Published in final edited form as:

J Neuropsychiatry Clin Neurosci. 2010 ; 22(3): 278–286. doi:10.1176/appi.neuropsych.22.3.278.

Neuropathic pain syndrome displayed by malingerers

José L. Ochoa, MD, PhD, DSc.¹ and Renato J. Verdugo, MD, MSc^{1,2}

¹ The Oregon Nerve Center at Legacy Health Systems and Departments of Neurology and Neurosurgery Oregon Health and Science University Portland, Oregon, USA

² Department of Neurology, Faculty of Medicine, Universidad de Chile, Santiago, Chile

Abstract

Among 237 patients communicating chronic pain, associated with sensory-motor and “autonomic” displays, qualifying taxonomically for Neuropathic Pain, there were 16 shown through surveillance to be malingerers. When analyzed through neurological methods, their profile was characteristically atypical. There were no objective equivalents of peripheral or central processes impairing nerve impulse transmission. In absence of medical explanation all 16 had been adjudicated, by default, the label Complex Regional Pain Syndrome (CRPS). We emphasize that CRPS patients may not only harbor unrecognized pathology (“lesion”) of the nervous system (CRPS II); or hypothetical central neuronal “dysfunction” (CRPS I); or conversion disorder; but may display a recognizable simulated illness without neuropsychiatric pathology.

INTRODUCTION

The task force on taxonomy of the International Association for the Study of Pain (Merskey and Bogduk, 1994) defined neuropathic pain as “pain initiated or caused by a primary *lesion* or *dysfunction* in the nervous system.” This definition has been rebutted explicitly (Max, 2002), and implicitly (Treede et al, 2008), and it has been shown that such pain patients dichotomize into two distinct categories by neurological evaluation: those with demonstrable and sufficient structural or physiological pathology versus those without it but with pseudoneurological presentation (Verdugo et al., 2004). Pseudoneurological signs (Shorter, 1995; Shaibani and Sabbagh, 1998; Krem, 2004) either reflect underlying unconscious conversion/somatization or fraudulent malingering (Ron, 2001), the clinical distinction between these categories being mandatory but difficult.

The American Psychiatric Association (DSM-IV-TR, 2000) defines malingering in the section “Additional conditions that may be a focus of clinical attention” as “the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs.” Halligan et al (2003) powerfully brought together medical, neuropsychological, legal and social perspectives on the subject. The suspicion of malingering naturally crosses the minds of clinicians facing patients with atypical complaints of various kinds, in absence of demonstrable medical bases. Prevalence of malingering for chronic pain was assessed as 31% in the robust study by Mittenberg et al (2002). Among 50 patients on compensation claims for musculo-skeletal pain treated in a Pain Clinic, Kay and Morris-Jones (1998) found 20% to be proven malingerers, as shown by covert video surveillance. However, malingering is seldom

reported formally in the literature on patients displaying chronic neuropathic pain, and there are no available statistics on malingered neuropathic pain.

Here we report 16 individuals communicating intractable chronic pain associated with psychophysical positive and negative motor and sensory displays, in absence of objective clinical or physiological equivalents of neuropathology, and proven to be malingerers by surveillance (Kurlan et al., 1997).

PATIENTS AND METHODS

This series emanated from a population of 237 patients referred to the Oregon Nerve Center between January 2000 and July 2007 for chronic, seemingly neuropathic pain. After informed consent, the subjects entered a standard clinical protocol described earlier (Verdugo and Ochoa, 1993; 1998), followed by nerve conduction measurements and electromyography for physiological evaluation of large caliber afferent and motor fibers; somatosensory evoked potentials (SSEPs) and transcranial magnetic motor stimulation for evaluation of central pathways; quantitative somatosensory thermotest (QST) to assess function of small caliber afferent pathways; infrared telethermography to evaluate function of sympathetic vasomotor activity and antidromic vasodilatation; and placebo-controlled (inert and active) somatic and sympathetic blocks.

Subjects entered this series if surveillance evidence of malingering became available to us. The video recordings were obtained in public places by professional agents appointed independently by the patient's third party agencies; we did not ourselves institute surveillance. Typically, surveillance had been implemented opportunistically in patients with medically unexplainable chronic symptoms and gross inconsistencies upon repeated medical evaluations. Traditional but non specific psychological tests intended to detect deception were not implemented (Faust, 1995). Two patients who underwent surveillance by naked eye were shown to function normally in public places on the day of a medical evaluation during which they displayed much illness behaviour. Criteria for diagnosis of malingering were a) misrepresentation of pain, hyperalgesia or sensory loss as reasons for functional disability and b) misrepresentation of weakness, paralysis or abnormal movements, all of which were assessed as medically incompatible with surveillance evidence of intact, able-bodied, sensory and motor performance. Only those patients displaying gross inconsistency between the volunteered complaints and the surveillance evidence were included.

RESULTS (TABLE)

Sixteen positive video recorded cases were analyzed. Fourteen indicated malingering and two records showed inconsistencies between the verbal and behavioural display in the clinic, versus the surveilled behaviors, but these were not regarded as gross enough. Therefore this series includes 14 video recorded subjects plus two monitored visually. The 16 malingerers constitute a small percentage (5.9%) of the overall population of "neuropathic pain" patients seen in our center. However, only a small proportion of the 237 patients were tested for malingering by initiative of the disability insurers. There were 8 women and 8 men, age 29 to 62 (average 38.3; TABLE). Four subjects complained of persistent pain and disability attributed to repetitive limb motion at work; 6 had suffered a minor work injury, and one a major injury; 4 followed a motor vehicle accident and one a trivial injury in a store. The time from injury to evaluation ranged 8 to 58 months (average 28.1 months).

Seven subjects complained of pain in the right and 4 in the left upper extremity; one in both uppers; one in the left sided extremities; one in the left lower extremity; one in the low back,

left shoulder and right ankle, and one communicated whole body pain after minor left ankle trauma. Subjective pain magnitude ranged 4–10 in a 10-point scale (average 7.3). Without exception every subject volunteered ongoing worsening of symptoms and displayed much pain behavior to physicians. Eleven were known to be on long-term narcotics by medical prescription. All were in litigation for alleged disability post injury.

On neurological examination non-anatomical and variable areas of cutaneous hypoesthesia to light touch, pinprick, cold and warm sensation, were found in odd combinations in 12 subjects; 11 displayed non-anatomical cutaneous mechanical hyperalgesia, of dynamic type in 10 and static type in 3 (Ochoa and Yarnitsky, 1993); 15 had give-way weakness and 8 displayed fixed dystonia or atypical abnormal movements. Tendon reflexes were regularly normal.

