Complex Regional Pain Syndrome After Hand Surgery

Zhongyu Li, MD, PhD, Beth P. Smith, PhD*, Christopher Tuohy, MD, Thomas L. Smith, PhD, L. Andrew Koman, MD

KEYWORDS

• Chronic pain • Upper extremity • Fracture

After an emergent or elective upper extremity surgery, complex regional pain syndrome (CRPS) may complicate recovery, delay return to work, diminish health-related quality of life, and increase the likelihood of poor outcomes and/or litigation. The clinical entity of CRPS is chronic pain that persists in the absence of ongoing cellular damage and is characterized by autonomic dysfunction, trophic changes, and impaired function. In the perioperative period, the physiologic consequences of CRPS in the upper extremity contribute to or create one or more of the following: clinically significant osteopenia, delayed bony healing or nonunion, joint stiffness, tendon adhesions, arthrofibrosis, pseudo-Dupuytren palmar fibrosis, swelling, and atrophy. This article discusses the diagnosis, physiology, and management of postsurgical CRPS that occurs after hand surgery.

INCIDENCE AND SIGNIFICANCE

Although the exact incidence and prevalence of CRPS after hand surgery is unknown, the reported incidence of CRPS is 5.5 to 26.2 per 100,000 person years, and the prevalence is reported as 20.7 per 100,000 person years.[1,2] Women are more frequently affected than men, with a ratio of 3:1 to 4:1; the upper extremity is involved more frequently than the lower extremity; and fracture is the most common causative event.[1,2] The following rates of upper extremity CRPS have been reported: 4.5% to 40% after fasciectomy for Dupuytren contracture,[3] 2% to 5% after carpal tunnel surgery,[4,5] and 22% to 39% after distal radius fracture.[6]

Current dogma states that early recognition and treatment of CRPS improves the prognosis for full recovery; however, the term "early" is not well defined. In a population-based study in Olmsted County, 74 patients were diagnosed with CRPS type I between 1989 and 1999 and 74% of these cases "resolved."[2] Another study that followed outcomes after distal radius fractures found that the stiffness noted at 12 weeks ("early" diagnosis of CRPS) correlated with residual symptoms at 10 years.[7] In addition, individuals who smoke have a poor prognosis compared with nonsmokers.[8]

DIAGNOSIS

CRPS is a clinical syndrome without a pathognomonic marker. CRPS type 1, also known as classic reflex sympathetic dystrophy, is defined as chronic pain without identifiable nerve involvement. CRPS type 2, also known as causalgia, includes pain with identified nerve involvement. In addition to pain, both syndromes are often associated with autonomic dysfunction (abnormal vasomotor activity, inappropriate piloerector activity, abnormal sweat gland activity, and inappropriate arteriovenous shunting) and functional impairment. Trophic changes may occur but vary with the severity of the precipitating event, the time after injury, or the degree of extremity compromise.

Department of Orthopaedic Surgery, Wake Forest University School of Medicine, Wake Forest University Health Sciences, Medical Center Boulevard, Winston-Salem, NC 27157, USA
* Corresponding author.
E-mail address: bpsmith@wfubmc.edu

Hand Clin 26 (2010) 281–289
doi:10.1016/j.hcl.2009.11.001

HISTORY AND PHYSICAL EXAMINATION

In patients diagnosed with postoperative CRPS, the details of the initiating injury or process are critical in developing a management plan. Therefore, the events leading to surgery should be reviewed to ascertain any preexisting conditions, past traumatic injuries or pain issues, and preexisting subclinical problems; all these variables may affect symptoms that occur after the surgery. For example, for patients with mild compression neuropathies (eg, carpal tunnel syndrome), quiescent CRPS may be exacerbated in the perioperative period and serve as a neuropathic event. Concomitant injuries or preexisting mechanical derangements may also potentiate nociceptive stimulation and contribute to the dystrophic process. There are no firm temporal relationships between the time of injury and the time of surgery, that is, early or late intervention has not been demonstrated to affect the incidence of CRPS.

