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SPECIAL SUPPLEMENT

• Spinal Cord Stimulation for Treatment of Chronic Regional Pain Syndrome

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• Clinical Case Report No. 2:

Development of CRPS After Hammertoe Surgery

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Severe Neuropathic Pain Caused by Tarsal Tunnel Syndrome

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Keywords: Spinal cord stimulation, dorsal column stimulator, chronic regional pain syndrome, reflex sympathetic dystrophy, neuroma, stump neuroma, tarsal tunnel syndrome, hammertoe surgery

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Spinal Cord Stimulation for Treatment of Chronic Regional Pain Syndrome

The benefits of and keys to using an implantable spinal cord stimulator to relieve neuropathic pain.

Diagnosing and treating neuropathic pain can be very difficult for healthcare providers, and even more frustrating for patients. Management of neuropathic pain varies widely, and options include oral adjuvant and topical analgesics, opioids, physical therapy, and regional anesthesia.

One of the most effective methods is a form of neuromodulation called spinal cord stimulation (SCS). SCS is a reversible procedure that is effective in reducing pain and beneficial in restoring function to the affected extremities. This article provides background on Chronic Regional Pain Syndrome (CRPS) and its treatments, and explores the benefits and practicalities of using spinal cord stimulation to treat patients suffering from this type of neuropathic pain.

Causes of Neuropathic Pain

Neuropathic pain of any origin can be debilitating to patients. Especially frustrating to patients is that neuropathic pain is one of the most difficult conditions to treat, and may require repeat office visits and extensive adjustments in medications.

Common causes of neuronal injury that cause neuropathic pain include complex regional pain syndrome types I and II, diabetic peripheral neuropathy, phantom limb pain, postlaminectomy syndrome, compression and nerve entrapment syndromes. However, neuropathic pain can also occur in the absence of any injury or known cause. Neuropathic pain may result from a lesion in any part of the nervous system.[1] It can involve uninjured adjacent neurons leading to peripheral sensitization and increased excitability of the brain and spinal cord due to central sensitization.

Typical manifestations of neuropathic pain include a burning sensation, throbbing pain accompanied by allodynia (pain with light touch), hyperalgesia (exaggerated pain from pinprick), and sensory deficits.[2]

Complex Regional Pain Syndrome Primer

The terminology of CRPS, formerly known as Reflex Sympathetic Dystrophy (RSD), was revised in 1994 by the International Association for the Study of Pain.[3] The following is a brief overview.

CRPS Type I (RSD) includes the following clinical findings: allodynia (such as sensory changes), regional pain, temperature abnormalities, edema, skin color alterations, and abnormal sudomotor activity. CRPS Type II (causalgia) includes all of the above findings along with a definitive peripheral nerve lesion. It is also important to note that not every criterion will be present in all patients with CRPS.

While the pathophysiology of the disease is not well understood, pain remains essential to the diagnosis. Therefore, it is important to consider the possibility of CRPS when treating a patient who has pain that is disproportionate to the inciting event. CRPS usually occurs after an inciting event such as surgery or an episode of trauma. It is important to note that CRPS has been reported event after trivial events such as venipuncture. In fact, there have been documented cases in which the patient may not even remember the event, because it seemed at the time such a minor, everyday occurrence.

Exclusion criteria for CRPS preclude patients with clinical findings that are temporarily proportionate anatomically and physiologically to an injury.[4]

Methods for Managing Neuropathic Pain

Recognizing neuropathic pain is the first step in treatment; attempting to medically manage the condition is often the second. Medications that have been used to successfully treat neuropathic pain include the adjuvant analgesics. These medications are referred to as adjuvant

analgesic because their primary indication is to treat conditions other than pain, such as depression or epilepsy. These medications include anti-epileptic drugs (such as gabapentin and pregabalin), tricyclic anti-depressants (such as amitriptyline and desipramine), and newer serotonin-norepinephrine reuptake inhibitors (such as duloxetine, venlafaxine).

Opioids such as tramadol are also believed to be effective when used in combination with other medications and aggressive physical therapy. The use of opioids in neuropathic pain was initially controversial, but is now recommended as first-line treatment for neuropathic pain.[1,5]

Topical analgesics such as capsaicin cream have been shown to be beneficial by depleting substance P from the peripheral terminals of sensory nerve fibers.[4,6] Selective serotonin reuptake inhibitors, on the other hand, have been shown to be no more effective in the treatment of neuropathic pain than placebo in patients who are not clinically depressed.[7,8]

In addition to medical management, mobilization of the affected extremity is essential in the treatment. The role of the physical therapist should not be underestimated when treating a CRPS patient because immobilization of the affected extremity is counterproductive.

Regional anesthesia as well as topical analgesics may be used in helping restore function to an effected extremity. Sympathetic blocks are useful in identifying pain that is sympathetically mediated. The procedure is performed by placing the tip of the needle under fluoroscopic guidance anterior to the L2 or L3 vertebral bodies. A local anesthetic is injected to interrupt the transmission of the lumbar sympathetic nerves. Objective determination of a successful block includes findings of vasodilation and increased temperature in the effected extremity.

If a patient reports reduced pain after the procedure, then the pain is classified as being sympathetically mediated. Sympathetic blocks performed before physical therapy may aid in facilitating mobilization of the effected extremity and greater participation in physical therapy. A series of lumbar sympathetic blocks are continued until either the patient receives resolution of their pain or no longer demonstrates continued benefit.

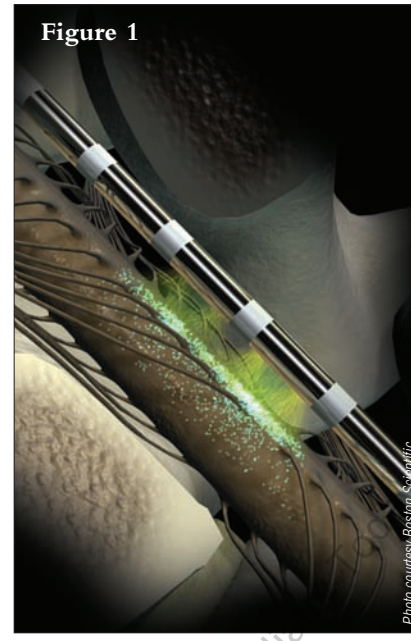

Figure 1. The implantable spinal cord stimulator masks pain signals by delivering pulses of electrical current directly to the spinal cord, thereby targeting pain in multiple areas.