Telethermography showed a hypothermic extremity in four cases, as has been described after disuse (Butler, 2001). All had available sympathetic vasomotor reflex responses. Nerve conduction studies, electromyography, SSEPs and transcranial motor stimulation were always normal. QST disclosed subjective cold hyperalgesia in three, and associated heat hyperalgesia in one. One subject reported erratic thermal thresholds; four reported warm and cold hypoesthesia in the affected extremity. Four patients expressed subjective pain relief in response to inert placebo injection prior to diagnostic block, two of them featuring additional telltale recovery of deficits: weakness and sensory loss.

Several neurological findings signified pseudoneurological dysfunction without organic substrate, namely: a) give-way weakness of commanded maximal voluntary muscle contraction, due to interrupted willful drive; b) bizarre abnormal movements; c) non anatomical and fluctuating areas of cutaneous hypoesthesia or hyperalgesia/allodynia; and d) relief of hypoesthesia, muscle weakness or abnormal movements by inert or active placebo intervention, (Lambert, 1956; Walters, 1961; Fahn and Williams, 1988; Verdugo and Ochoa, 1993; Lang, 1995; Wilbourn, 1995; Verdugo and Ochoa, 1998; Trimble, 2000; Verdugo and Ochoa, 2000; Schrag et al., 2004a). However, these signs were not cardinal enough to discriminate “hysterical” conversion/somatization from malingering. This is where surveillance proves essential in differential diagnosis.

CASE REPORTS

Case 1

A 34-year old man injured his left arm at work. X-rays were normal. Thereafter, pain invaded the whole extremity, neck and ear. He described swelling, numbness, hand tremor, and weakness. He was labeled with RSD/CRPS I. He received 9 stellate sympathetic ganglion blocks, which transiently relieved the self-reported pain. He eventually sued for personal injury. We evaluated him 34 months after injury. He volunteered severe pain in the entire left upper extremity. There was normal muscle bulk, tone and reflexes, and a distractible, irregular tremor of the left hand. He displayed give-way weakness behavior of the upper extremity. There was psychophysical hypoesthesia in the entire left upper extremity and posterior shoulder. Nerve conductions and needle EMG were normal. Transcranial motor stimulation and SSEPs were normal. Quantitative sensory thermotest was erratic. Telethermography showed symmetrical thermal emission profile of both hands, with normal sympathetic vasoconstrictor reactivity. A diagnostic placebo-controlled lidocaine block induced cutaneous anesthesia in median territory while abolishing the pain complaint in the upper extremity and normalizing of the limb paresis. A placebo-controlled sympathetic block with intravenous phentolamine vs. saline vs. sympathomimetic phenylephrine again induced recovery of muscle strength (paradoxically following phenylephrine) but no pain relief. We diagnosed a pseudoneurological painful syndrome,

possibly associated with a conversion/somatization disorder. A psychiatrist raised the possibility of malingering. Secret video surveillance, recorded on the dates of evaluation, caught the subject in street and airport carrying a bag on either shoulder and using either upper extremity naturally for manipulation, without pain behavior, weakness or abnormal movements.

Case 2

A 30-year old woman working as CNA in a nursing home was attacked by a demented resident who twisted her right middle and ring fingers. She immediately reported pain and swelling of the entire hand and wrist. At the emergency room, x-rays were normal. In the ensuing weeks, she reported persisting pain and swelling, blue discoloration, excessive sweating and abnormal hair growth in the right hand. She consulted a pain clinic where RSD/CRPS I was offered as a diagnostic label. She underwent a stellate ganglion block that “leaked into the spinal cord and paralyzed everything from the neck down” for several hours. A series of seven uncontrolled stellate ganglion blocks gave partial subjective relief. A spinal cord stimulator was self-reported to improve her subjective spontaneous pain from 9/10 to 2/10. Four days after such procedure, her pain returned and she was started on narcotics. She was evaluated in our clinic 8 months after the incident, referred by the claims adjuster. She kept the right hand in a guarded position complaining of spontaneous burning pain ranked as 8/10. She reported stiffness of the right hand and forearm, associated with weakness and “twitching” of the right shoulder. She mentioned marked “sensitivity” to touch in the right upper extremity up to the shoulder and “numbness” of all digits. On examination, there was limited passive but full active range of slow motion of the wrist and fingers. There was overt give-way weakness of the entire right upper extremity, even for painless movements. There was profound cutaneous hypoesthesia for all modalities, in glove, below elbow level. Nevertheless, she volunteered severe dynamic and static mechanical hyperalgesia in the same region. Vibration sense was absent in the right third and fourth digits, but two-point discrimination threshold was normal. Reflexes were normal and symmetrical. Quantitative sensory thermotest showed warm hypoesthesia diffusely in the right hand. Nerve conduction studies and needle EMG were normal except for intermittent voluntary recruitment of motor units, at low firing frequency. Transcranial magnetic stimulation of motor neurons to weak upper limb muscles was normal. Placebo-controlled phentolamine block resulted in no improvement of pain, but there was improvement of both voluntary muscle weakness and dystonic posture of the right fourth and fifth fingers. Diagnosis was pseudoneuropathy with a strong psychogenic component, and psychiatric evaluation was recommended. Some weeks later, a video record from the claim adjuster showed the woman nonchalantly driving a car while talking on a cell phone, then holding a cup, and then smoking using her right hand without splint and without any evidence of motor limitation or discomfort. She opened and closed the car door and personally loaded her fuel tank using the right hand, without restriction.

