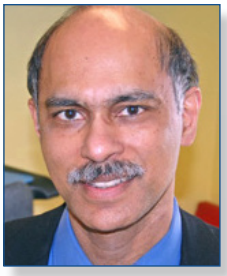
RHODE ISLAND
MEDICAL JOURNAL


Jon A. Mukand, MD, PhD

Pain Management Strategies & Therapeutic Options in the Rehabilitation Setting

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GUEST EDITOR

A variety of neurological and musculoskeletal conditions are treated in the rehabilitation setting, and people with disabilities are vulnerable to myriad causes of pain. Musculoskeletal structures (bone, ligaments, tendons) are often damaged in weight-bearing joints affected by osteoarthritis and in small joints of the hand and wrist in rheumatoid arthritis. Amputations often result in pain at the residual limb (due to neuromas and skin problems) as well as phantom pain in the part of the limb that has been removed. Disc disease of the spine can compromise small joints in the spinal column and also compress nerve roots. Patients with brain injuries are vulnerable to headaches. After a stroke, patients are at risk of the "hemiplegic shoulder" as well as painful contractures. These and other painful conditions lead to problems with function, sleep, and depression as well as a general decline in quality of life.

Treating pain in the rehabilitation setting often requires a combination of approaches: modalities such as ice, heat, electrical stimulation, and ultrasound; oral, topical, or intra-articular pharmacotherapy; neuromodulation with implanted neural stimulators; and referral for surgical procedures such as arthroscopies and arthroplasties. The authors who have contributed to this special issue of the *Rhode Island Medical Journal* provide a wide range of therapeutic options for pain management to help patients with disabilities. As we contend with the opioid epidemic, these options for optimal pain therapies need to be fully explored.

DR. JACOB MODEST and his co-authors note that the prevalence of post-amputation pain is rising, and almost all people with amputations experience residual limb and/or phantom pain. They discuss the mechanisms and review medication options (tricyclic antidepressants, gabapentin, opioids – with precautions, ketamine, local anesthetics); rehabilitation therapies including mirror visual feedback; cognitive behavioral therapy; and surgical approaches such as neuromodulation and surgery for neuromas.

ALEX HAN (Brown University medical student) and **DR. ALEXIOS G. CARAYANNOPOULOS** review spinal cord stimulators for chronic neuropathic pain disorders, including failed back surgery syndrome (FBSS), complex regional pain syndrome, neuropathy, and radiculopathy. A growing body of literature supports these minimally-invasive stimulators as a safe therapeutic option that can reduce pain and disability, especially in difficult cases of neuropathic pain that are complicated by medication side effects, drug tolerance, or the risks of re-operation.

DR. SHASHANK DWIVEDI and his co-authors (residents and attendings in Orthopedic Surgery at Rhode Island Hospital) have contributed articles on surgical approaches for osteoarthritis and rheumatoid arthritis. Both articles describe the mechanism of damage to joints and surrounding connective tissue structures, which sets the stage for a variety of surgical treatments. The wide-ranging article on osteoarthritis discusses therapeutic options including aerobic exercise, resistance training, weight loss, nonsteroidal anti-inflammatory drugs (NSAIDs, both oral and topical), and tramadol. In addition, Dr. Dwivedi and his co-authors did a literature search and found evidence that suggests a lack of efficacy for glucosamine, chondroitin sulfate, hyaluronic acid, acupuncture, and lateral wedge insoles. This information will help with educating patients about the scientific basis for various remedies that often turn up in internet searches. The article on rheumatoid arthritis focuses on surgical treatment and methodically explores options (tendon transfers, arthroplasty, arthrodesis) for joint damage at the wrist, metacarpophalangeal joints, and inter-phalangeal joints.

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Management of Post-Amputation Pain

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ABSTRACT

INTRODUCTION: The prevalence of amputation and post-amputation pain (PAP) is rising. There are two main types of PAP: residual limb pain (RLP) and phantom limb pain (PLP), with an estimated 95% of people with amputations experiencing one or both.

MEDICAL MANAGEMENT: The majority of chronic PAP is due to phantom limb pain, which is neurogenic in nature. Common medications used include tricyclic antidepressants, gabapentin, and opioids. Newer studies are evaluating alternative drugs such as ketamine and local anesthetics.

REHABILITATION MANAGEMENT: Mirror visual feedback and cognitive behavioral therapy are often effective adjunct therapies and have minimal adverse effects.

SURGICAL MANAGEMENT: Neuromodulatory treatment and surgery for neuromas have been found to help select patients with PAP.

CONCLUSION: PAP is a complex condition with mechanisms that can be located at the residual limb, spinal cord, and brain — or a combination. This complex pain can be difficult to treat. The mainstays of treatment are largely medical, but several surgical options are also being studied.

KEYWORDS: post-amputation pain, phantom limb pain, residual limb pain, amputation

INTRODUCTION

Limb amputations cause a major physical, economic, and psychological burden to patients and can result in chronic, often debilitating post-amputation pain (PAP). The most common causes of an amputation include diabetes, peripheral vascular disease, and trauma.[1] Amputation is a common operation; the prevalence of lower extremity amputation in the United States in 2005 was 1.6 million people, with projections of 3.6 million people by 2050.[2] Diabetic patients have an over thirty-fold increased risk of an amputation compared to non-diabetic patients, at a cost of $4.3 billion annually in the U.S.[3] While there are many detrimental effects of amputation including post-operative surgical site infection, the need for revision amputation, economic cost, and physical disability, one of the most debilitating results is chronic PAP. Comorbid conditions such as fibromyalgia, migraines, irritable bowel syndrome, irritable bladder, and Raynaud syndrome have all been associated with chronic PAP.[4] The management of PAP is primarily medical, but several surgical options also exist.

PATHOPHYSIOLOGY

There are several methods to categorize PAP. Acute post-amputation limb pain lasts less than two months and chronic post-amputation limb pain lasts more than two months.[5] Residual limb pain (RLP), often unfortunately referred to as "stump" pain, is pain at the surgical site or proximal remaining extremity. Phantom limb pain (PLP) is described as pain localized distal to the amputation level.[6] Ephraim et al. looked at 914 patients with limb loss and found that 95% had either RLP or PLP, and there was also a significant relationship with depression symptoms.[7]

While both types of pain may coexist, RLP is most common in the acute post-operative setting, while PLP may present later and with great chronicity. RLP is due to a variety of reasons including incisional pain, scar formation, poor prosthetic fit, or neuroma formation. While RLP has a more acute onset after surgery, it also improves more over time than PLP does.[8]

PLP is usually described as burning, aching, or stabbing and it can occur in the first postoperative week in as many as 25% of patients.[5] A study of 96 people with upper limb amputations found that there were two peaks for the development of phantom limb pain: acutely within 1 month and at 12 months after amputation.[8] Risk factors for phantom limb pain include female sex, upper extremity amputation, presence of pre-amputation pain, residual limb pain, and time after amputation.[9]

The mechanism for PLP is thought to be due to cortical reorganization of the somatosensory cortex that serves the sensation of the amputated limb. MRI studies of patients with hand amputations have shown that the cortical area corresponding to the amputated hand is activated and influenced by forearm movements.[10] Another clear mechanism of pain is from nerve damage, as treatment with lidocaine injection or nerve stimulation has been effective in certain

cases. A confounding issue with many cases of PAP is that a significant percentage of patients with amputations have underlying diabetes and diabetic neuropathy.

MANAGEMENT OF POST-AMPUTATION PAIN

Initial and General Approach

After evaluating for anatomic, vascular, infectious, or other pathologic conditions of the residual limb, nonsurgical management is the preferred initial approach to treat PAP. A wide variety of strategies have been studied for the treatment of chronic phantom limb pain. Given that chronic PLP is a type of neuropathic pain, these investigated modalities are often similar to those used to treat other neuropathic pain conditions. These range from pharmacologic, behavioral and rehabilitative therapies to nerve stimulation. The evidence for favoring one medication or modality above another is lacking, and trial data for most studies are based on small numbers of patients. Treatment decisions are often based on patient characteristics and co-morbidities such as depression, as well as side effect profiles. Coupling pharmacologic therapy with other therapies is often practiced. Here, we will review the various non-operative strategies employed for the management of PAP.