History of Spinal Cord Stimulation

Neuromodulation is a scientifically proven treatment used to alter the electrical signals sent to the brain and spinal cord. SCS, a form of neuromodulation, masks the pain signals by delivering pulses of electrical current directly to the spinal cord, thereby targeting pain in multiple areas (**see Figure 1**).

The use of SCS was first reported by Shealy in 1967.[9] Since then, the device has been used to treat a variety of painful conditions. SCS has been widely applied to a variety of neuropathic pain conditions over the past 30 years. Clinical studies show it is a safe, effective therapeutic modality for reducing chronic refractory pain of the trunk and limbs.[10]

The spinal cord stimulator itself is an implantable medical device that both generates and transmits an electrical impulse, thus creating an electrical field that stimulates the dorsal column fibers in the spinal cord. These electrical impulses convert pain signals into paresthesias, which are perceived by the patient as a tingling, pleasant sensation.

The potential advantages of SCS include the reduction or elimination of medication use in addressing patients' pain. In addition, SCS is a reversible procedure that is effective in not only providing anaglesia but also restoring function to the affected extremities.[4]

SCS Mechanism of Action

A variety of theories have been postulated as to the mechanism of action of spinal cord stimulation. In the gate-control theory, first published by Melzack

and Wall in 1965,[11] the authors postulated the existence of a proverbial gate in the dorsal horn of the spinal cord. This so-called gate controls the transmission of neural activity.[12] By stimulating the dorsal horn and thereby activating the large diameter afferent nerve fibers, the gate could be closed, resulting in suppression of painful inputs to the central nervous system.[12]

While the exact mechanism of SCS is still debated, recent studies have suggested that the effects of SCS are mediated by multiple mechanisms and are effective in neuropathic, sympathetically mediated pain and ischemic pain states.[12] SCS is thought to affect the neurotransmitters in the central nervous system, activate supraspinal circuits, modulate the spinothalamic tracts, suppression of sympathetic activity and activation of the descending inhibitory pain pathways.[13]

Indications for Use of SCS

SCS is indicated for pain in the trunk and limbs. It has been shown to be effective in painful conditions such as radicular pain syndromes, failed back surgery syndromes, and CRPS.[14] In addition to neuropathic chronic pain conditions in the trunk and limbs, other applications for SCS have been cited in the literature.[14-16]

Surgical Technique

A screening trial that lets the patient test drive the spinal cord stimulator device is performed before permanent implantation of the device. The trial can be done in the office under light intravenous sedation and local anesthesia while the pain medicine physician percutaneously inserts the leads. The trial leads are removed after the trial period regardless of their outcome. This lets the treating physician and patient adequately assess the benefit derived from the trial before proceeding to surgical implantation. Hence, removing the percutaneous leads makes the trial a reversible procedure and helps address patients' pre-operative concerns.

The patient is placed in a prone position. A 14-gauge epidural needle is typically inserted into the upper lumbar epidural space. The spinal cord stimulator leads are then inserted through the needle and advanced cephalad into the thoracic epidural space under fluoroscopic guidance. For the lower extremity, the lead tip is typically placed between T10 and T11. For bilateral leg pain, two leads are typically inserted. Each lead has eight contacts.

During the trial, the leads are taped to the skin and connected to an external battery via an extension cable (see **Figure 2**). The goal is to overlap the paresthesias produced by the device with the patient's painful areas. The device representative uses an external programmer to adjust the voltage, pulse width and frequency (see **Figures 3 and 4**). The patient is given several programs to choose from, the trial phase typically lasts between 3 and 7 days, which lets the patient assess the effectiveness of the stimulation under normal circumstances and in normal surroundings.[13] The patient is able to turn on and off the device using a wireless remote control. Upon completion of the trial, the patient returns to the physician's office to have the leads removed.

A trial is considered successful if a

Figure 2

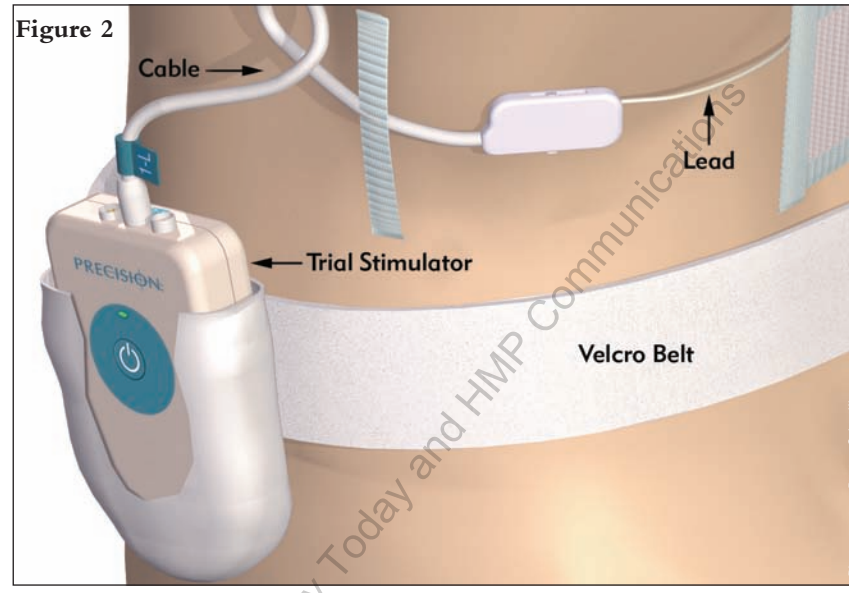

Figure 2. A screening trial lets the patient test drive the spinal cord stimulator device before permanent device implantation. During the trial, the leads are taped to the skin and connected to an external battery via an extension cable.

