The Role of Radiofrequency in the Management of Complex Regional Pain Syndrome

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Current Science Inc. ISSN 1069–5850
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The nomenclature, pathophysiology, and treatment modalities of complex regional pain syndrome (CRPS) are controversial. Thus far there are no specific, scientifically valid treatments for the management of CRPS. The numerous modalities of treatment range from sympathetic ganglion blocks, intravenous regional blocks, administration of a multitude of pharmacologic agents and behavioral interventions, to surgical sympathectomy. Minimally invasive radiofrequency lesioning for managing CRPS is a modality in its developmental stages. This article describes radiofrequency lesioning techniques in managing CRPS.

The evolution of the nomenclature, conceptual understanding, and management of complex regional pain syndrome (CRPS), formerly known as reflex sympathetic dystrophy (RSD) and causalgia, has not only been dynamic but has also been controversial, leading to contentious arguments between the proponents and opponents of this condition [1•,2–5•,6•,7–14,15••,16–20]. Well-known for over 125 years in the history of medicine, the established nomenclature for pain emanating from or related to the sympathetic nervous system has evolved in many stages with a multitude of names. Causalgia (1872), RSD (1943), sympathetically maintained pain (1986), sympathetically independent pain (1997), and CRPS (1995) encompass some of the commonly used nomenclature [1•,7–9,20]. In addition, neuropathic pain also has been confused with the previously mentioned terminology that essentially represents heterogeneous conditions, which neither can be explained by one single etiology nor by a particular anatomic lesion [21].

Although RSD syndrome or CRPS and its existence is supported by many proponents, opponents claim that RSD is neither reflex, nor sympathetic, nor dystrophy. They claim that a diagnosis of RSD or CRPS type I is invoked when the patient’s symptoms or pain are associated with sensory, motor, or vasomotor dysfunction and the physician does not find an authentic testable explanation. Or they claim that it is a psychosomatic disease, or that RSD and sympathetically maintained pain are iatrogenically induced and encouraged by the very physicians that are mistakenly using this diagnosis, when in fact these patients are either harboring psychiatric disease, suffer from conversion/somatization disorder, Munchausen Syndrome, or malingering [17–19].

However, the literature has shown that patients suffering with RSD or CRPS are not different from patients suffering with other chronic pain problems psychologically [2,3,12–14,15••]. In addition, Hendler et al. [22], to whom a number of “suspected psychosomatic” cases have been referred, found an organic origin of pain in 98% of the cases. Hendler and Kolodny [23] also estimated that the incidence of psychogenic pain is only 1 in every 3000 patients.

Similar to the previously mentioned controversies, diagnosis and management are contentious and controversial [2,5•,10,11,17–19]. Although there are many proposed theories explaining both CRPS types I and II, the reliability and validity of diagnostic tests applied in the diagnosis of the syndrome has been questioned [17–19]. Similarly, there are no specific, scientifically valid treatments for CRPS, but nevertheless, there are numerous modalities of treatment. They range from sympathetic ganglion blocks, intravenous regional blocks, regional anesthesia and analgesia, physical therapy, administration of a host of pharmacologic agents and behavioral interventions, to surgical interventions, claiming success rates varying from 70% to 100% [10]. However, there are no prospective, well-controlled, long-term studies to document the natural (untreated) course of this painful condition or long-term treatment outcomes, indicating the difficulty of conducting such studies in pain management in general and the management of CRPS in particular. This review discusses briefly the salient aspects of CRPS, with a major focus on the role of radiofrequency lesioning of sympathetic ganglia in the alleviation of pain and disability secondary to CRPS.
Epidemiology
It is extremely difficult to assess the prevalence of CRPS in the general population or even to estimate the incidence or prevalence of these conditions following trauma or surgery. Carron and Welsh [24] reported that they documented 123 patients who met rigid criteria for RSD among 1156 pain clinic patients treated over a 22-month period, thus yielding a prevalence among pain patients of 10.7%. Pak et al. [25] reviewed the etiology of 140 patients with RSD and found posttraumatic and postoperative etiology to be predominant. Kozin et al. [26] reported on a series of patients with RSD in a rheumatology practice and also found traumatic etiology to be predominant. Kleinert et al. [27] also examined precipitating cause in 560 patients and concluded that posttraumatic and postoperative etiologies were predominant. Davis et al. [28] found that 68 of 546 rehabilitation patients with hemiplegia were diagnosed as having "shoulder-hand syndrome" with an incidence of 12.5% in this group of patients. Veldman et al. [29], in a large prospective study, examined the symptoms and signs of civilian patients referred to a tertiary surgery clinic in the Netherlands who were diagnosed with CRPS. This study showed a predominantly female to male ratio. Allen et al. [30••], in a retrospective chart review, described the demographics and health care utilization of CRPS in 134 patients referred to a tertiary pain clinic in the United States. However, they failed to provide any epidemiologic data. Hooshmand and Hashmi [2] reviewed the records of 824 patients with CRPS over a period of 5 years describing diagnosis and therapy; however, once again they were unable to provide any epidemiologic data.

