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The Medico-legal Essentials of Complex Regional Pain Syndrome (CRPS)

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The litigation of a workers' compensation, personal injury, or claim arising in other legal/medical care/disability benefit systems which is based in whole or in part upon a diagnosis of CRPS is often difficult and confusing. This is mainly because the medical diagnosis and treatment of this subset of traumatic nerve injury disorders is in disarray, poorly defined, and variably treated. This article will simplify the clinical issues diagnostically, review the nomenclature and some of the rapidly evolving science, and, lastly, provide pointers pertinent to the different benefit/healthcare and disability benefit systems in which such claims arise.

The Confusing Nomenclature and Acronyms

Causalgia minor (in a peripheral nerve distribution) and Causalgia major (diffuse neuropathic pain) were early Civil War diagnoses. Reflex Sympathetic Dystrophy (RSD) was a term from the 1950s

IN THIS ISSUE..

Syndrome (CRPS) Type 2 (focal nerve) and Type 1 (diffuse neuropathic pain) was coined in the 1980s when the spectrum of signs and symptoms were examined. As a "syndrome" it is less specific and not a disease or specific disorder. Often, any neuropathic-like pain in any distribution can be labeled RSD or CRPS. Syndromal labels are, in fact, less distinct than diagnosing which nerves are injured and the severity, and delineating if the sympathetic system is involved. Complex Regional Pain Syndrome can be better understood used if users of the term are more specific, and include a classification of CRPS 1 or 2, with or without SMP (Sympathetic Maintained Pain)

Many of the CRPS “criteria" (e.g., AMA disability guidelines) focus on the dysautonomic features (sweating, temperature, color, edema, etc.) which may miss the fact that SMP is a spectrum of mild to severe involvement: so one can get a “CRPS” with SMP that is mild with or without autonomic changes. The important features are which nerves are injured and whether SMP present.

The Core Injury Phenomenology: The Traumatized Peripheral Nerve

Any part of the body, but particularly the limbs, can be traumatized (a blow, compression, burn, stretch etc.), and during the process nerves become injured, irritated, or damaged. Nerves coming from damaged boney or soft tissues will transmit pain (nociceptive pain) from the injury site to the central nervous system. If the nerves themselves, and the pain fibers within those nerves, are damaged, numbness and/or pain from the damaged nerves (neuropathic pain) can occur. The neuropathic pain will be in the peripheral neuroanatonic distribution of whatever nerve was damaged.

In order to diagnose and treat a traumatic nerve injury, the clinician must know and be able to examine the peripheral nerve system. For instance, a traumatic injury to the neck or anywhere along the upper limbs could cause neuropathic pain to the small finger of the hand. This neuropathic pain could be locally precipitated by damaging the ulnar nerve in the hand, wrist, or elbow; however, these same pain fibers coming from the hand go into the brachial plexus and eventually into the spinal cord by way of the C8 root. Injury anywhere along the route can cause neuropathic pain to the ulnar side of the hand. The clinician, to make a proper diagnosis and to assist in legal clarity, must be cognizant of the peripheral nerve anatomy and locate the site of injury. Sometimes the same nerve fibers can be injured in more than one site (e.g., ulnar nerve at the wrist and a stretch of the lower brachial plexus). If the nerve or nerves are not identified specifically, then medical treatment and

the nerve is injured. To do this one must understand that the nerves themselves have several functional types and sizes of nerve fibers. There are large caliber motor fibers with "insulation" myelin. If these are damaged one can see weakness and sometimes slowed electrical conduction (motor nerve conduction velocity/NCV) or change in the muscle the motor nerve goes to (electromyogram/EMG). There are also large myelinated A-Beta sensory fibers that transmit vibration and position (also can be seen with slowed sensory NCV). A nerve has to be damaged fairly significantly to injure these large motor and sensory fibers that are measured by standard EMG/NCV. Standard EMG/NVC does not measure damage to smaller pain fibers (A-delta and C-fibers). Standard EMG/NCV is often seen as “normal" or “negative” when there is actual painful nerve injuries. Complimentary nerve conduction studies of small myelinated A-delta pain fibers can be helpful.

The detailed clinical history and the clinical exam of the damaged nerves are still the gold standards. The clinician needs to test touch, pain with a pin, and cold in the damaged nerves. Damaged pain fibers can show specific "neuropathic pain" features. Non-painful pressure or light nonpainful touch, if experienced as painful, is "allodynia.” Increased pain to normally painful (but mild) stimuli (e.g., apin) is “hyperalgesia." Spontaneous pain generated and shooting up or down the nerve with no stimulus is “hyperpathia." Cold can also be experienced as painful "cold allodynia." History of these symptoms and examination of the findings in the distribution of the injured nerve is critical to diagnosis, treatment, and litigation of these cases.

