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**REFLEX SYMPATHETIC DYSTROPHY
CAUSED BY NERVE INJURY?
Electromyography in 75 patients**

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ABSTRACT

Reflex sympathetic dystrophy (RSD) has been considered as a pathological process developing after nerve trauma. 75 patients with RSD and neurologic signs and symptoms were investigated with EMG. In 40 patients, no disturbances were found. In 7 patients minor disturbances were present. In 28 patients, results indicated a compression neuropathy (11 x), a nerve injury the luxating event of RSD (8) or other coexistent pathology. For patients with disturbances found on EMG, the results of EMG could never offer an explanation for all neurologic signs and symptoms present. As both clinical signs and symptoms and EMG findings can not be attributed to nerve trauma, we do not support the concept of a - mechanical - nerve injury as the universal cause of RSD. A locally produced toxic factor which damages all structures seems more probable. In this latter concept, etiologic events, character and distribution of the signs and symptoms in RSD can all be explained. Decreased specialized sensory functions can be attributed to damage of receptors, increased pain sensation to sensitized nociceptors and effects of sympathetic blockade to α -adrenergic sensitization. Toxic oxygen radicals produced in an inflammatory (re) action to trauma or surgery are a possible cause.

INTRODUCTION

Many aspects of reflex sympathetic dystrophy (RSD) are still unknown. The syndrome is characterized by regional pain, edema, changes in skin color and skin temperature, limited active range of motion and increase of these signs and

symptoms after exercise. Many other signs and symptoms, especially neurologic disturbances, may be present. All structures can be affected and all functions lost (sensory, motor, sympathetic, hemodynamic) ³³.

Most physicians consider RSD as a result of nerve trauma ^{5 22}. In theories concerning pathophysiology of RSD, nerve trauma is the precipitating event leading to increased afference, crosstalk between nerves or sensitized wide dynamic range neurons and, as a consequence, increased efference or a vicious circle of ongoing activity. In that case one would expect alterations detected by EMG, showing impaired nerve conduction or signs of denervation or reinnervation. The purpose of this study is to report the results of EMG investigation performed in patients with RSD with neurologic complaints and to relate the findings to the pathogenesis of RSD.

PATIENTS AND METHODS

Since we instituted an outpatient clinic for RSD in november 1984, we have seen approximately 1500 patients - mostly referred from other departments or hospitals- with presumed or suspected diagnosis of RSD.

RSD has not been clearly defined in literature. The following criteria were used for diagnosis.

1. 4 or 5 of:
Unexplained diffuse pain
Difference in skin color relative to other limb
Diffuse edema
Difference in skin temperature relative to other limb
Limited active range of motion
2. Occurrence or increase of above signs and symptoms after use
3. Above signs and symptoms present in an area much larger than the area of primary injury or operation and including the area distal to the primary injury

These diagnostic criteria approximate those, utilized in other studies concerning RSD and are discussed in a previous report ³³.

Measuring nerve conduction and needle electromyographic examination was performed for various reasons but only when neurologic signs and symptoms were present.

RESULTS

75 patients fining into the criteria for RSD were investigated by EMG. In all patients, the classical signs and symptoms of RSD were accompanied by sensory

or motor disturbances. Included were 21 male and 54 female patients. The median age was 40 years (15-83 years). RSD was present since 5 days to 10 years (median 8 months).

EMG was performed for various reasons. Results were related to the indication for performing EMG (table 7.1).

Nerve injury

In 14 patients EMG was performed to diagnose or rule out a nerve injury as the luxating event of RSD. In 5 patients, results were normal. In 1 patient minor disturbances were present; an ulnar nerve injury was suspected in a 37 year old male with RSD after fracture of the radial head. EMG demonstrated only a slightly decreased conduction velocity (41 m/sec) of the ulnar nerve located at the elbow, with normal distal sensory and motor latencies.

In 8 patients, the suspected nerve injury could be objectivated by EMG. The cause of RSD was injury of the peroneal nerve in 2 patients, brachial plexus injury in 2 patients, median nerve injury in 3 patients and multiple nerve injuries in 1 patient.

