Pulsed Radiofrequency of the Sural Nerve for the Treatment of Chronic Ankle Pain

Lyudmil Todorov, MD

From: Memorial Hospital of Rhode Island, Pawtucket, RI

Dr. Todorov is with the Department of Anesthesiology, Center for Pain Management, Memorial Hospital of Rhode Island, Pawtucket, RI.

Address correspondence: Lyudmil Todorov, MD
Memorial Hospital of Rhode Island
111 Brewster Street
Pawtucket, RI
E-mail: ludmil@rocketmail.com

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The application of radiofrequency (RF) has been successfully used in the treatment of chronic pain conditions, including facet arthropathy, sacroiliac joint pain, groin pain, radicular pain, cervicogenic headaches, and phantom limb pain. Due to the neurodestructive effect of continuous RF ablation and possible deafferentation sequelae, only pulsed radiofrequency (PRF) has been applied to peripheral sensory nerves. There are no previous reports of successful PRF application to the sural nerve.

Objectives: To report on the successful use of PRF to the sural nerve for the treatment of ankle pain. To discuss current theories on the mechanism by which PRF produces pain relief.

Methods: The report presented here describes the case of a 39-year old patient who sustained injury to her ankle. The patient was complaining of pain in the distribution of the sural nerve, which was confirmed by electrodiagnostic studies. The pain did not respond to oral and topical analgesics. The patient had short-term relief with a sural block with bupivacaine and triamcinolone. The patient then underwent PRF application to the right sural nerve for 240 seconds at 45 volts.

Results: The patient reported complete relief. There was no pain recurrence 5 months after the procedure.

Limitations: This report describes a single case report.

Conclusions: It is conceivable that PRF may provide long-term pain relief in cases of sural nerve injury. The exact mechanism of the antinociceptive effect is still unknown. Possible mechanisms include changes in molecular structure by the electric field, early gene expression, stimulation of descending inhibitory pathways, and transient inhibition of excitatory transmission.

Key words: chronic ankle pain, sural neuropathy, radiofrequency, pulsed radiofrequency application

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A 39-year-old female patient developed sharp ankle pain after a fall. The pain started below the right lateral malleolus and radiated down the lateral side of the foot. The pain was associated with numbness and tingling in the affected area. The patient described the pain as constant, sharp, and aching. The pain was exacerbated by standing and walking and mildly decreased at rest. The pain did not respond to analgesics, including NSAIDs, hydrocodone, and acetaminophen. She was taking topiramate 50 mg daily for migraine headaches without any effect on her ankle pain.

The patient was referred to our pain clinic 6 weeks after the accident. The patient rated her pain at rest as 3/10 increasing to 7/10 with standing and walking. On physical exam there was dysesthesia below the right lateral malleolus. There was no discoloration of the foot, no trophic changes and no swelling. There was no difference in skin temperature between the left and the right foot. There were no motor deficits and no loss of sensation. The distal pulses were palpable.

The patient underwent EMG and nerve conduction studies of the right lower extremity. These suggested right sural neuropathy around the ankle.

The treatment options for neuropathic pain were discussed with the patient, including tricyclic antidepressants, reuptake inhibitors of serotonin and norepinephrine, and calcium channel alpha2-delta ligands such as gabapentin and pregabalin. She was concerned about the possible side effects and interactions with her current medications, which included quetiapine fumarate and citalopram for depression and topiramate and Fioricet (butalbital, acetaminophen, caffeine) for migraines.

This is why the patient only agreed to the administration of topical lidocaine. She was started on lidocaine 5% patches without any relief. She then underwent a right sural nerve block whereby a 25-gauge needle was placed between the right lateral malleolus and the Achilles tendon. This was followed by the injection of 5 mL 0.25% bupivacaine and 20 mg triamcinolone. The patient had complete pain relief after the injection but 2 days later the pain returned. Approximately 3 months after the onset of pain the patient underwent a PRF application to the sural nerve. A 22-gauge, 10 cm radiofrequency needle (Smith & Nephew, Andover, MA) with a 10 mm active tip was inserted subcutaneously between the right lateral malleolus and the Achilles tendon and advanced 1.5 cm in a caudad direction until sensory stimulation could identify the sural nerve at 0.4 volts and 50 Hertz. One mL 1% lidocaine was injected to decrease the impedance. This was followed by PRF application for 240 seconds at 45 V with the temperature never exceeding 42°C. Prior to removal of the needle, the area was injected with 5 mL of 0.25% bupivacaine and 20 mg triamcinolone. There were no complications. The patient experienced complete relief of the pain, both at rest and with activity. Five months post-procedure she has no pain in the ankle.

