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Muscle Spindle Injury Could Cause Tourniquet Paralysis.

Juhani V Partanen

University of Helsinki, Clinical Neurosciences, Clinical Neurophysiology, and University Hospital of Helsinki, Department of Clinical Neurophysiology, Finland

Corresponding author:
Juhani V Partanen, University of Helsinki, Clinical Neurosciences, Clinical Neurophysiology, and University Hospital of Helsinki, Department of Clinical Neurophysiology, Finland

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Abstract:

Objective:Knee surgery with a pneumatic tourniquet may result in quadriceps femoris muscle weakness. A patient with quadriceps femoris paresis was studied for two months after surgery.

Methods: Follow-up ENMG and lumbar magnetic stimulation (MEP).

Results: Six weeks after surgery, there was no discernible lesion of femoral or other nerve motor axons in ENMG or MEP. The paresis subsided after an eight-month follow-up, and the patient recovered completely in two years.

Conclusion: The tourniquet's high pressure may injure muscle spindles. Reduced Ia-afferent activity can significantly impair voluntary motor effort.

Keywords:
Tourniquet paresis; ENMG; Motor evoked potential; Muscle spindle; Ia-aerent Knee surgery

Introduction:

The benefit of using a femoral pneumatic tourniquet during knee surgery is a bloodless procedure. Tourniquet use, however, may result in hemodynamic, metabolic, or local problems [1]. Several clients suffer from postoperative thigh weakness, particularly when a strong tourniquet and a lengthy tourniquet period are employed [2]. When pre- and post-operative examinations were examined in a systematic ENMG research of patients who had anterior critical ligament procedures with tourniquet, the alterations in neurography were minimal. The vastus medialis muscle's EMG interference pattern of maximum voluntary effort had the most noticeable modification. In comparison to preoperative readings, the maximum voluntary activation was lowered to roughly 50% postoperatively. None of these individuals experienced any obvious quadriceps femoris muscle weakening. In needle EMG, there were no postoperative indications of axonal damage, such as fibriOOation potentials [3]. Six weeks following bilateral knee surgery with a pneumatic tourniquet, a patient developed virtually entire bilateral quadriceps femoris paresis. This case is documented.

Discussion:

Axonal compression syndrome has been linked to postoperative quadriceps femoris muscle weakness after procedures involving pneumatic tourniquets [2]. The patient presented here is an extreme occurrence, having nearly complete bilateral quadriceps femoris postoperative paresis for two months before gradually improving thereafter. The M-responses of the vasti muscles tested with lumbar MEP were normal despite the practically complete inability to engage these muscles voluntarily, proving that no specific axonal compression condition was present. There were only sporadic fibriOOation potentials; nevertheless, in six weeks, axonal loss should cause widespread fibriOOation activity [5].

The e saphenous nerve conduction values were normal, indicating that the femoral nerves' A beta fibres were in good condition. The remaining leg nerves showed normal nerve conduction, F- and H-responses, and no axonal involvement or conduction block of the motor nerve fibres or sensory A alpha or A beta nerve fibres. Thin sensory A delta and C nerve fibre involvement was indicated by pain and allodynia in the lower extremities. Eight months after surgery, there was a decent but not perfect interference pattern in the vasti muscles, and two years later, the right vastus medialis muscle's EMG had completely normalised. The arrangement or amplitude of the vastus medialis muscle's motor unit potentials showed no evidence of collateral reinnervation. The follow-up study's lumbar MEP latencies were normal but had lower amplitude. 9 months after surgery may show atrophy from inactivity.