In all but one patient the nerve injury could be related to the cause of RSD: direct trauma, insufficient positioning during surgery, inadequately applied plaster of Paris or misplaced injection. In one patient, results were not completely understood: a 52 year old female, known to suffer from hypereosinophilic syndrome, demonstrated signs of polyneuropathia and denervation potentials in the muscles innervated by the median nerve.

Entrapment

In 28 patients EMG was performed because of suspected entrapment of the median nerve (23x), ulnar nerve (3x) radial nerve (1x) or medial tarsal nerve (1 x). In 11 patients a carpal tunnel syndrome (CTS) (decreased conduction velocity located at the wrist with or without denervation and/or reinnervation) was found. In the other 17 patients, EMG demonstrated no disturbances.

In one of the patients with clinical and electromyographic evidence of a CTS, RSD had developed after a carpal tunnel release; renewed EMG showed improved - but still impaired - conduction velocity with signs of reinnervation (polyphasic potentials). Release of the median nerve was advised to all patients with evidence of a CTS. One patient refused surgery. Another, patient was seen only once on behalf of a second opinion. In 9 patients a carpal tunnel release was performed. This cured the CTS related symptoms in 6 patients and also RSD in 2 patients. In 3 patients complaints before and after surgery were the same.

Radiculopathy

A (pseudo)radiculopathy was suspected in 9 patients.

In 4 patients results of EMG were normal. In 2 patients, minor disturbances were found. In one of these two patients, who underwent laminectomy four times, H-reflexes were absent without signs of denervation or fasciculation. In the second patient, H-reflexes were normal but the distal peroneal superficial nerve showed slightly decreased compound motor action potential (CMAP) and a slightly decreased conduction velocity (44 m/sec; normal >45 m/sec).

In 3 patients evidence of radiculopathy was found, but again, clinical signs and symptoms could not explain the findings on EMG.

RSD

In 5 patients EMG was performed because RSD was diagnosed and the consulted physician thought this could be objectivated with EMG. In 2 patients we found disturbances.

Table 7.1 Indications and results of EMG

indication	n	results of EMG		
		normal disturbed	minor	disturbed
RSD	5	3		2
neurologic complaints not understood	13	9	4	
nerve injury	14	5	1	8
entrapment	28	17		11
radiculopathy	9	4	2	3
conversion	5	2		3
others	1			1

A 34 year old male with RSD of spontaneous origin in the left upper limb, had a slightly increased number of polyphasic potentials in both left and right deltoid muscles and in the left trapezoid muscle and a slightly decreased F-response over? the left ulnar nerve. Another patient, a 57 year old male, developed RSD in the left upper extremity after manipulation of the cervical spine, performed because of shoulder complaints. The EMG showed increased insertional activity in the biceps as well as the triceps muscle but not in the distal muscles of the extremity, and slightly impaired conduction of the median nerve at the level of the wrist on both sides, without signs of denervation. In both patients, the EMG disturbances could not be related to the loss of sensibility and the hyperpathia.

Neurologic signs and symptoms not understood

In 13 patients, EMG was performed because the neurologic complaints were not understood. The consulted physician could not relate the pain, sensory disturbances and/or paresis to a known disorder. No disturbances were found in 9 patients. In 4 patients, minor disturbances were found, which could not be related to the clinical symptoms. In patient 1, a minor conduction impairment of the ulnar nerve at the level of the elbow without denervation or reinnervation was found while clinically severe paresis of both flexor-and extensor-muscles in the forearm was present. In patient 2 who suffered from severe hypesthesia and hyperpathy, a few fibrillation potentials together with increased insertion was found. Patient 3, who complained of pain in the entire hand with hyperpathy, a minor increase in distal motor latency of the median nerve (4.7 msec) with normal conduction velocity at the level of the wrist (54 m/sec) was found. Increased insertion was present in the left and right anterior tibial muscles in patient 4 while RSD with severe sensory disturbances and paresis was present in only the left lower limb.

