Treatment of Complex Regional Pain Syndrome of the Lower Extremity

Christopher J. Hogan, MD, and Shepard R. Hurwitz, MD

Abstract

Complex regional pain syndrome, formerly known as reflex sympathetic dystrophy or causalgia, is a difficult therapeutic problem for the orthopaedic surgeon treating an affected lower extremity. Despite many divergent and often conflicting theories, the cause of the severe pain, alterations in regional blood flow, and edema is unknown. Interventions that have proved successful for treating similar conditions in the arm and hand frequently do not relieve pain similarly in the lower extremity. Common treatment regimens target individual components of this symptom complex, namely, sympathetic or afferent nerve hyperactivity, vasomotor instability, or regional osteoporosis. Despite widespread use of some of these treatments, few controlled clinical trials quantify their effectiveness. This challenging syndrome is best managed by a multidisciplinary team, including chronic pain management specialists, physical therapists, and orthopaedic surgeons.


The normal physiologic response of an extremity to a painful injury involves humoral and neural signals that give rise to increased blood flow, edema, limited joint excursion, and hypersensitivity to pain. These signs and symptoms are usually proportional to the severity of the injury and resolve with healing of the damaged tissues. When this injury response is prolonged, a chronic pain syndrome results. With time, painful symptoms may expand from the site of injury to involve the entire extremity, leading to a persistently swollen, painful, and ultimately functionless limb.

This progressive pain pattern has been recognized for over 100 years and has been described variously as causalgia, minor causalgia, major causalgia, mimicaisalgia, pseudocausalgia, reflex sympathetic dystrophy, algodystrophy, algoneurodystrophy, posttraumatic dystrophy, Sudeck's atrophy, and sympathetically maintained pain syndrome. To minimize the confusion in terminology, the International Association for the Study of Pain coined the term complex regional pain syndrome (CRPS) to describe this constellation of symptoms. Within this diagnosis are two subtypes, which differ only by the presence of a definable peripheral nerve injury in type II.

CRPS involving the lower extremity presents a diagnostic and therapeutic challenge to the orthopaedic surgeon. Often discomfort is severe and patients are unable to gain relief. Although this syndrome occurs in both the upper and lower extremities, it is of interest that the success of pharmacologic and other treatment modalities differs between the upper and lower extremities.1-5 Frequently, the symptoms in the lower extremity are more refractory to intervention than those in the upper extremity.6 This differential response has led some to suggest that CRPS of the legs and feet is a discrete clinical entity.7-10 Narcotic pain medications are of little restorative value and, if used, frequently result in drug dependence without improving limb function. Complicating management are social issues, such as liability or worker's compensation or disability, that may provide a financial disincentive for patients to report improvement. Often there is overlying psychological dysfunction, as well. Because of the many physiologic and psychological factors involved in pain, a multidisciplinary approach should be developed for each patient, integrating the efforts of the orthopaedic surgeon, anesthesiologist, physiatrist, physical therapist, occupational therapist, and psychiatrist.

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Pathophysiology

The pathophysiology of CRPS remains unknown, and little clinical evidence explains why certain treatment protocols appear to be successful. Divergent theories abound, likely because the spectrum of presentations of this syndrome is so diverse. This suggests that responses to treatment vary because heterogeneous conditions are being treated.

The older term reflex sympathetic dystrophy describes a pathologic reflex of the sympathetic nerves that causes blood flow irregularities, resulting in constant pain, muscle atrophy, and fibrosis. The proponents of this etiology cited the pain relief from sympathetic block as supportive evidence. They also advocated the use of calcium channel blockers to counteract the vasoconstriction of increased sympathetic-adrenergic activity. Sympathetic block, however, did not prove to be a reliable predictor of treatment response and for this reason, the term reflex sympathetic dystrophy was discarded.\textsuperscript{11,12}

Several other theories were offered based on response of peripheral or systemic antagonism to \( \alpha \)- or \( \beta \)-adrenergic agonists.\textsuperscript{1,3,13-16} The theories of injury-induced hypersensitivity to circulating catecholamines are based on many reports of positive clinical response to pharmacologic block using phenolamine, phenoxybenzamine, or propranolol. A subset of the catecholamine hypersensitivity theories suggested that the site of the pathologic receptors is in the skin rather than in the small vessels.