PHARMACOLOGIC TREATMENTS

Several classes of medications have been explored as possible management options in chronic phantom limb pain, including antidepressants, anticonvulsants, N-methyl-D-Aspartate (NMDA) antagonists, calcitonin, calcium channel blockers, beta blockers, and local anesthetics. (**Table 1**)

Antidepressants are a commonly employed class of medications for the treatment of neuropathic pain. The tricyclic antidepressant amitriptyline has been evaluated in the treatment of phantom limb pain. A placebo-controlled study did not show any improvement in analgesia after six weeks of treatment, while a 2005 study reported positive results in controlling PAP with daily amitriptyline.[11,12] However, amitriptyline was shown to produce undesirable side effects including drowsiness and dry mouth, making it a less desirable pharmacotherapeutic.[11] Urinary retention and weight gain are other problematic side effects of amitriptyline. Limited case reports and case series have also documented possible benefit with the use of mirtazapine and duloxetine. The advantages of duloxetine (Cymbalta) are that it is FDA-approved for both depression and neuropathic pain which are associated with diabetes.

Gabapentin is an anticonvulsant that is often used for the management of neuropathic pain, and it has also been

Table 1. Medical options for treating post-amputation pain

Medication	Mechanism of Action	Benefit	Adverse Effects
NMDA-antagonists			
Ketamine	Blocking hyperexcitability and sensitization via NMDA-receptors	Short-term benefit	Mood derangements, hallucinations, abusable
Dextromethorphan		Possible benefit, evidence insufficient	Well-tolerated; may cause abdominal symptoms, confusion
Memantine		Possible benefit, evidence insufficient	Well-tolerated
Antidepressants			
Amitriptyline	Serotonin and norepi-nephrine reuptake inhibitor	Possible benefit	Anti-cholinergic side effects: dry mouth, sedation
Opioids			
Morphine	Opioid receptor agonist	Beneficial	Dependency, respiratory depression
Injections			
Botulinum Toxin A	Transient focal nerve blockade	Short-term benefit	Low incidence of systemic toxicity, injection site reactions
Anticonvulsants			
Gabapentin	Voltage-dependent calcium ion channel inhibition	Likely beneficial	Good safety profile; may cause sedation
Hormone			
Salmon Calcitonin	Inhibition of centrally firing neurons	Possible benefit, evidence insufficient	Good safety profile; may cause nausea, abdominal pain, diarrhea
Antihypertensives			
Nifedipine	Calcium channel blocker	Possible benefit, evidence insufficient	Hypotension, flushing, peripheral edema
Propanolol	Beta blocker		Sedation, hypotension, bradycardia
Clonidine	Alpha2-adrenergic agonist		Sedation, syncope, hypotension, rebound hypertension

employed to control pain after amputations. The current literature is mixed as to the effectiveness of gabapentin in the treatment of PAP, and it has been associated with significant side effects in this population.[12]

The misuse of opioids continues to pose a nationwide problem, but they can be a useful tool in the management of acute pain, as they blunt the somatosensory cortical reorganization that is thought to lead to the phantom limb sensation.[11] Opioids such as high-dose morphine sulfate have shown a positive effect in improving PAP, but patients should be made aware of the risks of opioid dependence and tolerance.[6,13] Opioids do not represent a long-term solution for patients with PAP wishing to return to their pre-amputation lifestyles. Consequently, they are best suited for the acute management of PAP, along with stool softeners to prevent constipation. Patients should be advised of their short-term use and ideally opioids should be tapered off in the acute hospital or inpatient rehabilitation setting.

NMDA receptor antagonists evaluated for PAP include ketamine, memantine, and the over-the-counter drug dextromethorphan. These medications are thought to block the hyper-excitability and sensitization mechanisms of PAP.[14,15] Intravenous ketamine has been shown to improve such pain in the short-term, while a small case series has also demonstrated potential benefit for the use of oral ketamine.[16] It is important to note that ketamine has side effects ranging from alterations in mood to hallucinations, and it is a highly abusable drug.

Several other classes of drugs have been explored in the treatment of post-amputation and phantom limb pain. Salmon calcitonin is a hormone that reduces blood calcium levels; it has also been studied as an analgesic due to its inhibition of the central activation of neurons when they are stimulated peripherally.[6] Reports are mixed regarding calcitonin's ability to provide adequate analgesia in the setting of PAP. Calcium channel blockers such as nifedipine, beta blockers including propranolol, clonidine (an alpha 2-adrenergic agonist), and etanercept (tumor necrosis factor-inhibitor) are other possible treatments for PAP; however, the evidence to support the use of these agents is insufficient.[6,17]

REHABILITATIVE AND PSYCHOLOGICAL TREATMENTS

As the pathophysiology of the chronic PAP pathway is proposed to have a significant cortical component, various psychological treatment including hypnosis, biofeedback, cognitive behavioral therapy (CBT), and guided imagery have been investigated.[6,18] Other interesting interventions include eye movement reprocessing, mirror visual feedback (MVF), and the use of virtual reality.[18] Most of the literature on these topics comes from case series, and larger, long-term studies are required to better evaluate their appropriateness for this indication. They are safe and generally well-tolerated, however, and may help certain patient populations.

MVF utilizes the contralateral intact extremity to reflect an image in a position so that the patient perceives an intact limb, which may reverse the cortical reorganization proposed as a cause of PAP. This technique has shown short-term effectiveness for pain reduction in small studies.[6,18]

Various forms of physical therapy including massage, tapping, acupuncture and ultrasound have also shown modest relief of post-amputation pain. These low-risk, non-invasive treatment options should be offered before medications and invasive approaches.[18]

SURGICAL TREATMENTS

In general, the evidence supporting surgical management for the treatment of chronic PAP is limited, and surgery should only be performed if conservative management has failed. There are numerous procedures described in the literature, further emphasizing the lack of consensus for surgical treatment.[6,19–21] There are two basic types of surgical intervention, neuromodulatory and reconstructive.

Neuromodulatory procedures target maladaptive neuroplastic changes and are represented by various implantable and transcutaneous stimulators. Transcutaneous electrical stimulation (TENS) is theorized to provide pain relief after amputations by increasing blood flow, reducing muscle spasms, and activating large afferent nerves to block nociceptor neurons in the spinal cord. While small series have demonstrated its value, a recent Cochrane review study reported no appropriate studies to judge its effectiveness.[22] Other possible technologies include electric nerve block devices, myoelectric prostheses, residual limb covers, and laser systems.[21]

Deep brain, motor cortex, spinal or peripheral nerve stimulators attempt to modulate the neural pathways thought to be involved in chronic phantom limb pain. There are a few randomized controlled studies supporting their outcomes. As demonstrated by Corbett et al. in their systematic review, however, the evidence at best supports short-term benefits from repetitive transcranial magnetic or direct current stimulation.[19] There is limited evidence for implantable stimulators to treat chronic phantom limb pain, even though spinal cord stimulators are reimbursed by Medicare for this specific purpose.

Reconstructive options include surgery for peripheral mechanisms of pain, e.g. residual limb pain caused by neuromas or heterotopic ossification, which can occur at rates as high as 80% and 63% respectively.[6] There are many possible procedures for neuromas, but the general consensus is that after resection, the nerve end should be protected and implanted into soft tissues. Another surgical solution that has been investigated is upper extremity targeted muscle reinnervation (TMR), which transfers amputated nerves into remaining muscles in the residual limb in order to improve

myoelectric prosthesis control. A retrospective review showed that of the 15 patients who had post-amputation neuromas prior to the TMR, 14 had complete resolution of symptoms and the remaining patient experienced partial relief.[20] This evidence suggests that by altering the amputated nerve environment, perhaps neuroma prevention can produce better outcomes than treatment of problems that develop.