However, there was hypersensitivity of the A delta fibres presenting as altered cold pain threshold on the dorso-lateral foot areas two years after surgery. QST study was not conducted in the acute stage, but QST two years postoperatively did not reveal any residual neuropathy of the thin sensory nerve fibres.The normal saphenous nerve conduction results show that the femoral nerves' A beta fibres are intact. The remaining leg nerves showed normal nerve conduction, F- and H-responses, and no axonal involvement or conduction block of the motor nerve fibres or sensory A alpha or A beta nerve fibres. Thin sensory A delta and C nerve fibre involvement was indicated by pain and allodynia in the lower extremities. Eight months after surgery, there was a decent but not perfect interference pattern in the vasti muscles, and two years later, the right vastus medialis muscle's EMG had completely normalised. The arrangement or amplitude of the vastus medialis muscle's motor unit potentials showed no evidence of collateral reinnervation. The lumbar MEP latencies were normal, but the follow-up examination 9

months after surgery showed decreased amplitude, which might be a sign of atrophy from lack of use.

However, there was hypersensitivity of the A delta fibres presenting as altered cold pain threshold on the dorso-lateral foot areas as F- and H-responses were normal in the other leg nerves, without any signs of axonal involvement or conduction block of the motor nerve fibres or sensory A alpha or A beta nerve fibres. QST study was not performed in the acute stage, but QST two years postoperatively did not reveal any residual neuropathy of the thin sensory nerve fibres. Thin sensory A delta and C nerve fibre involvement was indicated by pain and allodynia in the lower extremities. Eight months after surgery, there was a decent but not perfect interference pattern in the vasti muscles, and two years later, the right vastus medialis muscle's EMG had completely normalised. The arrangement or amplitude of the vastus medialis muscle's motor unit potentials showed no evidence of collateral reinnervation. The lumbar MEP latencies were normal, but the follow-up examination 9 months after surgery showed decreased amplitude, which might be a sign of atrophy from lack of use.

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Experimental experiments with a clinically appropriate cu inflated pressure have shown localised necrosis of muscle fibres beneath the pneumatic tourniquet [6]. The potential amputation of muscle spindles beneath the tourniquet, however, was not investigated. Different muscle fibres may respond to pressure differently. The most vulnerable fibres appear to be Type IIB rapid glycolytic fibres [6]. Static beta motor axons innervate the fast glycolytic extrafusal muscle fibres as well as the long chain fibres of the muscle spindles [7]. Hus theEven at pressure levels that are clinically significant, long chain fibres may be particularly susceptible. It is likely that the pneumatic tourniquet's high pressure affected the proprioceptive aerent function, particularly the Ia and II-aerents of the patient's leg muscle spindles. Intrafusal capillaries are less permeable than extrafusal ones and are enclosed by a tight membrane in muscle spindles.

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The condition known as "compartment syndrome" may even result in severe hypoxia and complete necrosis of the muscle spindle under the tourniquet. Damage to the muscle spindles, which include somatic C-nerve fibres, may result in postoperative pain [9]. The sleeves of the muscle spindles are where the long nuclear chain fibres protrude [10]. If the spindles are in necrosis, the denervation of these tiny but crucial muscular fibre fragments may be the cause of the irregular fibriOOation potentials seen in the patient's quadriceps femoris muscles' EMG.

How might the inadequate quadriceps femoris postoperative voluntary activation be explained? Normal adjustments to motor neuron exitability are made by persistent inward currents (PICs). A descending neuromodulatory pathway that releases the monoamines serotonin and noradrenaline controls the PIC. The motor neurone exitability is very low without PIC. However, descending PICs have poor focusing and require local synaptic integration to meet the demands of various motor activities. The length sensitive muscle spindle Ia aerents and Ia interneurons are the intermediaries via which the PIC is extraordinarily sensitive to reciprocal inhibition [10].

Conclusion:

Hus, the first paresis of the quadriceps femoris muscles may result from reciprocal inhibition from the posterior thigh muscles, which lack the appropriate proprioceptive activation due to the reduced muscle spindle afferent firing. On the other hand, proprioceptive activity from the muscle may also be necessary for motor neuron activation.

As demonstrated in acute sensory axonal neuropathy, an initial drop in aerent firing appears to lead to a severe suppression of muscle activation in voluntary effort [11]. As in our patient, it takes several weeks for symptoms to go away. In knee procedures, it is advised to keep the tourniquet pressure as low as feasible. front cruciate It is even possible to operate on ligaments without using a tourniquet.

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