Local inflammatory factors were the rationale for the use of high-dose corticosteroids.\textsuperscript{17,18} This seemed a logical explanation for the swelling and pain in an area that was physically traumatized. The advocates of this theory suggested that the painful inflammation, in turn, disrupted local autoregulation of blood flow, and thus explained the classic phases of reflex sympathetic dystrophy.

A group of regional pain syndromes is associated with known nerve injury, previously referred to as causalgia. The concept of a painful local nerve injury establishing constant pain through a central mechanism in the spinal cord or spinothalamic network was an explanation that did not involve catecholamines or the sympathetic nerves. A variation on the centrally mediated pain mechanism was the proposal that peripheral nerve injuries created aberrant neuronal connections between peripheral sensory and sympathetic nerves. To support their theory, advocates of neural injuries cited success with membrane-stabilizing medications, such as bretylium, gabapentin, and calcium channel blockers.\textsuperscript{3,10,19-22}

Diagnosis and Clinical Course

The diagnosis of CRPS is based on physical examination findings because no laboratory or radiologic tests can reliably confirm or exclude the diagnosis. Traditionally, the clinical course of CRPS has been divided into three stages that describe the physical characteristics of the syndrome (Table 1). In the hand, these stages are relevant to the effectiveness of intervention, with earlier intervention more likely to result in successful outcomes. Whether early intervention im-

<p>| Table 1 |
| Stages of Complex Regional Pain Syndrome |</p>
<table>
<thead>
<tr>
<th>Stage</th>
<th>Usual Time Course (mo)</th>
<th>Clinical Features</th>
<th>Radiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>0 to 3</td>
<td>Warm, red, edematous extremity; aching, burning pain; intolerance to cold; altered sweat pattern; joint stiffness without any significant effusion; hyperesthetic skin; no fixed joint contractures</td>
<td>Normal plain radiographs; may have abnormal uptake of imaging agent on bone scan</td>
</tr>
<tr>
<td>Dystrophic</td>
<td>3 to 6</td>
<td>Cool, cyanotic, edematous extremity; shiny, hyperesthetic skin; fixed contractures; fibrotic changes occur in the synovium</td>
<td>Subchondral osteopenia; patellar and medial femoral condyle osteopenia on sunrise view; may have abnormal uptake of imaging agent on bone scan</td>
</tr>
<tr>
<td>Atrophic</td>
<td>6 to 12</td>
<td>Loss of hair, nails, skin folds; fixed contractures; muscle wasting</td>
<td>Bone demineralization</td>
</tr>
</tbody>
</table>

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proves the prognosis in CRPS of the lower extremity, however, is disputed.\textsuperscript{1,7-10} In the acute stage (within 3 months after the injury), patients report a burning or aching pain that generally does not respond to narcotics and is considerably more intense than expected for the degree of injury. Symptoms typically begin soon after the injury—within hours or days—but may develop more insidiously over several weeks. One of the first clinical signs is an extreme intolerance to cold, and patients often state that motion, dependency of the limb, and touch aggravate their symptoms. The skin is usually warm and hyperemic and frequently demonstrates an altered pattern of perspiration that ranges from dry to increased sweating. Early in its course, the pain may follow the distribution of a cutaneous nerve, but with time this may progress to involve the entire extremity. Mild edema around the joints is common, but true joint effusions are rare (Fig. 1). Although patients report a sensation of joint stiffness, examination under anesthesia usually reveals a full range of motion.

Plain radiographs help determine whether other definable lesions, such as stress fractures, are responsible for the symptoms. For CRPS in the acute phase, there are neither radiographic changes nor evidence of osteoporosis. Technetium 99m bone scans are commonly used to attempt to confirm the diagnosis of CRPS, but they have a specificity of 75\% to 98\% and a sensitivity of only 50\% for this condition.\textsuperscript{11} The most common finding is increased periarticular uptake of the imaging agent in each phase, although decreased flow has been reported in the acute setting.\textsuperscript{2,23} As a result, bone scan findings are generally reported as "abnormal" compared with those for the unaffected contralateral extremity.