Pathophysiology
In an attempt to define a taxonomy that more accurately describes conditions that fall under the umbrella term CRPS, the International Association for the Study of Pain Committee on taxonomy revised its previous descriptions of RSD and published those clinical features consistently found in these conditions. To satisfy a diagnosis of CRPS type I (RSD), the clinical findings include regional pain, sensory changes (eg. allodynia), abnormalities of temperature, abnormal sudomotor activity, edema, and an abnormal skin color that occurs after a noxious event. CRPS type II (causalgia) includes all the foregoing features in addition to peripheral nerve lesion [1••, 11].

However, the pathophysiology of these syndromes is poorly understood. The definitions of CRPS types I and II contain exclusion criteria that prevent the inclusion of patients with pain and clinical findings that are temporally proportionate anatomically and physiologically to an injury. Other conditions such as those that may constitute a myofascial pain syndrome are also excluded. Furthermore, a diagnosis of CRPS would be precluded by the existence of any known pathology that would otherwise account for symptoms and signs present in the distal parts of an extremity but outside the territory of an injured nerve.

The pathogenesis of type I CRPS has been the subject of much attention [31]. Many theories have been proposed to explain this disease, although none have proven conclusive. It is likely that the explanation may involve mechanisms from several of the current theories. Some studies provide convincing evidence that an initial noxious injury in the periphery results in a state of hyperexcitability in spinal cord neurons [32–34]. It is hypothesized that the fundamental abnormality in type I CRPS is an ongoing nociceptor input that maintains the central state of hyperexcitability for prolonged periods [35]. Thus, the principle difference between sympathetically maintained pain and sympathetically independent pain appears to be the mechanism of generation and maintenance of the nociceptor input to the spinal cord. In sympathetically maintained pain, the nociceptor input is maintained by an interaction between primary afferents and sympathetic efferents through an α-adrenergic mechanism [31]. However, in sympathetically independent pain, the maintaining nociceptor input arises from other causes, such as ongoing activity in a neuroma. Hence, both peripheral and central mechanisms play significant roles in the pathophysiology of the syndrome. In addition, an interaction between primary afferents and sympathetic efferents has been demonstrated in several animal models of nerve injury [36, 37].

Sympathetically maintained pain, by definition, is eliminated by an anesthetic blockade of the sympathetic efferents that serve the painful area [35–39]. It was noted that in sympathetically maintained pain, stimulation of the severed distal end of the sympathetic chain evoked pain in those diagnosed to have sympathetically maintained pain [40]. However, similar stimulation in other patients with sympathetically independent pain did not evoke pain. Hence, these observations suggest that efferent sympathetic fibers rather than afferent fibers account for sympathetically maintained pain. It is also postulated that sympathetically maintained pain results from exaggerated sympathetic efferent activity that causes an increased release of norepinephrine in the painful region [41, 42]. Thus, it is suggested that sympathetically maintained pain is a receptor disease that arises from activation of receptors sensitive to norepinephrine [43, 44].