Case 3

A 48-year old woman fell on a cement floor at work. She twisted the right ankle that reportedly became swollen and very painful. She also complained of pain in the left shoulder. At the emergency room, x-rays of the ankle were normal and she was put in a walking cast. In the ensuing three days, she reported increased pain in the left shoulder and restricted range of motion in spite of physical therapy. She underwent open surgery in the left shoulder, without cure. Pain in the ankle persisted and she reported “sensitivity” to touch in the foot. She underwent total surgical reconstruction of the right ankle. Thereafter she complained of numbness in the dorsum of the foot, but “sensitivity” to touch persisted. She also started complaining of low back pain. The diagnosis of RSD/CRPS I was entertained and she underwent two stellate sympathetic ganglion blocks without improvement of the

shoulder symptoms. Epidural steroid injections in the lumbar spine resulted in no improvement. She was evaluated in our clinic 34 months after the alleged injury. She volunteered severe limitation in active range of motion of the left arm. She also complained of tingling sensation in both hands. Pain in the right ankle was described as severe and constant, restricting her walking. She described intermittent painful spasms in the right toes that curled downward. She also complained of some loss of bowel and bladder control and profuse generalized sweating. On examination, she exhibited marked pain behavior including facial grimacing, gasping, withdrawal and guarding of the left arm. She also limped with a bizarre gait. When distracted, her motor performance improved dramatically. Give-way weakness, not due to pain, was present in the left hand. There was hypoesthesia in glove to pinprick in the left hand, and hypoesthesia to light touch, pinprick and warm in stocking distribution below the left ankle. Tendon reflexes were normal. Thermography was symmetrical in hands and feet. Comprehensive nerve conduction studies in upper and lower extremities and needle EMG were normal. Quantitative sensory thermotest recorded erratic warm hypoesthesia in the right foot. In the afternoon of the same day, personnel of our center observed her by chance in the street. She walked elegantly and without limping. She talked on a cellular phone held in her left hand without limitation of movements. No evidence of pain was observed. The diagnosis of malingering was made.

Case 4

A 29-year old woman complained of pain in both hands that she attributed to lifting and cutting stacks of cardboard at work. She stated that management pressured the employees to be more productive and she had to work very fast. After three weeks in this activity, she complained that the palms and fingers of both hands tingled and felt raw. She filed a claim for work-related injury. Non steroidal anti-inflammatories were prescribed. Complaints worsened to include "sensitivity" in the wrist. She complained of swelling, blotchy bluish discoloration, burning pain and weakness of both hands. Nerve conduction studies were normal. Thermograms were obtained, prompting the diagnosis of RSD/CRPS I. She underwent ten stellate ganglion blocks; five on each side with "dramatic relief" of all complaints for one month. Then she reportedly awoke with burning pain 10/10 in both hands, with weakness and muscle spasms of the hands. Further sympathetic blocks resulted in only partial relief. We evaluated her 14 months after the onset. She described total disability, even for writing, due to pain and allodynia of both hands. Muscle strength was difficult to evaluate because of pain. Atypical involuntary spasms and contortions of both hands were witnessed. There were inconsistent glove-like areas of profound hypoesthesia in both hands to cold, light touch and pinprick. A warm metal roll evoked severe hyperalgesia in the hands. She displayed severe dynamic mechanical hyperalgesia in both hands and static hyperalgesia in the wrists. Reflexes were normal. Thermography revealed hypothermia of the right hand with available sympathetic vasomotor reflex activity. Nerve conduction studies and SSEPs were normal. Transcranial magnetic stimulation of motor units was normal to both hands. Needle EMG showed reduced and interrupted firing of motor units on volition in muscles of both hands without evidence of denervation. A placebo-controlled somatic nerve block was followed by complete relief of the pain and hyperalgesia complaints, and total recovery of muscle weakness after saline injection. A placebo controlled (active and inert placebo) phentolamine block again led to complete relief of spontaneous pain and mechanical hyperalgesias after inert placebo. The diagnosis was chronic idiopathic pain syndrome and somatization/conversion disorder. A video surveillance record covering several days showed the subject carrying a backpack in her hands without limitation, driving a vehicle using both hands naturally, replacing a hood blown off by wind, and swimming without any evidence of pain, hyperalgesia or limitation of movements.

DISCUSSION

Hysterical conversion/somatization, malingering and surveillance

In Conversion Disorder, according to the American Psychiatric Association, the “somatic symptom represent[s] a symbolic resolution of an unconscious psychological conflict” (DSM IV-TR, 2000, page 494), obtaining or not a secondary gain through symptoms that “are not intentionally produced to obtain the benefits.” In conversion/somatization disorders pseudoneurologic symptoms are typically present (Shorter, 1995; DSM-IV-TR, 2000; Krem, 2004).

In contrast to hysterical conversion, malingering is conscious feigning of illness and the purpose is overt (Turner, 1999). It is a medical diagnosis but not a specific disease. Various clinical behaviours are particularly suspect of malingering (DSM IV-TR, 2000; Mendelson and Mendelson, 2004), but clinical psychological methods to detect deception are still not powerful enough (Bogduk, 2004). “It may be almost impossible for a court to conclude that the claimant is malingering without wholly convincing evidence, such as video observation of the claimant over a period of time” (Jones, 2003). Therefore surveillance is indispensable to assess whether an illness behaviour may be a lie (Kizorek, 1990; Kurlan et al., 1997; Kay and Morris-Jones, 1998) and constitutes a historical resource in the realm of chronic pain diagnosis (Jones and Llewellyn, 1917). Malingering is under-recognized because it requires opportunistic, covert, documentation of behaviours that refute veracity of the alleged symptoms (Rogers, 2008). Pain management physicians never recommend surveillance of pseudoneurological patients they label with CRPS I, and therefore only a minority of our CRPS I patients had been surveilled. When surveillance is incompatible with the reported or enacted disability, malingering is practically definite. False positives are inconceivable in non-malingers. Therefore the positive predictive power of surveillance must be very high (Hennekens and Buring, 1987) but the method is bound to substantial false negatives by chance, or because the subject may beware of surveillance.

Here we document that, among many other possible choices, malingerers may enact a fraudulent and fairly stereotyped clinical profile of unmeasurable pain associated with elements from the limited natural inventory for illness expression in a limb, namely displayed weakness, abnormal movements, sensory loss, allodynia, hyperalgesia, and changes in the physical appearance of the segment. We emphasize that, such clinical display amounts to subjective self-report, or is susceptible of wilful modulation or self infliction. In turn, the objective physical changes can result just from disuse rather than from autonomic neuropathology (Butler, 2001). This profile is often assessed concretely by the clinician as implying “neuropathic pain.”

Neuropathic pain and CRPS I: Taxonomy and hypothetical mechanisms

“Neuropathic pain” is a debated clinical attribution reserved by some authors to characterize patients with structural (“tissue”) neuropathology (Max, 2002), and broadened by others to encompass patients exhibiting atypical neurological dysfunction without demonstrable neuropathophysiology, but with assumed peripheral or central hyperexcitability (Jensen et al., 2002; Merskey, 2002). Whereas it is no surprise that such malleable profile may be simulated by malingerers, we remark that those who thus malingering invariably end labeled as CRPS I because they do not carry a neuropathological lesion to qualify for CRPS II and, we argue, because the descriptive category CRPS I flexibly accommodates atypical “neuropathic” clinical profiles without testable physical bases. Refreshingly, the new redefinition implicitly excludes CRPS I from “neuropathic pain” (Treede et al., 2008).