CONCLUSION

The number of people with amputations in the United States is increasing, and non-surgical practitioners will increasingly manage their chronic, residual limb and phantom pain. Post-amputation pain encompasses multiple complex pain syndromes after the amputation of an extremity. Currently, the specific pathophysiology is not well understood and is likely multifactorial, which explains the potential benefit of numerous non-operative and operative treatments. In general, approaches to treating chronic phantom limb pain are similar to those for other types of neuropathic pain, and there is no standard of care or strong evidence to support any particular method. However, there are several promising modalities, and further research on the mechanisms of PAP is likely to yield better evidence for some current treatments and new modalities.

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Spinal Cord Stimulation: The Use of Neuromodulation for Treatment of Chronic Pain

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KEYWORDS: neuromodulation, spinal cord stimulation, chronic pain, neuropathic pain

INTRODUCTION

Chronic Pain and the Role of Neuromodulation

Chronic pain, defined as pain persistent for more than 3–6 months, affects 100 million adults in the United States (US) and impacts all dimensions of health-related quality of life (QOL) and healthcare expenditures.[1] Low back pain is the leading cause of disability, with healthcare expenditures estimated to be as much as $560–$635 billion, more than the combined spending on heart disease and diabetes.[1] Despite lack of consistent evidence, rates of spine surgeries have increased, while other forms of chronic pain management, including narcotics, contribute to both adverse medical side effects and the ongoing opioid epidemic.[2]

As such, additional treatment options for chronic pain are being studied. One promising option is spinal cord stimulation (SCS), a form of neuromodulation used since the 1960s. Recently, a paradigm shift has occurred, with SCS increasingly accepted as a first-line therapy after noninvasive treatment (physical therapy, medications, etc). SCS reduces pain, decreases use of analgesic medication, and improves function. Level I evidence supports the use of SCS for chronic neuropathic pain from failed back surgery syndrome (FBSS), complex regional pain syndrome (CRPS), and painful diabetic peripheral neuropathy (DPN).[3] Recent guidelines suggest that appropriately selected patients who have failed conventional medical management (CMM) for neuropathic pain may benefit from SCS.[4]

Neuromodulation involves the application of electricity to the central or peripheral nervous systems for therapeutic benefit. The strengths of this approach include reversibility and a low-risk profile. Furthermore, studies demonstrate improved pain relief, health-related QOL, and functional status.[5,6] Multiple neuromodulatory techniques exist, including spinal cord stimulation (SCS), peripheral nerve stimulation (PNS), dorsal root ganglion (DRG) stimulation, and deep brain stimulation (DBS). Among these, SCS is most commonly performed in the US, with an estimated 50,000 implants annually, accounting for 70% of all neuromodulation treatments. The valuation of the SCS marketplace was $1.3 billion in 2014.[7]

Spinal Cord Stimulation and Mechanisms of Action

Spinal cord stimulation (SCS) involves the application of electricity to the spinal dorsal columns, which modulate pain signals relayed by ascending pain pathways to the brain. Although precise mechanisms are complex and not fully understood, the concept derives from the gate control theory, first described by Melzack and Wall.[8] This theory describes the presence of a "gate" in the dorsal horn, relaying neuronal signals from sensory afferent fibers to brain centers involved in pain perception. Ab fibers (myelinated) carrying non-nociceptive stimuli and C fibers (non-myelinated) relaying painful stimuli both synapse in the dorsal horn with the spinothalamic tract; the gate theory postulates that stimulating the faster Ab fibers leads to closure of nerve "gates," blocking the transmission of pain signals by slower C fibers (**Figure 1**). This theory provides insight into why non-nociceptive stimuli, such as tapping or massaging a painful area, provides temporary relief.

Figure 1. Gate theory of pain postulates that sending electrical current to the dorsal column leads to stimulation of faster, myelinated Ab fibers carrying non-nociceptive stimuli, resulting in closure of nerve "gates" and blocking slower, non-myelinated C fibers from transmitting pain signals via the spinothalamic tract.

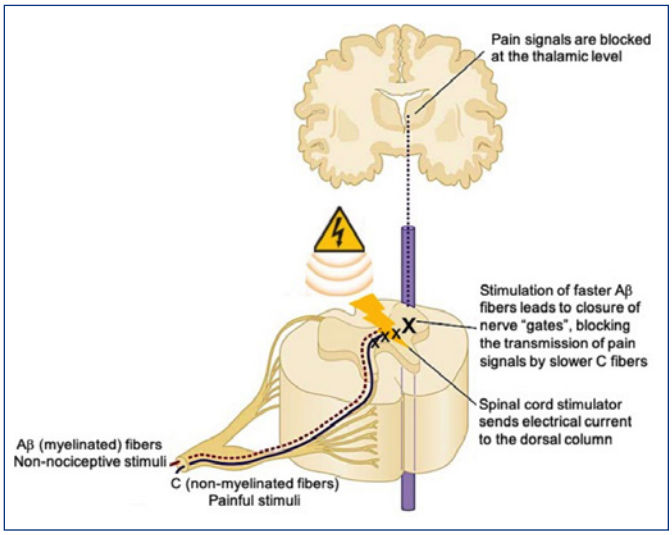
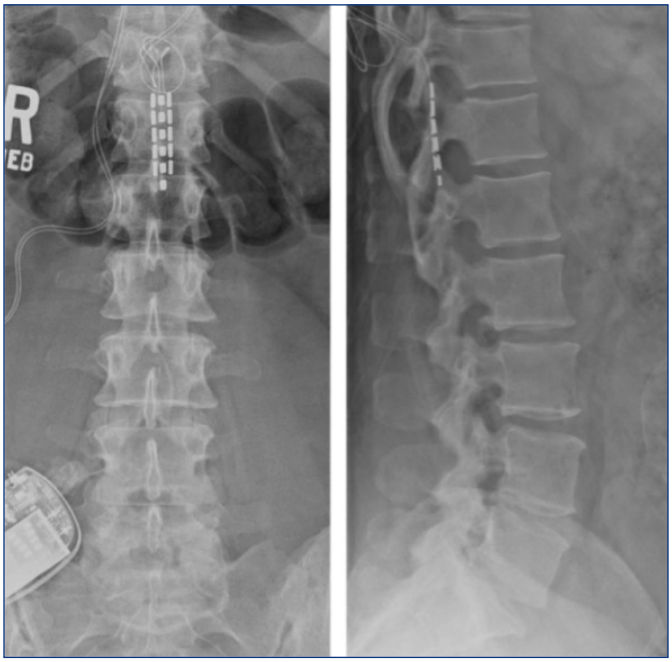


Figure 2. Plain AP and lateral radiographs of spinal cord stimulator implant in place, with pulse generator located within the right buttock subcutaneous tissue and leads placed in the dorsal epidural space at T12.



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In the spinal cord, Ab and C fibers are segregated from motor fibers, making the dorsal column an ideal target for neuromodulation. SCS involves placing electrical leads into the epidural space and sending currents to the dorsal columns (**Figure 2**). Historically, the goal was to replace nociceptive pain signals with more tolerable paresthesias in the distribution of the patient's pain. Recent devices employ higher frequencies, which minimizes paresthesias. Other stimulation paradigms deliver electricity in short bursts, clusters, and via closed-loop feedback.