Management Options
Stanton-Hicks et al. [11] described the guidelines for therapy of CRPS. These included pharmacologic management with nonsteroidal anti-inflammatory drugs, opioids, tricyclic and heterocyclic antidepressants, membrane stabilizers, corticosteroids, calcitonin bisphosphonates, capsaicin, and adrenergic drugs; physical therapy with reactivation, contrast baths, desensitization; increase of flexibility, edema control, peripheral electrical stimulation, isometric strengthening, gentle range-of-motion exercises, isotonic strengthening, aerobic conditioning, and ergonomics; regional anesthetic techniques to provide
sympatholysis, either by phenolamine infusion or regional anesthetic sympathetic blocks; neuromodulation with spinal cord stimulation or peripheral nerve stimulation; and psychiatric and psychological measures. Even though radiofrequency thermoneurolysis has not been described in this document, continuous regional anesthetic blocks have been described. Consipicuously, however, surgical sympathectomy has not been described.

Radiofrequency neurolysis is an extension of a continuous regional sympathetic block or a neurolytic block providing long-term relief with added safety. Percutaneous radiofrequency is a procedure for long-term interruption of the sympathetic chain, which involves less morbidity than open surgical techniques or chemical neurolysis and continuous sympathetic blocks or neural augmentation techniques with either spinal cord stimulation or peripheral nerve stimulation.

**Diagnosis**
Blockade of the sympathetic nervous system interrupts nociceptor visceral and somatic afferents and vasomotor, sudomotor, and viscero-motor fibers. Consideration of sympathetic blocks is to facilitate management of CRPS with analgesia commensurate with a program of functional restoration and sympatholysis to provide unequivocal evidence of sympathetically maintained pain [12,45]. In the absence of any clinical trial to demonstrate the relative efficacy of somatosensory conduction blocks with sympathetic blockade, there is a historical preference to use phenolamine infusion. Once it is established that sympatholysis is effective in relieving not only the burning dysesthesia but also allodynia or hyperalgesia, it is important to repeat the procedure to determine whether an increasing duration of effect can be expected in any particular patient. If this is the case, these individual blocks may be all that are necessary to enable a patient to regain function. When sympatholysis completely relieves the symptoms and facilitates exercise therapy but is limited to its duration of effect, it is appropriate to consider a prolonged block using radiofrequency neurolysis.

**Radiofrequency neurolysis**
The first use of radiofrequency for the treatment of chronic intractable pain started in the early 1930s, when Kirschner [46], using diathermy, produced high-current lesions of the gasserian ganglion for relief of trigeminal neuralgia. In the 1950s, the first commercially available radiofrequency generator was produced by Cosman and Aronoff [47]. Minimally invasive radiofrequency lesioning for pain of spinal origin was pioneered by Sluijter [47,48]. In the early 1980s, Sluijter developed refined techniques for cervical, thoracic, lumbar, and sacral spinal pain syndromes using percutaneous radiofrequency techniques. Kanta [49] pioneered the percutaneous radiofrequency lumbar sympathetic neurolysis. Subsequently, Wilkinson [50] developed an effective treatment for central hyperhidrosis using radiofrequency thoracic sympathectomy at the T2 and T3 levels, and at times the T4 levels. Sluijter [51] also developed the cervicothoracic radiofrequency sympatholysis.

Radiofrequency lesion generators produce high-frequency current that on application to neural tissue through a closed circuit produces neurolysis. Radiofrequency neurolysis depends on several variables, including tissue temperature, duration of lesion, and electrode size as the major determinants of lesion size. Throughout the years, it has been thought that radiofrequency works completely by blocking nociceptor stimuli that are conducted through a nerve with such a limited distribution area that there is no risk of deafferentation sequelae [52]. However, it has been shown that radiofrequency can also work by a selective action on myelinated nerve fibers in case the target is a larger nervous structure such as the dorsal root ganglion [52]. This is termed as pulsed radiofrequency, which is becoming much more popular and also may be used in neuropathic pain without adverse sequelae and safely in CRPS [53]. A well-controlled thermal lesion preferentially destroys unmyelinated C fibers and lightly myelinated A-δ fibers [54,55]. Theoretically, the greater the lesion size, the more profound the effects of the block. Theoretically, pain should return only when neural regeneration is complete or partially complete. At that time, it is not inappropriate to repeat the procedure. Thus, patients suffering with CRPS may benefit from radiofrequency neurolysis. Radiofrequency neurolysis may be performed at any level if a patient is suffering with CRPS.