As a diagnostic concept, “CRPS I” is also debated. It is taken to reflect neurological dysfunction; its localization and pathology are “unknown,” and there is no diagnostic test.

The task force on taxonomy of the International Association for the Study of Pain chose the following diagnostic criteria for CRPS I “1. The presence of an initiating noxious event, or a cause for immobilization, 2. Continuing pain, allodynia, or hyperalgesia with which (sic) the pain is disproportionate to any inciting event, 3. Evidence at some time of edema, changes in skin blood flow, or abnormal sudomotor activity in the region of the pain.” Criterion 4, one that “must be satisfied” reads: “This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction” (Merskey and Bogduk, 1994). Thus, the label CRPS I is by default; a left over nickname that lacks evidential medical power. It is assumed to be a syndrome, or even a discrete disease, when in fact it is a listing of non-specific symptoms and signs awaiting differential diagnosis, which is traditionally bypassed (Ochoa and Verdugo, 2001; Ochoa, 2003; Loeser, 2005). Neurophysiological testing is dismissed as futile because it is predictably normal, even in presence of displayed weakness, paralysis or sensory loss. In absence of differential diagnostic workup for these patients, management is abortive and only iatrogenic harm can follow (Ochoa, 2001; Schrag et al., 2004b; AMA, 2008).

Hypothetical neuropathophysiological mechanisms are also controversial for CRPS I. For Max (2002), in CRPS I there is no neural *lesion* and the evidence of neurological *dysfunction* is precarious as it relies on self-reported allodynia, pain, and abnormal motion, unaccompanied by true clinical and physiological signs of functional impairment. Contrary-wise, Merskey (2002) argues that the clinical resemblance of CRPS I and CRPS II implies that both profiles are “neuropathic” and states that CRPS I reflects “a dysfunctional set of neurons.” In reality, the resemblance is shallow. CRPS I patients express atypical neurologically unexplainable symptoms (N.U.S.), and there is no measurable biological evidence of neuronal dysfunction (Verdugo et al., 2004). Humans and experimental animals with documented nerve pathology do not display these atypical clinical profiles (Ochoa, 2004). All there is for putative neural mechanisms behind CRPS I is an unverifiable claim of sensitization of second-order neurons by nociceptor barrage, assumed by uncritical extrapolation to explain perpetuation and expansion of pseudoneurological symptoms (Woolf and Mannion, 1999; Jensen et al., 2001; Jänig and Baron, 2003).

The sympathetically maintained pain hypothesis can be dismissed on the grounds of negative current scientific medical evidence (for review see Ochoa, 1999; Ochoa and Verdugo, 2001). In turn, the hypothesis that these patients harbor subclinical neuropathology of peripheral unmyelinated fibers is not supportable by reliable data. Indeed, evidence-based differential diagnosis was not implemented for the eight chronic pain patients labeled CRPS I (RSD) and studied histopathologically by van der Laan et al (1998). Several of them displayed atypical dystonia. The nerve and muscle samples were obtained from amputated legs with severe recurrent infections or chronic edema, and the authors concluded that there was “no consistent pathology in the peripheral nerves.” A reduced myelinated fiber density was partly attributed by the authors to endoneurial edema. The changes in unmyelinated fibers reported in four of the eight cases (median age: 40.5 years) are explainable by aging alone (Ochoa, 1970; Ochoa 1978). To their credit however, the Dutch authors correctly recognize that the concept of SMP may be rejected as a placebo artifact and that the pathogenesis of RSD is unclear. Today, German authors argue against this peripheral hypothesis (Jänig and Baron, 2006, and Letter to the Editor that followed; Ochoa, 2006).

More than a few neurologists have regarded the clinical profile that many denominate RSD/CRPS I as potentially brain/mind driven (Walters, 1961; Weintraub, 1988; Rodriguez-Moreno et al, 1990, Ochoa and Verdugo, 1995; Landau, 1996). Determination of psychogenic origin should not be based just upon absence of detectable physiological anomaly. It should be based upon demonstration of neurophysiological normality of self-reportedly disturbed, subjective sensory, or willfully drivable motor functions...but most

importantly upon explicit detection of reputable *pseudoneurological* phenomena that are assessed as *psychogenic* by the American Psychiatric Association (DSM-IV-TR, 2000 page 493) and by learned authors (Shorter, 1995; Shaibani and Sabbagh, 1998; Krem, 2004). In the case of many subjects labeled “CRPS I,” pseudoneurological phenomena include: a) fluctuating cutaneous hypoesthesia or hyperalgesia/allodynia, spreading or metastasizing beyond neural, radicular or segmental territories (Walters, 1961; Trimble, 2000; Verdugo et al., 2004); b) punctual verbal denial, while blindfolded, of every brief natural stimulus within a reportedly anesthetic area (Collie, 1913; Verdugo and Ochoa, 1998; Lanska, 2006); c) normal two point discrimination of stimuli applied to areas of reportedly profound hypoesthesia; d) hypoesthesia reversed by placebo (Verdugo and Ochoa, 1998); e) muscle weakness with interrupted voluntary upper motor neuron drive, even in absence of inhibiting pain (Lambert, 1956; Verdugo and Ochoa, 1993; Wilbourn, 1995); f) atypical abnormal movements or dystonia that are of sudden onset, distractible, entrainable, or relieved with placebo (Fahn and Williams, 1988; Lang, 1995; Verdugo and Ochoa, 2000; Schrag et al., 2004a) and g) recovery of paralysis with placebo (Verdugo and Ochoa, 2000).

The symptom *allodynia* deserves special analysis in this context. The mechanical hyperalgesia prevailing in the present cohort of subjects was of dynamic type (TABLE) and is known to be mediated by myelinated fibers (Ochoa and Yarnitsky, 1993), which places such abnormal sensation in the category of *allodynia*, a term coined to specify a pain induced by stimuli that do not normally elicit pain (Merskey and Bogduk, 1994). For Lindblom and Hansson (1991) allodynia mediated by tactile myelinated fibers reflected CNS dysfunction, specifically sensitization of second order neurons within the spinal cord, a hypothesis still dominant among pain researchers (Woolf and Mannion, 1999; Jensen et al., 2001; Jänig and Baron, 2003). It is often ignored that allodynia might also be a clinical manifestation of hysteria, as pointed out by Mitchell (1872, page 180), or may be malingering as described by Jones and Llewellyn (1917) and as clearly shown in the present series of patients. We agree with Max (2002) that, in and by itself, the symptom allodynia does not necessarily indicate structural lesion nor dysfunction of the nervous system.