The time of onset of CRPS after surgery varies, with symptoms appearing as early as in the postanesthesia care unit (recovery room) or several weeks after surgery. Similar to nonsurgically related CRPS, the presentation may be obvious, with severe classic pain and swelling, or it may be insidious. Symptoms of CRPS are often nonspecific; pain, numbness, swelling, and stiffness are the normal symptoms reported by most postoperative patients. Therefore, clinical vigilance and acumen are crucial to discern CRPS within the context of nonspecific symptoms and signs and to evaluate the responses of these symptoms to the treatment and the passage of time. Recognizing the clinical character of CRPS is often crucial. In a classic presentation, this character includes pain that (1) is often described as burning, throbbing, and searing; (2) does not respond to narcotics; and (3) awakens patients at night or prevents normal sleep. Patients with CRPS are irritable and have difficulty with rehabilitation programs. In subtle or indolent presentations of CRPS, patients are often described as ''uncooperative.'' They may complain of stiffness, swelling, cold sensitivity, hyperalgesia, and allodynia. Return to work or normal activities is resisted, and patients may appear listless and forlorn. They are often irritated by family, coworkers, and medical providers, and this feeling is often mutual. In contrast, patients who present with massive swelling of their extremity, especially associated with a zone of demarcation, multiple sores, unexplainable wound breakdown, and/or abnormal hand clenching, need to be evaluated carefully to rule out a diagnosis of a fictitious disorder or malingering.[9] However, CRPS is not a psychiatric disease and is not related to any known psychological profile.[6] Because CRPS is, in effect, an abnormal prolongation of normal physiologic responses to injury in the periphery, in the spinal cord, and throughout the central nervous system, there is the potential for it to occur in any patient after surgical intervention.

PHYSICAL EXAMINATION

The physical examination of patients suspected of having postsurgical CRPS should be compared with their preoperative examination. Tight wound dressings or casts should be avoided. The examination needs to assess from the neck to the fingers, including all aspects of the affected extremity. The extremity inspection should include palpation; assessments of skin integrity, range of motion, joint stability, and motor power; and neurologic, vascular, and sensibility assessments. The extremity examination should also assess stiffness, edema, atrophy of hair and nails, hypersensitivity, and dexterity. Hand and extremity postures should be observed at rest and during activity or gait. Neuropathic or nociceptive contributors to the pain process should be investigated and identified. New exacerbations of preexisting subclinical compression neuropathy should be evaluated by motor examination, sensory testing, and mechanical indications (ie, Tinel signs).

Part of the physical examination should be focused on the identification of possible nerve injuries. Carpal tunnel syndrome may occur after distal radial fracture surgery or hand/wrist reconstruction, and it may require nonoperative or operative treatment. Iatrogenic nerve injury with neuromas-in-continuity and neuromas or perineural irritation of mixed nerves or sensory branches can also act as powerful drivers of CRPS. Commonly reported nerve injuries associated with CRPS include those that are associated with the palmar cutaneous branch of the median nerve, the superficial radial nerve and its branches, and the dorsal branch of the ulnar nerve and its branches. However, injury or irritation of any nerve can contribute to a dystrophic process.

Inspection and Observation

The hands of patients with CRPS may be swollen, obscuring the dorsal veins, and/or the hands may appear dry or damp; the posture of the hand is generally intrinsic minus (metacarpophalangeal [MP] joints extended and proximal interphalangeal [PIP] joint slightly flexed). Dystonia, posturing, or tremors are also occasionally observed. For a thorough examination, the upper limb should be observed at rest, during activity, and during

ambulation. Surgical sites should be inspected for signs of wound infection or surgical complications that vary from the expected usual complications.

Palpation

Patients may experience hyperpathia, allodynia, numbness, or hyperalgesia. Therefore, it is important to palpate the arm and hand to determine if these symptoms have a dermatomal distribution or if they are diffuse. If the symptoms are diffuse, the patient should be reevaluated after sympatholytic treatment.

Skin Integrity

The skin and any wounds around the affected area should be assessed carefully for signs of altered sensibility, abnormal autonomic function, and trophic changes.

