

# Treatment of Reflex Sympathetic Dystrophy Using the Stress-Loading Program

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**R**eflex sympathetic dystrophy (RSD) is a complication that may occur following any injury. Symptoms are out of proportion to the injury and include pain, swelling, vasomotor and sudomotor disturbances, trophic changes, stiffness and fibrosis, and decreased function. The symptoms often progress beyond the original site of injury, with pain radiating proximally up the arm. It is important to separate the RSD process itself from the resultant fibrosis. RSD is a condition varying in its clinical presentation. It may affect the entire extremity or be isolated to a single digit. Lankford<sup>1,1a</sup> describes the natural progression of RSD from a hot, erythematous, swollen extremity (Stage 1), to a cold, cyanotic limb with progressive trophic changes (Stage 2), and finally to an extremity with fixed fibrotic changes and atrophy (Stage 3). The stress-loading program has been reported to be effective for treating RSD.<sup>2</sup> The purpose of this paper is to describe the stress-loading program and to propose a theoretical rationale in light of current theories of RSD and exercise physiology.

## REVIEW OF CURRENT TREATMENT TECHNIQUES

Treatment of RSD has traditionally been directed toward interruption of the abnormal sympathetic

**ABSTRACT:** Reflex sympathetic dystrophy (RSD) may occur following any trauma to the upper extremity. Symptoms include pain; swelling; vasomotor, sudomotor and trophic disturbances; stiffness; and decreased function. Stress-loading is presented as an effective treatment of RSD. The stress-loading program consists of active exercises that require stressful use of the upper extremity with minimal joint motion. If necessary, other modalities are used to treat the residual fibrosis that occurs secondary to RSD, only after the pain and swelling subside. A theoretical rationale for the use of stress loading in RSD is presented in light of current theories of RSD and exercise physiology. Exercise, if of sufficient intensity, duration and frequency, places a demand on tissues that causes adaptive changes. Active loading of the involved tissues in RSD provides input to the central nervous system, which is hypothesized to alter the abnormal sympathetic output and break up the pain cycle.

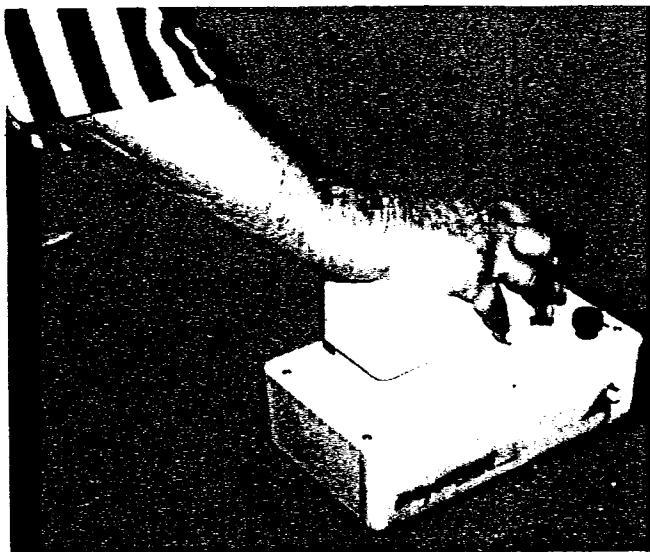
reflex<sup>1,1a,3-9</sup> through stellate ganglion blocks, sympathectomy, and, more recently, with regional intravenous blocks with guanethidine or reserpine. Treatment with corticosteroids has also been suggested.<sup>10-12</sup>

Techniques used in hand therapy include control of pain<sup>13-20</sup> with transcutaneous electrical nerve stimulation (TENS) or electroacupuncture. The concept of sensory overloading has also been advocated to decrease pain.<sup>1,1a,21</sup> Edema is reduced using elevation, intermittent compression, external pressure wraps, ice, retrograde massage and active exercise.<sup>22,23</sup> Motion and function are increased with active and passive range of motion, splinting, resistive exercise and functional activities.<sup>1,1a,23,24</sup> Temperature biofeedback has been used to help increase blood flow to the affected extremity.<sup>25,26</sup> Other reported modalities include connective tissue massage<sup>27</sup> and ultrasound.<sup>28</sup> Waylett emphasizes the importance of motivation and a structured, simplified approach in treatment of RSD patients.<sup>23</sup>