In the dystrophic stage, which begins approximately 3 months after the initial injury, pain is constant and aggravated by any stimulus. If the ankle is invovled, a fixed equinovarus deformity of the hindfoot may develop, with firm induration around the tibiotalar joint.\textsuperscript{11} The skin is frequently cool to the touch, cyanotic, and shiny, and hair may be scant. Radiographs of the involved extremity may reveal patchy subchondral osteopenia from both the disuse and the hyperemia of the acute stage (Fig. 2), although this finding may be absent in up to 20\% of patients who meet the diagnostic criteria for CRPS.\textsuperscript{11} If the knee is involved, osteopenia of the patella and medial femoral condyle on the sunrise view characteristically is present.\textsuperscript{7,24,26} Bone scan imaging at this time frequently demonstrates altered uptake of the imaging agent in the affected limb, particularly in the periarticular region.\textsuperscript{27} Synovial biopsies demonstrate increased fibrosis and synovial proliferation without any evidence of inflammatory changes.\textsuperscript{25} This degree of fibrosis increases with the duration of symptoms.

The atrophic stage begins about 6 months to 1 year after the onset of symptoms, when the skin and perfusion changes become fixed, leading to the clinical appearance of cyanotic, shiny, pale skin, with loss...
of the usual skin folds. The joint motion is severely restricted, and fixed contractures are common. Significant muscle wasting is apparent on inspection, and there is radiographic evidence of profound bone demineralization.

### Treatment

No current consensus exists regarding the most effective treatment regimen for CRPS of the lower extremity. Several studies report treatment results with the modalities used in upper extremity cases. However, these studies had either small numbers of patients or limited clinical follow-up.\textsuperscript{11,13,28} In addition, the heterogeneity of the patient populations precludes a single algorithm to guide treatment. Clinical trials for CRPS of the lower extremity are listed in Table 2.

The use of intravenous regional sympathetic blocks was first popularized by Hannington-Kiff in 1974, who suggested that guanethidine might relieve pain for patients with CRPS. The goal of this therapy is to reduce the sympathetic input to the limb, based on the theory that chronic pain results from either a central hyperactivity of the sympathetic nervous system\textsuperscript{33} or a peripheral hypersensitivity to circulating catecholamines.\textsuperscript{34,35} To achieve a block, the affected extremity is exsanguinated and placed under tourniquet control, and a sympathetic agent is infused into a vein. The medication suffuses the tissues via the venous pathways while the tourniquet prevents the circulation of the medication into the systemic vasculature. The drugs that have been used in intravenous regional blocks are guanethidine, reserpine, and bretylium, all of which inhibit release of norepinephrine by displacing it from neuronal storage vesicles. In the United States, intravenous preparations of guanethidine and reserpine are no longer available, leaving bretylium as the only option.

The efficacy of intravenous regional sympathetic blocks in the treatment of CRPS is unclear because separate studies dealing with very similar patient populations report contradictory levels of response to treatment. Some studies showed no difference between guanethidine, reserpine, and saline control,\textsuperscript{5,36,37} whereas some demonstrated pain relief in about half of the lower extremity patients,\textsuperscript{33,38} and others, improvement in approximately 75\% of patients.\textsuperscript{29} None of these studies mentioned the duration of benefit.

Similarly, the efficacy of bretylium for intravenous regional sympathetic blocks is uncertain. Although bretylium has been reported to be an effective treatment for CRPS, the largest published series does not discriminate between upper and lower extremity involvement.\textsuperscript{39} Of two studies that used bretylium,
comprising a total of five cases, one claimed total resolution of symptoms\textsuperscript{3} and the other, no benefit.\textsuperscript{19} Such conflicting reports of treatment response are difficult to reconcile, especially with the small number of patients included.

Several authors have used lumbar sympathetic blocks with either lidocaine or bupivacaine to manage CRPS of the knee.\textsuperscript{1,8,10,40} This technique involves introducing a needle into the region of the paravertebral lumbar sympathetic ganglia under either fluoroscopic or computed tomographic guidance and infiltrating the area with the local anesthetic. As an essential criterion for the diagnosis of CRPS, three of these protocols required that patients demonstrate pain relief following the administration of phenolamine, a criterion that may favorably bias the patient population toward this therapy. Although 93 of 112 total patients had some degree of pain relief, most had residual symptoms. In one series, only 12% of patients were pain free at 3 years' follow-up,\textsuperscript{1} whereas in another, only 57% of patients had a good response, although there was no mention of the duration of pain relief.\textsuperscript{4} With lumbar sympathetic block, in contrast to most other types of treatments, there appears to be no correlation between the duration of symptoms and the response to treatment.\textsuperscript{1,8,10,40}

Use of either continuous epidural anesthesia or an intrathecal narcotic pump allows either low-dose narcotics or local anesthetics to be administered locally, resulting in fewer systemic side effects than are seen with intravenous administration. When delivered locally, narcotics can help break the pain cycle, whereas the anesthetic agents provide a relatively selective sympathetic blockade. The narrow diameter of the unmyelinated sympathetic fibers allows for a block of these nerves with minimal inhibition of the motor or sensory fibers.