Figure 3

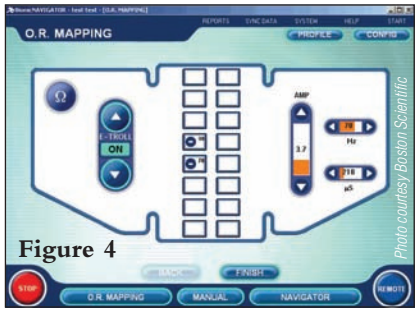

Figure 4

Figures 3 and 4. Once the trial device has been attached, the device representative uses an external programmer to adjust the voltage, pulse width and frequency. The patient is able to access several programs over the 3- to 7-day trial phase.

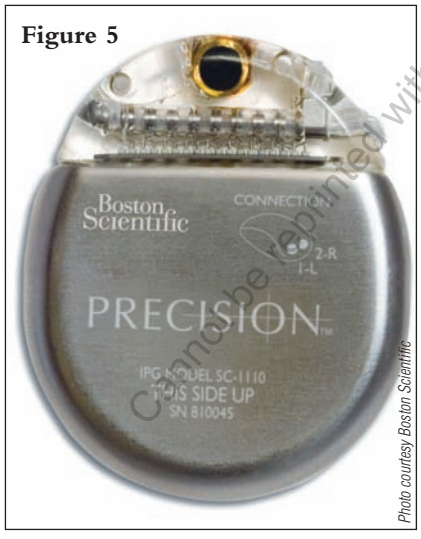

Figure 5

Figure 5. During surgical implantation, a subcutaneous pocket is created in the gluteal area for the impulse generator (battery). The leads are then tunneled to the pocket and connected to this rechargeable battery.

50% reduction in the intensity of the pain is achieved. If the trial is successful, the patient is then scheduled for implantation of the device in an operating room as an outpatient procedure. Implantation of the SCS can be performed either percutaneously or open. Open surgical placement involves the use of a paddle lead placed through an open laminotomy incision.

For permanent lead placement, a skin incision is made at the lead insertion site. Once the leads are placed in the epidural space, they are anchored to the fascia or supraspinous ligament. A subcutaneous pocket is created in the gluteal area for the impulse generator, or battery. The leads are then tunneled to the pocket and connected to this rechargeable battery (see **Figure 5**).

Summary

Spinal cord stimulation has a high initial acquisition cost, but reductions in medication usage, physician visits, emergency room visits, absence from work, surgeries, and additional hospitalizations offset overall costs.[13,17] Many studies have shown spinal cord stimulation is cost-effective in treating pain in comparison to other conservative therapies; Bell et al show SCS pays for itself within 2.1 years.[17,18]

In the following pages, we explore three real clinical cases in which SCS was used to treat different CRPS situations: after neuroma surgery (see p. 7); after hammertoe surgery (see p. 10); and after failed tarsal tunnel (see p. 12). ■

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Clinical Case Report No. 1: Residual Neuropathic Pain After Neuroma Excision

A patient who was successfully treated by spinal cord stimulation for a recurrent neuroma and residual neuropathic pain after previous surgical resection

Morton's intermetatarsal neuroma is a compression neuropathy that most commonly involves the digital nerves. The patient history, clinical findings and treatment guidelines have recently been published and are based on a consensus of current clinical practice and a review of the clinical literature. The guidelines were developed by the Clinical Practice Guideline Forefoot Disorders Panel of the American College of Foot and Ankle Surgeons.[1]

Neuromas are most commonly found in the 3rd interspace, but can occasionally occur in the other spaces as well. The patient history will typically describe a burning or tingling sensation and possible numbness that involves the associated digits. The patient may describe a "hot poker" or "wrinkle in the sock" sensation that is exacerbated in shoe gear and with ambulation. Relief is often felt upon removal of the shoe and with massage of the area.

The clinician will commonly be able to elicit pain and a palpable click (Mulder's sign) with manipulation of the interspace. X-rays are needed on the initial visit to exclude any bony pathology and MRI may be considered to aid in the diagnosis. Devices such as ultrasound and pressure-specified sensory device (PSSD) can be very helpful if available to the clinician.

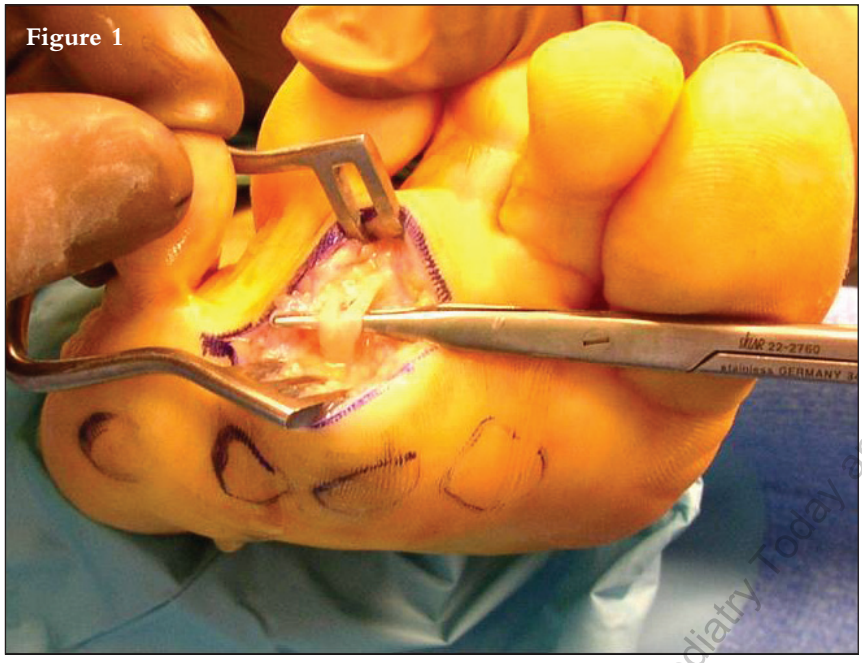
The diagnosis is made clinically; other ancillary tests should be reserved for atypical presentations or to exclude other types of pathology. Pathological conditions that mimic Morton's neuroma include stress fracture, metatarsal phalangeal joint bursitis, metatarsal phalangeal joint instability or predislocation syndrome (PDS), neoplasm, metabolic peripheral neuropathy, or other chronic pain syndromes.