Results of treatment in the literature emphasizing invasive techniques are best documented, with failure to achieve permanent relief in approximately 25% of patients. There are few documented group studies of patients treated with noninvasive methods. Subbarao and Stillwell<sup>29</sup> reported on 77 patients treated with a variety of modalities, including massage, TENS, contrast baths and sympathetic blocks. Thirty-five percent were on the disabled list at follow-up. Johnson and Pannazzo<sup>21</sup> reported good results based on functional activity in 82% of patients treated with sensory overloading, including exercise and pro-

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**FIGURE 1.** The Dystrophile provides stress-loading to the affected extremity. The timer and light are activated when the preset load is reached.

longed heat or cold. Improvement with physical therapy alone occurred in 80% of patients reported by Pak<sup>30</sup> and 46% of Kleinert's sample.<sup>31</sup> In a pediatric population, Bernstein<sup>24</sup> reported improvement in 96% of patients treated with weight-bearing (if there was lower extremity involvement) and active use.

Long-term follow-up results have been reported by us on 41 RSD patients treated with the stress-loading program.<sup>2</sup> Decreased pain occurred in 88% of patients. Active motion improved in 95% of patients. Grip strength increased in all. Ninety percent of patients previously employed returned to work. Direct comparison of the results of this approach with other conservative treatment regimes is difficult due to differences in methodology and patient population in the few documented studies available.

## STRESS-LOADING

The stress-loading program consists of active exercises that require stressful use of the entire upper extremity with minimal joint motion. Force and duration are graded according to the patient's ability. The following exercises are used in accordance with these principles.

- 1. Scrub:** The patient is positioned on the floor in a quadruped position. Holding a coarse bristled scrub brush in the affected hand, the patient scrubs a plywood board using a back and forth motion, applying as much pressure as possible. If possible, the shoulder should be directly over the hand for maximum pressure. If unable to tolerate this position, scrubbing can be done standing at a table. The average home program begins with 3-minute sessions of steady scrubbing performed three times a day. The duration is increased in accordance with the patient's physical capacity, generally up to 7 to 10 minutes.

## Dystrophile Program

(PLEASE BRING THIS SHEET WITH YOU TO EACH THERAPY SESSION)

**DYSTROPHILE** - On hands and knees, with body weight over hand, push down on the handle until the light goes on. While keeping the light on, move the Dystrophile in a back and forth motion, as if scrubbing or sanding the floor.

**CARRY** - Grasp purse in hand with arm down; increase weight as you can. Carry wherever you go.

DATE	PROGRAM		ACTUAL SESSIONS			WEIGHT Pounds
	Number per day	Minutes per session	Mins	Mins	Mins	

**FIGURE 2.** A record sheet is given to the patient to increase the structure of the home program.

The Dystrophile (Fig. 1) has recently been developed to provide additional structure and improve compliance with the scrub program. A timer and light are activated when the patient reaches the preset load. Visual cues assist the maintenance of muscle tension.<sup>32</sup> Duration and resistance are increased as the patient's physical capacity improves. At least 5 to 7 minutes of steady use is required before the resistance is increased.

- 2. Carry:** the patient carries a weighted briefcase or bag in the affected hand. The weight, or resistance, is graded according to the patient's ability. The weight should be carried throughout the day whenever the patient is standing or walking.

The stress loading program is a highly structured exercise program. The patient is instructed by demonstration as well as written instructions. A record sheet is used as part of the home program (Fig. 2). The patient, not the therapist or physician, controls the RSD process. The therapist plays an important supportive role and must provide enthusiastic encouragement. This is especially important during the first few days of stress-loading, when pain and swelling may actually increase and the effectiveness of treatment may not yet be evident. The frequency of clinic visits depends on the patient's need for reinforcement, supervision and treatment modification.