Contribution of the Sympathetic Nervous System to Traumatic Peripheral Nerve

Injury Pain

The sympathetic nervous system, which runs from the brain to the spinal cord to the peripheral nerve in the injured limb, normally runs functions like microcirculation, sweating, etc. In cases when the peripheral nerve is traumatized and damaged, the sympathetic nerve gets abnormally misconnected to pain fibers, worsening the neuropathic pain and sometimes allowing it to spread beyond the injured nerve distribution. This is like "adding gas to a fire." When these sympathetic nerves misconnect and stimulate pain fibers (sympatho-afferent misconnection), the sympathetic system then "maintains" or “mediates” the neuropathic pain called "Sympathetic

Diagnosis: Keep it Simple. The first diagnostic step is to document the bony and soft tissue injuries. The second step is to examine and delineate which peripheral nerves are damaged and how severely. And lastly, one must determine if the sympathetic nervous system is maintaining any of the pain (SMP).

As noted, the history and detailed peripheral nerve exam delineate which nerves are injured and the severity. One must temporarily block the sympathetic nervous system to determine if SMP is present. As an example, an injury to the upper extremity can be tested for SMP by blocking sympathetic system at the stellate ganglion, a T2 vertebral level sympathetic ganglion block, or an IV infusion of a sympathetic blocker like phentolamine. If SMP is present, sympathetic blocks will lessen SMP and, with antineuropathic medicines, will help for the SIP nerve injury pain. If the managing clinician can accurately delineate which nerves are damaged, and how severely and test to see if the sympathetic system is contributing, diagnosis and treatment, and therefore litigation, can be much simpler.

The Evolving Science

It is becoming clear that traumatic, painful, peripheral nerve injuries can cause sensitization at the peripheral nerve trauma site, as well as sensitization and pain enhancement at the spinal cord and/or the brain. Central nervous system neuroplastic changes can occur all the way up to the cortex of the brain. Peripheral nerve injury pain can worsen and expand even with no new nerve damage. Evolution of models of peripheral and central nervous system neuroinflammatory and neuromodulatory processes are helping us understand the development of a more severe and spreading neuropathic process which started with a relatively small peripheral nerve or soft tissue injury. Therefore the treating clinician needs to understand not only which nerve is damaged and if SMP present, but also secondary central nervous system sensitization processes.

The Legal

CRPS claims are present in all Healthcare/Disability Income Benefit Systems. Regardless of the "Benefit System," legal representatives and staff must take an active role in a claim involving the diagnosis of CRPS. This condition is known to be difficult to treat successfully and known to result in considerable claim expense for both medical care and disability/lost time benefits. Moreover, depression is often

predictors of the condition."[1] (Emphasis supplied).

Those representing plaintiffs or claimants must be thoroughly familiar with the current and rapidly evolving science in order to assist their clients in obtaining treatment from physicians and allied health providers who have had considerable experience with CRPS. Typically, pain management specialists and neurologists are likely to be familiar with the current and emerging science. Even when the CRPS diagnosis is initially accepted, the cost of treatment, and the protracted nature of the condition, often leads to litigation. Expect an aggressive defense which will spare no expense, will go to great lengths to attempt to exclude the diagnosis via "examination" by medical practitioners, who may claim that the condition, while present, has attained "MMI" and no longer requires treatment. Expect the defense to resort to the use of surveillance, to "FCEs," and other tactics to attack credibility.

Claims involving the CRPS diagnosis have long been viewed suspiciously by many; including medical practitioners. That suspicion is fostered--and exploited—by some who offer their services to perform examinations and to testify as experts in legal proceedings in which the condition has been misdiagnosed or is not present, and in which there is a medical (or psychiatric) condition present which better explains reported (subjective) symptoms. Those opinions are not necessarily limited to MDs and may be offered by "experts" with no medical degree or training. Some "experts" have been known to suggest that the plaintiff/claimant reports are consistent with Munchausen's Syndrome by Proxy or are simply motivated by "secondary gain," etc.

The Claims in which CRPS and other forms of neuropathic pain are pursued and often litigated include but are certainly not limited to: ERISA, Tort, Social Security Disability Insurance (SSDIB), and Federal and State Workers' Compensation. The system itself often determines the course litigation (if any) will follow. Of the benefit systems studied, DOL, ALJ, and BRB decisions are easily the most helpful as those decisions develop the facts thoroughly, include both objective and subjective symptoms and clinical signs, discuss the basis for the diagnosis, and describe chronologically the course of medical treatment, enabling those unfamiliar with CRPS litigation to learn quickly the science and case development, preparation and presentation techniques, and the pitfalls to avoid.