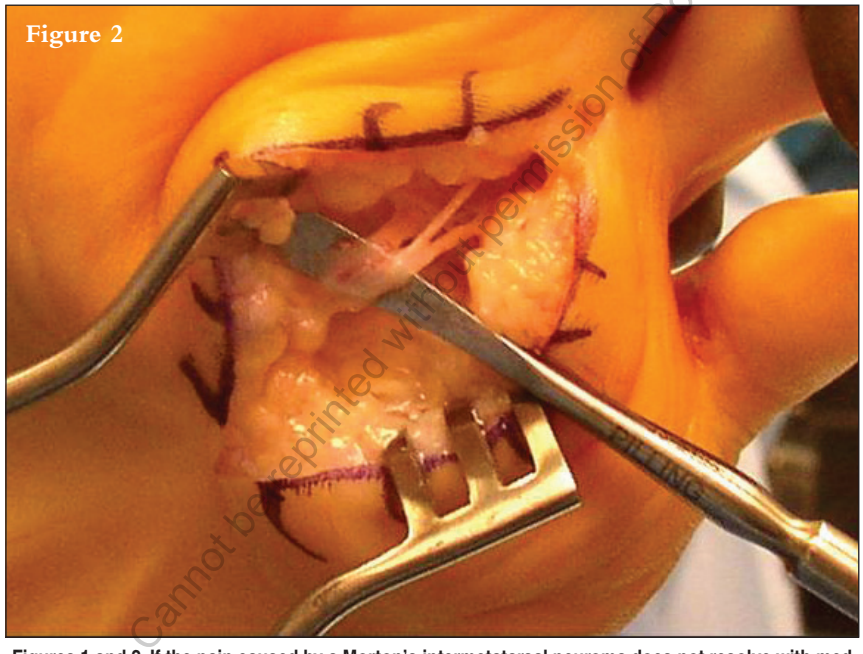
Initial treatment consists of the use of wider shoes, metatarsal padding, anti-inflammatory medications and injections of either steroids or 4% alcohol. If the pain does not resolve, the most common surgical procedure is excision of the neuroma (see **Figures 1 and 2**). Decompression surgery and cryogenic neuroablation are becoming more common in attempts to decrease the complications associated with resection such as, stump neuroma, seroma, wound dehiscence and infection.

Recurrent or stump neuroma can be a challenging entity for the clinician. It has a recurrence rate of 3% to 24% after dorsal excision,[2–12] and patients who have failed a primary nerve resection are often hesitant to undergo further surgery. The symptoms of recurrent neuroma can often overlap with multiple etiologies including orthopedic and neuropathic pain conditions.

Attempts have been made to decrease the incidence of recurrence with conservative and surgical techniques. More conservative treatments include injections (steroids or 4% alcohol), massage, ultrasound, orthotics, NSAIDS, and adjuvant analgesics. During initial surgery, the severed nerve is relocated into a local muscle belly during the initial surgery. This may aid in preventing localized trauma to the nerve, but there has been no proven treatment to prevent "regrowth" of the nerve.[13] Additional surgical treatment is commonly performed via plantar excision with further resection and muscular implantation with the possible need of an additional tarsal tunnel release.[13]

Pain and numbness to the plantar foot, under or between the metatarsal heads is the primary complaint. Accurate clinical diagnosis is paramount to successful treatment; consultation with a pain management physician may aid in accurate diagnosis and treatment. If





Figures 1 and 2. If the pain caused by a Morton's intermetatarsal neuroma does not resolve with medical management, the neuroma can be surgically excised.

there is any doubt that the pain is neurologically induced, one can consider an MRI for evaluation of a stress fracture or bursitis to the associated structures.

The neuroma is often located in close proximity to either the 3rd or 4th metatarsal head. PDS must be ruled out. Occasionally, a patient will present with PDS after nerve resection or due to possible misdiagnosis of PDS before the initial nerve resection. Palpation in the area will elicit pain in both recurrent neuroma and PDS, but the clinical history is quite different, thus the clinical history is essential in making the diagnosis.

Neuroma and recurrent neuroma pain are significantly reduced when the patient is barefoot or wears wide shoes, whereas PDS is often most painful when barefoot. A simple clinical test is to strap the toe in a slightly flexed position with a cross-tape or by buddy splinting the associated toes. Recurrent neuroma diagnosis can be aided by use of a PSSD.[13] Other neurological problems that may complicate the diagnosis of recurrent neuroma are diabetic peripheral neuropathy, idiopathic peripheral neuropathy, tarsal tunnel syndrome, and radiculopathy.

The following case reports a patient who was successfully treated by spinal cord stimulation for a recurrent neuroma and residual neuropathic pain after previous surgical resection.

Patient and Case

A 31-year old woman presented to the office 9 months after nerve resection for 2nd and 3rd interspace neuromas, originally performed by her orthopedic surgeon. Her pain was improved for the first 2 months. However, she had a recurrence of pain that worsened 3 months after the surgery.

The patient tried physical therapy and a TENS unit, both of which failed

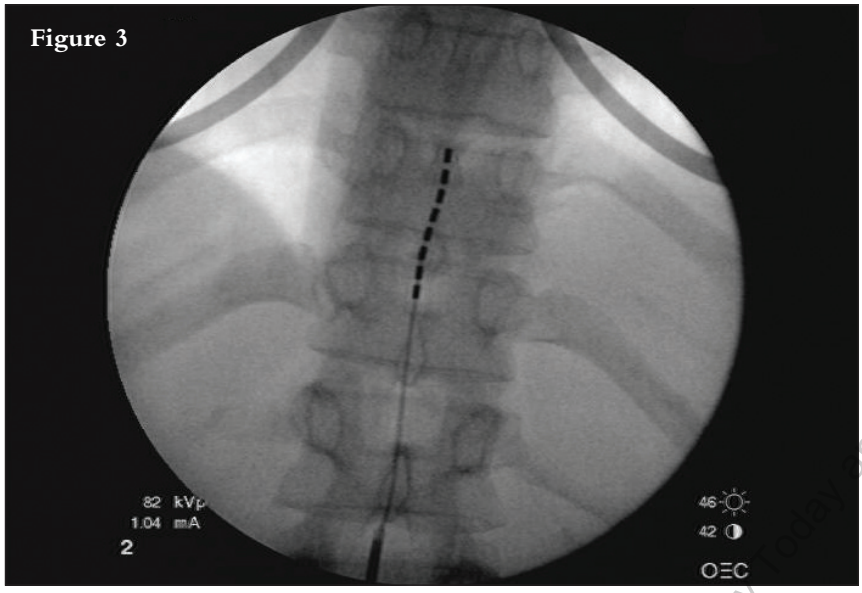


Figure 3. A 31-year-old woman who could not gain pain relief from more conservative treatments — such as orthotics and anti-neuropathic medications — for neuropathic pain after neuroma excision underwent permanent implantation of a spinal cord stimulator. After 8 months, she was able to discontinue all pain medications.

to improve her pain. She was then started on a combination of anti-neuropathic medications — gabapentin and nortriptyline — without relief. A series of local steroid injections and peripheral nerve blocks provided only temporary relief.