Generally, no other treatment modalities are used initially. The stress-loading program is the only treatment modality used until the pain and swelling begin to subside. This helps to improve patient compliance and avoids possibly increasing symptoms from other forms of treatment. Passive motion may increase synovitis and cellular damage and aggravate the active RSD process. General use of the affected extremity, however, is strongly encouraged. Pain normally begins to decrease first in those areas of the extremity farthest from the initial inciting trauma. The reso-

lution of trophic changes such as shiny skin can often be clearly demarcated anatomically. As the pain and swelling subside, other modalities, if necessary, are incorporated into the therapy program. During this second phase of therapy, the treatment of residual stiffness due to fibrotic changes is emphasized.

Ideally, early treatment will prevent the development of contractures and fibrosis. If fibrotic changes have occurred, however, additional therapy is needed to correct joint, muscle and tendon restrictions. Treatment focuses on gradual correction through prolonged splinting in conjunction with active/resistive exercise. Treatment must be carefully monitored to avoid a recurrence of the RSD process.

The stress-loading program may also be used in conjunction with other modalities if used on a preventative basis. Stress-loading should be used with any patient who is clearly more painful or swollen than the average patient following injury or surgery.

## THEORETICAL CONSIDERATIONS

Many theories have been proposed to explain RSD, but the physiological mechanisms involved are still unknown. Abnormal sympathetic activity is believed to be involved in maintaining the painful reflex arc. As described by Lankford,<sup>1</sup> a normal vasoconstrictive reflex occurs after injury. If this reflex fails to shut down, increased vasoconstriction producing tissue ischemia will occur. This causes additional pain and afferent activity, which perpetuates the sympathetic reflex arc.

Livingston<sup>33</sup> described a vicious circle of reflexes. Injury results in a focal irritation causing painful afferent input to the spinal cord, which sets up an abnormal state of activity in the internuncial pool of the spinal cord. This, in turn, results in abnormal sympathetic and somatic efferent output causing vasomotor changes and muscle spasms. These peripheral abnormalities become additional sources of abnormal afferent activity and pain, thus completing the abnormal reflex circle. The sympathetic output is believed to maintain the disturbance of the peripheral tissues. A change in central processing is needed to correct the disturbance.

To summarize, according to current theories of RSD it is assumed that the sympathetic nervous system is involved in perpetuating the painful cycle of reflexes in RSD. This abnormal sympathetic activity is due to alterations in afferent input and activity within the central nervous system. The effects of this abnormal activity include disturbances in vasomotor<sup>34-42</sup> and sudomotor<sup>43</sup> activity. Abnormal sympathetic activity may produce an increase in pain due to direct stimulation of sensory receptors and/or secondary to vasomotor changes.<sup>44-46</sup> Effective treatment must, therefore, change the pattern of afferent input sufficiently to cause a change in the central integration and sympathetic efferent activity. Stress-loading exercise is proposed as a method of providing the required stimulus for change.

Exercise results in increased afferent input to the

sympathetic nervous system from ergoreceptors in the active muscle, which are small myelinated Group III and unmyelinated Group IV afferents.<sup>47-51</sup> These afferents are stimulated by mechanical and/or metabolic changes in the active muscle. Descending activity associated with the activation of motor units, the "central command," may also affect the output of the sympathetic nervous system.<sup>47-50</sup> In the active muscle, sympathetic activity interacts with local metabolic vasodilation. At increased work loads or exercise to fatigue, local vasodilatory mechanisms may inhibit vasoconstriction by the action of metabolites on sympathetic nerve endings.<sup>49,52</sup> The immediate neurovascular response to exercise may be increased in proportion to the active muscle mass,<sup>47,50</sup> absolute tension,<sup>47,50,52</sup> duration of exercise,<sup>52</sup> and the effort required.<sup>50</sup> Long-term local and systemic neurovascular adaptations have been demonstrated to occur following physical training.<sup>53-55</sup>

The physiological effects of exercise on patients with RSD have not been studied. There is no direct physiological evidence that the temporary neurovascular adjustments to exercise can permanently change the abnormal neurovascular activity in RSD. However, the principle of creating a therapeutic effect by changing the pattern of afferent stimuli through exercise is not new. Techniques used in proprioceptive neuromuscular facilitation, for example, utilize proprioceptive input to alter neuromuscular activity. The stress loading approach utilizes a similar principle but theoretically targets the abnormal sympathetic reflex activity by changing the afferent input.