CRPS I. Brain component

A minority of patients provisionally labeled with CRPS I are shown to harbor unrecognized neuropathy (Ochoa, 2002). Most “classic” CRPS I patients display what seems at first sight a neuropathologically-based illness, but the profile is characteristically atypical and pseudoneurological as there is neither demonstrable peripheral nor central pathway pathology (Lacerenza et al., 1996; Verdugo et al., 2004). This clinical profile reflects brain origin (Shorter, 1995; DSM IV-TR, 2000; Krem, 2004; Lanska, 2006). Changes in the brain are increasingly recognized in CRPS II and CRPS I patients (Jänig, 2001). Indeed, functional imaging of the brain in patients with post-traumatic neuropathic, or post-herpetic neuralgia, compared with normal controls, show decreased activity of the contralateral thalamus (Iadarola et al., 1995). These are likely plastic changes evoked by abnormal afferent activity from injured primary sensory neurons. Using functional MRI in patients labelled CRPS I, Apkarian et al. (2001) reported widespread prefrontal hyperactivity, increased anterior cingulate limbic activity and decreased activity of the contralateral thalamus. These changes might signal injury to primary sensory neurons in unrecognized neuropathy (thus, CRPS II). We believe that in CRPS I such changes are primary and unlikely to reflect amplification of the assumed to be nociceptor-induced “spinal cord changes that accompany chronic pain conditions” (Apkarian et al., 2001). Effective placebo removes both symptoms and functional MRI anomalies (Apkarian et al., 2001). In hysterical conversion with sensory loss, striatohalamocortical neuronal circuits with important limbic input, believed to modulate sensory decoding, are pathologically deactivated (Vuilleumier et al., 2001). Moreover, in hysteric patients communicating chronic pain and sensory loss, the

areas of abnormal brain activity are the same as those reported in CRPS I (Mailis-Gagnon et al., 2003) and signify neuronal activation of the emotional limbic brain. Abnormal brain imaging does not necessarily signify neuropathology (Grande et al., 2004).

CRPS I: Discrete neurological disease, hysteria or malingering?

All these malingerers fit IASP criteria for the label CRPS I (Merskey and Bogduk, 1994). It might be argued that by virtue of criterion #4, CRPS I is excluded because of the existence of a condition that accounts for the symptoms: Malingering. Semantics aside, the undeniable existence of malingerers displaying what their physicians assessed as CRPS I proves that, at least a subpopulation of CRPS I-labeled individuals are brain-driven cases of pseudoneurological illness behavior. Thus, it becomes mandatory to include both malingering and hysteria in the differential diagnosis of patients who fit the CRPS I menu because “the current IASP criteria for CRPS have inadequate specificity and are likely to lead to overdiagnosis” (Bruehl et al, 1999).

Shaw (1964) reckoned malingering to be the cause of RSD, although he failed to distinguish feigning from unconscious hysterical somatization. Conversely, rejecting the potential deliberate psychogenic nature of CRPS I/RSD is equally biased, and challenged by previous reports (Rodriguez-Moreno, 1990; Chevalier et al., 1996; Kurlan et al., 1997; Mendelson and Mendelson, 2004; Verdugo et al., 2004; Taskaynatan et al., 2005) and by the present series of malingerers. Recently Mailis et al. (2008) report patients displaying the non-specific profile of CRPS I, plus self-inflicted cutaneous stigmata, implying a gross personality disorder. Intriguingly, the authors admit the patient’s factitious nature while stressing “the legitimacy of CRPS as a diagnosis.” The possibility that all CRPS I patients are malingerers is untenable given that many conversion/somatization-based unintentional pseudoneurological patients are truthful in the expression of their display (Ron, 1994). On the other hand malingerers might choose to amplify symptoms of a neuropathologically-based condition legitimately causing spontaneous pain and associated sensory and motor manifestations, but in the present series all subjects displayed CRPS I with only neurologically unexplained symptoms (Ron, 1994; Schrag et al., 2004b). Besides, as per surveillance, we only included *pure malingerers* (Resnick, 1984) who feigned the entire symptom complex rather than just exaggerating *bona fide* symptoms (Main, 2003). Moreover, malingerers may amplify an unconscious, psychogenic, pseudoneuropathic painful syndrome since malingering is often concomitant with conversion/somatization (Merskey, 1995; Weintraub, 1995). This satisfies the concept that conversion/somatization involves self-deception while malingering is deception of others (Spence, 1999). Using functional MRI, Spence et al. (2001) observed that in normal people who intentionally deceive there is activation of the ventrolateral prefrontal cortex which for Spence et al. (2001) may be engaged in generating lies or withholding the truth. It is striking that cortical areas activated in conversion/somatization (Marshall et al., 1997) are similar to areas activated in conscious deception. Thus, coexistence of conversion/somatization and feigned symptoms is in keeping with neuronal data.

We conclude that “neuropathic pain patients” classified in the provisional CRPS I category, who in reality may harbor an undiagnosed nerve injury (CRPS II), or unconscious hysterical conversion, alternatively or concomitantly may very well be malingerers. Unconscious conversion/somatization is a legitimate health disorder with no room for pejoratives. However, mistaking pseudoneurological displays, of one kind or another, for neuropathologically-based disease, fosters iatrogenesis by commission and by omission (Kouyanou et al., 1998; Bell, 1989; Ochoa and Verdugo, 1995; Ochoa, 2001; Butler, 2005) and rewards those who feign. We have all missed more than one malingerer in our clinics (Shorter, 1995; Ford, 1995).