Two published series report successful treatment with this intervention, either through placement of an indwelling morphine pump\textsuperscript{41} or administration of continuous epidural anesthesia in conjunction with continuous passive motion.\textsuperscript{30} Despite this success, the risks and costs of these treatments are considerable. Continuous epidural anesthesia requires hospitalization for the duration of treatment and carries the risks of urinary retention, skin breakdown, and hypotension. Intrathecal morphine pumps are relatively expensive and require periodic refilling of the narcotic reservoir.

The use of $\alpha$-adrenergic blocking agents is based on addressing the altered blood flow demonstrated in patients with CRPS, which is theorized to result from increased local secretion of norepinephrine and vascular endothelial hypersensitivity to this neurotransmitter. Normally, peripheral blood flow is determined largely by sympathetic activity at the $\alpha_1$-adrenergic receptors, with increased stimulation leading to vasoconstriction. Inhibition of the receptors leads to dilation of the arterioles and increased blood flow. Phenolamine is an $\alpha_1$-adrenergic sympathetic blocking agent with a very short duration of action. Pain relief following intravenous administration has been proposed as a diagnostic test for CRPS as well as a prognostic guide for favorable response to sympathetic block.\textsuperscript{15,42} The 15-minute plasma half-life of this medication, however, precludes its use as a therapeutic intervention. Phenoxybenzamine and prazosin have been used successfully in patients with lower extremity CRPS, although none of these reports states whether patients required long-term treatment.\textsuperscript{14,34} In contrast, another $\alpha$-adrenergic antagonist, droperidol, had no clinical benefit.\textsuperscript{45}

Intravenous or oral clonidine or the combination is commonly used as an intervention, likely on a theoretical basis because no published series documents its efficacy. One small series demonstrated temporary relief with topical use.\textsuperscript{42} There is some clinical evidence that an oral beta blocker may improve symptoms. Although the most dramatic property of propranolol is its peripheral beta blockade, this drug also demonstrates central nervous system activity via antagonism of serotonin. A total of five patients with acute CRPS treated with oral propranolol has been reported. Three of the patients were free of symptoms at their last follow-up,\textsuperscript{16,46} and two demonstrated no change in their symptoms.\textsuperscript{47} Pain relief occurred several weeks after beginning treatment in those who had relief. We found no reports of the use of intravenous beta blocker therapy or the use of any other beta blocker.

Oral calcium channel blockers oppose the vasoconstriction mediated by the sympathetic nervous system by causing smooth-muscle relaxation in arteriole walls, leading to increased peripheral blood flow. These drugs have been used successfully in vasospastic conditions, such as Reynaud's disease. The largest reported clinical series of CRPS patients treated with oral calcium channel blockers did not distinguish between the results of treating upper and lower extremity symptoms, although a high degree of pain relief was reported.\textsuperscript{43} Prough et al\textsuperscript{20} reported success in three patients with lower extremity CRPS with this intervention, but two required maintenance doses of nifedipine to prevent a recurrence of symptoms.

Reports of the efficacy of the selective serotonin blocker ketanserin in treating CRPS mostly have not differentiated between upper or lower extremity involvement. This use is based on the fact that serotonin demonstrates significant vasoconstrictive properties, and low concentrations of this neurotrans-
mitter applied topically have been shown to cause pain. Bounameaux et al.\(^{48}\) stated that, although oral ketanserin effectively increased blood flow to the involved leg in the majority of patients, none of the eight patients with lower extremity involvement reported any lasting pain relief. From this finding, the authors concluded that decreased peripheral blood flow might not be the crucial factor in some patients with CRPS.

Treatment with bisphosphonates is based on the concept that pain results from the osteopenia created by blood flow derangements and chronic disuse. Adami et al.\(^{49}\) reported that alendronate was effective in treating patients with long-standing CRPS, but their results did not distinguish between upper and lower extremity involvement. Rehman et al.\(^{50}\) showed that these patients have lower than normal bone density and that this relative osteoporosis improves with the use of pamidronate. The time to improvement in bone density in this study paralleled the time to improvement of symptoms seen by Cortet et al.\(^{31}\) and Devogelaer et al.\(^{32}\) These studies, comprising a total of 32 patients symptomatic for at least 6 months, reported improvement in symptoms in all cases but did not mention the duration of pain relief or whether patients required chronic therapy.