The stress-loading program is theoretically based on the ability of the body's tissues to adapt in response to demand. In physical training an overload is needed to achieve a training effect.<sup>56</sup> Similarly, it is suggested that exercise must be of sufficient intensity, duration and frequency to provide adequate afferent input to change the abnormal central nervous system activity characteristic of RSD. It is proposed that stress-loading exercise increases the demand on the neurovascular system by requiring sustained muscle tension throughout the involved upper extremity to fatigue. The resulting increased afferent input from ergoreceptors in the contracting muscle theoretically results in a change in the abnormal neural activity in the spinal cord and possibly the higher centers. Descending activity associated with the "central command" may also play a role. The actual physiological mechanisms involved in the response to stress loading in RSD are still unknown.

## CASE REPORTS

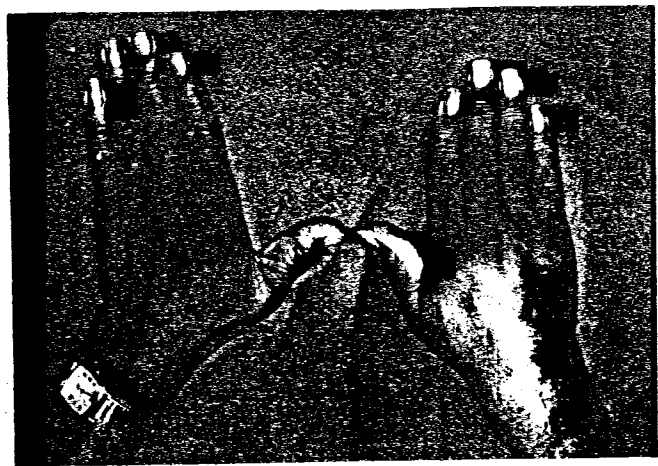
**Case 1.** A 52-year-old right-handed female machine operator was referred for therapy 3 weeks following a left index digital nerve laceration. Motion was avoided due to constant pain that she relieved by wrapping the finger. She also presented with localized swelling, cyanosis, decreased temperature, shiny skin and decreased pulp bulk. Flexion of the index finger lacked 7 cm to the distal palmar crease. Symptoms were resolved with 2 weeks of stress-load-



**FIGURE 3.** Prior to treatment, the right hand demonstrates severe edema, tenderness, erythema and decreased motion.

ing. The Dystrophile was initially set at 3 lb pressure. Even at this level, she could barely tolerate 2 minutes of scrubbing. Carrying was started at 2 lb. By the end of one week, tolerance had increased to 5 minutes on the Dystrophile and to 5 lb of carrying. Motion increased from a 7 cm lag to a 2.5 cm lag, with minimal pain complaints. The vasomotor and trophic picture was normal except for decreased pulp bulk. After 2 weeks on the Dystrophile, dynamic resistive exercises were added to the program. The stress-loading program was decreased to once a day for 7 minutes on the Dystrophile. At the end of a total of 3 weeks of treatment, she had no pain and full motion. She returned to work with no functional limitations.

**Case 2.** A 42-year-old right-handed male forklift operator was referred 6 weeks following a right wrist fracture. A 30% increase in swelling was present according to volumetric readings (Fig. 3). The clinical picture also included severe joint tenderness, erythema, increased temperature, shiny skin, flattened rugae pattern and increased nail curvature. Total active motion of the fingers was 31% of the unaffected side and the wrist was fixed at  $-32$  degrees extension. Grip was equal to 7 lb. The stress-loading program was started at 3 lb pressure for 3 minutes on the Dystrophile three times daily, and 8 lb of carrying; the program was increased gradually in resistance and duration on the Dystrophile to 12 lbs for 10 minutes and 30 lb of carrying. After one week of stress loading, swelling decreased to a 19% difference, appearance improved, and pain was minimal. The wrist was splinted at night in extension. Dynamic exercises were added after 2 weeks, as long as pain was not aggravated by joint motion. At the end of 1 month, finger motion increased to 76% of the unaffected side, and active wrist extension was present to 10 degrees. The patient was gradually weaned off the stress-loading program over the next month with no recurrence of the RSD. At 2 months, swelling was decreased to 5% and motion increased to 93% of the unaffected side. Grip increased to 50 lb and appearance was normal (Fig. 4). No functional limitations were noted in



**FIGURE 4.** Stress-loading produces a significant improvement in motion and appearance, with decreased tenderness and swelling.

normal daily activities. Work simulation was then emphasized, including lifting and driving, and the patient returned to work.