**Anatomic considerations of the sympathetic nervous system**
The sympathetic nervous system consists of central and peripheral components, the former including the hypothalamus, midbrain, pons, medulla, and lateral columns of the spinal cord extending from T1 to L2. Peripherally, the sympathetic nervous system consists of preganglionic and postganglionic efferent fibers and afferent fibers that are responsible for visceral sensation and reflex responses. It is the peripheral portion of the sympathetic nervous system that is amenable to regional block techniques, and thus also to radiofrequency neurolytic techniques.

Structurally, the two paravertebral sympathetic chains or trunks are connected segmentally by preganglionic neurons whose cell bodies are situated in the lateral spinal cord from T1 to L2, with exceptions in C7, C8, and L3, L4. Each preganglionic fiber may synapse with as many as 32 postganglionic fibers whose cell bodies are either in the ganglia of the sympathetic chain or in the more distal prevertebral ganglia. These preganglionic fibers travel in the emerging spinal nerve roots before branching as small white rami communicans, with the small nerves connecting the sympathetic ganglia in the chain to each segmental spinal nerve. However, some of the postganglionic neurons such as gray rami communicans return to the segmental nerve, whereas others proceed to viscera in the head, chest,
and abdomen. The sympathetic trunks extend from the second cervical vertebra to the coccyx. In the cervical region, the trunks are lateral to the vertebral column such that their ganglia lie ventral to the transverse processes; in the thoracic region the ganglia lie in front of the heads or necks of the ribs; in the lumbar region the sympathetic chain has moved to an anterolateral position with the ganglia lying on the vertebral bodies; and in the sacral region, the ganglia lie medial to the anterior sacral foramina. Both sympathetic trunks merge together in front of the coccyx to form the ganglion impar. The segmental paravertebral ganglia are present in the thoracic, lumbar, and sacral regions with 10 to 12 in the thoracic region, four to five in the lumbar region, and five in the sacral region. In the cervical region they fuse to form three ganglia, which are termed as superior, middle, and inferior. Thus, radiofrequency may be performed similar to regional sympathetic blocks in the cervical, thoracic, lumbar, and sacral regions.

Cervical sympathetic radiofrequency
Radiofrequency of the stellate ganglion or cervical sympathetic ganglion is typically performed for sympathetically maintained pain of the upper extremity. The cervical chain has three ganglia: the superior, middle, and inferior. The inferior ganglion is usually fused with the first thoracic ganglion to form the stellate ganglion in approximately 80% of the population. The stellate ganglion lies anterior to the neck of the first rib, posterior to the carotid sheath, lateral to the midline structures in the neck and recurrent laryngeal nerve, and superior to the dome of the pleura. Postganglionic fibers leave the stellate and middle cervical ganglia to provide the sympathetic supply to the arm either via the brachial plexus directly or via the subclavian parietal sympathetic network that continues distally along the branches of the subclavian artery in the upper extremity. However, the postganglionic fibers to the head and neck arise from the upper part of the stellate ganglion, the middle and superior cervical ganglia, as a parietal network around the internal and external carotid arteries and their branches. The pupillodilator fibers and fibers to the nonstriated muscle in the upper eyelids are located in the upper two-thirds of the stellate ganglion.