Acknowledgments

Supported by NIH grant R01 NS048932

References

- American Medical Association. Complex Regional Pain Syndrome Impairment. In: Rondinelli, R.; Genovese, E.; Katz, R.; Mayer, T.; Mueller, K.; Ranavaya, M.; Brigham, CR., editors. *Guides to the Evaluation of Permanent Impairment*. 2008. p. 450-451.
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4. Washington, DC: American Psychiatric Association; 2000. Text Revision, DSM-IV-TR
- Apkarian, AV.; Grachev, ID.; Krauss, BR.; Szevenyi, NM. Imaging brain pathophysiology of chronic CRPS pain. In: Harden, RN.; Baron, R.; Jänig, W., editors. *Complex regional pain syndrome*. Progress in pain research and management. Vol. 22. Seattle: IASP press; 2001. p. 209-25.
- Bell DS. "Repetition strain injury": an iatrogenic epidemic of simulated injury. *Med J Aust* 1989;151:280-4. [PubMed: 2671618]
- Bogduk N. Diagnostic blocks: a truth serum for malingering. *Clin J Pain* 2004;20:409-14. [PubMed: 15502684]
- Bruehl S, Harden RN, Galer BS, Saltz S, Betram M, Backonja M, et al. External validation of IASP diagnostic criteria for complex regional pain syndrome and proposed research diagnostic criteria. *International Association for the Study of Pain. Pain* 1999;81:147-54. [PubMed: 10353502]
- Butler SH. Primum non nocere-first do not harm. *Pain* 2005;116:175-176. [PubMed: 15982811]
- Butler, SH. Disuse and CRPS. In: Harden, RN.; Baron, R.; Jänig, W., editors. *Complex Regional Pain Syndrome*, Progress in Pain Research and Management. Vol. 22. IASP Press; Seattle: 2001.
- Chevalier X, Claudepierre P, Larget-Piet B, et al. Munchausen's syndrome simulating reflex sympathetic dystrophy. *J Rheumatol* 1996;23:1111-1112. [PubMed: 8782150]
- Collie, J. *Malingering and feigned sickness*. London: Edward Arnold; 1913.
- Fahn S, Williams DT. Psychogenic dystonia. *Adv Neurol* 1988;50:431-55. [PubMed: 3400501]
- Faust, D. The detection of deception. Malingering and conversion reactions. In: Weintraub, M., editor. *Neurol Clin*. Vol. 13. 1995. p. 255-266.
- Ford CV. Dimensions of somatization and hypochondriasis. *Neurol Clin* 1995;13:241-53. [PubMed: 7643823]
- Grande LA, Loeser JD, Ozuna J, Ashleigh A, Samii A. Complex regional pain syndrome as a stress response. *Pain* 2004;110:495-8. [PubMed: 15275803]
- Halligan, PW.; Bass, C.; Oakley, DA. Willful deception as illness behaviour. In: Halligan, PW.; Bass, C.; Oakley, DA., editors. *Malingering and illness deception*. Oxford Univ. Press; 2003. p. 3-28.
- Hennekens, CH.; Buring, JE. *Epidemiology in medicine*. Maynert, SL., editor. Boston: Little Brown and Co; 1987.
- Iadarola MJ, Max MB, Berman KF, Byas-Smith MG, Coghill RC, Gracely RH, et al. Unilateral decrease in thalamic activity observed with positron emission tomography in patients with chronic neuropathic pain. *Pain* 1995;63:55-64. [PubMed: 8577491]
- Janig W, Baron R. Is CRPS I a neuropathic pain syndrome? *Pain* 2006;120:227-229. [PubMed: 16426757]
- Jänig W, Baron R. Complex regional pain syndrome: mystery explained? *Lancet Neurol* 2003;2:687-97. [PubMed: 14572737]
- Jänig, W. CRPS-I and CRPS-II: a strategic view. In: Harden, RN.; Baron, R.; Jänig, W., editors. *Complex Regional Pain Syndrome*. Progress in Pain Research and Management. Vol. 22. Seattle: IASP press; 2001. p. 3-15.
- Jensen TS, Gottrup H, Sindrup SH, Bach FW. The clinical picture of neuropathic pain. *Eur J Pharmacol* 2001;429:1-11. [PubMed: 11698022]
- Jensen TS, Sindrup SH, Bach FW. Test the classification of pain: reply to Mitchell Max (letter). *Pain* 2002;96:407-8.

- Jones, AB.; Llewellyn, LJ. Malingering or the simulation of disease. Philadelphia: P. Blakiston's Son & Co; 1917.
- Jones, MA. Law, Lies and Videotape: Malingering as a legal phenomenon. In: Halligan, PW.; Bass, C.; Marshall, JC., editors. Malingering and Illness Deception. Oxford: Oxford University Press; 2003. p. 209-219.
- Kay NR, Morris-Jones H. Pain clinic management of medico-legal litigants. *Injury* 1998;29:305–8. [PubMed: 9743753]
- Kizorek B. Disability or deception: surveillance tapes helping doctors to decide. *J Disability* 1990;1:119–25.
- Kouyanou K, Pither CE, Rabe-Hesketh S, Wessely S. A comparative study of iatrogenesis, medication abuse, and psychiatric morbidity in chronic pain patients with and without medically explained symptoms. *Pain* 1998;76:417–26. [PubMed: 9718260]
- Krem MM. Motor conversion disorders reviewed from a neuropsychiatric perspective. *J Clin Psychiatry* 2004;65:783–90. [PubMed: 15291655]
- Kurlan R, Brin MF, Fahn S. Movement disorder in reflex sympathetic dystrophy: a case proven to be psychogenic by surveillance video monitoring. *Mov Disord* 1997;12:243–5. [PubMed: 9087986]
- Lacerenza M, Triplett B, Ochoa JL. Centralization in RSD/causalgia patients is not supported by clinical neurophysiological tests (abstract). *Neurology* 1996;46:A161.
- Lambert, EH. Mayo Clinic staff. Clinical examination in neurology. Philadelphia: WB Saunders; 1956. Electromyography and electrical stimulation of peripheral nerves and muscles.
- Landau WM. Reflex sympathetic dystrophy (letter). *Mayo Clin Proc* 1996;71:524–5. [PubMed: 8628039]
- Lang AR. Psychogenic dystonia: a review of 18 cases. *Can J Neurol Sci* 1995;22:136–43. [PubMed: 7627915]
- Lanska DJ. Functional weakness and sensory loss. *Seminars in Neurology* 2006;26:297–309. [PubMed: 16791776]
- Lindblom, U.; Hansson, P. Sensory dysfunction and pain after clinical nerve injury studied by means of graded mechanical and thermal stimulation. In: Besson, JM.; Guilbaud, G., editors. Lesions of primary afferent fibers as a tool for the study of clinical pain. Amsterdam: Elsevier; 1991. p. 1-18.
- Loeser, JD. Introduction. In: Wilson, PR.; Stanton-Hicks, M.; Harden, RN., editors. CRPS: Current Diagnosis and Therapy. Progress in Pain Research and Management. Vol. 32. Seattle: IASP Press; 2005. p. 3-7.
- Mailis-Gagnon A, Nicholson K, Blumberger D, Zurowski M. Characteristics and period prevalence of self-induced disorder in patients referred to a pain clinic with the diagnosis of complex regional pain syndrome. *Clin J Pain* 2008;24(2):176–185. [PubMed: 18209524]
- Mailis-Gagnon A, Giannoylis I, Downar J, Kwan CL, Mikulis DJ, Crawley AP, et al. Altered central somatosensory processing in chronic pain patients with “hysterical” anesthesia. *Neurology* 2003;60:1501–7. [PubMed: 12743239]
- Main, CJ. The nature of chronic pain: a clinical and legal change. In: Halligan, PW.; Bass, C.; Oakley, DA., editors. Malingering and illness deception. Oxford: Oxford University Press; 2003. p. 171-83.
- Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RSJ. The functional anatomy of a hysterical paralysis. *Cognition* 1997;64:B1–8. [PubMed: 9342933]
- Max MB. Clarifying the definition of neuropathic pain (letter). *Pain* 2002;96:406–7. [PubMed: 11973017]
- Mendelson G, Mendelson D. Malingering pain in the medicolegal context. *Clin J Pain* 2004;20:423–432. [PubMed: 15502686]
- Merskey H. Clarifying definition of neuropathic pain (letter). Reply to Max *Pain* 2002;96:408–9.
- Merskey, H. Understanding conversion and dissociation. 2. Glasgow, Bell & Bain Ltd; 1995. The analysis of hysteria.
- Merskey, H.; Bogduk, N. Task force on taxonomy, International Association for the Study of Pain. 2. Seattle: IASP press; 1994. Classification of chronic pain. Descriptions of chronic pain syndromes and definitions of pain terms.