**Case 3.** A 41-year-old right-handed male teacher was seen 2½ months following multiple metacarpal fractures of the left hand. Previous therapy included whirlpool and range of motion. He also had a history of sympathetic hyperactivity, including migraines and fainting spells. He presented with periarticular swelling, severe pain, erythema, increased temperature, hyperhidrosis, and severe trophic changes, including thin shiny skin, increased nail curvature, flattening of the cuticle base, and flattening of the rugae pattern (Fig. 5). Thickened bands were present in the palm and diffuse osteoporosis was evident on x-ray. Active range of motion was 27% of the unaffected side and grip was equal to 10 lb. The stress-loading program was started with 5 minutes on the Dystrophile three times daily and 4 lb of carrying, and gradually increased in resistance and duration as tolerated, up to 10 minutes on the Dystrophile at 12 lb pressure and 25 lb of carrying. After 1 month, pain decreased to a mild level and erythema was limited to the proximal interphalangeal joints. Active motion increased to 44% of the unaffected side and grip increased to 26 lb. Splinting and dynamic exercise were added to treat the fibrotic changes secondary to the RSD. The patient continued on a home program of stress-loading and splinting with monthly checks for two more months, after which the vasomotor and trophic picture were normal, motion had increased to 68% of the unaffected side, and grip increased to 49 lb (Fig. 6). The patient later had surgery, which included intrinsic release and capsuloplasties of the proximal interphalangeal joints, with no recurrence of the RSD after surgery.

**Case 4.** A 48-year-old right-handed male laborer was referred over a year following wrist reconstruction. Previous therapy included TENS, ROM and resistive exercise. Pain throughout the extremity and trophic changes were the most significant clinical findings, including shiny skin, a flattened rugae pattern and increased nail curvature. Volume was de-



**FIGURE 5.** Prior to treatment, the left hand demonstrates severe stiffness and trophic changes as well as erythema, peri-articular swelling and pain.

creased due to generalized tissue atrophy despite periarticular swelling. Total active motion of the fingers was 84% of the unaffected side. The stress-loading program was started with the Dystrophile at 3 lb pressure for 3 minutes, three times daily and carrying at 7 pounds. After 1 week, pain in the shoulder decreased, and exercises were added to increase shoulder motion. The Dystrophile was increased to 7 minutes and the carrying to 12 lb. After 2 weeks, improvement was noted in the ring and little finger. By 3 weeks, pain was localized to the wrist and metacarpophalangeal joints. Intrinsic stretching exercises were added as well as splinting of the MP joints into extension to correct the fibrotic changes that had occurred secondary to the RSD. After 2 months, the patient's pain was localized to the radial side of the wrist due to an impingement syndrome. The patient later had further surgery to correct the wrist problem and was placed back on the stress loading program due to a temporary recurrence of symptoms.

## CONCLUSION

The actual mechanisms involved in the etiology and treatment of RSD are still unknown. Despite our poor understanding of the process, RSD can be successfully treated using the stress-loading program. Using this treatment approach, load, not motion, is required. Stress-loading can resolve RSD even in chronic cases; however, full function is also dependent on the extent of the residual fibrosis. This is a two-part problem. The fibrosis should be treated only after the RSD process is under control. Additional research is needed to increase our understanding of the physiological processes involved in RSD and the effects of therapeutic exercise.

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**FIGURE 6.** Stress-loading results in increased motion, improved appearance and decreased pain. Full motion did not occur due to the fibrotic changes occurring prior to stress-loading treatment, which are secondary to the RSD process.

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