Complex Regional Pain Syndrome: 
An Update Prepared at the Request of the American 
Academy of Orthopaedic Surgeons, 2014

The American Academy of Orthopaedic Surgeons has authorized Dr. Barth to share this chapter with you “for educational purposes only”.

This material cannot be distributed or used for any reason without Dr. Barth’s permission.
16th Annual AAOS Workers’ Compensation and Musculoskeletal Injuries: Improving Outcomes with Back-to-Work, Legal and Administrative Strategies

Volume 2 - Friday Breakout

November 7-9, 2014
Las Vegas, NV

J. Mark Melhorn, MD
Course Director
Ian Blair Fries, MD
Course Co-Director

Copyright © 2014 by the American Academy of Orthopaedic Surgeons™
All rights reserved. No part of the booklet may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise without the prior written permission of the publisher.
Chapter L
Complex Regional Pain Syndrome – What’s New

Robert J. Barth PhD

Preview: What you will find in this chapter...

I. Noteworthy recent (2013-2014) publications regarding complex regional pain syndrome

II. Modern diagnostic protocols

III. The 20th Anniversary of the death of reflex sympathetic dystrophy (the undead diagnosis)

IV. A fact-based definition of complex regional pain syndrome, created with a focus on the pervasiveness of legal claims in cases which involve this diagnosis

I. Noteworthy recent (2013-2014) publications regarding complex regional pain syndrome


In this general discussion of the scientifically established risk factors for chronic pain in the context of medical-legal claims, CRPS is specified as an example of chronic pain presentations for which scientific findings have indicated all of the following as risk factors:
Notes:
litigation/compensation, personality disorders, other forms of pre-existing psychopathology, and narcotic medications.


The abstract is provided below, with emphases added:

“This study examines validity findings in a particular behavioral pain disorder. We examined two types of validity scores in 73 participants with a primary diagnosis of the controversial Complex Regional Pain Syndrome Type I (CRPS-1). All participants were incentivized by a disability-seeking context. Failure rates on performance validity tests ranged from 23% (Test of Memory Malingering) to 50% (Reliable Digit Span). Positive findings on symptom validity tests (MMPI-2 or MMPI-2-RF) ranged from 15% to 50% of subsamples. At least 75% of the sample failed one performance validity indicator and over half showed at least one positive symptom validity score. This initial study suggests that CRPS-I could serve as a good patient model for studying the role of simulation in pain-related disability.”


- An editorial from the Secretary General of the European Wrist Arthroscopy Society, and an editor for the Journal of Hand Surgery (European volume), published in the Journal of Hand Surgery (European volume).

- Notable passages:
  - “So even at the risk of being wrong, and putting my reputation at stake, I repeat: (CRPS I) RSD does not exist.”
  - “In my view, accepting that this condition (CRPS type one) does not exist may benefit patients enormously. By the same token, a clear disservice would be made to the patient and the progress of science by doing otherwise. We need to be much more self-critical when assessing our bad results (not only those of others). It is our obligation as doctors to search for tangible cause for what went wrong, as opposed to laying the blame on a mysterious condition.”
Notes:

- "I must confess that I dream that we have tackled the condition named RSD/CRPS I etc. and that the mystery and bad doctoring that hide behind the acronyms are over."

- "Why is it bad to diagnosis CRPS I? It is both bad science and may also be harmful. I (simplistically) divide patients into two groups: those with a problem; and those who wish to have a problem, when they have none (in their hands). Both need a doctor but in different ways. The first one needs a doctor to make a diagnosis and offer treatment, whilst the others need the doctor to find a non-existing condition that justifies their complaints. Both sets of patients are hurt by a diagnosis of CRPS I: the first are prevented from receiving treatment for the cause of the problem as CRPS I has no treatable cause. The second group suffers from the nocebo effect: the opposite of a placebo. The nocebo gives/reinforces in a patient a nonexistent disease. We are all aware of malingeringers who feign a disease for secondary gain. This may be financial or psychological. These patients benefit immensely from a diagnosis, especially one as non-specific as RSD."

- "Be fully aware that there are malingeringers, and that conversion disorders may mimic CRPS I. Both need us to fuel their process. CRPS I is ideal for their goals and it is down to us to prevent this from progressing further."

- "This is not to say that I do not have patients with pain after I have operated on them. Rather I search diligently for a cause and usually find one so that in the last 15 years I have not referred a single patient to a pain doctor. I do see patients for second opinions in the miserable throes of a “mysterious painful process” under the care of a pain doctor, who have not had adequate investigation/treatment. I find I can nearly always make a diagnosis and successfully initiate treatment."

**NOTE:** this publication is consistent with a 2010 contribution to the Evidence-Based Medicine column in the American edition of the *Journal of Hand Surgery*:


- Notable passages:
  - (in cases which have been diagnosed as CRPS) “there is nearly always another diagnosis that might account for the degree of pain and dysfunction, particularly when mood disorders, somatoform disorders, and personality disorders are considered.”
“It has been consistently and reliably shown that pain intensity and disability correlate as much or more with psychological distress (e.g., depression or anxiety), psychopathology in general (e.g., personality disorders), and ineffective coping strategies (e.g., pain catastrophization) than with measures of pathophysiology or impairment. In addition, the influence of sociological factors such as secondary gain (the concrete benefits that are conferred upon individuals who become sick, including comfort, support, attention, and disability compensation) is familiar to hand surgeons and psychologists alike.”

“Cognitive behavioral therapies with a strong evidence base may be underused in part because they are stigmatized, and in part because labeling patients with biomedical diagnostic labels, such as CRPS, leads primarily to biomedical treatments.”

“Biomedical constructs such as CRPS are likely overdiagnosed and applied to patients with secondary gain, psychological distress, psychopathology, ineffective coping strategies, and heightening illness concern.”

“The biomedical approach to disproportionate pain and disability has resulted in subjective and unsubstantiated disease paradigms without any clear effective treatments. In contrast, there is strong empirical support for cognitive behavioral therapy in the treatment of somatoform disorders, ineffective coping strategies, and psychological distress. As long as an underlying pathophysiology remains speculative and the psychological aspects of illness remain stigmatized, it may be preferable to use nonspecific descriptive terms ("disproportionate pain and disability") rather than illness constructs such as CRPS that imply discrete pathophysiology and effective biomedical treatment.”

Note: The Del Piñal publication is consistent with the ODG and ACOEM Guidelines in calling for the avoidance of healthcare which involves a direct focus on pain in the absence of consideration of the psychological and social issues that are the scientifically established risk factors for such presentations (e.g., as Del Piñal says, “a pain doctor”).

I.D. Talmage JB, Melhorn JM, Ackerman WE, and Barth RJ. Musculoskeletal Disorders:
Note: The first edition of this book highlighted CRPS as an example of non-injury-related/non-work-related issues which have been inappropriately imposed upon the workers compensation system, and otherwise explained that there is no credible scientific basis for claiming work-relatedness or injury-relatedness for a CRPS diagnosis.

Notable passages of the new 2014 edition:

- “many of the signs of this illness (referring to issues that were mislabeled as “signs” in the Budapest protocol for CRPS) are not objective findings (not really signs). Anyone who wishes to pretend to have CRPS can simulate enough “signs” to meet point three in these criteria (referring to the portion of the Budapest criteria which are mislabeled as “signs”).”

- “In most clinical cases, and in most case series, a thorough differential diagnosis process, including consideration of the mental disorders that can create similar symptom complexes, has not been performed.”

- “An interesting and medically unexplainable concern is that occupational injury (Worker’s Compensation) involves a minority of the total number of injuries that occur, and yet in published case series CRPS seems to be concentrated in compensation settings.”

- “CRPS I associated with minimal or no injury is typically where causation disputes arise. These cases are not usually detailed separately in the case series that are published but are buried in the epidemiological statistics of CRPS in general and retrospective case series. CRPS cases are rare enough that no quality literature exists to provide relative risk or hazard ratio statistics. The events these patients allege as being associated with the condition do not explain the unknown pathophysiology of their illness and are not consistent with the known biology of humans healing after exposure(s). This coupled with the extremely common rate of minor trauma in the population at large, and the extremely low rate of these cases in the population, makes conclusions that a minor incident or exposure should be legally considered to be the cause of the syndrome scientifically illogical.”

- “The clustering of these cases in compensation settings is problematic for considering this a physical injury in disputed cases.”

This “comprehensive and critical review” incorporates information from 441 references. The abstract is quoted below, with emphases added:

“Complex regional pain syndrome (CRPS) is a term used to describe a variety of disorders characterized by spontaneous or stimulus-induced pain that is disproportional to the inciting event and accompanied by a myriad of autonomic and motor disturbances in highly variable combinations. There are no standards which can be applied to the diagnosis that would fulfill definitions of evidence-based medicine. Indeed, there are almost as many diagnostic criteria as there are names to this disorder. The umbrella term CRPS has been subdivided into type I and type II. CRPS I is intended to encompass reflex sympathetic dystrophy and similar disorders without a nerve injury; while CRPS II occurs after damage to a peripheral nerve. There are numerous etiological pathophysiological events that have been incriminated in development of CRPS, including inflammation, autoimmune responses, abnormal cytokine production, sympathetic-sensory disorders, altered blood flow and central cortical reorganization. However, the number of studies that have included appropriate controls and have sufficient numbers of patients to allow statistical analysis with appropriate power calculations is vanishingly small. This has led to over-diagnosis and often excessive pharmacotherapy and even unnecessary surgical interventions. In this review we provide a detailed critical overview of not only the history of CRPS, but also the epidemiology, the clinical features, the pathophysiological studies, the proposed criteria, the therapy and, in particular, an emphasis that future research should apply more rigorous standards to allow a better understanding of CRPS, i.e. what it is, if it is, and when it is.”