Anatomic considerations for radiofrequency neurolysis include that the stellate ganglion is a spread out structure anterior to the base of the 6th and 7th transverse processes; thus, radiofrequency lesioning can only affect a partial interruption. However, the partial interruption has the obvious advantage that this procedure does not carry the risk of a long-lasting Horner’s syndrome such as other neurolytic procedures like a phenol injection do. At the same time, it is expected that the neurolytic effect seems to be great enough to relieve pain in a number of situations. In addition, it also has the advantage that it is a simple procedure with very little morbidity, so that it can be repeated from time to time if necessary.

Technical considerations of stellate ganglion neurolysis include that no stimulation response can be elicited in the stellate ganglion. The proper localization of the electrode is confirmed by the injection of contrast. However, motor stimulation should be carried to ensure that the electrode is at a safe distance from the brachial plexus, without eliciting a response below. The procedure is performed in the supine position on the operating table with a straight anteroposterior projection. During the procedure, the large vessels are kept aside with two fingers of one hand while a radiofrequency cannula is inserted with the other hand to make contact with the base of the transverse process. The position of the radiofrequency cannula is confirmed by injection of less than 1 mL of contrast, which should show the typical cranial caudal spread. After the motor stimulation, 1 mL of 2% Xylocaine (Astra, USA, Westborough, MA) may be injected and the radiofrequency lesion made subsequently; however, to ensure the sympathectomy is in fact secondary to neurolysis one may proceed without injection of local anesthetic.

Sluijter [51] reported that the procedure was most helpful in a number of patients suffering with pain due to sympathetically involved pain. In a number of miscellaneous conditions such as posttraumatic dystrophy, intractable cardiac angina, and residual pain after a cordotomy for a Parinaud tumor and Raynaud’s phenomenon. No other studies are available describing the effectiveness or lack thereof of this technique.

Radiofrequency of the thoracic sympathetic chain
The upper thoracic sympathetic chain is an extension of the cervical sympathetic. The merging of the first thoracic ganglion with the inferior cervical ganglion forms a stellate ganglion as previously described. The thoracic sympathetic chain continues in a caudal fashion and lies along the periosteum of the vertebral body in a much more posterior position as compared with that of the lumbar sympathetic chain.

The technique of thoracic sympathectomy with a radiofrequency cannula usually involves ablation of the thoracic sympathetic chain at the T2 and T3 levels, and occasionally T4 has been described for the treatment of upper extremity sympathetically maintained pain. However, thus far there are no studies comparing techniques directed toward the stellate ganglion versus procedures directed toward the upper thoracic sympathetic chain. With a local anesthetic block the quality of relief should be differentiated between the cervical sympathetic block and the thoracic sympathetic block. If a patient achieves higher grade relief with the thoracic sympathetic chain blockade, then radiofrequency lesioning of these fibers is advocated.

Radiofrequency lesioning of the thoracic sympathetic chain is performed in a prone position by executing two lesions at each level with the active tip turned cephalad for the first lesion and caudal for the second. After appropriate positioning of the cannula in the correct position, this should be confirmed by injection of contrast, as well as by sensory electrostimulation, which typically produces the experience of a deep aching feeling in the chest and back of
the patient, whereas with motor stimulation intercostal fasciculations are noted, but for appropriate positioning they should be absent at 3 V. Local anesthetic may be injected at this level; however, neurolysis may be performed without the injection of local anesthetic to ensure effectiveness of radiofrequency sympathectomy.

In a retrospective study involving 145 cases treated with radiofrequency thoracic sympathetic lesioning for over 15 years, Wilkinson [56] described effective sympathetic interruption in 96% of the patients at 2 years. In this retrospective study, patients were suffering from sympathetically maintained pain, hyperhidrosis, vasculopathy, and Raynaud's disease. Three lesions were performed at each level in question, using an 18-g cannula with a 10-mm active tip. Results showed a complication rate of 4% with pneumothorax, and successful sympathetic interruption in 96% of the patients at 2 years.