Motor Testing

An evaluation of motor power must be performed to determine if there are weakness or endurance issues. Computerized endurance testing may be beneficial for collecting this information. Both intrinsic and extrinsic motor testing must be evaluated.

Range of Motion

The range of motion of all joints, including the shoulder, should be evaluated and recorded. Adhesive capsulitis or restricted range of motion of the shoulder is common in patients with long-standing CRPS. In patients with long-standing CRPS of the hand, intrinsic muscle contractures are also common. Therefore, the hand should be evaluated for any evidence of contracture.

Joint Stability

Joints should be assessed for global stability. Joint instability may elicit or contribute to nociceptive stimuli that contribute to CRPS. Instability of ulnohumeral, radiohumeral, distal radioulnar, radiocarpal, intercarpal, and finger joints may all contribute to significant nociception. In addition, tears in the triangular fibrocartilage complex or chronic sprains and strains may contribute to CRPS.

Neurologic Examination

A careful neurologic examination should be performed to identify any possible neuropathic involvement, especially spinal cord or brachial plexus involvement. In addition, the patient should be evaluated for movement disorders, such as dystonia.

Vascular Examination

Vascular examination is important to identify any vascular deficiencies and should not be neglected.

Sensibility Examination

Assessments of pain threshold (monofilaments) and innervation density (2-point discrimination) may be beneficial and can suggest a neuropathic component to the pain syndrome (CRPS type 2).

DIAGNOSTIC EVALUATION

Mechanical Testing

Pain threshold evaluations may be performed using algometers, dolorimeters, computer-assisted stimulation devices, and thermal threshold machines.[10] It is difficult to obtain these evaluations except in large medical centers; however, when available, they provide clinically useful, objective information. Von Frey monofilaments can also be used to determine pain thresholds.

Autonomic Function Evaluation

Autonomic function of the hands may be assessed by an evaluation of vasomotor control after the application of a stressor (ie, exposure to cold) as part of an isolated cold stress testing.[5] Sweat production may be determined by measuring resting sweat output and by the quantitative sudomotor axon reflex test.[11] Thermography, when used with a physiologic stressor, provides valuable confirmatory information.[12] These tests provide objective measures of autonomic function; however, they are only available at selected tertiary referral centers.

Radiologic Testing

Many investigators have recommended 3-phase bone scans as a diagnostic tool for CRPS. Positive scan results support the clinical diagnosis of CRPS. A positive 3-phase bone scan result in patients with CRPS is characterized by increased third phase bone scan periarticular uptake in all joints. Evidence of vasomotor instability and abnormal patterns of flow distribution may also be evident on phase I and phase II bone scans. However, these scans document vasomotor abnormalities, and therefore, they are not diagnostic for CRPS. The difficulty in using bone scans as a diagnostic tool for CRPS is that the scans may have insufficient sensitivity and specificity in patients with partially treated CRPS or in patients with variant presentations of the syndrome. These findings have led to Lee and Week's[13] analysis of the existing literature and their conclusion that ''a three phase bone scan is not a prerequisite for

the diagnosis of complex regional pain syndrome.'' Quantitative scintigraphies (bone scans) have demonstrated that there are both cortical and cancellous osteopenia that appear in excess of that observed with entry-matched controls treated with casting.[14]

Sympatholytic Challenge Testing

Sympathetically maintained pain (SMP) may be differentiated from sympathetically independent pain by a sympatholytic challenge provided by an intravenous injection of phentolamine, which is a combination of α_1- and α_2-adrenergic receptor blockers. In patients with SMP, the injection results in transient pain relief.[15] Similarly, stellate ganglion blocks, single cervical epidural blocks, sympathetic brachial plexus blocks, or scalene blocks may provide temporary relief of pain in patients with SMP. In patients who respond during the block, pain is relieved, whereas motor function remains. Although single blocks provide a useful diagnostic test, they are associated with significant false-negative rates. However, they are helpful in differentiating patients with SMP from patients with sympathetically independent pain.

TREATMENT

Early recognition of CRPS and the prompt initiation of treatment seems to improve patient outcomes. However, diagnosis and treatment of CRPS within 6 to 12 weeks of the onset of symptoms is not common in most patients; delays in diagnosis and treatment are especially common in patients with milder variants of CRPS. In addition, patients who develop CRPS after fractures, especially of the distal radius, seem to have a worse prognosis even when the CRPS is discovered early and treated promptly.