Additional notable passages:

- “For the time being, the choice of a specific set of diagnostic criteria is arbitrary”

- (page 17) “What is even more peculiar than the explicit exclusion of a diagnosis of CRPS by the existence of other conditions that would otherwise account for the degree of pain and dysfunction, is the invention of a CRPS-NOS (not otherwise specified) category by the Budapest consensus panel. This category is intended for patients who partially (partially is not ever defined!) fulfill the diagnostic criteria, but whose symptoms are “not better explained by any other condition.” What could better illustrate that CRPS is a default label rather than a
diagnosis. Accordingly, it has been shown that “CRPS I” can be mononeuropathy, nerve compression or entrapment; dysfunctional posture; somatoform disorder, self-inflicted or factitious disorder, or malingering. Of course, these are almost all part of the differential diagnosis, but obviously are not always recognized by many treating physicians. This clearly indicates that “CRPS” is simply a catch-all label and, therefore, should not be considered a valid diagnostic endpoint... Therefore, both CRPS I and II should be considered to indicate the urgent need for extensive exploration of the differential diagnosis.”

(NOTE: Thanks to AAOS faculty member Dr. Talmage for bringing this publication to my attention)

The following points from this publication are well-referenced in the original source material:

“Several new diagnostic criteria have been proposed, but they are not sufficiently objective or reliable. For example, criteria such as “continuing pain that is disproportionate to the inciting event,” allodynia (pain to light touch), and weakness or tremor are common and non-specific. Any patient with these characteristics could attract a diagnosis of CRPS when “disproportionate pain” would be a more appropriate description. This term (“disproportionate pain”) does not medicalise the symptoms with a “disease” label and has less potential for iatrogenic harm.”

“Not only is the diagnosis difficult to confirm but also it has been shown that brief immobilisation or prolonged casting of a limb can produce symptoms and signs that mimic CRPS.”

“One of the unfortunate consequences of this diagnosis is that once established it has the potential to cause considerable disability, especially in vulnerable people. The “disease” label may also have a profound adverse effect on patients’ beliefs and behaviour: the adoption of a label such as CRPS affords legitimacy but may be disempowering and encourage the adoption of the sick role.”

“As a consequence these patients often become excessively bodily focused and the suspicion of “disease” heightens bodily awareness and reinforces the belief that the patient is ill. Disability often ensues. Key psychosocial factors are ignored and neither acknowledged nor tackled by the interviewer. This is important, because considerable evidence now shows that catastrophic thinking and abnormal health beliefs and expectations are the main determinants of chronic pain after injury, as most of these cases are.”

“Indeed, abundant evidence now shows that it is these aspects of the psychosocial
environment and not biological factors that are associated with a higher likelihood of developing chronic painful disorders such as back pain, whiplash neck injury, and fibromyalgia or chronic widespread pain. It is invariably antecedent psychosocial factors, as well as beliefs or expectations about recovery after an injury, that are more likely to determine whether the pain becomes chronic, rather than the nature or severity of the injury.

“there is a case for abandoning the term CRPS altogether because of its potential for iatrogenic harm. Terms such as “non-specific arm pain” have been advocated, and good examples in the English language include headache and backache, why not armache or legache?”

II. Modern Diagnostic Protocols for Complex Regional Pain Syndrome

II.A. Background

Complex regional pain syndrome (CRPS) is a construct that was created in the 1990’s in response to the complete scientific failure of the concept of reflex sympathetic dystrophy (RSD) (e.g., Merskey & Bogduk 1994; Stanton-Hicks; Biller). Apparently, the people who created this concept perceived a need for a construct to replace RSD, even though the scientific failures of the concept of RSD were so extreme (e.g., Biller) that it seems it would have been better to simply discontinue it without any replacement. The shortcomings of the conceptualization of CRPS also indicate that it might have been better to avoid replacing RSD with anything (Bass; Borchers; Biller; Barth & Bohr a & b; Barth 2006, 2009, 2011; Del Piñal; Ring).

Because of the complete nature of the scientific failings of RSD, any construct that was created for the purpose of replacing it had to be drastically different from RSD, so that the replacement would not be vulnerable to RSD’s failures. Consequently, the concept of CRPS was deliberately created in a fashion that was free from issues that defined RSD (e.g. reflexive etiology, involvement of the sympathetic nervous system, dystrophy, diagnosis through sympathetic block, etc.). In fact, the avoidance of such RSD definitional issues, in combination with the lack of a scientific basis for the creation of the construct of CRPS, resulted in CRPS being created in a fashion that causes it to be extremely ambiguous (Barth, 2006).

Given the intentionally vague nature of this concept, it is not surprising that scientific efforts to validate it as a specific health condition have failed (Barth 2009, 2011; Borchers; Biller; Rondinelli). Because of such lack of scientific validation, the American Medical Association published a clear statement that CRPS is not a disease, but is instead a constellation of complaints that have no known cause (Melhorn 2008). Consequently, it is important to note that CRPS is simply a construct, rather than actually representing a specific health condition.
The construct of CRPS definitionally involves disproportionality to any claimed inciting event (such as a claimed injury) (Harden 2005, 2007; Merskey & Bogduk 1994, 2012). This disproportionality creates an obstacle to credibly claiming injury-relatedness for a CRPS-like presentation, because scientific standards for causation analysis call for proportionality to be demonstrated if a causative relationship exists (Melhorn, 2014). Consistent with the lack of injury-relatedness which is inherent to such definitional disproportionality, insinuations of injury-relatedness which were involved in RSD have been specified, from the beginning of the history of the construct of CRPS, as not being essential for that construct (Merskey & Bogduk 1994). As the construct of CRPS has evolved over time, it has moved even further away from insinuations of injury-relatedness. For example, the “initiating noxious event” criterion which was non-essential in the original diagnostic criteria for CRPS (Merskey & Bogduk 1994) is not mentioned at all in the modern diagnostic criteria sets that are discussed in this chapter.

In spite of the definitional inconsistency of CRPS with injury-relatedness, cases which receive the diagnosis are dominated by legal claims in general (Allen) and especially workers compensation claims (Verdugo; Talmage). The manner in which such cases are dominated by legal claims has been highlighted in the American Medical Association’s causation Guides (Talmage), along with the lack of scientific credibility for such legal claims of injury-relatedness (Talmage; Melhorn 2008). The paradox of such a non-injury-related concept being dominated by legal claims of injury-relatedness also prompted the Medical Director of the American Academy of Orthopaedic Surgeons to highlight CRPS as a primary example of issues which have been inappropriately forced upon the workers compensation system (Haralson 2008).

It is the paradoxical prominence of the CRPS construct in legal claims that warrants this discussion of modern diagnostic protocols within the AAOS workers compensation curriculum, in spite of the concept’s ambiguity, lack of scientific validity, and definitional lack of injury-relatedness.

II.B. History of Diagnostic Chaos

Consistent with the ambiguous nature of CRPS, and consistent the lack of scientific validity for the construct, there is no objective basis on which such a diagnosis can be based (Bass; Borchers; Biller; Barth 2006, 2009, 2011; Barth & Bohr a & b; Barth & Haralson a & b; Del Piñal; Ring). Consequently, a variety of diagnostic protocols have been proposed which are based exclusively on description (rather than etiology, physiology, or any other aspects that are typical for general medical concepts which have been scientifically validated). Historically, such protocols have been “non-standardized, idiosyncratic, or incompatible” (Hardin 2001), and the existence of this multitude of protocols has highlighted the extreme ambiguity and unreliability of the CRPS construct (Barth 2006).
An example of the problematic scenarios which have been caused by this history of diagnostic chaos was widely reported in 2013 (e.g., Judicial View; Vermont Workers Comp Bulletin; government posting of the court ruling - http://info.libraries.vermont.gov/supct/current/op2011-270.html). Reportedly, Vermont was still requiring impairment ratings to comply with the 5th Edition of the Guides to the Evaluation of Permanent Impairment (Cocchiarella), even though that edition had already been obsolete for years. A court ruled that a claim of CRPS warranted an impairment rating as determined by the CRPS protocol from the 5th Edition, even though the diagnosis was not based on the diagnostic requirements which are specified within that Edition (the diagnosing doctor reportedly used something other than the 5th Edition’s diagnostic requirements as the basis for the diagnosis). In other words, the court forced the use of a 5th Edition impairment rating onto an individual claim of CRPS which, according to the 5th Edition’s requirements, was not actually eligible for that impairment rating. The result was the use of a 5th Edition impairment rating within a context for which such ratings were never intended (a clear misuse of the 5th Edition impairment evaluation method). This judicial mandating of a misuse of the 5th Edition would have had less of an opportunity to develop if a single standard protocol for diagnostic evaluation of CRPS claims had been established (instead of the history of diagnostic chaos).

II.B.1. The International Association for the Study of Pain’s original attempt to introduce some standardization

In 1994, the International Association for the Study of Pain (IASP) attempted to introduce some standardization through formal endorsement of a specific diagnostic protocol (Merskey & Bogduk 1994). However, that attempt was a failure on multiple levels.

