

# **THE ASSOCIATION OF PERIPHERAL NERVE COMPRESSION AND REFLEX SYMPATHETIC DYSTROPHY**

**J. J. MONSIVAIS, J. BAKER and D. MONSIVAIS**

From the Hand and Microsurgery Center of El Paso, and the Orthopaedic and Hand Center, El Paso, Texas, USA

35 patients who presented with reflex sympathetic dystrophy (RSD) are reported. Peripheral nerve compression was present in 86% of the patients (30). 50% of the patients (15) had a single nerve compression, and 50% had multiple nerve compressions. The high incidence of these entrapments should alert the clinician to check for this treatable problem early in the course of RSD.

**Journal of Hand Surgery (British and European Volume. 1993)18 B: 337-338**

The aetiology of reflex sympathetic dystrophy (RSD) has puzzled clinicians and research workers since 1864, when Mitchell et al, during the American Civil War, first observed this disease in soldiers suffering from nerve wounds to the extremities (Mitchell et al, 1869 in Rutkow, 1989). The disease can occur following surgery, trauma, or even minor injuries. It is characterized by pain, swelling, discolouration and stiffness in an extremity (Lankford, 1988). The true incidence of the disease is unknown (Merritt, 1990). It is known, however, that the diagnosis is often missed in the early stages of the disease, and when left untreated the end results are incapacitating and often irreversible. This delay in diagnosis stems from the fact that the aetiology is unknown, and the presenting symptoms are varied, a treatable component of this condition may be the presence of peripheral nerve compression. Grundberg and Reagan (1991) studied a series of 22 patients with reflex sympathetic dystrophy who were not improved by standard treatment and found that they had carpal tunnel syndrome in the affected hand. Some patients had more than one associated compressive neuropathy. When the compression was released, there was an improvement in the patient's condition. Chodoroff and Ball (1985) described a patient who developed RSD in the left leg, and had an associated lumbar radiculopathy and compressive lesion of the posterior tibial nerve in the tarsal tunnel. Following surgical decompression, the pain improved.

This study reports 35 patients who presented with RSD. 30 patients or 86%, were found to have entrapment neuropathies of one or more nerves in the affected limb. Adding data which points to the idea that this treatable condition may commonly occur in conjunction with RSD.

## **MATERIALS AND METHODS**

This is a prospective evaluation of 35 patients seen over a 2-year-period. The patients presented with a painful syndrome which had incapacitated them for between 24 and 36 months. Of the 30 with entrapment neuropathies of the affected limb, 18 were female and 12 were male. 26 of the patients were right-handed, and four were left-handed. All cases were unilateral. The right hand was affected with RSD in 18 cases (60%), and the left hand in 12 cases (40%). 21 of the patients had jobs involving repetitive tasks, while nine were involved in non-repetitive work. The age range was between 20 and 70 years, with the majority in the 30 to 50 range. Five criteria were established for diagnosis (adapted from those proposed by Pak et al, 1970):

1. Pain out of proportion to the injury
2. Oedema
3. Prolonged disability
4. Trophic changes of the skin
5. Vasomotor disturbances.

Each patient included in the study met at least four of the five criteria. After the diagnosis of RSD was made, three-phase bone scans were carried out. Nerve compressions were then sought clinically by established methods:

1. Percussion test
2. Compression test
3. Tinel's sign
4. Phalen's test
5. Provocative testing.

Further diagnostic studies included sensory testing using the Semmes-Weinstein monofilament test, vibratory testing and moving two-point discrimination. Electromyography was done and nerve conduction velocities measured. The patients were then placed on a modified programme of progressive stress loading and carrying for pain control as described by Watson and Carlson (1987). If progressive neuropathy was documented, even if the RSD symptoms had improved. Surgical exploration was then carried out.

## **RESULTS**

30 out of 35 (or 86%) of the patients with RSD were found to have concomitant nerve compression in the affected limb. This results in a P value of <0.0001. Single nerve compression was found in 15 out of 30 patients (50%), and the other 15 had multiple nerve compressions. These multiple compressions are shown in Tables 1 and 2.

---

Table 1

---

No. of patients	No. of compressions
10	2
4	3
1	4

---

Table 2

---

Number of patients	Site of compression
21 (70%)	Median nerve at wrist
14 (47%)	Ulnar nerve at elbow
11 (37%)	Posterior interosseous nerve at elbow
2 (6%)	Ulnar nerve at wrist
1 (3%)	Superficial radial nerve at wrist

---

Three-phase bone scans showed increased uptake in 17 out of 25 patients (680/0). Eight scans were 'cold" 32%). in five patients, the three-phase bone scan was not performed. Electrical diagnosis was positive for nerve compression in 27 patients (90%), and negative in three (10%). 21 of the patients who had RSD and compression neuropathy had jobs involving repetitive tasks. This was found to be statistically significant, with a P value of 0.0286.