The exact mechanism for the modulation of neuronal pain in the dorsal columns is probably complex. In animal models, SCS leads to enhancement of inhibitory neurotransmitters such as GABA and the release of acetylcholine, which acts on muscarinic M4 spinal cord receptors involved in analgesia.[9,10] SCS may also modulate supraspinal pathways to mediate analgesia, as functional MRI demonstrates changes in the thalamus and somatosensory cortex corresponding to altered pain processing.[11]

Common Indications for Spinal Cord Stimulation

SCS is approved by the Food and Drug Administration (FDA) for chronic neuropathic pain disorders of the trunk and extremities, including failed back surgery syndrome (FBSS), complex regional pain syndrome, neuropathy, and radiculopathy. SCS is also used for refractory angina pectoris, peripheral limb ischemia, and irritable bowel syndrome.[10]

SCS has been most widely studied in FBSS, which broadly encompasses pain that persists despite spine surgery. Randomized controlled trials (RCTs) have studied the efficacy of SCS, particularly in cases of leg-predominant symptoms. A landmark RCT compared patients undergoing SCS with those who had re-operation of the lumbar spine.[12] Significant pain relief, defined as 50% reduction, was achieved in 39% of the SCS group vs. 12% in the re-operation group ($p<0.01$); in addition, there was an 87% reduction in opiate analgesic use with SCS vs. 58% with re-operation ($p=0.025$). Furthermore, after a 6-month post-operative period, 54% of re-operation patients elected to cross-over and undergo SCS, vs. 26% of SCS patients who crossed over from SCS to re-operation ($p=0.02$). A recent review including 2 RCTs and 9 prospective observational trials demonstrated efficacy of SCS for FBBS, with the pooled 1035 observational patients experiencing between 48–77% treatment efficacy at 1-year follow-up.[13]

Evidence also supports the use of SCS in complex regional pain syndrome (CRPS), which involves pain dysregulation and nerve dysfunction in both the sympathetic and central nervous systems, leading to sensory, vasomotor, and sudomotor changes as well as pain and weakness. Management is difficult and symptom-based, with limited structural pathology to target. A systematic review of SCS in CRPS (1 RCT and 25 observational studies) found that in addition to reducing analgesic use and improving QOL, SCS led to a pooled decrease of 47% on the visual analog pain scale.[14]

Beyond current FDA-approved indications, RCTs have shown that SCS can benefit ischemic vascular disease, including chronic inoperable limb ischemia and treatment refractory angina.[3] The mechanism of action is thought to involve modulation of the sympathetic nervous system, levels of prostaglandin, and nitric oxide production. Compared to CMM, SCS therapy leads to greater pain relief, improved ankle-brachial pressure indices, and higher rates of limb salvage.[15] In patients with treatment refractory angina, no differences were found in exercise function and QOL measures when comparing SCS and coronary artery bypass grafting (CABG), although total mortality was increased in the CABG group at 6-month follow-up.[16] There is also Level I evidence supporting SCS use in peripheral neuropathic pain syndromes, including diabetic and post-chemotherapy neuropathy.[3,17] Other conditions involving nerve pathology such as post-herpetic neuralgia, post-thoracotomy neuralgia, and phantom-limb pain have been successfully treated with SCS but with less robust evidence.

Complications of Spinal Cord Stimulation and Patient Selection

SCS is widely reported to be a safe procedure, owing to its reversibility and minimal invasiveness.[10] Catastrophic surgical complications are rare, with neurologic injury reported as 0.25% in one review of 44,587 cases.[18] Some causes include formation of epidural hematoma (0.19%) and spinal cord

contusion (0.1%). Minor complications mostly occur within 12 months after implantation and are generally reversible.

The most common mechanical complication is lead migration (14%), which may require revision if it results in loss of targeted stimulation.[19] Lead migration typically occurs within several days of implantation. Post-operative scarring keeps leads in place longitudinally. Migration is mitigated by appropriately anchoring leads. Recently, a lower incidence of migration is attributed to improvements in lead design and anchoring systems. Hardware malfunction or lead fracture necessitating device removal occurs less frequently (7-10%). Subcutaneous implantation of the SCS impulse generator may lead to incisional or pocket pain (10%).

The most common medical complication is infection (3.8%), which is generally superficial and minimized with pre-operative broad-spectrum antibiotic use. There is no evidence supporting routine post-operative antibiotics.[10] Other medical complications are less common (2.5%) and include hematoma, seroma, epidural fibrosis, dural puncture, and neurologic injury.

Appropriate patient selection is crucial for reducing SCS complications and treatment failures.[20] SCS candidates should undergo behavioral assessment before a trial of therapy. Untreated depression, major psychiatric illness, and unrealistic expectations lead to less improvement of pain and disability scores. Obesity, younger age, and male gender also predict early failure.[21]

MRI Compatibility and Cost-Effectiveness

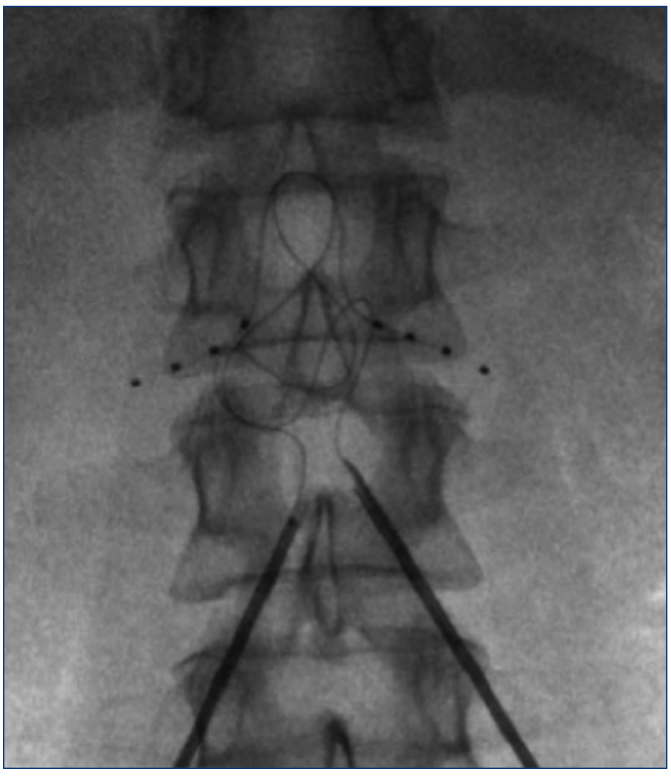
Historically, SCS was not compatible with magnetic resonance imaging (MRI). Recent technological advancements allow use of MRI under specific conditions. Although the initial costs of SCS are high, they are offset by improved QOL measures and decreased use of drug and non-drug therapies over time.[19] SCS is particularly cost-effective in long-term studies (>6 months) and for back and leg pain refractory to CMM; SCS was shown to be cost-effective as an adjunct to CMM and when compared to re-operation, with >80% greater likelihood of cost-effectiveness versus CMM and re-operation in predictive models.[22] In FBSS patients, SCS decreased annual healthcare costs by an average of 40% over 9-year follow-up compared to CMM.[23] Additionally, newer high-frequency stimulators and rechargeable battery systems have led to increased cost-effectiveness.

Emerging Approaches in Neuromodulation

Traditionally, SCS stimulates dorsal columns in the 50-100 Hz range, creating perceptible paresthesias in the distribution of the patient's pain. Recently, the application of sub-threshold high-frequency stimulation (HFS) or burst stimuli has reduced or eliminated parasthesias while providing similar or improved pain relief versus traditional SCS.[7] HFS was FDA-approved in 2015 for chronic refractory trunk and/or limb pain. Its unique waveform at 10kHz preferentially blocks large-diameter nerve fibers (responsible for paresthesias) while recruiting smaller and medium fibers (involved in vibration and pressure signaling), resulting in paresthesia-free SCS. Burst stimulation delivers higher-frequency stimulation in closely-spaced bursts, rather than constant lower-frequency tonic stimulation used in traditional SCS, which reduces pulse amplitudes and provides subthreshold stimulation with minimal paresthesia. This approach may improve the tachyphylaxis and physiologic tolerance seen with constant stimulation.

Neuromodulation techniques targeting the dorsal root ganglion (DRG) and peripheral nervous system (PNS) allow for greater anatomic specificity. Stimulation of the DRG, which is located at the transition between the spinal cord and peripheral nervous system and contains the primary sensory neuron cell body, can block or reduce painful peripheral signals to the CNS. Anatomically, the vertebral foramens housing the DRG form an ideal enclosure for leads, reducing migration and positional issues seen with SCS (**Figure 3**). Minimal cerebrospinal fluid at the foramens also allows for lower energy requirements, improving battery longevity. Targeting the DRG of a specific dermatome provides focal pain relief, particularly for refractory CRPS and focal neuralgias that are less effectively treated by traditional SCS, such as the groin and foot.[10]

Figure 3. AP fluoroscopic image of bilateral dorsal root ganglion stimulation, with leads in place at L1.