- Mitchell, SW. American Academy of Neurology reprint series. New York: Dover; 1965. Injuries of nerves and their consequences (1872).
- Mittenberg W, Patton C, Vanyock EM, Condit DC. Base rates of malingering and symptom exaggeration. *J of Clinical and Experimental Neuropsychology* 2002;24:1094–1102.
- Ochoa JL. Pseudoneuropathy: Conversion versus malingering. *Painful Pain Patients. American Academy of Neurology Annual Course Syllabus* 2008:73–103.
- Ochoa J. Letter to the editor. In reply to: “Is CRPS I a neuropathic pain syndrome? *Pain* 2006;123:332–335. [PubMed: 16781072]
- Ochoa, JL. The irritable human nociceptor under microneurography: from skin to brain. In: Hallet, M.; Phillips, LH.; Schomer, DL.; Massey, JM., editors. *Advances in clinical neurophysiology*. Vol. 57. Amsterdam: Elsevier; 2004. p. 15-23.
- Ochoa JL. Quantifying sensation: “look back in allodynia. *Eur J Pain* 2003;7:369–74. [PubMed: 12821409]
- Ochoa, JL. Pathophysiology of Chronic “Neuropathic Pains. In: Burchiel, KJ., editor. *Surgical Management of Pain*. Thieme Medical and Scientific Publishers; New York: 2002. p. 25-41.
- Ochoa JL. Neuropathic pain and iatrogenesis. *Am Acad Neurol Continuum* 2001;7:91–104.
- Ochoa JL. Truths, errors, and lies around “reflex sympathetic dystrophy” and “complex regional pain syndrome”. *J Neurol* 1999;246(10):875–879. [PubMed: 10552232]
- Ochoa JL. Recognition of unmyelinated fiber disease: morphologic criteria. *Muscle & Nerve* 1978;1:375–387. [PubMed: 233736]
- Ochoa, JL. Ph.D. Thesis. University of London; 1970. The structure of developing and adult sural nerve in man and the changes which occur in some diseases. A light and electron microscopic study; p. 202
- Ochoa JL, Verdugo RJ. Mechanisms of neuropathic pain: nerve, brain, and psyche: perhaps the dorsal horn but not the sympathetic system. *Clin Auton Res* 2001;11:335–9. [PubMed: 11794712]
- Ochoa JL, Verdugo RJ. Reflex sympathetic dystrophy: a common clinical avenue for somatoform expression. *Neurol Clin* 1995;13:351–64. [PubMed: 7643830]
- Ochoa JL, Yarnitsky D. Mechanical hyperalgesias in neuropathic pain patients: dynamic and static subtypes. *Ann Neurol* 1993;33:465–472. [PubMed: 8388678]
- Resnick PJ. The detection of malingered mental illness. *Behav Sci Law* 1984;2:21–37.
- Rodriguez-Moreno J, Ruiz-Martin JM, Mateo-Soria L, Rozadilla A, Roig-Escofet D. Munchausen’s syndrome simulating reflex sympathetic dystrophy. *Ann Rheum Dis* 1990;49:1010–2. [PubMed: 2270960]
- Rogers, R. An introduction to response styles. In: Rogers, R., editor. *Clinical assessment of malingering and deception*. The Guilford Press; New York: 2008. p. 3-13.
- Ron MA. Explaining the unexplained: understanding hysteria. *Brain* 2001;124:1065–66. [PubMed: 11353722]
- Ron MA. Somatization in neurological practice. *Brain* 1994;57:1161–4.
- Schrag A, Trimble M, Quinn N, Bhatia K. The syndrome of fixed dystonia: an evaluation of 103 patients. *Brain* 2004a;127:2360–72. [PubMed: 15342362]
- Schrag A, Brown RJ, Trimble MR. Reliability of self-reported diagnoses in patients with neurologically unexplained symptoms. *J Neurol Neurosurg Psychiatry* 2004b;75:608–11. [PubMed: 15026507]
- Shaibani A, Sabbagh MN. Pseudoneurologic syndromes: recognition and diagnosis. *Am Fam Physician* 1998;57(10):2485–94. [PubMed: 9614416]
- Shaw RS. Pathologic malingering. The painful disabled extremity. *N Engl J Med* 1964;271:22–6. [PubMed: 14148225]
- Shorter E. The borderland between neurology and history: conversion reactions. *Neurol Clin* 1995;13:229–40. [PubMed: 7643822]
- Spence SA. Hysterical paralyses as disorders of action. *Cognitive Neuropsychiatry* 1999;4:203–26. [PubMed: 16571507]