Treatment decisions are guided by the patients' symptoms and whether their pain is sympathetically maintained or sympathetically independent. In addition, the presence of an identifiable nociceptive or neuropathic component may require surgical intervention. Treatment is often multifactorial and involves a combination of therapy, oral medications, parenteral medications, and surgery. Hand therapy and various treatment modalities are usually combined with oral medications. Commonly, hand therapy including active and passive range of motion, splinting, and contrast baths (alternating heat and cold) is used. Other interventions that are beneficial for some patients are transcutaneous nerve stimulators, H-wave therapy, and stress loading.[10]

Oral pharmacologic intervention is used frequently to manage the symptoms of CRPS. Currently, there are no drugs for CRPS that are labeled by the Federal Drug Administration (FDA), and few drugs are approved for chronic neuropathic pain. However, based on clinical experience and the literature, the following classes of drugs with a sympatholytic component have been recommended for use in patients with CRPS: antidepressants, anticonvulsants, membrane stabilizing agents, and adrenergic agents. The most common oral agents in these categories are listed in **Table 2**. The types of oral medications that are prescribed depend, in part, on the patient's presentation. For a hot, swollen hand, an antidepressant combined with an anticonvulsant is prescribed in conjunction with hand therapy. Commonly used drugs include a tricyclic antidepressant in low doses (ie, amitriptyline [Elavil], 25 mg three times a day, or amitriptyline, 50 mg, at bedtime for a normal-sized adult) combined with phenytoin (Dilantin), 100 mg three times a day, or pregabalin (Lyrica), 75 to 100 mg twice a day or three times a day. For patients with cold, stiff hands, a mild antidepressant is often combined with a calcium channel blocker. Another common drug regimen is amitriptyline combined with amlodipine (Norvasc).

For patients with acute hyperalgesia or allodynia and mild to moderate edema, an adrenergic agent (eg, clonidine [Catapres]) may be of benefit. A clonidine patch (0.1 mg) is applied over the most sensitive area; the application of the patches is combined with hand therapy, including a stress loading program. Steroid dose packs are used by many physicians and have demonstrated efficacy in reducing symptoms. Corticosteroids act to stabilize membranes and to decrease inflammatory pain (**Table 1**).

Prophylactic Treatment

Vitamin C taken prophylactically at a dose of 500 mg/d has been shown to decrease the incidence of CRPS in patients who sustain distal radius fractures.[16] However, the role of other vitamins in the treatment of CRPS is unclear.

Parenteral Agents

Generally, parenteral agents are administered by anesthesiologists. However, recent controlled studies have not demonstrated the efficacy of intravenous treatment of CRPS.[17,18] Intravenous agents, such as guanethidine, cortisone, reserpine, lidocaine, and bretylium, have been used previously. Of these drugs, bretylium tosylate was the only drug labeled by the FDA for this

Table 1
Definitions

Nociception	Detection of an unpleasant (noxious) stimulus that produces pain in human subjects under normal circumstances
Allodynia	Pain in a specific dermatomal or autonomous distribution associated with light touch to the skin; a stimulus that is not normally painful
Hyperalgesia	Increased sensitivity to pain (includes allodynia and hyperesthesia)
Hyperesthesia	Increased sensitivity to stimulation (pain on response to a mild nonnoxious stimulus)
Sympathetic pain	Pain in the presence of and/or associated with over action of the sympathetic pain fibers; by definition, the pain is relieved by sympatholytic interventions
Hypoesthesia	Decreased sensitivity to stimulation
Hyperpathia	Abnormally painful reaction to a stimulus (especially repetitive); often includes extended duration of pain, frequently with a delay
Dysesthesia	An unpleasant, abnormal sensation
Paresthesia	An abnormal sensation

purpose; however, marketing of bretylium in the United States has been discontinued.

