

Complex Regional Pain Syndrome: Understanding Reflex Sympathetic Dystrophy (RSD)

By Ted Priebe

The first clinical description of reflex sympathetic dystrophy occurred in 1863, when Dr. S.W. Mitchell described a burning pain called causalgia in *Gunshot Wounds and Other Injuries of Nerves*.¹ Reflex sympathetic dystrophy is now known as a neurogenic disease with a multisymptom medical condition that affects one or more extremities.

RSD was officially recognized and given an ICD-9 code (337.2) in 1993.⁸ It is described as an intense, severe burning pain, usually with swelling, color changes to the skin, and intense sensitivity to touch and temperature.^{2,3,4}

Generally, it is caused by a slight injury; repetitive motion injury; surgery; venipuncture; laceration; burns; degenerative joint disease; compression due to casting; infection; and myocardial infarction. Many patients may have accompanying neuromas; peripheral neuropathies; temporal mandibular joint pain; nerve entrapments such as carpal tunnel or thoracic outlet syndrome; and peripheral nerve compression. RSD will affect up to five percent of these patients.^{2,6,7}

RSD is an autonomic nervous system dysfunction that involves a sympathetic component in the early stages, which may frequently progress to include the somatic nervous system. Sensory input of the somatic system terminates in the post-central parietal sensory cortex of the brain and is perceived as a conscious, focalized, well-defined sensation felt in the nerve root distribution (i.e., radiculopathy). Very small sensory nerve fibers (C-fibers) transmit hyperpathic pain, while visceral and neuropathic pain are generated in the afferent portion of the sympathetic reflex arc. This nociceptive propagation, in contrast to the somatic pathway, generates a spreading and referred type pain by the sympathetic (A-delta) nerves, which follow the arteries and small arterial branches and result in a dermatomal distribution of the pain. Sympathetic input

terminates in the limbic part of the brain, giving rationale to symptoms of agitation, insomnia and depression. Vasoconstriction and pain are caused by stimulation of wide dynamic range neurons in the intermediolateral cell column of the spinal cord, increasing sympathetic activity to the periphery with the release of norepinephrine. This leads to increased release of substance P, prostaglandin and nociceptive activity.^{2,5,9,10}

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Stage one of RSD diagnosis usually consists of sympathetically maintained pain in one region, and includes sensory changes, allodynia, hyperpathia, edema, and sudomotor and vasomotor changes.

Stage two CRPS or causalgia increases the symptom profile and includes inflammatory changes in the skin, neurodermatitis, bruising, tremors, swelling of joints, insomnia and emotional disturbances. Stage three includes spontaneous severe skin ulcers, infection, atrophy of muscles, flexion deformities, osteoporosis and depression, with varying degrees of severity. Diagnostic nerve blocks in the early stages (one and two) are used to confirm RSD. Stages two and three usually progress to sympathetically independent pain.^{2,3,5,12,13,14}

Management

RSD is best treated in an interdisciplinary pain program in which the patient is evaluated and treated with appropriate medication and procedures such as nerve blocks, epidural injections, infusion pumps, acupuncture, cognitive psychological intervention, exercise programs adjusted and monitored to a patient's individual needs, and biofeedback. Inactivity, immobilization, ice, braces, crutches, wheelchairs, casts, surgery and sympathectomy accelerate disease progression.^{15,16,17}

Acupuncture

With RSD patients, acupuncture should be correlated with rehabilitative exercises on the same or alternating days. This treatment protocol blocks A-delta and C-fibers to decrease sympathetic efferent outflow and allow the patient to voluntarily control hypertonic muscles as a method of controlling pain and disability. Utilizing this protocol improves range of motion and flexibility while managing the barrier pain, allowing mobilization and strengthening to return the patient to preinjury status. Needling therapy works primarily because of its interaction with the spinal afferent processing system, which involves somatic nociceptive (pain), proprioceptive (muscle static load, length and position) and autonomic fibers, as well as other nerves of the body that provoke local, spinal and centrally mediated control. The principal effect of this descending

control is to restore autonomic balance and reduce pain.^{9,10}

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