Ten patients showed resolution of symptoms with conservative management. 20 patients eventually under-went surgical decompression of the affected nerves. Of the 30 patients, 18 returned to work, seven were improved, and five were unchanged.

Among those who had surgery, 15 returned to work, three were improved, and two were unchanged.

## **DISCUSSION**

30 of the 35 (86%) patients with RSD in this study were shown to have one or more nerve entrapments in the affected limb. A single entrapment was as likely to occur in conjunction with RSD as multiple entrapments. While it is not known whether these entrapments are a cause or a symptom of the disease. The high incidence should alert clinicians to look for this treatable problem.

Our experience has been that when RSD continues for more than 6 months it becomes intractable. Even if the first episode resolves, the prognosis is often poor because recurrence is the rule. Since chronic nerve compression eventually causes permanent nerve damage, a link between this damage and the pathology of RSD is possible. Studies by Mackinnon and Dellon (1986) showed that the degree of nerve injury is related to both the amount of force and the length of time the force is applied. Early intervention to decompress the nerve is therefore indicated. Further research is needed to study how decompression affects the long-term course of RSD. and to find diagnostic methods which are useful before permanent changes occur. The three-phase bone scan has shown a trend for increased uptake when RSD Symptoms are most intense. This correlates with a study by Werner et al (1989) who found that the number of hot scans decreased as the duration of Symptoms increased. They suggested that the diagnosis of RSD remains largely a clinical one, and the three-phase bone scan must be interpreted with caution.

Additionally, the role of peripheral neurotransmitters in the inflammatory and pain response of RSD remains to be explored (Merritt, 1990). Because the early presentation of reflex sympathetic dystrophy is non-specific. Treatment is often delayed until permanent damage has occurred. Our study has demonstrated a high incidence of nerve compression in extremities affected with RSD. Increased awareness of this treatable problem may allow earlier intervention improving the potential for recovery.

## **Acknowledgments**

Maily Christensen for assistance with data collection.

## References

CHODOROFF. B. and BALL, RD. (1985). Lumbo-sacral radiculopathy. reflex sympathetic dystrophy and tarsal tunnel syndrome: Unusual presentation. Archives of Physical Medicine and Rehabilitation, 66. 185-187.

GRUNDBERG. A. B. and REAGAN, D. (1991). Compression syndromes In reflex sympathetic dystrophy. Journal of Hand Surgery, 16A: 4: 731-736.

LANKFORD, L. L. Reflex Sympathetic Dystrophy. In: Green. D. P, (Ed.): Operative Hand Surgery. 2nd Edn. New York. Churchill Livingstone. 1988: 633-663.

MACKINNON. S. H. and DELLON. A. L. (1986), Experimental study of chronic nerve compression: Clinical implications. Hand Clinics. 2: 4: 639-650.

MERRITT. W. H. Reflex Sympathetic Dystrophy. In: J.W. May and J.W. Littler. (Eds.): McCarthy's Plastic Surgery. The Hand. Philadelphia. W. B. Saunders. 1990: vol. 1: 4884-4921.

MITCHELL. SW. MOREHOUSE. G. R., and KEEN, W.W. Gunshot Wounds and Other Injuries of Nerves. In: Rutkow. I. M. Ed.): Civil War. Surgery Series. San Francisco. Norman Publishing. 1990: 95- 3,

PAK. T. I., MARTIN. G M., MACNESS. L., and KAVANAUGH. C. J. (1970). Reflex sympathetic dystrophy: Review of 140 cases. Minnesota Medicine, 53:507-512.

WATSON. H. K. and CARLSON. L. (1987). Treatment of reflex sympathetic dystrophy of the hand with an active "stress loading program". Journal of Hand Surgery. 12A: 5(1)779-785.

WERNER. R., DAVIDOFF. C., JACKSON, M. O., CREMER. S., VENTOCILLA. C., and WOLF. L. (1989). Factors affecting the sensitivity and specificity of the three-phase technetium bone scan in the diagnosis of reflex sympathetic dystrophy syndrome in the upper extremity. Journal of Hand Surgery, 14A: 3: 320-523.

Accepted: 3 September 1992

Jose J. Monsivais, MD. 716 Twin Hills. El Paso, Texas 79912. USA.