Lumbar sympathetic radiofrequency
Radiofrequency lesioning of the lumbar sympathetic chain is performed for a number of conditions benefiting from lower extremity sympathetic neurolysis. In the lumbar region the sympathetic chain and ganglion extends from the first to the fifth vertebra. These chains are continuous with the thoracic sympathetic chain from above and the pelvic sympathetic chain from below. In the lumbar region, the sympathetic chain and its ganglia lie on the anterior and lateral surface of the vertebral body. However, there is some variability and the chain can lie along the vertebral body as much as 5 mm anterior or 5 mm posterior to the most anterior border of the vertebral body itself. The chain lies close to the anterior lateral aspect of the vertebral body and anterior to the psoas sheath.

Radiofrequency denervation of a lumbar sympathetic chain has been shown to be most effective if it is performed at multiple levels. Lesioning of the sympathetic chain at a single level (L3) only produced a 25% lasting response [57]. However, when the procedure was performed at multiple levels it resulted in a 75% response at 8 weeks [57]. Clinical experience suggests that the lesions may be required at four levels or at least three levels for lasting relief, specifically foot pain requiring lesions at L2, L3, L4, and L5.

Technical considerations include an in-depth knowledge of the anatomy of the sympathetic chain and the surrounding structures. The aorta and inferior vena cava are located anterior to the body of the vertebra. The ureters and somatic nerves are also located in close proximity to the sympathetic chain. At the level of L1 and L2, the genitofemoral nerve lies in close proximity to the sympathetic chain. This procedure is performed in a prone position under fluoroscopy. The manner in which the contrast spreads is crucial to the identification of accurate needle placement in the facial compartment bounded by the anterolateral aspect of the vertebral body, the psoas sheath, and retroperitoneal fascia. The contrast will spread in a linear fashion beneath the facet joints in a cephalocaudal direction visualized on an anteroposterior view. In a lateral view it will spread in a linear fashion covering the anterior third of the vertebral body, again in the cephalocaudal direction. However, if the contrast is noted laterally in the anteroposterior view it indicates the placement of the needle tip in the psoas muscle. Thermoablation with the needle in this position could result in damage to the lumbar plexus nerves [49]. In addition, spread of contrast anterior to the anterior part of the vertebral body indicates an intraperitoneal needle/sip placement [49].

As one can expect, thermoablation with a needle in an intraperitoneal placement could cause damage to a viscus. Resistance to injection indicates that the needle tip is either in the psoas muscle, the periosteum of the vertebral body, the fibrous tunnel around somatic nerves around the body of the vertebra, intervertebral disc, the abdominal viscus, or the wall of the aorta [49]. Electrode stimulation performed to test for proximity to sensory and motor nerves is performed at 50 to 70 Hz and 2 to 5 Hz, respectively. Tingling in the anterior compartment of the thigh and knee is tested with a 70 Hz and 1.5 V stimulation. If no tingling occurs, motor nerve stimulation at 2 Hz and 3 V is carried out. Low-frequency stimulation should not cause muscle contraction of the anterior compartment of the thigh and knee area. Because the sympathetic nerves carry afferent pain fibers, patients might complain of tingling; however, this would not be in a dermatomal distribution, but in a quadrantal distribution [49].

Significant success with long-lasting relief has been reported anecdotally. However, there are no clinical studies evaluating either the quality or the duration of relief with radiofrequency neurolysis of the lumbar sympathetic chain, or for that matter any comparisons with neurolytic blocks or sympathetic blocks.

Radiofrequency for neuropathic pain
Neuropathic pain is usually considered as a contraindication to the use of radiofrequency nerve lesioning, except in the case of pulsed radiofrequency in which only short bursts of radiofrequency are applied to the nerve by the use of a modified radiofrequency machine. It was stated that there was no clinical evidence of neural damage and little postoperative soreness such as is experienced often after conventional high temperature radiofrequency lesioning [53]. Munglani [53] reported that four patients who suffered with predominantly neuropathic pain and responded poorly to prior treatment with local anesthetic and steroid placed epidurally or near the spinal root/dorsal root ganglion, did extremely well with thoracic spinal root pulsed radiofrequency and lumbosacral pulsed radiofrequency.
Complications of Radiofrequency

1. Potential complications with cervical sympathetic radiofrequency include damage to phrenic or recurrent laryngeal nerves, neuritis, and vertebral artery injury.