Conversion

In 5 patients, a conversion disorder was suspected and the consulted physician thought EMG was necessary to rule out other disorders. In a later stage we made the diagnosis RSD in all 5 patients. In 2 patients EMG examination was unrevealing, in 3 patients disturbances were found. In a 56 year old female suffering from RSD after a fracture of the fifth metatarsal bone, EMG showed polyphasic potentials in the muscles innervated by the peroneal nerve, as a sign of reinnervation. Probably an old nerve injury was present because of an inadequately applied plaster of Paris. Nevertheless, these results could not explain the severe pain, hypesthesia and severe paresis of extensor and flexor muscles. The second patient was a 40 year old female suffering from RSD in the left lower limb after an injection placed in the left gluteal area. EMG showed signs of polyneuropathy for which we found no cause. The third patient was a 31 year old female with a previous history of lumbar spondylodesis. At first, low back pain decreased but 1 year after surgery she developed RSD in the right lower limb. H-reflexes could not be elicited and signs of denervation were found in the muscles innervated by the 5th lumbar and first sacral nerve roots. The findings had improved in comparison with the EMG performed before surgery.

As a consequence, these disturbances could not be related to the newly developed signs and symptoms, such as pseudoparalysis of foot- and ankle muscles, severe pain and hypesthesia and hyperpathy in a stockinglike area (exceeding dermatomes L5 and S1).

Others

In one patient with RSD and known to suffer from amyotrophic lateral sclerosis we found a radiculopathy.

In 2 patients complaints increased after needle electrode investigation. One patient told us complaints temporarily decreased the days after EMG investigation.

DISCUSSION

Ever since the description of causalgia by Mitchell et al in 1864²⁵, many authors consider - sequelae of - nerve injury as the main pathogenetic factor of RSD. But, in many cases RSD results from minor injury in which nerve injury is not likely to occur and in 5 to 10% no precipitating event can be identified. Also the incidence of RSD after a proven nerve injury' and after a fracture of the distal radius without nerve lesions is both approximately 5%^{4 21 26}; one would expect a higher incidence of RSD after proven nerve injury.

Neurologic signs and symptoms in RSD

Sensory disturbances in RSD are frequent and consist of decreased sensation for touch (hypesthesia), temperature (hypothermesthesia) and position. In contrast, pain sensation is increased (hyperalgesy and hyperpathy). Also normally non-painful stimuli will be felt as pain (allodyny). In severe cases, anesthesia dolorosa (pain felt in an anesthetic area) may occur. Motor disturbances are also frequently found Paresis is invariably present, sometimes progressing until patients can not move actively at all (pseudoparalysis)¹⁴ Incoordinated voluntary movements or involuntary movements, such as increased physiological tremor⁶, dystonia^{2 20} and localized muscle spasms are found infrequently^{14 24 28} In most patients these signs and symptoms are not present at onset of RSD, but develop after a variable length of time.

The distribution pattern of motor and sensory disturbances in RSD is not limited to areas distributed by nerve roots or peripheral nerves. They are present in a glove- or stocking-like area. If nerve injury is present, the neurological disturbances are of course more prominent in the distribution area of that particular nerve, but they always exceed its borders. Thus the clinical symptoms fit best in a concept of a regional polyneuropathy.

Electromyography in RSD

Electromyographic findings in RSD have been reported in a few studies. Dumoulin reported increased distal latency with polyphasic potentials without signs of denervation and normal conduction velocity in some (number of patients not reported) of 22 patients⁹. Doury⁸ reported decreased conduction velocities of sensory and/or motor nerves in 9 of 17 patients with RSD. Uematsu found decreased conduction velocity and/or fibrillations in 5 of 32 patients with RSD³². Unfortunately these studies have poor diagnostic criteria for RSD and the EMG-findings are not related to the clinical signs and symptoms. Especially no remarks are made concerning the possibility of nerve injury or entrapment neuropathy. Hyman et al¹⁹ reported normal somatosensory evoked potentials after median nerve stimulation in 5 patients with cold RSD. Doury suggested that early electromyograms would show anomalies more frequently, but this was never proven⁸.

RSD as a complication of carpal tunnel release has been reported¹¹. Stein reported 6 patients with CTS and RSD after distal radius fractures or a stab wound surrounding the median nerve³⁰. Carpal tunnel release or adhaesiolysis relieved all complaints. Grundberg¹⁶ saw nerve compression syndromes in 22 patients with therapy resistant RSD; this was confirmed with EMG in most patients. Decompression improved the complaints in all patients. We found compression syndromes too, but only in a minority of patients. Decompression relieved the compression-related complaints in 6 of 9 patients and also cured RSD in 2 patients. Thus in 7 of 9 patients, decompression of the median nerve could not cure the RSD, which suggests that the CTS was not the cause of RSD.