The rationale for the clinical use of anticonvulsants in patients with CRPS is that injured neurons have abnormal sensitivity and may send impulses spontaneously that lead to the perception of pain. Although review articles have reported treatment of lower extremity CRPS with carbamazepine, clonazepam, valproic acid, and phenytoin,\(^{11,12}\) we have uncovered no clinical studies substantiating the effectiveness of these drugs.

The only anticonvulant studied clinically and reported in the literature to date is gabapentin, which acts both peripherally and centrally to depress the excitatory pathways and stimulate the inhibitory pathways. This drug increases central nervous system levels of serotonin, which is an inhibitory neurotransmitter. Mellick and Mellick\(^{21}\) reported that gabapentin relieved pain in a series of six patients with CRPS, two of whom had symptoms in the lower extremity for 3 years. They did not mention the duration of pain relief or whether these patients required chronic treatment with the medication.

Antiarrhythmic medications also have been used to suppress the spontaneous discharge of injured neurons and depress C fiber–mediated reflexes at the level of the spinal cord. The efficacy of bretylum, a class III antiarrhythmic, has been discussed. Mexiletine is an oral agent similar in action to lidocaine, which was used by Chaubet et al.\(^{22}\) to treat three patients with chronic type II CRPS of the lower extremity. They found improvement in two of three patients but did not mention the duration of benefit or whether the patients required long-term treatment.

Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit the enzyme cyclooxygenase. This inhibition leads to a decreased production of prostaglandins and thromboxanes, substances that increase the sensitivity of nociceptors to painful stimuli and promote vascular constriction. Despite the widespread use of this class of medications, no published trials report the use of oral NSAIDs to treat CRPS. The benefits of intravenous ketorolac were reported in a single small series.\(^{17}\) All three patients with lower extremity symptoms demonstrated temporary pain relief, which increased in duration with serial doses. The average duration of benefit was 15 days, allowing most patients to begin edema control measures and attempt to regain joint motion.

The potent anti-inflammatory effects of corticosteroids make these drugs an attractive option for treatment of CRPS because, theoretically, decreased tissue edema will lessen pain and improve joint motion. Use of prednisone, prednisolone, or methylprednisolone is commonly cited in the treatment of lower extremity CRPS,\(^{11,13,28}\) but most studies supporting their use combine the results of patients with upper and lower extremity involvement or include only those with upper extremity symptoms.\(^{18}\)

Poplawski et al.\(^{9}\) however, reported on a series of 27 patients with CRPS, 8 of whom had lower extremity involvement. Using intravenous regional lidocaine and methylprednisolone, they found that 60% of patients with upper extremity involvement had a good or excellent result compared with 50% of those with lower extremity symptoms. No definitive studies support the benefit of oral corticosteroids in the treatment of lower extremity CRPS.

Gentle physical therapy both to control edema and prevent joint contracture is beneficial in all stages of CRPS. Activity is thought to improve function, but it has little proven effect on pain. There is no compelling evidence that any exercise or activity is curative. Overly aggressive therapy should be avoided because this can exacerbate the patient's sense of loss of control in the treatment process. Although the use of transcutaneous electrical nerve stimulation (TENS) units is widespread, their usefulness has been demonstrated only in a small series in the pediatric literature,\(^{51}\) and there is no published report of the efficacy of this intervention in adults. In a small study, other modalities, such as daily ultrasound treatments, have been shown to be beneficial in treatment of CRPS of the foot.\(^{40}\)
Treatment Overview

Several conclusions can be drawn about CRPS of the lower extremity. There are no defined risk factors for developing CRPS, and this syndrome has been described after both major and incidental trauma to the lower extremity. The pain and edema generally do not resolve without treatment, and patients frequently have residual symptoms, despite prompt intervention. The treating orthopaedic surgeon who suspects CRPS should keep in mind that these patients may have other mechanical derangements contributing to their pain, such as a stress fracture or degenerative arthritis. A careful, detailed history and physical examination should be done in every case to eliminate the possibility of concomitant pathology. Intrarticular pathology requiring surgical intervention should be addressed after the symptoms of CRPS have subsided; even in this setting, CRPS recurs after surgery in up to 47% of patients. Because of this high reported risk of recurrence, exploratory surgery should be avoided. Radiographs and nuclear bone scans are helpful in confirming the diagnosis but may be falsely negative early in the course of the syndrome. Magnetic resonance imaging has no proven diagnostic value.