The 1994 IASP protocol has been subjected to much criticism, including criticism that was published by the IASP itself (Wilson). Aspects of the protocol which are vulnerable to criticism include its inherently self-defeating nature: it calls for CRPS to be excluded from diagnostic consideration if the case involves other potential explanations, such as a somatoform disorder or malingering, but it completely overlaps with diagnostic protocols for somatoform disorders, and most cases will overlap with the diagnostic protocol for malingering (Barth & Bohra). Clinicians and researchers continued to use competing diagnostic systems (van de Beek; Reinders). The protocol’s adoption by scientists has been characterized as “sporadic at best” (Harden 2007; American College of Occupational and Environmental Medicine). Additional diagnostic protocols continued to emerge (Cocchiarella; Hardin 2005; Rondinelli; American College of Occupational and Environmental Medicine; ODG Treatment). Most importantly, field testing of the 1994 IASP protocol demonstrated that it leads to excessive diagnosis (Harden 2007). This vulnerability to excessive diagnosis was demonstrated to be so severe that a diagnosis of CRPS was found to be incorrect in the majority of research cases (Hardin 2005).
II.B.2. The American Medical Association’s first few attempts to overcome the shortcomings of the original IASP protocol

In spite of the lack of scientific validity for the concept of CRPS, the scientific and professional failure of the IASP’s 1994 protocol, and the definitional lack of injury-relatedness for CRPS, the paradox of CRPS cases being dominated by legal claims (especially workers compensation claims) necessitated the American Medical Association addressing this concept in its Guides Library.

The Guides Library’s contributions to the discussion of the concept of CRPS apparently began with a 1997 Guides Newsletter article (Ensalada). Because of the flawed nature of the 1994 IASP protocol (e.g., it’s vulnerability to excessive diagnosis of CRPS), the 1997 article called for clinicians to avoid utilization of the IASP’s protocol, in favor of an extensive differential diagnostic process seeking to eliminate alternative diagnoses (the specified differential diagnostic issues were all of a psychological nature: somatoform disorders, malingering, and factitious disorder), followed by objective determination of whether a list of physical signs and imaging findings were relevant to the examinee. In other words, a diagnostic approach was recommended which involved determining whether a recognizable syndrome of objective findings emerged, in the context of a total lack of relevant alternative explanations for the clinical presentation. This was the beginning of a history of the Guides Library presenting protocols which attempted to remedy the inadequacy of the IASP’s 1994 protocol (with the remainder of that history to be discussed later in this text). That protocol from the 1997 AMA article was re-emphasized in the AMA’s 1999 Guides Casebook (Brigham).

An important element of the protocol that was recommended in the 1997 AMA article and the 1999 Casebook was the avoidance of any insinuation of injury-relatedness for CRPS-like presentations. This avoidance of such insinuations contrasts against the impression of injury-relatedness that could be construed from the strange formatting of the 1994 IASP protocol (Merskey & Bogduk 1994). The 1994 IASP protocol starts with the criterion, “The presence of an initiating noxious event, or a cause of immobilization”, but then strangely reverses itself by noting that this criterion is not actually required in order for a diagnosis of CRPS to be made. In contrast to that confusing set of circumstances and the insinuation of injury-relatedness that could be construed from it, the protocol that was recommended in the 1997 Guides Newsletter article did not contain any such language regarding an inciting event. This elimination of any misleading implication of injury-relatedness for the concept of CRPS was a precursor for research publications which indicated the need to abandon such implications (Harden, 1999), the development of other protocols which similarly removed insinuations of injury-relatedness (Rondinelli; Harden, 1999, 2005, 2007; American College of Occupational and Environmental Medicine; ODG Treatment) and for later Guides Library publications which highlighted the lack of injury-relatedness that is inherent to the concept of CRPS (Rondinelli; Barth & Bohr a; Melhorn 2008).
Notes:

The AMA approach was re-emphasized and expanded in the 2001 *Guides to the Evaluation of Permanent Impairment, 5th Edition* (Cocchiarella). The *Guides 5th Edition* re-emphasized the directive for clinicians to avoid utilization of the IASP’s 1994 protocol, in favor of an extensive differential diagnostic process seeking to eliminate alternative diagnoses (again specifying primary differential diagnostic issues which were all of a psychological nature), followed by objective determination of whether a list of physical signs and imaging findings applied to the examinee in the absence of relevant alternative diagnoses. The specifics of the protocol which had been presented in the previous *Guides* Library publications were expanded. The new protocol required a more conservative approach, by specifying that a longer list of objective findings needed to be present before a diagnosis of CRPS could be endorsed. That newly expanded protocol was the second major step in what would become a continuing process of the *Guides* Library presenting protocols which attempt to remedy the inadequacy of the IASP’s 1994 protocol for CRPS, specifically including attempts to reduce the empirically established excessive diagnosis of CRPS.

II.C. Modern Protocols

The above discussion of the history of IASP and AMA protocols did not include the details of those protocols, because they are all now obsolete. The following discussion provides a more detailed discussion of the modern protocols which have replaced them.

There is a common element in all of the modern protocols: some adoption of criteria from what has been called “the Budapest protocol”.

II.C.1. The emergence of the Budapest protocol

“Budapest” refers to the city in which “a closed workshop (by invitation only) was held” in the fall of 2003, to discuss diagnostic and treatment considerations for CRPS (Harden 2005, 2007). The results of that closed meeting included an endorsement of a new protocol from the by-invitation-only group, and a proposal that the IASP consider adopting this new protocol (as is noted below, the IASP failed to adopt anything from this new protocol until 2012, and then adopted only parts of it).

A published discussion of the Budapest meeting (Harden 2007) refers to a book that has been based on that meeting (Wilson), and the book reveals sponsorship by a medical device manufacturer who stands to benefit financially from people receiving a diagnosis of CRPS. Personal communication with the reported primary organizer of the meeting confirmed such industry sponsorship of the meeting (Stanton-Hicks M. Personal communication, 12-2-2009). This is a concern, given aspects of the Budapest protocol which stray from the original IASP and AMA protocols in a manner that creates a risk of a severe exacerbation of the empirically established excessive diagnosis of CRPS (details discussed below). Remarkably, the
sponsorship by a commercial entity that will financially benefit from such excessive diagnosis has not been acknowledged in more recent published discussions of the Budapest protocol (Harden 2007, 2010a, 2010b; Merskey & Bogduk 2012).

The Budapest protocol is largely based on research (Bruehl; Harden 1999, 2005, 2010a) that has been conducted in an effort to overcome the complete lack of scientific support for, and field test failures of, the 1994 IASP protocol. Although it contributes the significant benefit of avoiding the self-contradictory implication of injury-relatedness that was written into the 1994 IASP protocol, and adds a minimal level of scientific support, it is none-the-less afflicted by several significant concerns. Such concerns have repeatedly been published in AMA Guides Library and American Academy of Orthopaedic Surgeons publications (Barth & Bohr b: Barth 2009, 2011), and are therefore only being discussed here in a highly summarized fashion. They include:

- The relevant research papers have been self-contradictory, misleading, and inaccurate in their use of the concepts of “sensitivity” and “specificity” for a health concept that the researchers acknowledge has no definitive diagnostic mechanism (Harden 2010a). Therefore, in reality, there is no credible basis for making determinations of sensitivity or specificity. The research reports of sensitivity, specificity, and diagnostic accuracy are consequently meaningless.

- The research designs that have been used for purposes of claiming some validation for the Budapest protocol are of a nature that can artificially inflate the resulting claims of diagnostic accuracy (this was acknowledged in an early research publication; Harden 2005), but, as is the case for the early admission of corporate sponsorship, such acknowledgement of this flaw in the research design has been remarkably missing from more recent published discussions (Harden 2007, 2010a; Merskey & Bogduk 2012).

- Despite this risk of producing artificially inflated claims of diagnostic accuracy, the results revealed that this protocol does not reliably distinguish between claims of CRPS, and cases that are known to not involve CRPS (Harden 2005, 2007, 2010a). The numbers are improved compared to similar research for the 1994 IASP protocol, but the research based on the Budapest protocol demonstrated that the diagnostic process is still unreliable, and the Budapest protocol continues the history of excessive diagnosis. Furthermore, originally reported rates of diagnostic accuracy have decreased in later research (Harden 2010a).

- The original research publications that reported some field testing of the Budapest protocol concluded with statements which specified that further attempts to validate the protocol were needed (in other words, the research design was not of a nature which credibly allowed for a claim that the findings
had validated the protocol) (Harden 2005, 2007). The more recent claims of validation for the Budapest protocol involved the same almost meaningless research design that had been reported for prior research, and involved an emphasis on comparing the Budapest protocol to the 1994 IASP protocol (Harden 2010a). None of this provides a sound basis for a claim of validation, given the inadequacies of the research design, and because the 1994 protocol was already known to be deeply flawed, unreliable, and a field test failure. The resulting impression is that the original acknowledgement of a need for validation has been forgotten due to new research which is not actually capable of providing such validation.

- The research which has focused on the Budapest protocol has not been of a nature, in terms of design or results, that could actually validate the existence of a health condition that corresponds to the concept of CRPS.

- The overlap with somatoform disorders and malingering that is inherent to the 1994 IASP protocol is repeated in the Budapest protocol (e.g. disproportionality, lack of diagnostic pathophysiology).

- The protocol involves an acknowledged reliance on the subjective reports of the examinee, and on the subjective impressions of the examiner, rather than on anything objective (Harden 2010c).

- The protocol involves a misclassification of subjective examinee responses as objective signs (e.g. all of the following subjective issues which are dependent upon reports or demonstrations from the examinee are mischaracterized within the protocols as “signs”: hyperalgesia, allodynia, range of motion, weakness).

- The protocol allows for examiner subjectivity to compromise the potential objective value of signs. For example, there is no requirement for abnormalities of temperature, skin color, sweating, or edema to be objectively evaluated. The protocol allows for all such issues to be subjectively evaluated by clinicians. Scientific findings have revealed that such subjective clinical impressions are not reliable, and are frequently inaccurate (Dijkstra; Harden 2001; Oerlemans; van de Vusse).

- The Budapest protocol characterizes motor abnormalities as a diagnostic criterion for CRPS, in spite of scientific findings which have indicated that this issue is uniquely psychological when it occurs within the context of a CRPS claim (Verdugo).