Epidural clonidine and corticosteroids have been used successfully in patients with refractory symptoms.[19,20] The blockade may be achieved by indwelling catheters located in the epidural space or contiguous with portions of the brachial plexus or peripheral nerves. These continuous blocks may be used for 1 to 5 days as an outpatient treatment when used to target peripheral nerves.

Some of the newer treatments for CRPS include the use of free radical scavengers, including dimethyl sulfoxide, vitamin C, and *N*-acetylcysteine.[16,21] Their mechanism of action is hypothesized to be related to their ability to interfere with oxygen radical–mediated inflammatory response.[22] Calcitonin,[23] bisphosphonates,[24–26] and *N*-methyl-D-asparate antagonists (eg, ketamine, memantine)[27,28] have been used to treat patients with CRPS, with reported reduction in symptoms. However, controlled trials will be required to determine the efficacy of these drugs.

Surgical Treatment

Surgery to correct neuropathic or nociceptive sites plays an important role in the treatment of patients with CRPS. If indicated, surgery may be performed early if the CRPS symptoms cannot be controlled medically. The dictum that surgery is inappropriate and doomed to failure in patients with CRPS is incorrect and deprives patients of valuable and necessary interventions. However, surgical procedures should be used with caution, and care should be taken in determining the appropriate choice of surgical intervention. Before surgery, it is important to confirm that the pain is predominantly SMP and that it can be controlled with medications or continuous blocks.

Surgery to release compressed nerves, to correct or cushion neuromas-in-continuity, or to relieve perineural fibrosis may offer significant benefits to some patients.[29,30] Another option, the surgical ablation of sympathetic nerves by reversible means, such as phenol injections or radiofrequency ablation, may provide symptom relief. In other patients, neurolysis and blockade of peripheral nerves may be efficacious. Spinal cord stimulation provides effective pain relief in 50% of patients.[31] Use of implantable nerve stimulators and dorsal column stimulators are valuable as salvage procedures in selected patients with refractory symptoms.[6,32,33] Other salvage procedures for the most difficult cases include the use of dorsal column stimulators and gray matter stimulators and cingulotomy.[34]

Psychological Treatment

Counseling, biofeedback, and adaptive therapy can be beneficial for certain patients with CRPS. Although CRPS is not a psychiatric disease, chronic pain does affect health-related quality of life. Patients with CRPS may experience reactive depression as a result of their symptoms.

LATE MANAGEMENT OF COMPLICATIONS RELATED TO CRPS

Surgery on the extremities of patients with CRPS is appropriate if pain cannot be managed using other methods. Assuming that perioperative pain control is possible using sympatholytic interventions,

Table 2
Oral medications

Drug	Usual Dosage	Mechanism	Major Short-Term Disadvantage or Side Effects	Contraindications
Amitriptyline hydrochloride (Elavil)	25 mg tid or 50 mg qhs	Inhibits amine pump-decreased norepinephrine reuptake	Drowsiness	With guanethidine sulfate
Amlodipine (Norvasc)	5–10 mg qd	Ca^{++} channel blocking agent; prevents arteriovenous shunting; increases nutritional flow	Headache Postural hypotension	
Corticosteroids	20–80 mg/d; prednisone equivalents × 5–40 d	Stabilizes membranes; increases nutritional flow; decreases inflammatory pain	Adrenal suppression Avascular necrosis (dose related)	
Fluoxetine (Prozac)	20 mg/d AM	Serotonin reuptake inhibitor	Minimal drowsiness	
Gabapentin (Neurontin)	300–600 mg tid		Dizziness Somnolence Ataxia	Renal disease; patients must be carefully monitored
Nifedipine (Procardia)	10 mg tid, may increase slowly to 30 mg tid	Ca^{++} channel blocking agent; prevents arteriovenous shunting; increases nutritional flow	Headache Postural hypotension	
Phenoxybenzamine hydrochloride (Dibenzyline)	40–120 mg/d	α_1-receptor blocking agent	Orthostatic hypotension	
Phenytoin (Dilantin)	100 mg tid	Decreases resting membrane potentials; inhibits amine pump; stabilizes synaptic membrane	Ataxia Liver damage Convulsion	Liver disease Pregnancy
Pregabalin (Lyrica)	50–200 mg tid		Dizziness Somnolence Peripheral edema	