When the patient has no apparent mechanical derangement, ancillary services should be recruited within 4 weeks to 3 months to break the pain-impairment cycle of CRPS. A multidisciplinary approach, using physical therapy and pain control measures, is preferred to a single physician dispensing treatment. Physical therapy is useful in all stages, but forced motion should be avoided because patients' sense of losing control can be exacerbated by overly aggressive manipulation. Edema control can most effectively be accomplished with compression, gentle motion, and distal-to-proximal massage. Ice or ultrasound may be helpful, but often patients do not tolerate extremes of heat and cold well, limiting the usefulness of these modalities. Progressive tactile desensitization may provide a useful adjunct, although this is less important than in patients with upper extremity CRPS because ambulation commonly provides adequate stimulation.

Many different medications have been used in the treatment of CRPS of the lower extremity, with varying degrees of success. Narcotic pain medications should come from one physician to minimize the risk of drug dependence and drug interactions. Orthopaedic surgeons do not commonly prescribe these medications and therefore should consult early with a specialized pain clinic. Patients should be referred to pain clinic services within 3 months of the diagnosis of CRPS. Gabapentin, prazosin, propranolol, nifedipine, and me trendine all have been used in successful treatment protocols, but studies with these drugs involved fewer than five patients with lower extremity symptoms, making it difficult to draw conclusions regarding the efficacy of each intervention.

The greatest clinical experience is with the use of intravenous regional administration of sympatholytic agents, NSAI ds, or corticosteroids. Considered as one large group, patients treated with guanethidine, reserpine, or bretylium responded similarly to those treated with ketorolac, prednisolone, or methylprednisolone, with approximately half reporting relief. This suggests that other mechanisms may contribute to the clinical response.

Some of the controversy surrounding intravenous regional techniques comes from studies suggesting that the pain relief results, not from the infusion of a sympatholytic agent, but from the tourniquet-induced ischemia. Rocco et al and Blanchard et al demonstrated no difference in results between guanethidine, reserpine, and normal saline infusion administered to patients with lower extremity CRPS, a finding supported by Jadad et al, who demonstrated no difference between guanethidine and saline infusions. These authors suggest that tourniquet-induced ischemia may provide enough pain relief to allow for increased physical therapy, but no studies have tested this hypothesis.

More encouraging results were seen after paravertebral sympathetic blocks and after treatment with continuous epidural anesthesia. All of the studies of these two interventions used response to phentolamine as a diagnostic criterion for CRPS, a factor that may select for a better response to treatment with anti-α-adrenergic agents.

Pamidronate, a bisphosphonate, has demonstrated encouraging results in the treatment of CRPS of the lower extremity. Although it is difficult to ascribe all of the clinical features of CRPS to osteopenia, this medication reportedly led to improvement or resolution of symptoms in all patients in the study populations.

Summary

CRPS of the lower extremity poses a management challenge. The etiology is unclear, and theories to explain the condition, as well as proposed therapies, abound. Therapies that may be effective for upper extremity CRPS are often not as effective for lower extremity CRPS, leading some to conclude that it is a different entity.

No laboratory or radiologic tests can reliably confirm the diagnosis. In the acute stage, burning or aching pain that cannot be controlled by narcotics is the major feature. In the dystrophic stage, beginning approximately 3 months after injury, bone scans often demonstrate altered up-
take of the imaging agent in the affected limb. In the atrophic stage, 6 months to 1 year after injury, fixed contractures, significant muscle wasting, and profound bone demineralization are common.

The greatest clinical experience in managing CRPS is with intravenous regional administration of sympatholytic agents, NSAIDs, and corticosteroids. Numerous systemic medications have been studied for relief of pain in CRPS, but no clear evidence advocates the use of any of these drug therapies for lower extremity CRPS. Published evidence seems best to support the use of the anticonvulsant gabapentin, the α-adrenergic blocking agent prazosin, the oral beta blocker propranolol, the calcium channel blocker nifedipine, and the antiarrhythmic mexiletine. Results with bisphosphonates have been encouraging.

Gentle physical therapy is beneficial in all stages of CRPS both to control edema and prevent joint contracture, but it has little proven effect on pain. Overly aggressive therapy can increase pain. A multidisciplinary team approach to management is recommended, including chronic pain management specialists and physical therapists as well as orthopaedic surgeons.

References

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