their aggravation was no longer evident.

Discussion

The difficulty here is that it is not very easy to obtain a precise clinical picture of what we are describing in this type of syndrome in paraplegic patients. The present patient was not aware of any burning pain in the soles of the feet because of the thoracic cord lesion, but a flexion-withdrawal reflex was elicited instead. This phenomenon is very similar to the experimental observation by Gilliat[1] in that patients with spastic paraplegia or hemiplegia, ischaemia of the leg may be followed by a hyperexcitable phase, during which an extensor plantar response with reflex flexion withdrawal of the leg is very easy to elicit by light tactile stimuli, which were previously ineffective. In the present case, painless stimuli could elicit the flexion withdrawal reflex when applied to the limited skin area innervated by the posterior tibial nerve. Although it has been reported that relief of pain and paraesthesiae can be obtained by

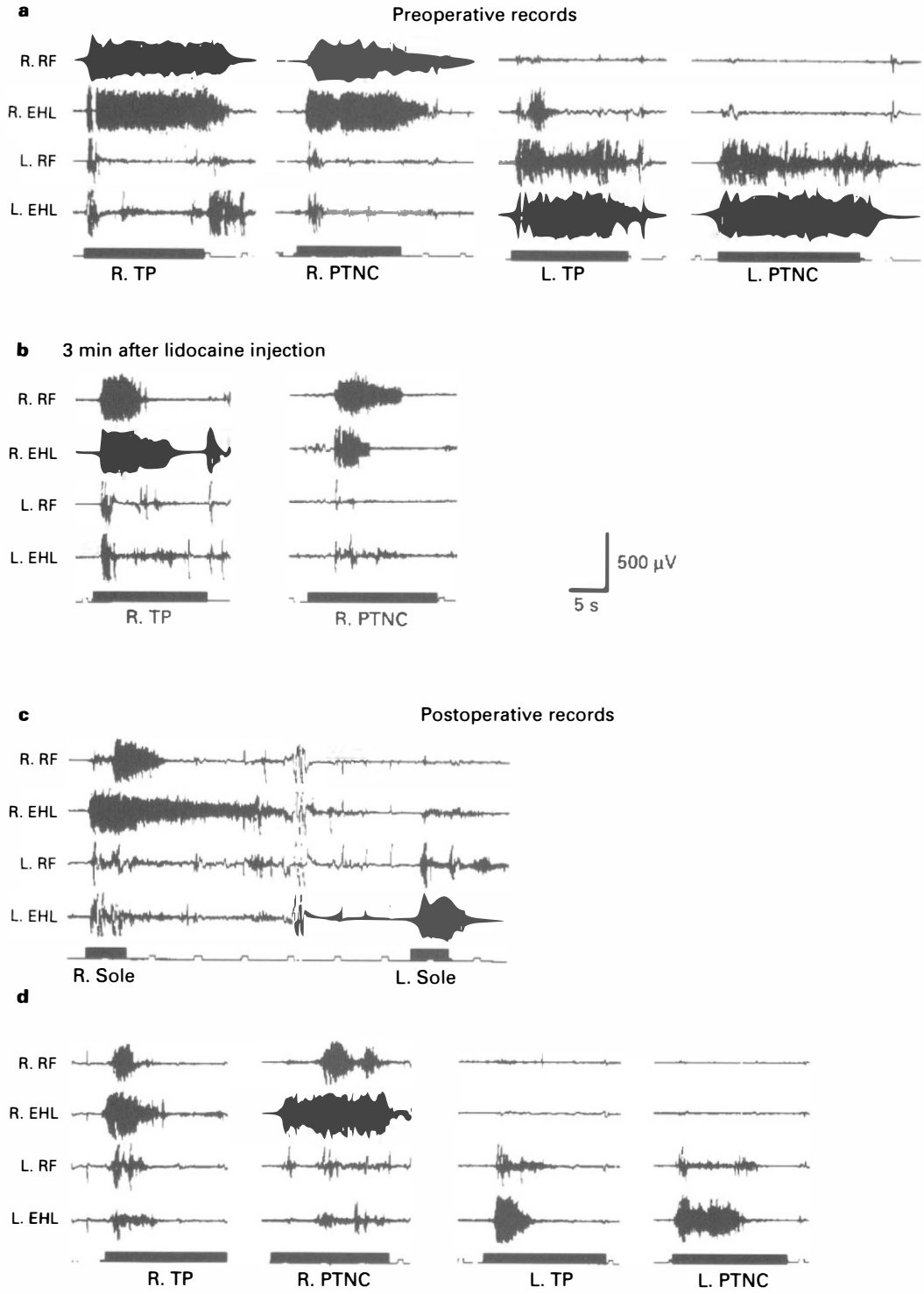

Figure 1 (a) Preoperative records of the flexor-withdrawal reflex. Pairs of surface electrodes were placed over the right and left rectus femoris (RF) and the extensor hallucis longus (EHL) muscles. Light pinching of the big toe (TP), or sustained compression over the posterior tibial nerve at the tarsal tunnel (PTNC) elicited marked flexor-withdrawal reflexes. (b) The reflex was markedly reduced 3 min after lidocaine injection into the tarsal tunnel. (c) Scratching of the lateral part of the sole still elicited distinct flexor-withdrawal reflexes in both legs 2 weeks after surgical decompression. Solid bars at the bottom indicate scratching the sole. (d) The same tests of toe pinching and sustained compression over the posterior tibial nerve in the tarsal tunnel were repeated 2 weeks after the surgical decompression. Marked reduction of the reflex activity in either leg is obvious

a nerve block distal to a lesion,[2] in the present case, the result of the lidocaine test strongly suggests that an irritative afferent impulse was generated in or distal to the tunnel.[3–5] This was also confirmed by the fact that surgical decompression of the posterior tibial nerve in the tarsal tunnel was followed by an immediate and long lasting successful result. Aggravation of symptoms at night has been described elsewhere,[6] although their enhancement after taking a hot bath has not previously been reported.

The pathophysiological mechanisms underlying paraplegic flexor spasms are still controversial, but they can easily be induced by noxious stimuli, not only to the sole of a foot but also to considerable areas of the leg.[7] Apart from the stimulation itself, several indirect influences on the reflex have been studied such as the temperature of the foot,[8] tonic neck reflex,[9] etc. Aggravation of the flexor-withdrawal reflex some time after taking a hot bath has been suggested to be due not only to the local pathology but also to an enhancement effect of warming the foot.[8] In this patient, hyperexcitability appears to have coexisted both in the spinal cord and in the peripheral nerve. The hyperexcitability of the peripheral nerve in the tarsal tunnel was reduced by surgical decompression. However, the intraspinal pathology remained unresolved and a minimal, albeit distinct flexor-withdrawal reflex remained capable of being elicited by noxious stimuli to the sole of the foot.

In conclusion, we would like to stress that latent entrapment neuropathy may aggravate pathological reflexes and restrain the normal activities of a patient. In such cases surgical decompression of the nerve may result in a favourable outcome.

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