- The Budapest protocol was actually intentionally created to be two protocols – one for clinical work and a different one for scientific research (Harden 2005,
Notes:

2007). If this aspect of the protocol is widely adopted, then the future of research on this subject will be largely irrelevant to health care. Because the clinical protocol is different from the research protocol, attempts to apply scientific findings to health care will be analogous to expecting that findings regarding apples will universally apply to oranges.

- The differential diagnostic requirement is softened (compared to the 1994 IASP and historical AMA protocols) in a manner that creates an even greater vulnerability to excessive diagnosis than that which had been empirically demonstrated for the 1994 IASP protocol. Amazingly, the protocol also introduces an option of making a CRPS diagnosis in the absence of any diagnostic requirements, as is explained below.

- Published descriptions of the protocol do not agree with one another regarding the details of the protocol, even though the different versions of the protocol are both claimed as having been “adopted and codified” at the by-invitation-only, corporately sponsored meeting for purposes of “being proposed to the Committee for Classification of Chronic Pain of the IASP for inclusion in future revisions of their formal taxonomy and diagnostic criteria for pain states” (Harden 2005, 2007). I have attempted to read every publication that I can find regarding the protocol, but I have never found an explanation for this published contradiction regarding the details of the protocol (discussed further in the following text).

Readers are strongly urged to keep the above limitations in mind as they review the following details of the Budapest protocol (Harden 2005, 2007). Because publications regarding the Budapest protocol are contradictory of one another in regard to the specifics of the protocol (Harden 2005, 2007), the more recently published description is provided below, along with notes regarding contradictory information from the previously published description. Oddities of formatting (e.g. bold print, missing punctuation, etc.) are original to the source material (Harden 2007).

"Proposed clinical diagnostic criteria for CRPS"

General definition of the syndrome:

CRPS describes an array of painful conditions that are characterized by a continuing (spontaneous and/or evoked) regional pain that is seemingly disproportionate in time or degree to the usual course of any known trauma or other lesion. The pain is regional (not in a specific nerve territory or dermatome) and usually has a distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. The syndrome shows variable progression over time.

To make the clinical diagnosis, the following criteria must be met:
Notes:

1. Continuing pain, which is disproportionate to any inciting event

2. Must report at least one symptom in three of the four following categories:

   **Sensory:** Reports of hyperesthesia and/or allodynia

   **Vasomotor:** Reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry

   **Sudomotor / Edema:** Reports of edema and/or sweating changes and/or sweating asymmetry

   **Motor / Trophic:** Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. Must display at least one sign at time of evaluation in two or more of the following categories:

   **Sensory:** Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement)

   (NOTE: The option for this sensory criterion to be satisfied by temperature sensation was missing from the 2005 publication, and then introduced without explanation in the 2007 publication, with both publications claiming that its version of this criterion had been “adopted and codified” at the closed meeting in Budapest in 2003.)

   **Vasomotor:** Evidence of temperature asymmetry (>1° C) and/or skin color changes and/or asymmetry

   (NOTE: The requirement that temperature asymmetry must exceed one degree centigrade was missing from the 2005 publication, and then introduced without explanation in the 2007 publication, with both publications claiming that its version of this criterion had been “adopted and codified” at the closed meeting in Budapest in 2003.)

   **Sudomotor / Edema:** Evidence of edema and/or sweating changes and/or sweating asymmetry

   **Motor / Trophic:** Evidence of decreased range of motion and/or motor
dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. There is no other diagnosis that better explains the signs and symptoms

For research purposes, diagnostic decision rule should be at least one symptom in all four symptom categories and at least one sign (observed at evaluation) in two or more sign categories.”

The associated literature (Harden 2007) specifies:

- “Current distinctions between CRPS type I and CRPS type II subtypes, reflecting, respectively, the absence and presence of evidence of peripheral nerve injury, were retained”.

- “A third diagnostic subtype called CRPS-NOS was recommended that would capture those patients who did not fully meet the new clinical criteria, but whose signs and symptoms could not better be explained by another diagnosis”.

Readers can note that the following two passages contribute to the increased risk of excessive diagnosis that is associated with the Budapest protocol (Harden 2007):

- “4. There is no other diagnosis that better explains the signs and symptoms.”

  - This new wording can be compared to a corresponding passage from the 1994 IASP protocol (Merskey & Bogduk 1994): “This diagnosis is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.” The new wording in the Budapest protocol puts the possibility of a diagnosis of CRPS on the same footing as any of the differential diagnostic issues, while the 1994 IASP protocol emphasized that differential diagnostic issues had a preferential standing compared to the possibility of a CRPS diagnosis. This might not be a significant difference for any case in which a comprehensive differential diagnostic process is completed, because the overwhelmingly more common nature of many relevant differentials (Barth and Haralson a & b) will almost always result in the identification of another “diagnosis that better explains the signs and symptoms”.

- “A third diagnostic subtype called CRPS-NOS was recommended that would capture those patients who did not fully meet the new clinical criteria, but
whose signs and symptoms could not better be explained by another diagnosis”.

- The risk of over-diagnosis is obvious in the above passage, as it indicates that a clinical presentation does not actually have to satisfy any diagnostic requirements in order for a CRPS diagnosis to be adopted. In competent clinical practice (involving a comprehensive differential diagnostic process), this might not be a significant issue, because the overwhelmingly more common nature of many relevant differentials (Barth and Haralson a & b) will almost always result in a realization that “signs and symptoms” can be better explained by “another diagnosis”.

It is important to note that an early passage within this protocol (quoted above) specifies that the construct of CRPS is not intended to imply that a specific health condition has been identified, but is instead an umbrella construct which is potentially applicable to “an array of painful conditions”. This is consistent with the historical purpose for which the CRPS construct was created (Stanton-Hicks 1995). However, it is contradictory of other literature from authors of the published descriptions of the protocol, in that such other literature actually claims (without explanation) that the CRPS construct represents a single health condition (Harden & Bruehl 2010b). This contradiction is another indication of the unreliability of the CRPS construct.

II.C.2. 2008: Criteria from the Budapest protocol are adopted for the 6th Edition of the AMA’s Guides to the Evaluation of Permanent Impairment

2008 is the original copyright year for the 6th Edition of the Guides to the Evaluation of Permanent Impairment (Rondinelli). The Guides 6th continued the tradition of the AMA’s Guides Library attempting to overcome the shortcomings of the 1994 IASP protocol for CRPS. In this regard, one of the published versions of the criteria set from the clinical portion of the Budapest protocol was adopted, for the 6th Edition, in a fashion that was consistent with the Guides Library’s historical emphasis on differential diagnosis (in other words, the Budapest protocol was not adopted in its published form from either 2005 or 2007, both of which set the stage for an expansion of the empirically documented excessive diagnosis of CRPS).

This adoption of a criteria set from the Budapest protocol by the Guides 6th appears to be the first endorsement, of any kind, of any aspect of the Budapest protocol by any health science academy, or by any entity that was not a recipient of the corporate sponsorship that led to the protocol’s creation. Consistent with my impression in that regard, personal communication with the reported primary organizer of the by-invitation-only Budapest meeting which endorsed the Budapest protocol reported that he was not aware of any other endorsement of any other aspect of the protocol by any health science academy prior to this adoption of a criteria set for the Guides 6th (Stanton-Hicks M. Personal communication, 12-2-2009).
Notes:

The 6th Edition incorporated the criteria from the clinical version of the Budapest protocol for several reasons, including all of the following (NOTE: I am reporting the following based on my direct experience as a contributor and reviewer for the Guides 6th, including contributing to the creation of the Guides 6th discussions of CRPS):

- While the Guides 6th reiterates that CRPS has not been scientifically validated as an actual health condition, and the construct was created in a fashion which causes it to be inherently non-injury-related (and therefore, non-work-related), workers compensation claims and personal injury claims continue to dominate cases in which this diagnosis is utilized (and the purpose of the Guides is to assist administrators with such claims). Due to the paradox of this non-injury-related condition being dominated by legal claims, the only means by which the contributors to the 6th Edition (including me) could construe a workers compensation-relevant example was to discuss a scenario in which an assumption of work-relatedness was mistakenly forced onto the case by administrative officials (since there would not be any credible basis for concluding work-relatedness). But the need for CRPS to be addressed with the 6th Edition was made clear by the prominence of this construct in medical-legal claims, and the resulting need from administrators for help with such claims.

- Although the scientific “validation” research that has been conducted for the Budapest protocol is of a largely misleading and meaningless nature (as was discussed above), its existence provides a basis for claiming that the Budapest criteria has been subjected to more study than prior protocols.

- The Budapest protocol avoids the self-contradictory insinuations of injury-relatedness that had compromised the 1994 IASP protocol.

- Reviewers/contributors who were involved in the creation of the Guides 6th predicted that the Budapest protocol would eventually be more widely utilized within health care than other protocols would be, and, consequently, the Guides 6th would have the best chance of being consistent with health care long-term if it utilized the Budapest criteria in some manner (even if the Guides 6th did so in a fashion that avoided the risk of excessive diagnosis that was inherent in the Budapest protocol).

In order to emphasize scientific credibility, the Guides 6th did not adopt the “General definition” portion of the Budapest protocol. Instead, the Guides 6th provided a far more extensive discussion which emphasizes all of the following...

- the lack of scientific credibility for the CRPS construct
- the inherent lack of injury-relatedness
- the role of disuse in the creation of relevant presentations
- almost all differential diagnostic issues are far more probable than CRPS is said to be
Notes:

- the unreliable nature of a diagnostic claim of CRPS (no matter what diagnostic protocol is utilized)
- the necessity of a comprehensive differential diagnostic process
- the greater importance of differential diagnosis relative to diagnostic criteria
- the elimination of CRPS from diagnostic consideration if any potentially relevant differential cannot be ruled out.