nociceptive and neuropathic foci may be managed by appropriate neurolysis of compressed nerves, repair of damaged nerves, or perineural fibrosis in correction of mechanical lesions. In addition, patients with CRPS may develop MP and PIP joint contractures that can be managed by surgical release. Contracted intrinsic muscles are common in these patients, and intrinsic releases may be beneficial. In addition, bony malunion or nonunion may occur because of the CRPS-induced osteopenia. For these patients, osteotomies may be warranted to gain the necessary correction. Neuropathic foci are managed using the same techniques that are used for patients with neuropathic foci without CRPS. Nerve repair, neurolysis, nerve wrapping, resection of neuromas, and temporary blockade with phenol or cryoablation are various techniques that can be used successfully in these patients.

SPECIFIC ENTITIES

Distal Radius Fracture

The reported incidence of CRPS after distal radius fracture ranges from 2% to 39% and is one of the leading causes of poor outcomes and malpractice claims.[6] The incidence of CRPS is increased as a result of improper casting. The presence of CRPS and the resultant osteoporosis (in excess of similarly treated non-CRPS fracture) contributes to delayed healing and nonunion. The delay in healing and the occurrence of nonunion is secondary to bone demineralization.[14] Onset of CRPS after distal radius fracture is variable and often delayed; stiffness, difficulty sleeping, burning pain, and cold sensitivity are the common symptoms. Median nerve involvement, often slow and insidious, may provide a neuropathic component, and for these patients, surgical release of the median nerve may be beneficial. In patients who are treated with external fixators or who have radial incisions, irritation or injury of the superficial radial nerve may create a neuropathic focus. Overdistraction, radioulnar joint instability, intercarpal instability, unstable triangular fibrocartilage complex, and/or chondral injury may contribute as nociceptive drivers of the pain process.

The most common presentation of CRPS after distal radius fracture is a warm, swollen hand; hyperpathia or allodynia; stiffness in which edema is responsive to elevation; and pain that is refractory to narcotics. The hot, swollen presentation usually occurs at 1 to 3 days after surgery, with associated median nerve symptoms reported by many patients. A more problematic alternative presentation of CRPS occurs when pain and stiffness are the predominate symptoms. The extremities of these patients are often cool, and swelling may be minimal. Patient compliance is compromised if active range of motion is restricted. These patients tend to request additional narcotics, and acquiescence to their demands by their treating physicians is rarely beneficial. Delayed union and osteopenia are often observed. Phase I and phase II bone scans may show hyperperfusion (hot/warm hand) and hypoperfusion (cool stiff hand); 3-phase scans show increased uptake at the fracture site and in the periarticular areas of the hand and wrist.

In patients with CRPS after distal radius fractures, awareness of the vagaries of presentation is crucial. Because the onset of CRPS may occur later, patients may develop symptoms during the 2- to 3-week period between their scheduled follow-up physician appointments. Therefore, office staff and other providers (especially nurses, nurse practitioners, physician assistants, and therapists) need to understand the importance of early diagnosis of CRPS based on symptoms reported by the patients. In patients reporting pain and stiffness, a chart notation that addresses the presence or absence of CRPS is an important safeguard. If CRPS is suspected, this concern should be noted, a sympatholytic medication should be prescribed, and consultation with a physician should be arranged. In patients who have coexistent carpal tunnel neuropathy, neurolysis of the median nerve should be performed.

Carpal Tunnel Surgery and CRPS

Severe chronic pain that occurs after carpal tunnel release surgery is classified as type 2 or neuropathic CRPS. In this instance, CRPS may be associated with nerve irritations related to the procedure (idiopathic), perineural fibrosis, or nerve injury (neuropraxia, neuromas-in-continuity, nerve transection). Onset of symptoms may be rapid, and patient symptoms can be apparent in the recovery room. However, in most cases, symptoms occur at 1 to 3 weeks after surgery. Injury to the palmar cutaneous nerve branch may occur and may be problematic in patients with coexistent CRPS. Patients present with pain, cold sensitivity, variable swelling, and difficulty sleeping. Nonsurgical management is the first line of treatment; however, for patients with refractory SMP, surgical intervention may be needed after an appropriate period of medical intervention fails to control pain.