In all patients in this study, there was a discrepancy between the severe and often impressive neurologic complaints on one hand, and the absence of obvious disturbances on EMG on the other hand. EMG could never adequately explain the motor and sensory disturbances found on physical examination. One would expect decreased conduction velocities or fibrillation potentials and positive waves, as signs of denervation, or polyphasic potentials as a sign of reinnervation. In our highly selected patients - only those with neurologic symptoms were investigated by EMG - EMG was of no value in explaining the neurologic signs and symptoms. As classical EMG is very sensitive in testing large diameter nerve fibers (motor and A α -sensibility) we conclude that these fibers are not disturbed in RSD. Unfortunately, conventional EMG can not detect disturbances of small sensory or autonomic fibers¹⁰. Autonomic function in the extremities can not be tested reliably at this moment²⁹. We do not know if and how these fibers are (dys) functioning. On the other hand, these fibers are not considered to conduct motor function and epicritic sensibility, which are both disturbed in RSD.

Pathogenesis of RSD

Many authors consider - partial - nerve injury and its sequelae as the cause of RSD. As stated above, analyzing the precipitating events does not support this

hypothesis. Also the character, distribution and development of clinical neurological signs and symptoms in RSD do not support the concept of a nerve injury, but fit in the concept of a unilateral regional polyneuropathy. If mechanical nerve injury would be the cause of RSD, large diameter fibers should be the first to dysfunction, and this can be objectivated with EMG. This study showed no or minor abnormalities in EMG recordings. So we do not consider - mechanical - nerve injury as the cause of RSD.

More probably, a more peripheral lesion - at cellular level - is responsible.

A locally produced toxic factor may be the cause of damage. Such a metabolic pathogenesis explains why neurological signs and symptoms are of a stocking- or glovelike distribution, as these factors are locally produced and therefore result in regional damage not related to a peripheral nerve or nerve segment. These factors can damage receptors and in this way explain how sensibility for touch, thermal stimuli and proprioception are decreased (chemodenervation). Damage to nociceptors can result in increased sensitivity¹⁷. Cline et al have proven that chronic hyperalgesia in RSD is caused by chronic sensitization of the C polymodal nociceptors³. Others reported α -adrenergic sensitivity^{17 23}, which explains spontaneous pain and increased pain sensation after mechanical- and thermal stimuli and after sympathetic arousal. These disturbances can not be objectivated by EMG.

This hypothesis also explains how sympathetic blocks can - temporarily - relieve pain, but are not permanently successful. As there is increased α -adrenergic sensitivity, circulating catecholamines and those which are locally produced by sympathetic efferents, can activate these damaged nociceptors and in this way aggravate pain. Blocking sympathetic stimuli prevents excess stimulation but can not take away the abnormal sensitivity.

In RSD all structures and functions are damaged. In a previous study³³ we regrouped signs and symptoms of RSD and found that in the early stage signs of inflammation are the most prominent. Toxic oxygen radicals, locally produced in an - exaggerated - inflammatory (re) action, can very well explain the signs and symptoms found in RSD. Other reports support this concept. For instance, RSD is characterized by increased permeability for macromolecules²⁷ and impaired oxygen metabolism^{12 18}. Also accumulation of lipofuscin - a product of lipid peroxidation by toxic oxygen radicals - can be found in longstanding RSD³¹ and scavengers of oxygen radicals are therapeutically successful^{13 15}

We do not know why RSD-patients react to trauma or surgery with an increased production of toxic oxygen radicals.

CONCLUSION

We conclude that EMG is not useful in establishing the diagnosis RSD, because the peripheral nerve functions seem to be intact in most patients. For RSD patients, EMG can be helpful when accompanying compression syndromes are present or nerve injury as a luxating event of RSD is suspected.

The results of this study are in accordance with our previous stated opinion that RSD has a metabolic pathogenesis. Damage to receptors by toxic oxygen radicals can explain both neurologic signs and symptoms and the absence of electromyographic disturbances.

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