One hundred-fourteen decisions were found when using the search term, "Complex Regional Pain Syndrome" on the website of

While defense of LHWA and DBA cases are often similar to those in state workers' compensation systems.[3] Conspicuously absent is the use of the AMA Guides—particularly Table 16-16 of the 5th Ed. —as diagnostic criteria. The LHWA and DBA cases also illustrate other characteristics of the CRPS diagnosis, e.g., that the condition can "spread" to other body parts and be a lifelong malady.[4]

Another characteristic of CRPS litigation is that case development expenses are likely to exceed those of other medical conditions[5]and, because of the often "subjective" complaints, speakers of English as a second language may find it more difficult to communicate their symptoms adequately through an interpreter.[6]

Decisions from State Workers' Compensation Administrative Law Judges and Appellate Courts regularly apply the AMA Guides, Table 16-16 of the 5th Ed. Of the Guides to the Evaluation of Permanent Impairment, for a purpose not intended by the AMA: diagnostic criteria. Ignoring the 2003 "Budapest" IASP diagnostic criteria, validated in 2010 by Harden, Bruehl, et al., "Validation of Proposed Diagnostic Criteria (the "Budapest Criteria") for Complex Regional Pain Syndrome.[7]

State Workers' Compensation Administrative Boards and agencies as well as Appellate Courts routinely use the AMA Guides 4th and particularly 5th Ed. to exclude the CRPS diagnosis resulting in denial of treatment – unless provided by some other medical benefit system[8]That the AMA Guides are being used tactically to exclude the condition is illustrated by *Chester Oak Fire Ins. Co. v. Swanigan**[9]*.

An encouraging departure from the harm occasioned by reliance upon Table 16-16 of the AMA Guides 5th Ed: *Brown v. W. T. Martin Plumbing & Heating, Inc.**[10]***

Resources and Recommendations for Further Reading:

- http://www.rsds.org/organization.html
- Chapter 25 Complex Regional Pain Syndrome, Harden and Bruehl, Bonica's Management of Pain, Scott M. Fishman, Jane C. Ballantyne and James P. Rathmell Lippincott Williams & Wilkins; Fourth edition;
- Meta-analysis of Imaging Techniques for the Diagnosis of Complex Regional Pain Syndrome Type I, Capello, et al, J Hand Surg 2012; 37A:288-296

- Treatment of complex regional pain syndrome in adults: A systematic review of randomized controlled trials published from June 2000 to February 2012, Cossins, et al., Eur J Pain 17 (2013) 158-173
- Complex regional pain syndrome in adults UK guidelines for diagnosis, referral and management in primary and secondary care, Royal College of Physicians, May 2012: http://www.rcplondon.ac.uk/sites/default/files/documents/complex-regional-pain-full-guideline.pdf
- Clinical Features and Pathophysiology of Complex Regional Pain Syndrome, The Lancet, Vol 10, July 2011
- The Diagnosis of CRPS: Are we there yet? Harden, Pain 153 (2012) 1142-1143
- Complex Regional Pain Syndrome: State-of-the-Art Update, Henson and Bruehl, Current Treatment Options in Cardiovascular Medicine (2010) 12:156-167

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[1] Jessica A. Lohnberg & Elizabeth M. Altmaier, *A Review of Psychosocial Factors in Complex Regional Pain Syndrome*, Source: 20 J. Clin. Psychol. Med. Settings 247 (2013).
[2] http://www.oalj.dol.gov/search/oalijsearch.aspx
[3] Martin, Jeff v. McGinnis, Inc., 1998LHC00950, (Mar 18, 1999), 121741 CADEC SD.
[4] Ellison, Debra v. Naval Military Perso, 2004LHC 01927 (Jul. 27, 2005) 181907 MODIS SD. [See, Spreading of Complex Regional Pain Syndrome: Not a Random Process, Rijn, et al., J. Neural Transm (2011) 118:1301-1309]
[5] . Knight, Casper A. v. Atlantic Marine, Inc., 2002LHC 000219 (Mar. 07, 2005) 145035 ATDEC SD;
[6] Gonzalez-Delgado, Isr v. Huntington Ingalls I, 2012LHC 01202 (May 08, 2014) 112342 CADEC SD.
[7] 150 Pain 268 (Aug. 2010).
[8] See, e.g., Bradley v. Ohio Dept. of Transportation, 2012 – Ohio – 451; 2012 Ohio App. LEXIS 384; Heinzman v. Cendant Corp., 129 Haw. 105; 294 p.3d 1091 (2013); Westmoreland Reg. Hosp. v. Workers' Compensation Appeal Bd., 29 A. 3d 120 (2011).
[9] 2012 Tex. App. LEXIS 3312.
[10] 2013 Vt. 38, 72 A. 3d 346 (2013).

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