The Guides 6th introduced several mechanisms for the purpose of minimizing the increased risk of excessive diagnosis that is inherent to the Budapest protocol:

- The Guides 6th did not accept the CRPS NOS concept (as was discussed above, this is the “subtype”, from the Budapest protocol, which allows a CRPS diagnosis to be made for cases which do not actually satisfy diagnostic requirements for CRPS type one or type two).

- Because of the lack of scientific credibility for the concept of CRPS, and because almost all differential diagnosis issues are going to be far more common than CRPS is said to be (Barth & Haralson a & b), and because all of the objective physical findings that have been written into the concept of CRPS can be created simply through disuse of the involved body part (Butler), the 6th Edition specifies that the criteria set from the Budapest protocol is only to be utilized after a comprehensive differential diagnostic process has been completed in a fashion that rules out all differentials. By emphasizing the necessity of such a differential diagnostic process, the 6th Edition largely overcomes the increased vulnerability to excessive diagnosis of CRPS that is built into the Budapest protocol.

In regard to that differential diagnostic requirement, the Guides 6th references another American Medical Association publication (Barth and Haralson a) which emphasizes that, because a CRPS-like presentation can be created simply through disuse, and because a CRPS-like presentation can easily be created through deliberate attempts to create a fraudulent medical-legal claim (and, according to scientific findings, often is a product of such deliberate fraud), any issue which might be contributing to such disuse or deliberate fraud must be ruled out before a diagnosis of CRPS can be credibly considered. At a minimum, the list of differential diagnostic issues which must be evaluated for and ruled out before a CRPS diagnosis can be credible, according to that American Medical Association publication (and also according to a parallel publication from the American Academy of Orthopaedic Surgeons) (Barth and Haralson a & b) includes (but is not limited to):

- mental illness that would have traditionally fallen into the category of somatoform disorders
- factitious disorder
- malingering
- personality disorders
Notes:

- mental illness that would have traditionally fallen into the category of anxiety disorders
- mental illness that would have traditionally fallen into the category of mood disorders
- mental illness that would have traditionally fallen into the category of substance-related disorders
- psychotic disorders
- Raynaud’s phenomenon/disease
- cellulitis
- thromboangiitis obliterans
- thrombosis
- traumatic vasospasm
- nerve entrapment
- herpes zoster
- rheumatologic conditions
- stress fracture
- diabetic neuropathy

If a comprehensive differential diagnostic process rules out all potential differentials, then the Guides 6th calls for a version of the Budapest criteria to be applied to the clinical presentation. The criteria set which is presented in the Guides 6th is as follows:

1. **Continuing pain, which is disproportionate to any inciting event**

2. **Must report at least 1 symptom in 3 of the 4 following categories:**

   **Sensory:** Reports of hyperesthesia and/or allodynia

   **Vasomotor:** Reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry

   **Sudomotor / Edema:** Reports of edema and/or sweating changes and/or sweating asymmetry

   **Motor / Trophic:** Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. **Must display at least 1 sign at time of evaluation in 2 or more of the following categories:**

   **Sensory:** Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement)
Notes:

Vasomotor: Evidence of temperature asymmetry and/or skin color changes and/or asymmetry

Sudomotor / Edema: Evidence of edema and/or sweating changes and/or sweating asymmetry

Motor / Trophic: Evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. There is no other diagnosis that better explains the signs and symptoms

This criterion set follows the 2005 published version of the Budapest protocol (Harden 2005) more closely than the 2007 version (Harden 2007) in the following ways:

- Temperature sensation has not been included as a physical examination tool that can be used for the hyperalgesia criterion.

- The requirement that a finding of temperature asymmetry must exceed 1°C in order to satisfy the relevant diagnostic requirement was not included.


2008 is also listed as the year in which a revision was undertaken for the American College of Occupational and Environmental Medicine’s (ACOEM) Occupational Medicine Practice Guidelines chapter which is entitled “Chronic Pain” (American College of Occupational and Environmental Medicine).

A pre-publication draft of that chapter was circulated for review, and the American Academy of Neurology asked me to represent it in that review process. That pre-publication draft included endorsement of the criteria set from the Budapest protocol in a form that included an increased risk of excessive diagnosis. The written response to reviewing that chapter, which I provided to the American Academy of Neurology, advised of the need to highlight the corporate sponsorship and associated financial conflict of interest that was associated with the Budapest protocol, and the consequent increased risk of excessive diagnosis that was inherent to that protocol.

The American Academy of Neurology shared that recommendation with ACOEM, and the editor-in-chief of the ACOEM Guidelines (AAOS faculty member Kurt Hegmann) consequently offered me an opportunity to help develop a modification for those Guidelines which could minimize the risk of excessive diagnosis. Our efforts in this regard led to a
Notes:

modification of the differential diagnosis language from the protocol (see item #4 below), so that it was more consistent with the 1994 IASP protocol and with the 6th Edition of the AMA’s Guides to the Evaluation of Permanent Impairment (and, consequently, less prone than the Budapest protocol to expanding the tradition of excessive diagnosis).

The ACOEM protocol also rejected the CRPS NOS subtype from the Budapest protocol (the subtype that involves a CRPS diagnosis being made in cases which do not satisfy diagnostic requirements for either CRPS type one or type two).

The ACOEM protocol also differs from the Budapest protocol in that the “General definition” portion of the Budapest protocol was not adopted.

The ACOEM protocol’s criteria set is consistent with the 2005 criteria set that was published for the Budapest protocol (Harden 2005), and is consistent with the 6th Edition of the AMA’s impairment Guides (Rondinelli). The ACOEM criteria set does not match the 2007 version of the Budapest criteria (Harden 2007), in that the following issues were not included: temperature sensation as a physical examination tool that can be used for the hyperalgesia criterion; the requirement that a finding of temperature asymmetry must exceed 1°C in order to satisfy the relevant diagnostic requirement.

The resulting ACOEM protocol reads as follows:

1. Continuing pain, which is disproportionate to the inciting event

2. At least one symptom in three of these four categories:

   Sensory: hyperesthesia and/or allodynia

   Vasomotor: temperature asymmetry and/or skin color changes and/or skin color asymmetry

   Sudomotor / edema: edema and/or sweating changes and/or sweating asymmetry

   Motor / trophic: decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. At least one sign at time of evaluation in two or more of the following categories:

   Sensory: evidence of hyperalgesia to pinprick and/or allodynia to light touch and/or deep somatic pressure and/or joint movement

   Vasomotor: Evidence of temperature asymmetry and/or skin color changes
Notes:

and/or asymmetry

Sudomotor / edema: evidence of edema and/or sweating changes and/or sweating asymmetry

Motor / trophic: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. Diagnosis: CRPS is excluded by the existence of conditions that would otherwise account for the degree of pain and dysfunction.

The ACEOM Guidelines warn that the above protocol “may be inadequate” because the protocol fails to mention objective measurement of the supposed “signs” (a shortcoming that is also of relevance to all of the other protocols that are discussed in this article; see Harden 2010c). Consequently, within the ACOEM Guidelines, objective measurement of such signs is “required” (the Guidelines offer examples such as temperature probes and volumetry).

Consistent with the emphasis on psychological differentials from the AMA Guides 6th (and all prior relevant Guides Library publications), the ACOEM Guidelines also report: “The threshold for concomitant psychological consultation and psychometric testing in such circumstances should be quite low.”


While the 2003 closed meeting in Budapest was reportedly focused on recommending the Budapest protocol for adoption by the International Association for the Study of Pain (IASP) (Harden 2007), the IASP has not adopted the protocol, and did not even adopt one of the published versions of the criteria set from the Budapest protocol until 2012 (Merskey & Bogduk 2012).

The modern IASP protocol has the following characteristics:

- The “General definition” portion of the Budapest protocol (Harden 2007) has been eliminated in favor of a more expansive discussion.

- The use of temperature sensation as a physical examination tool for the hyperalgesia criterion, which is part of the 2007 published version of the Budapest protocol (Harden 2007) was not adopted (consistent with the 2005 publication of the Budapest protocol, and with the AMA and ACEOM versions of that criterion).
Notes:

- The requirement that a finding of temperature asymmetry must exceed 1°C in order to satisfy the relevant diagnostic requirement, which is part of the 2007 published version of the Budapest protocol (Harden 2007) was not adopted (consistent with the 2005 publication of the Budapest protocol, and with the AMA and ACEOM versions of that criterion).

- The CRPS NOS subtype from the Budapest protocol is included in the IASP version, thereby allowing a CRPS diagnosis to be made in cases which do not satisfy diagnostic requirements for CRPS type one or type two.

- The differential diagnosis criterion is of a nature that is consistent with the 2005 and 2007 publications of the Budapest protocol (Harden 2005, 2007), and consequently eliminates the historical IASP and AMA principle that CRPS was excluded from diagnostic consideration by the presence of any other issues which could explain the clinical presentation (like the inclusion of the CRPS NOS subtype, this element of the modern IASP protocol increases the risk of excessive diagnosis that was empirically established for the 1994 IASP protocol).