Source: Giordano et al. (Case Rep Anesthesiol 2018), licensed under Open Access and the Creative Commons Attribution.

CONCLUSIONS

Growing Level I evidence supports the use of SCS as a safe and cost-effective therapeutic option for numerous chronic pain conditions. SCS is a minimally-invasive procedure that may be implemented earlier in the treatment continuum to reduce pain and disability, particularly in cases of refractory neuropathic pain complicated by analgesic side effects, drug tolerance, or the need for re-operation. Advancements such as high-frequency, burst, and dorsal root ganglion stimulation have further improved pain relief and patient satisfaction, while mitigating risk and improving outcomes.

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Osteoarthritic Pain: A Brief Review of Nonsurgical, Surgical, and Alternative Treatment Approaches

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KEYWORDS: osteoarthritis, pain, inflammation, arthroplasty, radiofrequency ablation, arthroscopy, alternative medicine

INTRODUCTION

Osteoarthritis (OA) is thought to be the most common chronic joint disease, with the hip, knee, and hand most commonly affected.[1] Although estimates of prevalence vary, 67% of women and 55% of men demonstrated radiographic signs of hand OA in a cohort of nearly 4000 individuals older than 55.[2] Among those older than 80, 53% of women and 33% of men had radiographic knee OA. It is well known that not all patients with radiographic OA experience pain, and the estimates of the prevalence of osteoarthritic pain are varied. One study reported that 26% of women and 13% of men experienced pain due to OA of the hand, and, in general, 17% and 10% of people experience pain due to OA of the knees and hips, respectively.[3,4] Pain from OA is known to have a significant effect on quality of life, with women more affected than men.[5] This effect is modulated negatively by depression and poverty, and positively by education and treatment of underlying disease. Along with back pain, OA accounts for the two most common causes of chronic pain in the US.[6]

The mechanisms of osteoarthritic pain can be generally divided into two pathways, peripheral and central. Early controlled studies demonstrated that pain was correlated with joint space narrowing and other structural abnormalities.[6] Later studies pointed towards synovium and bone as more likely pain generators, because cartilage lacks nerve endings. It is now understood that the intraarticular milieu of growth factors and cytokines also contributes significantly to the experience of pain. Notably, nerve growth factor (NGF), which promotes axon growth and survival of peripheral neurons, has been implicated in osteoarthritic pain. NGF decreases the firing threshold of vanilloid receptor 1, causing depolarization of nociceptive neurons. Its expression is increased in cartilage in response to mechanical stress and leads to haphazard innervation of previously aneural cartilage with peripheral sensitization to pain. Cytokines such as Tumor Necrosis Factor α (TNF–α) and Interleukin-6 (IL-6) are also upregulated in arthritis, leading to inflammation and pain.[6]

The central mechanism of osteoarthritic pain deals mainly with the spinal cord. Increased glutamate receptor sensitivity leads to the formation of closed synaptic loops, which widens the receptive field for noxious stimuli and enhances both temporal and spatial summation of pain signals. The impairment of descending inhibitory pain pathways further exacerbates pain. Finally, involvement of central pain processing pathways as well as the limbic system, which is responsible for the fear response to pain, also aggravates osteoarthritic pain.[6]

The numerous treatment options for painful OA all involve modulation of these pathways in some manner, although all the effects of some individual treatments are not fully understood.[6] The purpose of this review is to explore the traditional treatments for painful OA, including nonsurgical and procedural or surgical options, as well as nontraditional methods.

NONSURGICAL MANAGEMENT

There is a robust body of literature describing the nonsurgical management of osteoarthritic pain. In 2013, the American Academy of Orthopaedic Surgeons (AAOS) released updated guidelines for the treatment of the osteoarthritic knee (**Table 1**).[7] Current evidence demonstrates that exercise

Table 1. AAOS Consensus Guidelines for Nonsurgical Management of Knee Osteoarthritis[7]

Recommendations For	Strength
Self-directed exercise and strengthening program	Strong
NSAIDS and Tramadol	Strong
Weight loss	Moderate
Recommendations Against	**Strength**
Acupuncture	Strong
Glucosamine and chondroitin	Strong
Lateral wedge insoles	Moderate
Electrotherapy (e.g. TENS, etc.)	Inconclusive
Manual Therapy (e.g. chiropractic therapy, myofascial release)	Inconclusive
Medial compartment unloader brace	Inconclusive
Acetaminophen, opioids, pain patches	Inconclusive

is strongly associated with decreased pain and self-reported disability in patients with knee arthritis.[7] A systematic review of 13 randomized controlled trials compared aerobic exercise with resistance training and found that both modes of exercise produced significant improvements in pain and disability, with no difference between the two.[8] Patients may be encouraged to choose their preferred mode of exercise in order to maximize adherence, which is a key predictor of good outcomes.[8] Home-based and clinic-based physical therapy are equally effective at producing clinically significant improvement in pain, although previous studies have found that clinic-based therapy can lower medication utilization and increase patient satisfaction.[9]

Another common treatment measure with moderate evidence is weight loss for obese patients.[7] These patients have excess adipose and muscle tissue and their strength is generally inadequate to bear the load placed through their joints; contributing to this lack of strength is intramuscular fat deposition, which is associated with a 10-fold increase in systemic pro-inflammatory cytokines.[10] Together, these changes alter the composition of cartilage and likely contribute to osteoarthritic progression and worsening pain. Studies have demonstrated significant pain reduction in the hip, knee, ankle, spine, neck, shoulder, elbow, wrist, and hand after weight loss in obese individuals.[10] Weight-loss options include exercise, FDA-approved medications like Orlistat and Sibutramine, and bariatric surgery in the form of laparoscopic gastric banding, sleeve gastrectomy, and vertical banded gastroplasty.[10]

There is strong evidence for the use of NSAIDs (oral and topical) and tramadol for arthritic pain in the knee.[7] Topical NSAIDs such as diclofenac and ketoprofen are associated with a 50% pain reduction in 60% of OA patients, with the benefit of avoiding gastrointestinal side effects, while several placebo-controlled trials also demonstrated good efficacy of oral NSAIDs.[11] There is strong evidence in support of tramadol,[7] but a direct comparison showed that NSAIDs produced a superior analgesic effect[11] (and lack the former drug's risk of dependence).

On the other hand, there is inconclusive evidence in support of acetaminophen, opioids, pain patches, and corticosteroid injections.[7] Intra-articular steroid injections are frequently utilized for their anti-inflammatory and analgesic effects, with effects lasting anywhere from 1 week to 24 weeks.[11] Despite their widespread use, more research is necessary to validate their role in the treatment of osteoarthritic pain. Other treatments with inconclusive evidence include growth factors, platelet-rich plasma, stem-cell injections, electrotherapeutic modalities, manual therapies like joint manipulation and chiropractic therapy, and valgus-directed force braces.[7] Treatments with evidence suggesting a lack of efficacy include acupuncture, glucosamine, chondroitin sulfate, hyaluronic acid, and lateral wedge insoles.[7]

SURGICAL MANAGEMENT

Surgical management plays an important and well-established role in the treatment of osteoarthritis when non-surgical modalities have failed. Among all joints treated for osteoarthritis, hip and knee arthroplasty remain the most prevalent. There is a preponderance of data demonstrating success in reducing pain and improving function in the short- and long-term, with the most significant improvement within the first three months.[12] Certain predictive factors have been shown to affect post-surgical outcomes in surgical candidates. In a comprehensive study of patient-reported outcomes[13] after total hip (THA) and knee arthroplasty (TKA), age and gender did not have any predictive value, whereas high expectations of pain relief had a positive effect in both hip and knee patients. Interestingly, severe radiographic evidence of OA had a positive predictive effect for TKA patients, but no predictive effect for THA patients.