If CRPS symptoms are only partially resolved after combinations of oral or parenteral pharmacologic treatment and therapy, surgery for neuropathic CRPS may be considered. Treatment options for perineural fibrosis include: (1) neurolysis alone,

(2) neurolysis and nerve wrapping, (3) neurolysis and local adipose transposition, and (4) neurolysis and muscle or fascial transposition or flaps. In the case of nerve injury, the injured nerves may be explored and neuromas may be repaired, resected, or transposed. However, palmar cutaneous nerve injuries are often difficult to repair. For the cases in which nerve repair is not possible, resection within soft tissue muscle implantation or grafting into an uninjured area is an option. As a salvage procedure, implantable nerve stimulations are available.[35]

Mechanical Derangements/Arthritis/ Cartilage Injury

Nociceptive events that contribute to dystrophic processes include intercarpal dissociations, osteochondral fractures with loose bodies, radiocarpal subluxation, triangular fibrocartilage complex injury, distal radioulnar joint instability, and chondrolysis. If symptoms are controlled incompletely by nonoperative interventions, surgery is appropriate to address these mechanical events.

SUMMARY

CRPS after hand surgery is not uncommon and may complicate postoperative care. Early diagnosis and treatment of CRPS is critical for optimal patient outcomes.

REFERENCES

1. de Mos M, de Bruijn AG, Huygen FJ, et al. The incidence of complex regional pain syndrome: a population-based study. Pain 2007;129(1–2):12–20.
2. Sandroni P, Benrud-Larson LM, McClelland RL, et al. Complex regional pain syndrome type I: incidence and prevalence in Olmsted County, a population-based study. Pain 2003;103(1–2):199–207.
3. Reuben SS. Preventing the development of complex regional pain syndrome after surgery. Anesthesiology 2004;101(5):1215–24.
4. Lichtman DM, Florio RL, Mack GR. Carpal tunnel release under local anesthesia: evaluation of the outpatient procedure. J Hand Surg Am 1979;4(6): 544–6.
5. MacDonald RI, Lichtman DM, Hanlon JJ, et al. Complications of surgical release for carpal tunnel syndrome. J Hand Surg Am 1978;3(1):70–6.
6. Koman LA, Poehling GG, Smith BP, et al. Complex regional pain syndrome. In: Green D, Hotchkiss R, Pederson W, et al, editors. Green's operative hand surgery. 5th edition. Philadelphia: Churchill Livingstone; 2005. p. 2015–44.
7. Field J, Warwick D, Bannister GC. Features of algodystrophy ten years after Colles' fracture. J Hand Surg Br 1992;17:318–20.
8. An HS, Hawthorne KB, Jackson WT. Reflex sympathetic dystrophy and cigarette smoking. J Hand Surg Am 1988;13:470–2.
9. Grunert BK, Devine CA, Sanger JR, et al. Thermal self-regulation for pain control in reflex sympathetic dystrophy syndrome. J Hand Surg Am 1990;15:615–8.
10. Li Z, Smith BP, Smith TL, et al. Diagnosis and management of complex regional pain syndrome complicating upper extremity recovery. J Hand Ther 2005;18(2):270–6.
11. Chelimsky TC, Low PA, Naessens JM, et al. Value of autonomic testing in reflex sympathetic dystrophy. Mayo Clin Proc 1995;70(11):1029–40.
12. Bruehl S, Lubenow TR, Nath H, et al. Validation of thermography in the diagnosis of reflex sympathetic dystrophy. Clin J Pain 1996;12(4):316–25.
13. Lee GW, Weeks PM. The role of bone scintigraphy in diagnosing reflex sympathetic dystrophy. J Hand Surg Am 1995;20(3):458–63.
14. Bickerstaff DR, Kanis JA. Algodystrophy: an underrecognized complication of minor trauma. Br J Rheumatol 1994;33:240–8.
15. Raja SN, Treede RD, Davis KD, et al. Systemic alpha-adrenergic blockade with phentolamine: a diagnostic test for sympathetically maintained pain. Anesthesiology 1991;74(4):691–8.
16. Zollinger PE, Tuinebreijer WE, Breederveld RS, et al. Can vitamin C prevent complex regional pain syndrome in patients with wrist fractures? A randomized, controlled, multicenter dose-response study. J Bone Joint Surg Am 2007;89(7):1424–31.
17. Lake AP. Intravenous regional sympathetic block: past, present and future? Pain Res Manag 2004; 9(1):35–7.
18. Livingstone JA, Atkins RM. Intravenous regional guanethidine blockade in the treatment of post-traumatic complex regional pain syndrome type 1 (algodystrophy) of the hand. J Bone Joint Surg Br 2002; 84(3):380–6.
19. Dirksen R, Rutgers MJ, Coolen JM. Cervical epidural steroids in reflex sympathetic dystrophy. Anesthesiology 1987;66(1):71–3.
20. Rauck RL, Eisenach JC, Jackson K, et al. Epidural clonidine treatment for refractory reflex sympathetic dystrophy. Anesthesiology 1993;79(6):1163–9.
21. Perez RS, Zuurmond WW, Bezemer PD, et al. The treatment of complex regional pain syndrome type I with free radical scavengers: a randomized controlled study. Pain 2003;102(3):297–307.
22. Oyen WJ, Arntz IE, Claessens RM, et al. Reflex sympathetic dystrophy of the hand: an excessive inflammatory response? Pain 1993;55(2):151–7.
23. Hamamci N, Dursun E, Ural C, et al. Calcitonin treatment in reflex sympathetic dystrophy: a preliminary study. Br J Clin Pract 1996;50(7):373–5.
24. Manicourt DH, Brasseur JP, Boutsen Y, et al. Role of alendronate in therapy for posttraumatic complex