- The 2012 IASP text provides a differential diagnostic discussion which is remarkably minimal compared to the AMA and American Academy of Orthopaedic Surgeons discussions (Rondinelli; Barth & Haralson a & b). The IASP discussion in this regard fails to consider most of the strongest scientific findings, such as the prominence of legal claims among people who obtain a CRPS diagnosis (Barth, 2013 a & b; Talmage), the prominence of personality disorders and other pre-existing forms of psychopathology among people who obtain a CRPS diagnosis (Barth & Haralson a & b; Barth 2013 a & b), and the prominence of people who obtain a CRPS diagnosis responding to objective testing in a malingering-like manner (Greiffenstein). In fact, the IASP discussion inexplicably avoids consideration of most of the scientific findings that have been discussed in relevant AMA and American Academy of Orthopaedic Surgeons publications (Barth and Haralson a & b; Barth 2013 a & b). This avoidance of science has been an intentional feature of the CRPS construct since it was first created (Stanton-Hicks 1995).

- The three bullet points directly above this one indicate that the 2012 IASP protocol creates an extreme risk of over-diagnosis, including a greater risk of excessive diagnosis than the risk that was created by the 1994 IASP protocol (which reportedly prompted the creation of the Budapest protocol because it was demonstrated to be so vulnerable to over-diagnosis that field trials revealed most CRPS diagnoses to be false).

- The 2012 IASP protocol strays from the Budapest protocol’s distinction between CRPS type one and CRPS type two. The difference between the IASP and Budapest protocols in this regard is demonstrated by a comparison of these two passages:
The 2012 IASP adoption of a modification of the criteria set from the Budapest protocol reads as follows:

"Diagnostic Criteria"

There are two versions of the diagnostic criteria: a clinical version meant to maximize diagnostic sensitivity with adequate specificity, and a research version meant to more equally balance optimal sensitivity and specificity.

Clinical Diagnostic Criteria for CRPS

1) Continuing pain, which is disproportionate to any inciting event.

2) Must report at least one symptom in three of the four following categories: Sensory: Reports of hyperalgesia and/or allodynia. Vasomotor: Reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry. Sudomotor/Edema: Reports of edema and/or sweating changes and/or sweating asymmetry. Motor/Trophic: Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nails, skin).

3) Must display at least one sign* at time of evaluation in two or more of the following categories:
Notes:

Sensory: Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or deep somatic pressure and/or joint movement).
Vasomotor: Evidence of temperature asymmetry and/or skin color changes and/or asymmetry.
Sudomotor/Edema: Evidence of edema and/or sweating changes and/or sweating asymmetry.
Motor/Trophic: Evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nails, skin).

4) There is no other diagnosis that better explains the signs and symptoms.

*A sign is counted only if it is observed at time of diagnosis.

**Research criteria for CRPS are recommended that are more specific, but less sensitive than the clinical criteria; they require that four of the symptom categories and at least two sign categories be present.

Subtypes of CRPS

CRPS I (old name: Reflex Sympathetic Dystrophy): As defined above.
CRPS II (old name: Causalgia): Defined as above with electrodiagnostic or physical evidence of a major nerve lesion.
CRPS-NOS* (Not Otherwise Specified): Partially meets CRPS criteria, not better explained by any other condition."

Readers can also note the following confusing elements of the modern IASP protocol. The passage which reads “CRPS I (old name: Reflex Sympathetic Dystrophy)” could be misconstrued as an indication that CRPS type one and reflex sympathetic dystrophy are new and old names for the same construct, when in fact, the construct of CRPS type one is drastically different from the discontinued construct of reflex sympathetic dystrophy. The passage which reads “CRPS II (old name: Causalgia)” could be misconstrued as an indication that the concept of causalgia has been discontinued in favor of CRPS type two, when in fact, the IASP’s system (Merskey & Bogduk 2012) actually maintains causalgia as a diagnostic entity.

II.C.5. 2013: ODG Treatment in Workers Compensation references the 2007 version of the Budapest criteria set.

In 2013, these Guidelines (ODG Treatment in Workers Comp) were modified to include referencing of the 2007 version of the Budapest criteria set. Key issues from the ODG text include (http://www.odg-twc.com/):
Notes:

- An emphasis on differential diagnosis, consistent with the emphasis in this regard from the AMA (Barth and Haralson a; Rondinelli), American Academy of Orthopaedic Surgeons (Barth & Haralson b), and American College of Occupational and Environmental Medicine literature (American College of Occupational and Environmental Medicine). This emphasis is markedly different (more thorough) than the originally published versions of the Budapest protocol (Harden 2005, 2007) and the modern IASP protocol (Merskey & Bogduk 2012). Relevant passages from ODG Treatment include:

  - “Financial gain (such as that involved with litigation) has been found to increase the risk of CRPS.”

  - “There should be evidence that all other diagnoses have been ruled out. A diagnosis of CRPS should not be accepted without a documented and complete differential diagnostic process completed as a part of the record.”

  - “It is suggested that in the absence of a differential diagnostic evaluation for patients with a suggested diagnosis of CRPS, management can be abortive and iatrogenic harm may follow.”

  - “The importance of establishing a correct diagnosis and to prevent potentially harmful and/or unwarranted treatment cannot be emphasized enough.”

  - “the objective physical signs of CRPS, including imaging, can be created with disuse and or physical manipulation.”

- While the overall approach of the 2005 or 2007 published versions of the Budapest protocol was not adopted into ODG Treatment, a criteria set reportedly based on the Budapest protocol was “recommended”, and the text specifically recommends the 2007 published version (Harden 2007) of that set (including the use of temperature sensation for the sensory “sign” criterion, and the requirement that the temperature asymmetry sign involve a discrepancy of greater than one degree centigrade, all of which is absent from the 2005 published discussion of the Budapest protocol, the AMA protocol, the ACOEM protocol, and the IASP protocol).

- In regard to the CRPS NOS diagnosis, the ODG text specifies: “This diagnosis is not endorsed by ODG.”

The new ODG criteria set parallels the criteria from the 2007 published version of the Budapest protocol, and specifically reads as follows:

“The diagnostic criteria are the following: (1) Continuing pain, which is disproportionate to any inciting event; (2) Must report at least one symptom in three of
the four following categories: (a) Sensory: Reports of hyperesthesia and/or allodynia; (b) Vasomotor: Reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry; (c) Sudomotor/Edema: Reports of edema and/or sweating changes and/or sweating asymmetry; (d) Motor/Trophic: Reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin); (3) Must display at least one sign at time of evaluation in two or more of the following categories: (a) Sensory: Evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch and/or temperature sensation and/or deep somatic pressure and/or joint movement); (b) Vasomotor: Evidence of temperature asymmetry (> 1°C) and/or skin color changes and/or asymmetry; (c) Sudomotor/Edema: Evidence of edema and/or sweating changes and/or sweating asymmetry; (d) Motor/Trophic: Evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin); (4) There is no other diagnosis that better explains the signs and symptoms.”

II.D. Recommendations

Scientific and professional publications have provided strong support for a decision to avoid utilization of CRPS as a diagnostic possibility (Bass; Biller; Borchers; Del Piñal; Ring). Scientific findings have also indicated that, even when CRPS is considered in the diagnostic process, other diagnostic options are going to be far more probable (Barth and Haralson a & b). Scientific findings have also revealed that there is going to be a high probability of any patient who obtains a diagnosis of CRPS demonstrating an invalid clinical presentation when objective testing is administered (Greiffenstein). Consequently, any clinician or scientist who decided that he or she was going to completely avoid consideration of CRPS as a diagnostic option would find a wealth of support for that decision in scientific findings and professional literature.

However, if a clinician or scientist wanted to entertain the possibility of finding some value in the construct of CRPS, then the following recommendations are offered.

II.D.1. For those unique situations in which an impairment rating is required within a legal jurisdiction that mandates use of the Fifth Edition of the Guides to the Evaluation of Permanent Impairment:

II.D.1. a. The evaluator should, first and foremost, note that scientific findings have indicated that the prognosis for CRPS-like presentations is excellent, and that there is consequently a lack of justification for claiming that such a case involves permanence (Sandroni), and a corresponding lack of justification for a permanent impairment rating.

II.D.1.b. The 5th Edition's diagnostic protocol for CRPS must be adhered to (this direction is only being provided for the purposes of that impairment evaluation, e.g. not
Notes:

for scientific, any other clinical purpose, or any other forensic purpose). The impairment rating will only be valid if the 5th Edition’s diagnostic protocol is adhered to, because the remainder of the 5th Edition’s protocol for creating an impairment rating was not designed or intended for use with any other diagnostic method. The clinician who creates the impairment rating should document, clearly and in the same document that provides the impairment rating, that legal requirements of that jurisdiction have forced the evaluator to utilize obsolete methods for impairment evaluation and diagnosis.

II.D.1.c. The diagnostic protocol from the 5th Edition of the Guides to the Evaluation of Permanent Impairment should not otherwise be used (because it is now officially obsolete). Even an evaluator that is forced to use that protocol for impairment rating due to the legal requirements of a specific jurisdiction should avoid using that protocol for any other scientific, clinical, or forensic purpose. Instead of using that protocol for any other purpose, the following recommendations should be considered.

II.D.2. The 1994 IASP protocol should be avoided, because it was empirically demonstrated to lead to excessive diagnosis, and because it is now officially obsolete.

II.D.3. The modern IASP protocol, and all of the published versions of the Budapest protocol, should be avoided, because they all have aspects which actually increase the risk of excessive diagnosis that was empirically established for the 1994 IASP protocol. Readers are also reminded of the additional problematic aspects of these protocols, as was discussed previously in this text, as well as in AMA Guides Library publications, and previous AAOS material (Barth & Bohr a & b; Barth 2009, 2011).

II.D.4. The protocol that is specified in the 6th Edition of the Guides to the Evaluation of Permanent Impairment is the protocol of choice. Advantages of this protocol include the emphasis on differential diagnosis as the first step in the diagnostic process, with the criteria from the Budapest protocol being reserved as the last part of the diagnostic process (to be considered only after all potentially relevant differential diagnostic issues have been ruled out). Readers who want to insure that they understand this AMA protocol should study not only the 6th Edition itself, but also the differential diagnosis publication which it references (Barth and Haralson a), and a more recent AMA publication which provides historical and scientific context for that protocol (Barth 2009).