Initial presentation was for "foot pain after surgery." The patient's history also revealed lower back pain. Localized tenderness plantar and slightly lateral to the 3rd metatarsal head was observed with "shooting pain" proximally. X-ray findings were negative for osseous pathology. A tentative diagnosis of recurrent neuroma was made and the patient was instructed to use pregabalin 75 mg PO qhs for 3 days then BID on day 4. Over-the-counter orthotics were customized to offload the area, and she was given a prescription for an MRI. She was then instructed to follow up in 2 weeks.

The patient denied any relief with orthotics or with pregabalin. Her MRI was negative for stress fracture or bursitis, but showed inflammation to the plantar foot between the metatarsal heads. The patient refused any further invasive procedures and was referred to a pain management physician for further care.

The patient was then initiated on duloxetine, hydrocodone and diclofenac, all of which failed to improve her symptoms. A lumbar sympathetic block also failed to alleviate her symptoms. After failing conservative treatment and continuing to suffer from lower extremity pain, the patient agreed to a 5-day spinal cord stimulator trial using the Precision Plus™ (Boston Scientific Corporation, Valencia, CA).

During the trial, the patient reported greater than 80% reduction in her pain. She underwent permanent implantation of the spinal cord stimulator (see **Figure 3**). At the 8-month follow-up, the patient has been able to discontinue all pain medications. For exercise, she has even returned to her prior routine of long-distance running. ■

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Clinical Case Report No. 2: Development of CRPS After Hammertoe Surgery

A patient who was successfully treated with spinal cord stimulation for neuropathic pain resulting from a second hammertoe

Hammertoe correction is a part of every surgical practice. These digital contractures can occur in all three planes of motion including the frontal, sagittal, and transverse planes.[1] Initial classifications of the digital deformities we often encounter will include claw toe, mallet toe, curly toe, and the classic hammertoe. The specific term to be used depends on the location of hyperextension and hyperflexion. With the classical hammertoe deformity,[2] we see hyperextension at the metatarsal phalangeal joint as well as at the distal interphalangeal joint and an associated hyperflexion at the proximal interphalangeal joint.

The etiology of the deformity includes intrinsic musculature imbalance, length irregularities of the respective lesser metatarsals, an elongated plantar plate, progressive contracture secondary to an arthropathy and the list goes on from there. From a biomechanical standpoint, we often refer to the causes of hammer toes to be flexor stabilization,[3] extensor substitution,[4,5] or flexor substitution.[1] Regardless of the etiology, when digital deformities lead to pain or tissue breakdown, they must be addressed.

Treatment generally falls into two categories: conservative care and surgical intervention. Conservative options, especially with a rigid digital contracture, are often limited to padding and shoe modifications. While different manufacturers have produced more varieties of pads then most of us will ever use, their goal is often the same: alleviating pressure from the area of irritation. A biomechanical argument for orthotics can always be made when addressing conservative treatment of hammertoes. If conservative measures fail to adequately address the problem and the patient is a surgical candidate, then surgical intervention is a viable option.

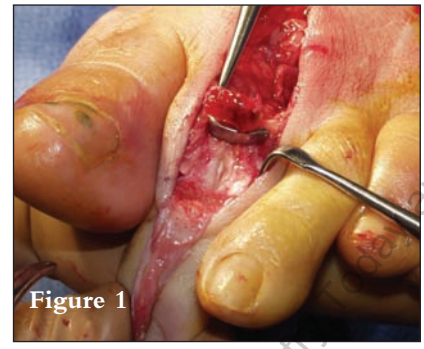
Figure 1

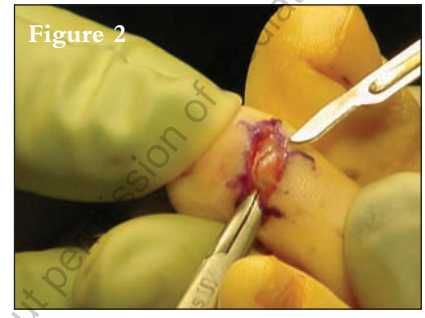
Figure 2

Figures 1 and 2. Many surgical options are now available for hammertoe, including digital arthroplasty, digital arthrodesis, tendon transfers, and tendon lengthening.

Many surgical options are now available (see **Figures 1 and 2**). Surgical options include digital arthroplasty, digital arthrodesis, tendon transfers, tendon lengthening, flexor tenotomies, capsular rebalancing, capsulotomies, plantar plate procedures, and implant arthroplasties, as well as various combinations of those procedures.

As with any surgery, there is a chance of complications — hammertoe surgery is no exception. Surgical complications of hammertoe surgery were well outlined by Judge.[6] Neuritis was discussed among them; however, Complex Regional Pain Syndrome (CRPS) was not, because it is not generally considered to be a complication specific to or common with digital surgery.

The following case reports on a patient who was successfully treated with spinal cord stimulation for neuropathic pain (due to CRPS) resulting from a second hammertoe surgery.