- Spence SA, Farrow TF, Herford AE, Wilkinson ID, Zheng Y, Woodruff PW. Behavioural and functional anatomical correlates of deception in humans. *Neuroreport* 2001;12:2849–53. [PubMed: 11588589]
- Taskaynatan MA, Balaban B, Karlidere T, et al. Factitious disorders encountered in patients with the diagnosis of reflex sympathetic dystrophy. *Clin Rheumatol* 2005;24:521–526. [PubMed: 16010448]
- Treede R-D, Jensen TS, Campbell JN, Cruccu G, Dostrovsky JO, Griffin JW, Hansson P, Hughes R, Nurmikko T, Serra J. Neuropathic pain: redefinition and a grading system for clinical and research purposes. *Neurology* 2008;70:1630–1635. [PubMed: 18003941]
- Trimble, MR. Behavior and personality disturbances. In: Bradley, WG.; Daroff, RB.; Fenichel, GM.; Marsden, CD., editors. *Neurology in Clinical Practice*. 3. Newton, MA: Butterworth-Heinemann; 2000. p. 89-104.
- Turner M. Malingering, hysteria and the factitious disorders. *Cognitive Neuropsychiatry* 1999;4:193–201. [PubMed: 16571506]
- van der Laan L, ter Laak HJ, Gabreëls-Festen A, Gabreëls F, Goris RJA. Complex regional pain syndrome type I (RSD). *Neurology* 1998;51:20–25. [PubMed: 9674773]
- Verdugo RJ, Bell LA, Campero M, Salvat F, Triplett B, Sonnad J, Ochoa JL. Spectrum of cutaneous hyperalgesias/allodynia in neuropathic pain patients. *Acta Neurol Scand* 2004;110:368–76. [PubMed: 15527449]
- Verdugo RJ, Ochoa JL. Abnormal movements in complex regional pain syndrome: assessment of their nature. *Muscle Nerve* 2000;23:198–205. [PubMed: 10639611]
- Verdugo RJ, Ochoa JL. Reversal of hypoaesthesia by nerve block, or placebo: a psychologically mediated sign in chronic pseudoneuropathic pain patients. *J Neurol Neurosurg Psychiatry* 1998;65:196–203. [PubMed: 9703170]
- Verdugo RJ, Ochoa JL. Use and misuse of conventional electrodiagnosis, quantitative sensory testing, thermography, and nerve blocks in the evaluation of painful neuropathic syndromes. *Muscle Nerve* 1993;16:1056–62. [PubMed: 8413358]
- Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain* 2001;124:1077–90. [PubMed: 11353724]
- Walters A. Psychogenic regional pain alias hysterical pain. *Brain* 1961;84:1–18. [PubMed: 13782957]
- Weintraub MI. Chronic pain in litigation. What is the relationship? *Neurol Clin* 1995;13:341–9. [PubMed: 7643829]
- Weintraub MI. Regional pain is usually hysterical. *Arch Neurol* 1988;45:914–15. [PubMed: 3395266]
- Wilbourn AJ. The electrodiagnostic examination with hysteria-conversion reaction and malingering. *Neurol Clin* 1995;13:385–404. [PubMed: 7643832]
- Woolf CJ, Mannion RJ. Neuropathic pain: aetiology, symptoms, mechanisms, and management. *Lancet* 1999;353:1959–64. [PubMed: 10371588]

TABLE

Clinical characteristics of subjects.

Subject/Age/sex	Alleged cause	Location of pain	Illness duration	Spont. pain (magnitude)	Mechanical hyperalgesia/allodynia	Cutaneous hypoesthesia	Muscle weakness	Other
1/34/M	Fall at work	Left upper extremity	34 m	Burning (6)	No	Pinprick entire L upper extremity & shoulder	Give way weakness left lower ext.	Atypical tremor left hand
2/30/F	Motor injury at work	Right upper extremity	8 m	Burning & shooting (8)	Dynamic & static: right upper extremity	Light touch, vibration, pinprick, cold & warm: long glove left upper ext.	Give way weakness right upper ext.	Normal two point discrimination, right hand hypothermic
3/48/F	Fall at work	Low back, left shoulder, right ankle	34 m	Constant acute (10)	No	Pinprick left hand glove. Light touch, pinprick & warm right stocking	Give way weakness left hand	Painful spasms, variable bizarre limping.
4/29/F	Repetitive hand motion at work for 3 weeks	Both hands	14 m	Burning (4)	Dynamic & static: hands and wrists	Light touch, pinprick & cold in glove, both hands	Give way weakness both hands	Warm hyperalgesia, spasms left upper extremity, left hand hypothermic
5/36/F	Hit by falling object (stone)	Left upper extremity	42 m	Burning (9)	Dynamic: left upper extremity	Pinprick, left upper extremity glove	Give way weakness left upper ext.	
6/36/F	Use of hands at work	Right hand	24 m	Burning (6)	Static: right upper extremity	No	Give way weakness left thumb	Cold hyperalgesia left hand
7/46/M	Severe fall from a fishing ship	Right lower extremity	18 m	Burning (7)	No	Light touch, vibration, pinprick, cold & warm: long stocking	Give way weakness right lower extremity	Normal two point discrimination
8/45/F	Motor vehicle accident	Right upper extremity	23 m	Burning (7)	Dynamic: right upper extremity	Vibration, two point discr, cold in long right glove	Give way weakness right upper extremity	Spasms right upper extremity
9/62/M	Motor vehicle accident	Right hand	12 m	Burning (10)	Dynamic: right hand	Pinprick right upper ext. glove	Give way right dorsal interossei	Spasms right hand
10/49/M	Use of hands at work	Right upper extremity	47 m	Burning (6)	No	All modalities left upper ext.: glove	Give way right upper ext.	
11/42/F	Motor vehicle accident	L upper & lower extremities	34 m	Burning & lancinating (8)	Dynamic: left upper extremity	Light touch, pinprick left upper and lower extremities	Give way left upper and lower extremities.	
12/33/M	Minor work injury	Whole body	34 m	Burning (7)	Dynamic generalized	No	No	Bizarre pseudospastic gait
13/33/M	Minor work injury	Left hand	11 m	Burning (7)	Dynamic & static: right hand	No	Give way left hand	Dystonic posture left hand. Self inflicted edema.
14/29/F	Motor vehicle accident	Left lower extremity	46 m	Crushing (8)	Dynamic left lower extremity	Pinprick left lower extremity	Give way left lower extremity	

Subject/Age/sex	Alleged cause	Location of pain	Illness duration	Spont. pain (magnitude)	Mechanical hyperalgesia/allodynia	Cutaneous hypoaesthesia	Muscle weakness	Other
15/31/M	Use of hands at work	Right upper extremity	28 m	Constant acute (5)	Dynamic right upper extremity	Pinprick right upper extremity	Give way right hand	Spasms right upper extremity
16/30/M	Minor work injury	Right upper extremity	58 m	Burning (9)	No	Pinprick right upper extremity, neck and trunk	Give way right shoulder	

f = female, M = male, m = months.