II.D.5. A decision to utilize the protocol from the 6th Edition of the Guides to the Evaluation of Permanent Impairment can be additionally supported by referencing the similarity of its principles with the ACOEM protocol (American College of Occupational and Environmental Medicine) and ODG Treatment, as well as referencing support that can be found in other publications that have been referenced for this chapter (e.g., American Academy of Orthopaedic Surgeons publications such as Barth and Haralson b, and Barth 2011).
Notes:

II.D.6. Specifically for impairment rating purposes, unless the jurisdiction requires impairment ratings to be based on something other than the 6th Edition of the *Guides to the Evaluation of Permanent Impairment*, the protocol from the 6th Edition must be adhered to. The remainder of the impairment evaluation protocol from the 6th Edition will not be valid for any claim of CRPS which is based on some other diagnostic protocol. Additionally, the evaluator should, first and foremost, note that scientific findings have indicated that the prognosis for CRPS-like presentations is excellent, and that there is consequently a lack of justification for claiming that such a case involves permanence (Sandroni), and a corresponding lack of justification for a permanent impairment rating.

III. The 20th Anniversary of the death of reflex sympathetic dystrophy (the undead diagnosis)

The construct of reflex sympathetic dystrophy (RSD) was formally discontinued as of 1994, because it was found to be a complete scientific failure (extensive referencing for this fact is provided below). Consequently, any clinical, scientific, or otherwise professional work which uses this construct is inherently non-credible (except for discussing RSD as part of history). This discussion (Section III) is being provided so that readers/participants...

- can understand the need to avoid any use of this construct (other than within history lessons)
- can understand the need to reject any clinical, scientific, or professional work that utilizes the construct (other than history lessons)
- will have extensive referencing available to them if they face resistance on these issues.

III.A. The diagnostic construct of reflex sympathetic dystrophy was formally discontinued, and removed from diagnostic taxonomy, as of 1994.

The following list provides examples of publications (listed in chronological order) which can be reviewed in order to independently confirm the fact that the reflex sympathetic dystrophy construct was formally discontinued as of 1994. These references are only being offered for purposes of verifying the history of the RSD construct having been formally discontinued (it is not recommended that these publications be relied upon for all other potential issues).

- *Classification of Chronic Pain, Second Edition*  
  - See pages 40-41 for verification of the fact that the reflex sympathetic dystrophy construct had been discontinued by the time of this book’s publication.
  - Year of publication: 1994
Notes:
  • Editors: Merskey H and Bogduk N
  • Publisher: International Association for the Study of Pain
    • NOTE: A more recent revision of this text is available (and is discussed elsewhere in this chapter, for different reasons). The 1994 edition is specified in this list of historical references, because the 1994 publication was the specific documentation of the formal removal of the RSD construct from diagnostic taxonomy. That history of RSD having been formally discontinued is specified in the referenced edition, but was not mentioned in the most recent edition (apparently because the discontinuation of the RSD construct had occurred 18 years prior to the publication of the most recent edition, and had already been documented). Again, this is an example of reasons for referencing the 1994 edition for purposes of the current discussion.

    o See page 128 for verification of the fact that the reflex sympathetic dystrophy construct had been discontinued by the time of this article’s publication.
    o Year of publication: 1995
    o Authors: Stanton-Hicks M, et al.
    o Publisher: International Association for the Study of Pain

  • *Reflex Sympathetic Dystrophy, a Reappraisal*
    o See pages 6, 79, and 91 for examples of verification of the discontinuation of the RSD construct.
    o Year of publication: 1996
    o Editors: Janig W and Stanton-Hicks M
    o Publisher: International Association for the Study of Pain

  • *Complex Regional Pain Syndrome*
    o See pages 292 and 303 for examples of verification of the discontinuation of the RSD construct.
    o Year of publication: 2001
    o Editors: Harden RN, Baron R, and Janig W
    o Publisher: International Association for the Study of Pain

    o Pages 102 and 103 specify that “RSD was eliminated from medical taxonomy” in the 1990’s, and that RSD was a “meaningless diagnostic category” that was scientifically debunked.
    o Year of publication: 2001
    o Chair: Biller, J.
Notes:

- Publisher: American Academy of Neurology

- *Bonica’s Management of Pain, Third Edition*
  - See page 389 for verification of the discontinuation of the RSD construct.
  - Year of publication: 2001
  - Editor: Loeser JD
    - NOTE: A *Fourth Edition* of this book also specifies the obsolete nature of “reflex sympathetic dystrophy” (and that *Fourth Edition* is discussed elsewhere in this chapter). However, that later edition did not repeat the *Third Edition*’s detailed discussion of reasons for the discontinuation of “reflex sympathetic dystrophy” (apparently due to the fact that the RSD construct had been discontinued for 16 years by the time of the publication of the *Fourth Edition*, and that history of the RSD construct being discontinued had already been documented). Therefore, this *Third Edition* is referenced for the purposes of the current discussion.

- *CRPS: Current Diagnosis and Therapy*
  - See page 45 for an example of verification that the RSD construct was removed from diagnostic taxonomy by 1994.
  - Year of publication: 2005
  - Editors: Wilson PR, Stanton-Hicks M, and Harden RN
  - Publisher: International Association for the Study of Pain

- *Differential Diagnosis for Complex Regional Pain Syndrome, Chapter 15 in: Melhorn JM and Shields NN. 9th Annual Occupational Orthopaedics and Workers Compensation: A Multidisciplinary Perspective*
  - See page 406 for verification that the RSD construct had been discontinued as of 1994.
  - Year of publication: 2007
  - Authors: Barth RJ and Haralson R
  - Publisher: American Academy of Orthopaedic Surgeons

- *Differential Diagnosis for Complex Regional Pain Syndrome, AMA Guides Newsletter*, pages 1-16.
  - See pages 1-2 for verification that the RSD construct had been discontinued as of 1994.
  - Year of publication: 2007
  - Authors: Barth RJ and Haralson R
  - Publisher: American Medical Association

- *Guides to the Evaluation of Permanent Impairment, 6th Edition*
  - See page 538 for an example of text which verifies the discontinuation of the RSD construct.
Notes:

- Year of publication: 2008, reprint 2009
- Editor: Rondinelli RD
- Publisher: American Medical Association

- A Historical Review of CRPS in The American Medical Association’s Guides Library, The AMA Guides Newsletter
  - See page 2 for an example of verification that the RSD construct had been formally discontinued as of 1994.
  - Year of publication: 2009
  - Author: Barth RJ
  - Publisher: American Medical Association

  - See page 1345 for text which verifies that the RSD construct had been discontinued.
  - Year of publication: 2010
  - Authors: Ring D, et al.
  - Publisher: Elsevier / American Society for Surgery of the Hand

  - See pages 736 and 756 for examples of text which verifies the formal discontinuation of the RSD construct as of 1994.
  - Year of publication: 2011.
  - Author: Barth RJ
  - Publisher: American Academy of Orthopaedic Surgeons.

III.B. The diagnostic construct of reflex sympathetic dystrophy was formally discontinued, and removed from diagnostic taxonomy, as of 1994, because of the numerous and severe inadequacies of the construct.

The following list references...

- publications from before the discontinuation of “reflex sympathetic dystrophy” construct which provide information regarding why that construct needed to be discontinued

- publications from the time of the discontinuation which further explain the reasons why the construct was discontinued
Notes:

- later publications which further explain the reasons why the construct was discontinued, and which discuss the inadequacy of professional work which is based on that obsolete construct.

In 1991, the International Association for the Study of Pain published an article, in its journal entitled *Pain*, which summarized the status of the construct of reflex sympathetic dystrophy at that time. Key points from that article included:

- “the minimal criteria for clinical diagnosis of RSD and for its subclassifications are neither clear nor accepted”
- “the pathophysiology of RSD (notably the involvement of the sympathetic nervous system) is unclear”
- “there are no generally accepted therapeutic approaches to RSD”
- “under these circumstances it is difficult to develop hypotheses which are relevant for the pathophysiology of RSD which are capable of being verified in animal experiments, on patients and in vitro.”

Note: The final bullet point above is of direct relevance to the scientific principle, and the “Daubert” legal principle, of falsifiability.

Reference information:
  - See pages 242-243 for the section containing the passages that are quoted above.
  - Year of publication: 1991
  - Author: Janig W
  - Publisher: International Association for the Study of Pain

In 1992, the *Clinical Journal of Pain* published a discussion which was triggered by the above referenced publication, and which elaborated on issues that had been raised in the above publication. This 1992 publication again highlighted the inadequacies of the construct of reflex sympathetic dystrophy, including the construct’s falsifiability problem, by explaining that a diagnosis of reflex sympathetic dystrophy “cannot be proven wrong because there are neither objective nor valid subjective criteria for it. Of the same token, it cannot be proven right either.”

Reference information:
  - See page 365 for the section containing the passage that is quoted above.
  - Year of publication: 1992
  - Author: Ochoa JL

In 1994, the *Second Edition* of the International Association for the Study of Pain’s
Notes:

*Classification of Chronic Pain* was published, and explained that the construct of reflex sympathetic dystrophy had been removed from diagnostic taxonomy because of the construct’s misleading nature. Misleading aspects of the construct which are specified in that text include the facts that, in contradiction to the title of the construct (reflex sympathetic dystrophy), cases which involved that diagnosis did not reliably involve sympathetically mediated pain or dystrophy.