2. Complications related to thoracic sympathetic neurolysis include pneumothorax, which was reported in 4% of the patients [56] and postprocedure neuritis in 10% to 15% of the patients, which can last for approximately 3 to 6 weeks.

3. Genitofemoral neuralgia may occur secondary to inadvertent lesioning of the genitofemoral nerve following an inappropriately produced radiofrequency lumbar sympathetic lesion, either at L3 or L4. The genitofemoral nerve arises from the first and second segmental nerves and penetrates the psoas muscle at the L3 and L4 interspace. If proper technique is not used, inadvertent lesioning of the genitofemoral nerve may lead to permanent pain in the distribution of that nerve.

4. Following a radiofrequency sympathetic neurolysis in which intense waves of spasm were previously present, a reflex hyperemia may result in profound pain and swelling of the extremity. However, this is usually of short duration (1 to 2 weeks) and easily managed with pain medication.

5. Spinal cord infarction, Brown-Séquard syndrome, paraplegia, loss of motor functioning, loss of proprioception, urethral and anal sphincter control loss, and postcordotomy mirror image pain may develop secondary to inadvertent lesioning of the spinal cord or its constituents.

Advantages of Radiofrequency

1. Potential advantages compared with chemical sympathectomy include:
   - Radiofrequency neurolysis obviates the spread of neurolytic solution to the genitofemoral nerve with resultant complication of neuritis. Chemical neuritis causes severe pain and hyperesthesia in the groin region, in addition to numbness in the approximately 5% to 10% of the patients. This occurs much less frequently with radiofrequency lesioning.
   - The neurolytic solution can spread posteriorly between the slips of origin of the psoas major, not the long winding communicating ramus in a fibrous tunnel. The solution could then backtrack and cause neuritis of the third lumbar nerve producing pain, dysesthesia, and numbness around the knee area. Solution can also enter a dural cuff and cause paraplegia. Motor and sensory loss can occur when the L1, L2, L3, and L4 nerves are affected.
   - Intravascular absorption of phenol can cause tinnitus and blindness. Intravascular absorption of alcohol can cause thromboembolic phenomena. Both are avoided with radiofrequency thermo-neurolysis.
   - Urethral strictures may result when neurolytic agents are injected in the vicinity of the uterus. Hypotension can occur with chemical sympathectomy.

2. Advantages of radiofrequency lesioning compared with surgical sympathectomy include:
   - Amelioration of risk of surgery and anesthesia.
   - Radiofrequency lesioning is performed on an outpatient basis.
   - Avoidance of all the surgical complications, including thromboembolic phenomena.
   - Ability to perform this procedure bilaterally if necessary.
   - Ability to repeat the procedure with minimal mobility.

3. Other advantages of radiofrequency lesioning include:
   - There is an effective sympathetic blockade with a warm extremity but without swelling.
   - No urethral strictures occur following radiofrequency neurolysis.
   - Hypotension is less frequent.
   - Post-sympathectomy sympathalgia is virtually absent after radiofrequency neurolysis.
   - The patient will be able to ambulate early.
   - Impotence is rare and limited.
   - The lesion produced by the radiofrequency current is controllable and discreet.
   - The lesion does not spread across the psoas muscle, to the somatic nerves, or to the subarachnoid space.

Conclusions

Radiofrequency neurolysis is an effective modality of treatment in managing CRPS, specifically due to developments and advances of radiofrequency neurolysis using the pulsed mode. Radiofrequency neurolysis is a safe and effective technique with a minimal complication rate, (if performed appropriately) when specifically compared with neurolytic blocks or surgical sympathectomy.
References

And Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

• Of importance

• Of major importance


Assessed epidemiologic variables in 134 patients with CRPS evaluated at a tertiary chronic pain clinic in the United States. This study demonstrated that most patients are referred to a pain specialty clinic after several years of symptoms and many failed therapies.