Patient and Case

A 39-year old female presented to the clinician with a painful second hammertoe. Initial conservative measures included padding, digital splints and shoe modifications. Unfortunately, the patient failed to respond favorably to conservative care

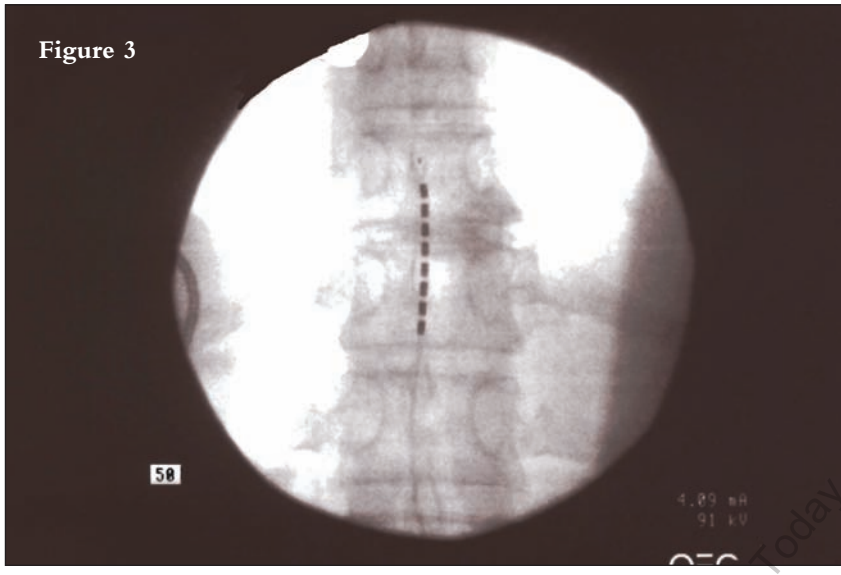

Figure 3. After implantation of the permanent spinal cord stimulator with a single lead at T-10, the patient no longer needs any medications to address her pain.

and progressed on to surgery.

The isolated digital surgery went well and the patient initially appeared to be recovering in an uneventful manner. Unfortunately, approximately 3 weeks post-operatively, the patient called requesting "more" and "stronger" pain medications. Given the abrupt change in the patient's desire for additional opioids, she was instructed to return to clinic where she could be re-evaluated.

The initial clinician concern was for the potential for abuse of the prescription pain medication. What was instead discovered upon her arrival was an even worse case scenario. The previously ambulatory patient presented to the clinic on crutches. During the evaluation, the patient was noted to be guarding the foot. She was also noted to have edema to the second digit as well as the entire forefoot. Examination revealed a temperature difference between the affected and the contra-lateral lower extremity.

She was educated on concerns related to neuropathic pain and the probability of CRPS. Initial medical management included additional opioids and an escalating dose of pregabalin. After an extended discussion with the patient, she revealed that she previously suffered from "something similar" when she had injured her upper extremity, but related that the physician had called it "something different" at the time. When the term reflex sympathetic dystrophy (RSD) was mentioned, she immediately related that to be the condition for which she had been treated after what turned out to be a lower-trunk brachial plexopathy.

Given the prior history of CRPS, this patient was immediately referred to a pain medicine practitioner for further treatment. The patient was initiated on pregablin and duloxetine by the pain management physician. Three separate lumbar sympathetic nerve blocks where performed. Each block was performed one day before physical therapy to facilitate patient participation.

The patient initially reported a 60% reduction in pain following the sympathetic nerve blocks, but the favorable results where not sustained for any significant duration. The sympathetic nerve blocks where discontinued because the last procedure failed to provide even temporary reduction in her pain. Additional medical management included oxycodone, methadone, hydrocodone, and clonazepam, all without benefit to this patient.

The patient then underwent a 7-day spinal cord stimulator trial, using Precision Plus™ (Boston Scientific Corporation, Valencia, CA). The patient reported a 90% reduction in her foot pain during the trial and subsequently progressed to implantation of a permanent spinal cord stimulator (see **Figure 3**) with a single lead at T-10. She has since returned to all activities and no longer needs any medications to address her pain. At 10-month follow-up, the patient continues to maintain reduction in pain and remains a productive, working member of society. ■

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Clinical Case Report No. 3: Severe Neuropathic Pain Caused by Tarsal Tunnel Syndrome

A patient who was successfully treated by spinal cord stimulation after tarsal tunnel decompression failed.

Tarsal tunnel syndrome refers to compression of the posterior tibial nerve as it courses along the posterior-medial aspect of the ankle and into the foot. Keck and Lam were the first to describe the condition in 1962.[1,2] The classic symptoms include paresthesias along the medial aspect of the heel associated with a burning sensation into the ankle. Pain is often aggravated by prolonged standing or walking and frequently includes pain at night. Upon examination, the clinician can elicit a positive Tinel's sign with percussion of the tibial nerve. In rare cases, a space-occupying lesion may even be palpated.

Accurate diagnosis of tarsal tunnel syndrome can be challenging. Many conditions mimic findings of tarsal tunnel syndrome. Examples of these conditions include lumbosacral radiculopathy and peripheral neuropathy. Although controversial, electrodiagnostic studies are still the most common objective test used for evaluation. The use of a pressure-specified sensory device (PSSD) has become an alternative method of nerve conduction testing.[3] An MRI should be ordered before treatment to evaluate for a space occupying lesion as well as to exclude entities such as intrafascicular ganglions[4] and neurilemmomas,[5] which may mimic tarsal tunnel symptoms.

Tarsal tunnel syndrome may be the result of both intrinsic and extrinsic factors. Intrinsic factors include overpronation resulting in a traction injury or direct compression within the flexor retinaculum. Extrinsic factors include lumbosacral radiculopathy, diabetic neuropathy, ganglions, varicosities, and other space-occupying lesions.

Conservative care should always be attempted when possible. This may include orthotics, injections, anti-inflammatory medications, and physical therapy. Obvious exceptions to prolonged conservative measures would include malignancy or a large space-occupying lesion causing direct nerve compression, as expedient surgical intervention would be indicated.

Surgical decompression (see **Figures 1 and 2**) is associated with a high rate of complications. Gundring reported that 100% of patients developed positive Tinel's signs and abnormal nerve studies after surgery.[6] Raikin also noted that revisional tarsal tunnel surgery rarely yielded additional benefit to the patient.[7]

Chronic tarsal tunnel pain that fails to respond to conservative treatment or surgical intervention poses a significant obstacle to both the patient and physician. Extrinsic factors such as diabetic neuropathy or lumbosacral conditions can be of such severity that a tarsal tunnel decompression provides only minimal benefit to the patient — and may even create an exacerbation of their pain. In these cases, a timely referral to an interventional pain management specialist for spinal cord stimulation might be indicated.