regional pain syndrome type I of the lower extremity. Arthritis Rheum 2004;50(11):3690–7.
25. Robinson JN, Sandom J, Chapman PT. Efficacy of pamidronate in complex regional pain syndrome type I. Pain Med 2004;5(3):276–80.
26. Varenna M, Zucchi F, Ghiringhelli D, et al. Intravenous clodronate in the treatment of reflex sympathetic dystrophy syndrome. A randomized, double blind, placebo controlled study. J Rheumatol 2000; 27(6):1477–83.
27. Goldberg ME, Domsky R, Scaringe D, et al. Multiday low dose ketamine infusion for the treatment of complex regional pain syndrome. Pain Physician 2005;8(2):175–9.
28. Sinis N, Birbaumer N, Gustin S, et al. Memantine treatment of complex regional pain syndrome: a preliminary report of six cases. Clin J Pain 2007; 23(3):237–43.
29. Jupiter JB, Seiler JG III, Zienowicz R. Sympathetic maintained pain (causalgia) associated with a demonstrable peripheral-nerve lesion. Operative treatment. J Bone Joint Surg Am 1994;76(9):1376–84.
30. Koman LA, Smith BP, Smith TL. Stress testing in the evaluation of upper-extremity perfusion. Hand Clin 1993;9(1):59–83.
31. Taylor RS, Van Buyten JP, Buchser E. Spinal cord stimulation for complex regional pain syndrome: a systematic review of the clinical and cost-effectiveness literature and assessment of prognostic factors. Eur J Pain 2006;10(2):91–101.
32. Barolat G, Schwartzmann R, Woo R. Epidural spinal cord stimulation in the management of reflex sympathetic dystrophy. Appl Neurophysiol 1987;50:442–3.
33. Nashold BS Jr, Friedman H. Dorsal column stimulation for control of pain. J Neurosurg 1972;36:590–7.
34. Santo JL, Arias LM, Barolat G, et al. Bilateral cingulumotomy in the treatment of reflex sympathetic dystrophy. Pain 1990;41(1):55–9.
35. Strege DW, Cooney WP, Wood MB, et al. Chronic peripheral nerve pain treated with direct electrical nerve stimulation. J Hand Surg Am 1994;19(6):931–9.