Resurfacing operations for hip osteoarthritis have also been studied. Unlike total hip arthroplasty, in which the femoral head is removed, resurfacing techniques for hip arthritis involve reshaping the femoral head in order to fit a metal cap. The acetabulum is trimmed to accept a metal shell with which the femoral cap articulates. This technique preserves bone compared to, and may be easier to revise than, total hip arthroplasty. It has a decreased risk of dislocation and leads to more normal walking patterns than total hip arthroplasty as well, and was initially developed for younger and more active patients with hip arthritis. However, disadvantages of hip resurfacing include a greater risk of femoral neck fracture and adverse local tissue reactions related to metal-on-metal wear and the resulting buildup of metal ions in the local tissues, followed by lymphocytic inflammation and pain.[14] Hip resurfacing remains less frequently performed when compared to total hip arthroplasty in part due to this complication profile.[14]

Unfortunately, some patients are poor candidates for arthroplasty for various reasons, including medical comorbidities. An alternative to arthroplasty in patients with hip and knee osteoarthritis is radiofrequency peripheral nerve ablation. Radiofrequency ablation (RFA) is a two-step procedure that is typically performed under light sedation, and is also used in the treatment of lumbar facet pain. In the treatment of knee arthritis, the genicular nerves are targeted for ablation. The first step is a diagnostic anesthetic injection to the peripheral extraarticular sensory branches of the genicular nerves. If this trial provides significant pain relief, then a radiofrequency probe is used to apply thermal energy to ablate the peripheral nerve endings. In a double-blind, randomized controlled trial[15] comparing radiofrequency genicular neurotomy to sham surgery, the neurotomy group showed significant improvement in both visual analog (VAS) and Oxford Knee Scores (OKS). Nearly 60% of all patients reported at least 50% pain reduction at 1, 4, and 12 weeks. Similarly, a prospective observational study[16] demonstrated

significant improvement in VAS and Western Ontario and McMaster Universities Arthritis Index (WOMAC) scores following radiofrequency genicular neurotomy. Although the therapeutic effect declined after 6 months, 32% of patients reported at least 50% improvement in pretreatment VAS scores after 1 year.

Patients with hip osteoarthritis may also benefit from radiofrequency ablation of articular branches of the obturator nerve, femoral nerve, or sciatic nerve dependent on the location of pain. Reported techniques are similar to those for treatment of knee arthritis, with a diagnostic anesthetic injection followed by application of thermal energy to ablate the targeted nerve endings. A recent systematic review reported pain relief ranging from 30% to 90% from baseline scores, although the durability of this effect remains unclear, with some studies reporting recurrence of pain within 1–2 years.[17]

Complications following radiofrequency ablation are rare. Kumar et al. described loss of cutaneous sensation in the distribution of the lateral femoral cutaneous nerve in a small number of patients treated with RF ablation for hip arthritis.[17] In a recently published systematic review of RF ablation for knee arthritis, Jamison et al. discovered no major adverse complications reported in the literature, although they refer to unpublished cases of skin burns in some patients with nerve branches in near proximity to the skin.[18] Strand et al. recently published a case report of medial thigh hematoma after RF ablation for knee arthritis.[19] Longer-term follow-up studies are required to better characterize the role of nerve ablation in treating pain associated with OA.

Arthroscopy is an alternative to arthroplasty and nerve ablation, especially for the treatment of knee osteoarthritic pain. The mechanism of pain improvement after arthroscopy of the knee is thought to be related to lavage as well as removal of debris. Lavage of the knee decreases the concentration of inflammatory cytokines and degradative enzymes in the synovial fluid, leading to diminished inflammation of the cartilage and surrounding synovium. Removal of tissue debris, inflamed synovium, and damaged cartilage and meniscus also removes sources of synovial and cartilage inflammation, which is thought to improve pain.[20] Although arthroscopy was previously used more widely in the treatment of OA, it has received more scrutiny in recent years. A review of arthroscopic management of osteoarthritis found that it tends to benefit young, active patients with mild to moderate arthritic changes without deformity.[21] Thus, arthroscopy may play a role in the management of early OA in select patients. However, a paucity of high-quality studies makes it difficult to produce thorough guidelines with respect to the treatment of OA with arthroscopy.

ALTERNATIVE TREATMENTS

A number of alternative therapies have tried to address osteoarthritic pain (**Table 2**). These include practices that have a rich tradition in other cultures, like acupuncture and yoga, as well as nontraditional pharmacological interventions. OA frequently leads to a sedentary lifestyle, which further exacerbates joint stiffening, pain, and muscle weakness.[22]

Table 2. Alternative Therapies for Treatment of Symptomatic Knee Osteoarthritis[7]

Alternative Therapy	AAOS Recommendation	Rationale
Yoga (Hatha or Chair)	For; Strong	Improved WOMAC and SF-36 Physical Functioning and Bodily Pain Subscales in yoga + physical therapy treatment group when compared to physical therapy alone
Acupuncture	Against; Strong	Based on evidence of lack of efficacy rather than evidence of harm
Glucosamine/ Chondroitin	Against; Strong	Based on evidence of lack of efficacy rather than evidence of harm

Hatha yoga (HA) is a form of yoga that reduces pain and stiffness in OA patients by realigning the skeleton, strengthening muscles around joints, and stretching out joints.[23] For patients unable to perform HA, chair yoga is an option. A significant reduction in pain was appreciated in patients with lower extremity OA who performed yoga for 3 months.[22] Regardless of the specific form, yoga has been shown to increase sleep quality and decrease sleep disturbances in patients with OA.[22] Coupled with breathing and relaxation, yoga can help patients cope with the reactive aspects of chronic pain.[23]

Similarly, acupuncture has been shown in some studies to help patients with OA pain. The proposed mechanism is that small diameter muscular afferent nerves are stimulated to send impulses to the spinal cord to release endorphins and monoamines involved in the pain messaging pathway.[24] Studies examining the immediate effects of electroacupuncture and manual acupuncture on pain and mobility in patients with knee OA have demonstrated a significant reduction in pain intensity and time-to-run test scores in both groups compared to controls.[25] Acupuncture has consistently been shown to be an effective treatment modality for pain and dysfunction associated with knee OA.[24]

Alternative pharmacologic interventions, such as glucosamine, chondroitin, capsaicin, and cannabidiol, are gaining popularity among patients with painful OA. While both glucosamine and chondroitin are over-the-counter supplements in the United States, they are registered as medications in Europe.[26] In-vitro models suggest that chondroitin sulfate and glucosamine sulfate exert a beneficial effect on the metabolism of synovial joint cells, including chondrocytes, synoviocytes, and cells from subchondral bone.[26] They increase the expression of type II collagen and stimulate proteoglycan synthesis in human articular chondrocytes while

reducing the production of pro-inflammatory cytokines and proteases.[26] Studies using MRI have demonstrated that these supplements reduce the loss of cartilage volume and associated joint space narrowing.[26] The Osteoarthritis Research Society International (OARSI) considers glucosamine and chondroitin to be low-risk medications with a moderate to high effect. There is a favorable risk/benefit ratio for the treatment of older patients with OA and co-morbidities, in contrast to the long-term administration of drugs like NSAIDs and acetaminophen.[26] It should be noted, however, that the 2013 AAOS consensus guidelines for the treatment of knee osteoarthritis recommend against the use of acupuncture or glucosamine/chondroitin due to lack of efficacy, although they do not conclude that either treatment modality is harmful.

Capsaicin, a highly lipid-soluble compound available in topical formulations, is thought to help alleviate osteoarthritic pain through the "defunctionalization" of nociceptive nerve fibers, a process that involves temporary loss of membrane potential, inability to transport neurotrophic factors, and a reversible retraction of epidermal and dermal nerve fiber terminals. According to the 2019 American College of Rheumatology/Arthritis Foundation Guideline for the Management of Osteoarthritis of the Hand, Hip, and Knee,[27] capsaicin is conditionally recommended for the treatment of osteoarthritic knee pain due to low effect sizes in the available literature, and conditionally recommended against in terms of its use in osteoarthritic hand pain due to lack of available literature in hand OA.