The following case reports a patient who was successfully treated by spinal cord stimulation to alleviate neuropathic pain when surgery for tarsal tunnel syndrome failed.

Patient and Case

A 57-year-old female with multiple comorbidities, including diabetes mellitus, obesity and fibromyalgia, presented to the office with severe pain in both feet. She was initially treated by her primary care physician for diabetic neuropathy and by another podiatrist, who performed cortisone injections, initiated physical therapy, and provided biomechanical support.

Despite this care, she denied any improvement in her intensity of heel pain

Failed Tarsal Tunnel Surgery

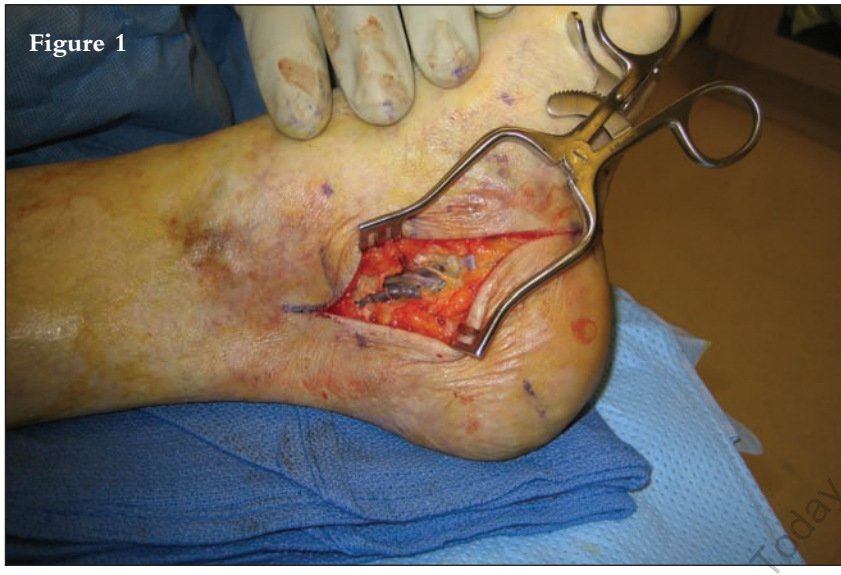

Figure 1

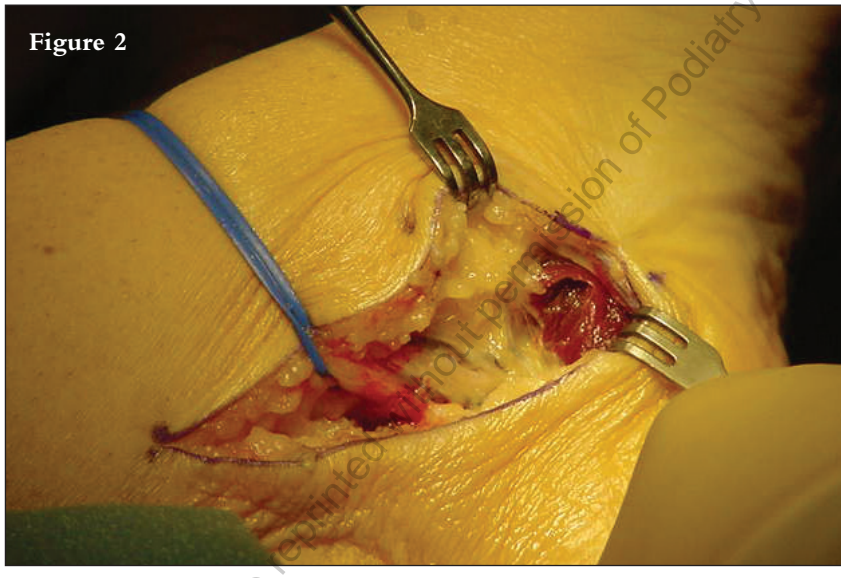

Figure 2

Figures 1 and 2. Surgical decompression is associated with a high rate of complications: One study reports that 100% of patients developed positive Tinel's and abnormal nerve studies post-surgery.

and continued to describe a burning and tingling sensation in her feet. After a historical review and physical examination, the initial working diagnosis included heel spur syndrome, tarsal tunnel syndrome, and diabetic neuropathy.

Over the next 5 months, conservative care included CAM Walker immobilization, anodyne treatments, additional physical therapy, a home TENS unit, anxiolytics, nutritional and diabetic education, and opioids. Despite the additional intervention, the patient was unable to ambulate for longer than 20 minutes at a time. A nerve conduction study revealed severe diabetic peripheral neuropathy with tarsal tunnel syndrome and infracalcaneal nerve entrapment.

The patient continued to describe bilateral foot pain, but her left foot remained most symptomatic. Physical examination revealed a positive Tinel's sign. She also demonstrated a gastrocequinus deformity and heel pain at the insertion of the plantar fascia with associated infracalcaneal nerve pain.

She underwent surgical intervention to the left foot including gastrocnemius recession, tarsal tunnel decompression, and partial plantar fascietomy with decompression at the infracalcaneal nerve. She initially reported significant pain reduction to the heel but continued to have bilateral pain associated with her diabetic neuropathy and fibromyalgia.

Her neuropathic pain as well as her left lower-extremity pain remained well controlled over the following year, but she developed progressive pain in her right Achilles tendon. Radiographs showed a large retrocalcaneal spur to the Achilles tendon and insertional calcification of the Achilles tendon. MRI did not reveal a tear to the tendon. After 6 months of conservative care, she underwent surgical intervention including a gastronemius recession, posterior calcaneal spur resection, Achilles detatchment, debridement and reattachment with screw and washer.

She progressed well after surgery, including removal of her hardware 6 months after surgery. Nine months after the surgery, she was doing well from a post-operative pain standpoint, but developed a loss of balance, possibly associated with her progressive diabetic neuropathy.