This is the first report of successful RF application to the sural nerve for the treatment of neuropathic pain. The sural nerve is a sensory nerve providing innervation to the lateral surface of the foot and ankle. Sural neuropathy is an uncommon electrophysiological diagnosis. Trauma is the most frequent cause (21).

Radiofrequency application (pulsed or continuous) has been used in the treatment of facet arthropathy, sacroiliac joint pain, groin pain, radicular pain, cervicogenic headaches, and phantom limb pain (1-5).

RF is an alternating current with an oscillating frequency of 500,000 Hz (22). The main advantage of PRF over conventional (continuous) RF (CRF) is that its effect does not rely on thermal destruction of nerve tissue. In PRF the current is delivered in pulses, each lasting 20 milliseconds, followed by a period of no activity for 480 milliseconds, allowing the heat to dissipate, thus avoiding temperature increases. CRF causes coagulative necrosis leading to Wallerian degeneration, whereas PRF only causes transient mild edema without affecting the structural integrity of the nerve (1). Even when CRF is performed at 40°C, there is endoneurial edema and severe damage to transverse myelin fibers. These changes can be seen with PRF, but the severity of the lesions is much lower (23).

The sural nerve is a sensory nerve. Applying CRF to a sensory nerve could result in neuroma formation and worsening pain (2). This is why only PRF could be used for this patient. PRF has been applied successfully to the ilioinguinal, genitofemoral, and supraspinal nerves (2,18).

It is still unclear by what mechanism PRF produces pain relief. According to Sluijter et al (24), PRF produces a very weak magnetic field without any significant biologic effects. However, the active ( uninsulated) tip of the radiofrequency needle produces an electric field with a very high current density (2 x 104 A/m²) (24). PRF has a much stronger electric field than CRF. The electric field can induce charges on tissue and distort and dislocate charged molecular structures, thus disrupting cell func-
tion without substantial elevations in temperature (25). Higuchi et al (26) showed that PRF induces early gene expression in pain-processing neurons in the dorsal horn, demonstrated by an increase in the nuclear-binding cFOS protein. The effect is not mediated by tissue heating (26). In the dorsal root ganglion PRF induces activation transcription factor 3 (ATF3), a marker of neuronal response to injury (27). PRF applied to rat sciatic nerves can enhance noradrenergic and serotonergic descending pain inhibitory pathways (28). Cahana et al (29) showed that PRF causes transient inhibition of evoked excitatory transmission with full recovery of synaptic activity within a few minutes, whereas CRF produces a long-lasting blockade. Cells very close to the tip of the electrode (<500 µm) were damaged by both modalities. At 1,000 µm there was cell destruction only in the CRF group, even though in both groups the voltages were adjusted to reach similar temperatures. The authors concluded that CRF results in a neurodestructive effect, whereas PRF produces neuromodulation (29).

Erdine et al (30) showed that PRF produces enlargements in the endoplasmatic reticulum cisterns and increased number of cytoplasmic vacuoles of treated DRG cells. These changes were visible using electron microscopic analysis but not with light microscopy. There was no structural pathology in the cell membranes (30).

There are several limitations to this case report. It involves only one individual and may not be representative of all patients suffering from sural neuropathy. Confounding factors cannot be excluded. First-line treatments for neuropathic pain, including tricyclic antidepressants, reuptake inhibitors of serotonin and noradrenergic and serotonergic descending pain inhibitory pathways (28). Cahana et al (29) showed that PRF causes transient inhibition of evoked excitatory transmission with full recovery of synaptic activity within a few minutes, whereas CRF produces a long-lasting blockade. Cells very close to the tip of the electrode (<500 µm) were damaged by both modalities. At 1,000 µm there was cell destruction only in the CRF group, even though in both groups the voltages were adjusted to reach similar temperatures. The authors concluded that CRF results in a neurodestructive effect, whereas PRF produces neuromodulation (29).

**Conclusion**

It is conceivable that PRF may provide long-term pain relief in cases of sural nerve injury. The exact mechanism of the antinociceptive effect is still unknown.

**References**