Finally, the effects of cannabidiol on the treatment of osteoarthritic pain are poorly studied. Cannabidiol is the most abundant non-psychoactive compound in extracts of the *Cannabis sativa* plant, and various cannabinoids have been shown to have anti-inflammatory, anti-tumorigenic, and analgesic effects through action on a variety of receptors, but mainly cannabinoid receptors 1 and 2 (CB1, CB2). In animal models cannabinoids have shown to modulate OA pain manifestations and stress-related responses, and CB2 receptor activation has been shown to attenuate the development of pain and sensitization in an OA rat model.[28,29] However, the only known study to assess the effects of cannabidiol on human articular chondrocytes demonstrated that exposure to high levels of cannabidiol induces apoptosis in chondrocytes in-vitro.[28] No clinical trials exist studying the effects of cannabidiol in the treatment of osteoarthritis in humans.

CONCLUSION

Osteoarthritic pain is relatively common and is mediated by both peripheral and central pain mechanisms. Treatment options for osteoarthritic pain are numerous and include nonsurgical treatments such as in-home or clinic-based physical therapy, NSAIDs, and tramadol. Joint arthroplasty for appropriate patients is a durable and successful surgical treatment option. Other surgical options such as radiofrequency genicular nerve ablation and arthroscopy have also shown to be beneficial in certain patient populations. Finally, alternative treatment options such as yoga, acupuncture, and over-the-counter supplements like chondroitin and glucosamine have also shown benefits in the treatment of OA pain.

Although the literature surrounding arthroplasty and traditional nonsurgical treatment options is robust and continues to grow, the research surrounding alternative treatments is relatively sparse. Future research is needed in this field as many patients with either cultural preferences or those with contraindications to traditional approaches could benefit from alternative treatments. Osteoarthritis (OA) is a common cause of disability and pain. A patient-centered approach to the treatment of osteoarthritis is necessary as there are myriad nonoperative and operative treatment options for osteoarthritic pain.

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Surgical Management of Rheumatoid Arthritis of the Hand

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KEYWORDS: rheumatoid arthritis, hand, pain, surgery, deformity, arthroplasty, athrodesis

INTRODUCTION

Rheumatoid arthritis (RA) is a painful autoimmune disease that affects about 1% of the population.[1] A comprehensive epidemiological study of global disease burden in 2015 found that the prevalence of RA was about 25 million individuals, with an overall increase by 23.8% from 2005.[2] RA usually causes bilateral joint pain, stiffness, and swelling, which is typically worse after periods of inactivity or in the morning. While RA is characterized by joint involvement, other inflammatory manifestations include fever, anemia of chronic disease, pericarditis, and pulmonary fibrosis. RA can also be associated with other autoimmune diseases, including systemic lupus erythematosus and psoriatic arthritis.

The disease process of RA is a Type III hypersensitivity reaction that involves autoimmune cellular activation and immune complex formation in joints. These deposits lead to thickening of joint capsules, cartilage and bone damage due to inflammatory cascades, and rupture of tendons and ligaments. Chronic RA can lead to pannus formation, due to inflammation and proliferation of the joint synovium.[3] Damage to ligaments and tendons in the hands causes deformities such as boutonniere, swan neck, and ulnar deviation of the fingers as well as tendon ruptures affecting the ulnar digits.[4]

Rheumatoid arthritis has a multivariable inheritance pattern, and environmental factors influence the severity of the disease. Risk factors associated with RA include female sex and family history while environmental factors include exposures to cigarette smoke and silica dust.[1]

The diagnosis of rheumatoid arthritis is both clinical and serological. RA can be classified as a seropositive disease process with rheumatoid factor (RF), an autoantibody against the Fc portion of IgG, or as a seronegative form. Other autoantibodies in RA include anti-citrullinated protein antigens (ACPAs) as well as antinuclear antibody (ANA) and other, less specific antibodies. While RF was classically associated with the disease, newer data show that ACPA is the most specific antibody for RA.[5] Serological markers may be present before clinical symptoms; there is a group of patients in whom RF is not present in the early stages of the disease but does develop later, so its sensitivity for early detection is somewhat limited.[6]

Definitions	
Synovectomy	Surgical resection of inflamed and hypertrophied synovial tissue within a joint. This is generally performed through an open or arthroscopic approach.
Arthrodesis	A surgical procedure on a joint in which the bones comprising the joint are fused. This is generally accomplished through removal of the articular cartilage of the joint, resection of cortical bone on all sides of the joint, and the application of surgical hardware to apply compression across the prepared joint for a period of time.
Anthroplasty	A broadly defined surgical procedure on a joint in which the joint is reconstructed or replaced in order to maintain function while treating pain. This may be accomplished through implanting an artificial prosthesis or resecting the joint with or without interposing a biological or artificial spacer

There are multiple classification systems for RA, and the most recent one (2010) defines "definite RA" based on a score generated by four domains: number and site of joint involvement, serological markers, elevated acute-phase response, and duration of symptoms.[7]

Involvement of the hands in patients with RA can lead to pain, significant limitations in function, and concerns about cosmesis. Every joint can be affected, from the wrist to the individual interphalangeal joints. Although medical management may be appropriate for many, some patients may not tolerate medications or be treated until their joint deformities are advanced. In these patients, surgical treatment may be beneficial. The purpose of this article is to describe the manifestations and surgical treatment options for painful rheumatoid arthritis of the hand.

WRIST

The wrist is affected in approximately 80% of patients with RA.[9] All three articulations of the wrist (distal radioulnar (DRUJ), radiocarpal, and midcarpal joints) can be affected by RA, with the DRUJ being the most commonly involved

(**Figure 1**). The surgical goals for the rheumatoid wrist are to reduce pain, improve function, and prevent progressive deformity.

Synovial hyperproliferation characterizes early rheumatoid disease and is therefore a primary surgical target. Synovectomy and tenosynovectomy are indicated in relatively early disease in which wrist motion and radiocarpal joint space are preserved, but absolutely contraindicated in advanced degenerative disease.[10] These procedures reduce wrist pain through denervation of nociceptive nerve fibers, but do not slow the overall disease process.[11] Synovectomy can be performed via open or arthroscopic approaches, with comparable pain relief, but a higher risk of recurrence and radiographic progression after the arthroscopic technique.[12]

Following early synovitis, RA frequently involves the ligamentous structures of the distal ulna. When the synovial-lined ulnar carpal ligaments and other stabilizers of the distal radial ulnar joint (DRUJ) are affected, patients may experience dorsal subluxation and eventual dislocation of the distal ulna ("caput ulna syndrome"). In these patients, various surgical interventions to treat the DRUJ may be considered. A common surgery is the resection of the ulnar head, or Darrach procedure, but this may be complicated by excessive ulnar translation of the carpus. To prevent this complication, the Suave-Kapanji procedure creates a radioulnar fusion (arthrodesis) following partial ostectomy of the ulna just proximal to the DRUJ.[9] Ulnar head replacement (arthroplasty) has also been used in the RA wrist. It can confer improved stability and function compared to ulnar head resection and can also salvage a failed Darrach procedure.[13] While good outcomes have been reported in small case series, larger studies with longer follow-up are necessary to determine its long-term efficacy and safety.[10]

With progressive degenerative changes of the wrist, patients may experience involvement of midcarpal and radiocarpal joints. Surgical options include partial arthrodesis, complete arthrodesis, and

Figure 1. Wrist involvement in the Rheumatoid Hand

PA and lateral radiographs of the wrist in a 91-year-old patient with largely untreated rheumatoid arthritis. Note the extensive joint space destruction in the radiocarpal, ulnocarpal, and distal radioulnar joints, as well as auto-fusion of the carpus.