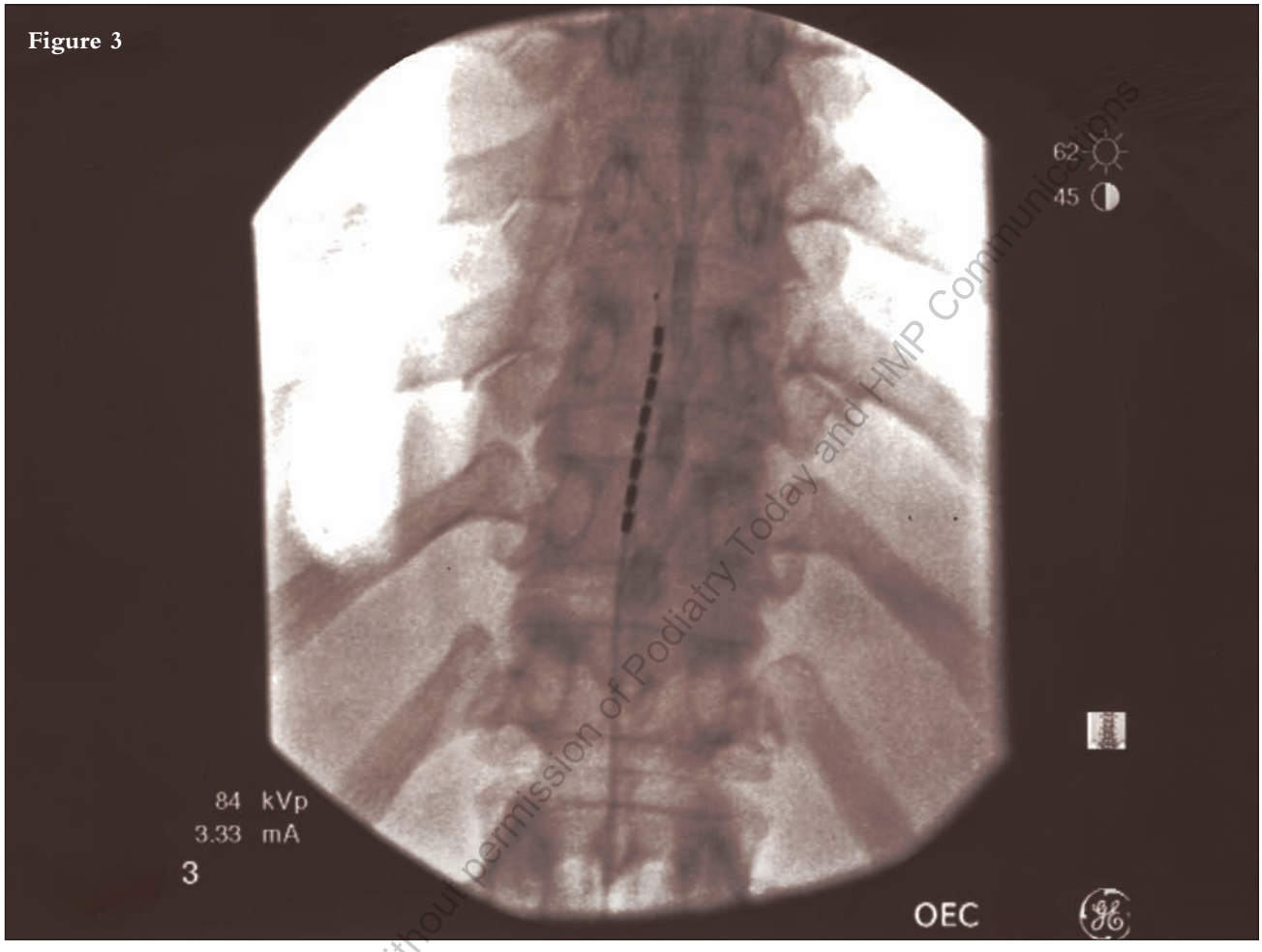

Figure 3. Two weeks after the trial, the patient underwent implantation of the device with dual spinal cord stimulator leads. Two years later, she continues to use the spinal cord stimulator and has been able to discontinue the majority of the pain medications.

Her left footVAS was originally reported as 10/10, now a 2/10. The right foot also improved from the original 8/10 to 2/10. Her neuropathic and fibromyalgia pain remained under the care of her primary care physician.

She was referred to physical therapy for her loss of balance and underwent gait therapy and anodyne treatments. Several weeks into this therapy she presented with burns on her right foot and ankle from the anodyne machine. She was treated with local wound care, and the wounds healed.

The patient subsequently missed several of her scheduled appointments for personal reasons and after a 3-month gap, presented with severe foot pain. She denied benefit with anodyne therapy and related an exponential increase in the intensity of her pain to both feet after the burn from the therapy. Clinical presentation included bilateral edema, an antalgic gait and loss of balance.

Upon lower-extremity physical examination, the patient was noted to demonstrate pain with light touch to both heels. She also demonstrated limited ankle joint range of motion due to the swelling of her legs. A venous Doppler was ordered and did not demonstrate the presence of a deep venous thrombosis to either lower extremity.

NSAIDs and opioids were prescribed and adjustments were made to her anxiolytic medications. Despite this treatment, she returned with severe burning and tingling in both feet. Her pain was reported to be worse at night, limiting

her ability to sleep for any duration.

Clinical examination was noted to demonstrate allodynia and hyperalgesia to the bilateral heels with muscle weakness and peripheral edema. A strong suspicion of chronic regional pain syndrome (CRPS) was documented, and the patient was referred to an interventional pain medicine physician. Upon examination of the patient, the diagnosis of CRPS was confirmed. The patient failed medical management and ultimately went on to receive a spinal cord stimulator.

The patient was initiated on baclofen to address lower-extremity muscle spasms and celecoxib to address pain secondary to osteoarthritis. Additional medical management also included the following anti-neuropathic medications: duloxetine, venlafaxine, gabapentin, and pregabalin, which all failed to alleviate her pain. Opioids where discontinued due to intolerable side effects.

After failing conservative treatment, the patient underwent a 5-day spinal cord stimulator trial. During the trial the patient was able to ambulate without difficulty. She also reported improved sleep and a 95% reduction in the intensity of her pain. Two weeks after the trial, the patient underwent implantation of the device with dual spinal cord stimulator leads (see **Figure 3**).

The patient continues to use her spinal cord stimulator 2 years after implantation and has been able to discontinue use of the majority of the pain medications. As demonstrated in the patient described in this case report, implantable SCS can provide a safe modality for the treatment of chronic pain in the lower extremity, and may be an option that is under used by the podiatric profession. ■

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