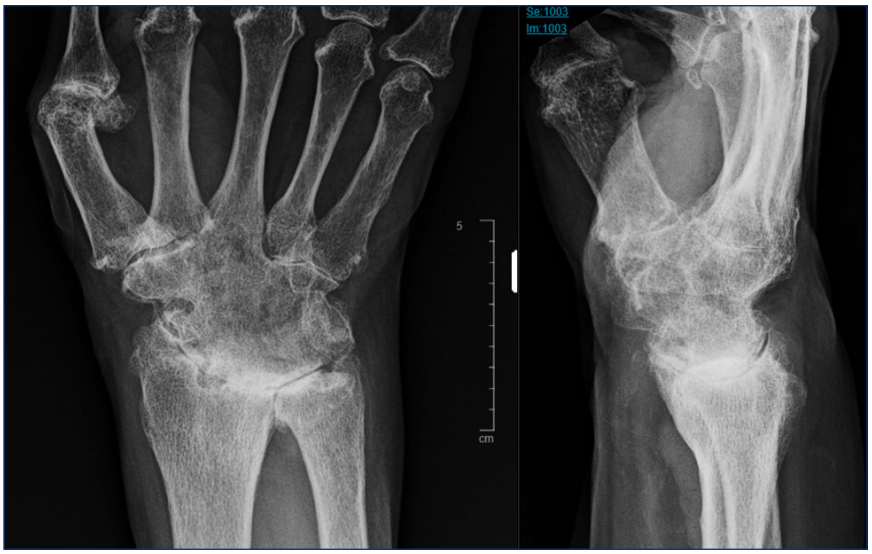
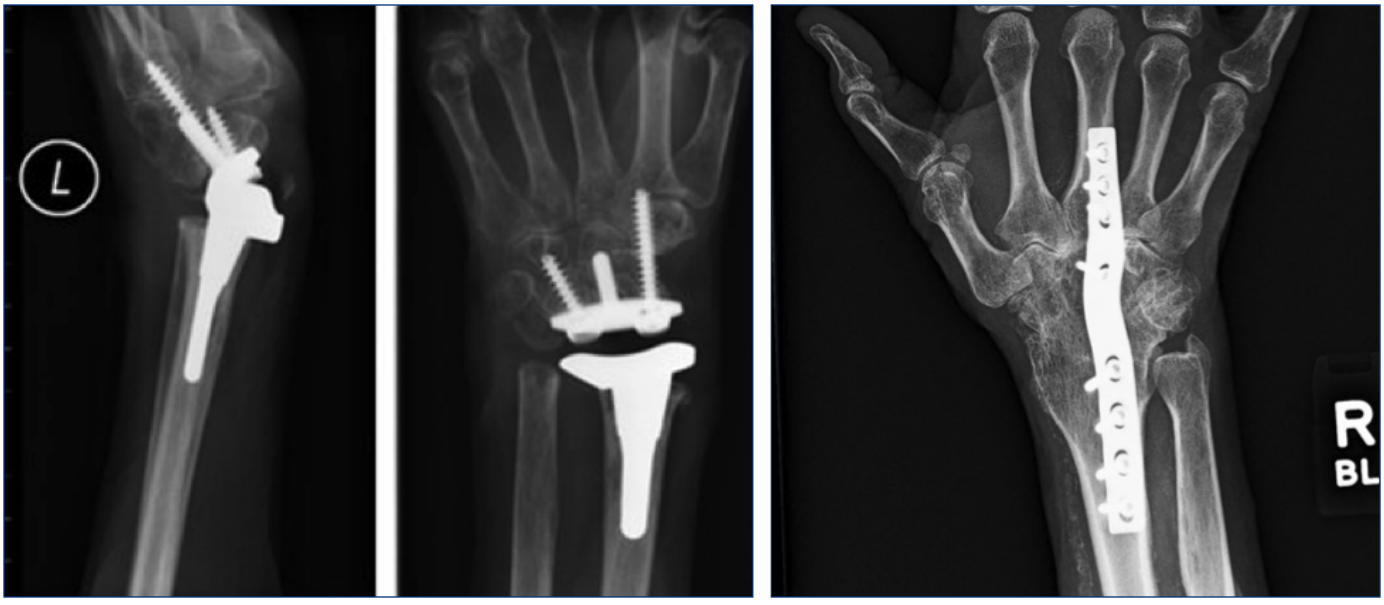


Figure 2. Treatment Options for End-Stage Wrist Arthritis

Left: Total wrist arthroplasty. Right: Total wrist arthrodesis.



arthroplasty. Partial arthrodesis of the radiolunate joint prevents ulnar drift of the wrist and digits. Results are promising, with reports of decreased pain, improved stabilization, and grip strength; however, destruction of the midcarpal joint may persist. Other partial fusions can be attempted, depending on the specific joints involved, such as radioscapholunate arthrodesis for radioscaphoid arthritis. In cases of severe destruction of the wrist joint, total arthrodesis is the preferred treatment.[14] Complications of arthrodesis surgeries are not uncommon, and in the case of total wrist arthrodesis may be as high as 29%, with a 4.4% rate of nonunion. Among these, major complications, such as deep infection, carpal tunnel syndrome, symptomatic hardware, extensor tenosynovitis, and ulnocarpal impaction may occur in 19% of wrists.[1] Total wrist arthroplasty can also be considered for carefully selected patients with an end-stage rheumatoid wrist (**Figure 2**). This treatment option may be used for low-demand patients with good bone stock for distal fixation, who require maintenance of wrist range of motion. Patients must be well educated on the lifelong restrictions related to joint arthroplasty, as well as the potential complications including infection, implant failure, and requirement for fusion in the future.[15]

METACARPOPHALANGEAL JOINT

Although the wrist is frequently involved, the most common deformity affecting the rheumatoid hand involves the metacarpophalangeal (MCP) joint; volar and ulnar subluxation of the proximal phalanx on the metacarpal causes ulnar displacement of the finger (**Figure 3**).[16] Early theories, which have mostly been discounted, attributed this ulnar deformity to the effect of gravity on patients with hands held in the neutral position during rest, atrophy of the interossei leading to muscle imbalance, and pain-related flexor muscle spasm.[17] The current understanding of ulnar drift at the MCP joint is that chronic synovitis damages the radial fibers of the MCP capsule and sagittal bands of the extensor hood leading to ulnar extensor subluxation, radial joint laxity, and radial forces with functional activities.[17]

Ulnar drift is more than a cosmetic deformity, although aesthetic considerations are indeed important to patients. Functional limitations include difficulties with gripping large objects and performing tip-to-tip pinch. In addition, extensor tendon subluxation leads to weakness with digit extension.[16]

Treatment of the MCP joint in patients with RA is challenging. Unlike the distal interphalangeal joint, for example, fusion is not commonly performed due to the significant loss in range of motion. In the early stages of disease with comparatively less deformity, synovectomy and/or crossed-intrinsic transfer may be beneficial. As rheumatoid arthritis is a disease of the synovium, the rationale behind synovectomy involves reducing the burden of inflammatory cells and tissue, thereby also treating pain. Crossed intrinsic transfer involves detaching the intrinsic muscle tendons at the ulnar aspect of each digit and rerouting them to the radial proximal phalanx of the adjacent ulnar digit, thereby providing a radially deviating force to restore position of each digit. This treatment is not ideal for patients in whom the disease is not well controlled medically, as further synovitis and joint destruction can stress the repaired connective tissues and predispose the patient to recurrent deformity.[18]

In patients with more advanced disease or more rigid deformities, arthroplasty is a treatment option. The main advantages to arthroplasty are maintenance of joint range of motion and shortening of the joint, which relaxes deforming tendon stresses and allows for easier joint repositioning. As in other hand joints, silicone implants are commonly used and have been well studied in the literature. A longitudinal analysis of 325 silicone arthroplasty cases with an average 7-year follow-up demonstrated good short-term outcomes and 95% revision-free survival at 15 years. Although recurrent coronal plane deformity > 10 degrees and implant fracture were common at 15 years, neither was associated with diminished function or the need for revision.[19]

Figure 3. Ulnar Deviation of Digits in the Rheumatoid Hand

PA radiographs of bilateral hands in a patient with rheumatoid arthritis demonstrating the characteristic ulnar deviation deformity at the metacarpophalangeal joints along with extensive joint destruction in the digits and wrist.