Reference information:
- *Classification of Chronic Pain, Second Edition*
  - See pages 40-41 for the discussion of the misleading nature of the reflex sympathetic dystrophy construct, and the removal of that construct from diagnostic taxonomy.
  - Year of publication: 1994
  - Editors: Merskey H and Bogduk N
  - Publisher: International Association for the Study of Pain

In 1995, the International Association for the Study of Pain, in its journal entitled *Pain*, published an article which elaborated on the reasons why the reflex sympathetic dystrophy construct had been discontinued. Key points from that article included:

- “Confusion exists with regard to what is meant by RSD.”
- “The term RSD has lost usefulness as a clinical designation because it has been used so indiscriminately that it is no longer clear what it means.”

Reference information:
  - See page 128 for the passages that are quoted above.
  - Year of publication: 1995
  - Authors: Stanton-Hicks M, et al.
  - Publisher: International Association for the Study of Pain

Also in 1995, the journal *Neurologic Clinics* published a history of the concept of reflex sympathetic dystrophy, including a summary of research which had been funded by the federal government for purposes of studying people who had obtained that diagnosis. That summary highlighted reasons why the RSD construct needed to be discontinued, including the following key points:

- “Patients diagnosable with RSD are not a homogenous population, they have any of multiple possible disorders generating what on the surface appears to be a specific clinical expression; a majority of them neither have nerve injury nor other organic dysfunction to explain their symptomatology.”
- “From all these observations it transpires that the clinical picture of many CPSMV (chronic pains associated with various and variable combinations of negative and positive sensory, motor and vasomotor phenomenon) patients without nerve injury, traditionally diagnosed with RSD, is specific and corresponds to a pseudoneuropathy of psychogenic origin, akin to the pseudoseizures of psychogenic origin.”
Reference information:
- Reflex Sympathetic Dystrophy, a Common Clinical Avenue for
  o See page 369 for the passages that are quoted above.
  o Year of publication: 1995
  o Authors: Ochoa JL and Verdugo RJ

In 1996, the International Association for the study of pain published a book which
documented details of the process which resulted in the removal of the construct of reflex
sympathetic dystrophy from diagnostic taxonomy. Key points, from that book, regarding the
reasons for the discontinuation of the RSD construct include:
- “Participants in the workshop on reflex sympathetic dystrophy felt the term and its
descriptors were no longer appropriate in the light of current knowledge.” (Page 91)

- “Participants (of the meeting) agreed that use of the term RSD has lost its usefulness as
  a clinical designation and had also become an indiscriminate diagnosis for patients
  showing elements of neuropathic pain or resistance to therapy.” (Page 79)

- “the term RSD is inappropriate mainly because it may a priori imply, first, that the
  sympathetic nervous system is causally involved and, second, that the clinical
  phenomenology is the result of a reflex activation of sympathetic neurons. However, in
  many patients there is no proof that the sympathetic activity is elevated and there are
  indications that an indistinguishable clinical picture (including the pain!) can be
generated by different mechanisms.”(Pages 5-6)

Reference information:
- Reflex Sympathetic Dystrophy: A Reappraisal
  o Year of publication: 1996
  o Editors: Janig W and Stanton-Hicks M
  o Publisher: International Association for the Study of Pain

In 1998, the results of a scientific investigation were published in The Clinical Journal of
Pain, which explained that the unreliability of the RSD construct was another reason for its
removal from diagnostic taxonomy. The publication highlighted the unreliability of the RSD
construct by specifying that reasons for its discontinuation included “the lack of international
agreement as to the definition and diagnostic criteria of RSD” (page 49).

Reference information:
- IASP diagnostic criteria for complex regional pain syndrome: a
  preliminary empirical validation study. The Clinical Journal of Pain,
  14:48-54.
  o Year of publication: 1998
  o Authors: Galer BS, et al.

In 2001, the International Association for the Study of Pain published a book which similarly
explained that reasons for the discontinuation of the RSD construct had included the
Notes:

unreliability of that construct. The book also emphasized the manner in which the problematic aspects of the RSD construct had actually prevented scientific progress. For example, that book explained:

- “Lack of consensus regarding standardized diagnostic criteria for RSD resulted in serious problems with the comparability of patient samples across various studies addressing diagnosis and treatment of the disorder. The resulting lack of generalizability of results across studies made progress more difficult in identifying optimal treatments and treatment sequences for these patients.”

- By discarding the RSD construct, “we have discarded old ideas that were impeding progress”.

Reference information:

- Complex Regional Pain Syndrome
  - Year of publication: 2001
  - Editors: Harden RN, Baron R, and Janig W
  - Publisher: International Association for the Study of Pain

In 2001, the Third Edition of a prominent book entitled Bonica’s Management of Pain was published. The first edition of this book had suggested the utilization of the RSD construct for a wide variety of older diagnostic constructs, because of a belief “that the major underlying etiology of RSD was a disturbance in the sympathetic nervous system” (this passage, and a discussion of the history described above, can be found on page 388 of the Third Edition).

This Third Edition was the first edition of that book to be published since the removal of the RSD construct from diagnostic taxonomy. The book provides a relatively thorough discussion of the reasons why the RSD construct needed to be discontinued (see page 389). Key points from that discussion include:

- “confusion continued because no single diagnostic criterion had been widely accepted by clinicians and researchers worldwide. Hence, patients evaluated by one physician or researcher diagnosed with RSD may or may not have obtained the same diagnosis from another evaluating physician. This lack of consensus and the confusion with regard to RSD and causalgia diagnostic criteria have retarded clinical research.”

- “little agreement existed among those from different medical fields and among those from different parts of the world as to the diagnostic criteria and appropriate therapies for RSD”

- “In 1993, a consensus group of pain medicine experts (a special consensus workshop of the International Association for the Study of Pain) gathered with the defined task of reevaluating the clinical syndromes of RSD and causalgia. It was agreed by this group that these disorders were characterized by controversy and confusion with regard to diagnostic criteria, pathophysiologic mechanisms, and effective therapies. Therefore, this international consensus group agreed to dismantle the terms RSD and causalgia, to admit the field’s ignorance”.

Reference information:
Also in 2001, the American Academy of Neurology produced a publication, and formal continuing medical education program, which highlighted reasons why the RSD construct needed to be discontinued. Key points from that project include:

- “Objectives” of this publication included: “To review several taxonomic terms, such as “reflex sympathetic dystrophy”, “sympathetically maintained pain”, and “complex regional pain syndrome”, and understand them as purely descriptive, devoid of evidential medical power, and evasive of the refutability principle.” (page 91).

- On page 92, reflex sympathetic dystrophy is characterized as a “mythical diagnostic term” (NOTE: the same characterization is applied to complex regional pain syndrome).

- In regard to this characterization of “RSD” as a mythical diagnostic term, page 94 explains further: “When testable diagnostic hypotheses are ruled out, and the treating doctor does not understand the case, a mythical diagnosis is entertained. Mythical diagnoses are characterized by a wishful descriptive term and a hypothetical underlying mechanism that cannot be tested. Therefore, the hypothesis cannot be validated, but neither can it be ruled out. The "diagnosis" therefore becomes permanent and condemns a patient to chronic illness behavior and iatrogenesis.”

- Page 102 specifies that, “in the 90s, the very concept of RSD was eliminated from medical taxonomy by peer review.”

- On page 103, “RSD” is specified as a “meaningless diagnostic category” that was debunked.

Reference information:

  - Year of publication: 2001
  - Chairperson: Biller J.
  - Publisher: American Academy of Neurology

In 2005, the International Association for the Study of Pain published a book that reviewed the “changes brought about by the diagnostic reclassification and redefinition” that resulted in the RSD construct being removed from diagnostic taxonomy. This text provided considerations such as the following in regard to why the RSD construct was discontinued:
Notes:

- “What is reflex sympathetic dystrophy if it isn’t reflex and it isn’t sympathetic and it isn’t dystrophy?” (page 15) NOTE: This passage highlights the comprehensive nature of the failures of scientific efforts to validate the RSD construct—every element of the construct was a failure (e.g., reflex, sympathetic, and dystrophy).

- “the diagnosis of RSD generally continues to be made by gestalt, rather than by the application of consistent criteria that can be quantified” (page 20) NOTE: This passage highlights several issues of relevance to “Daubert” legal principles—e.g., the “gestalt” basis of RSD diagnosis—as opposed to a standardized and validated method; the lack of consistent/reliable methodology; the lack of objective quantification of diagnostically relevant information.

- Exceptionally thorough scientific research which was published shortly prior to the formal discontinuation of the RSD construct produced results which indicated (see pages 22-23):
  - “differentiation of RSD from other clinical conditions was difficult”
  - “the pathogenesis of RSD was still speculative”
  - “the authors believed that their data... could not support any sympathetic neural involvement”.

- “The name of reflex sympathetic dystrophy... is problematic. If there is a reflex involved... it is complicated, multisynaptic, and not fully characterized. The sympathetic autonomic changes may be an epiphenomenon and may not be causative or perpetuating, and true dystrophy occurs in only about 10% of cases.” (page 45) Note: This passage highlights the comprehensive nature of the scientific failures for the construct of reflex sympathetic dystrophy.

Reference information:

- CRPS: Current Diagnosis and Therapy
  - Year of publication: 2005
  - Editors: Wilson PR, Stanton-Hicks M, and Harden RN
  - Publisher: International Association for the Study of Pain

For the 2007 American Academy of Orthopaedic Surgeons’ formal continuing medical education curriculum focused on “Occupational Orthopaedics and Workers’ Compensation”, I was asked by the Medical Director of the American Academy of Orthopaedic Surgeons (Robert Haralson) to create a nearly comprehensive discussion of the differential diagnostic process for complex regional pain syndrome. The resulting program was accepted into the curriculum of the American Academy of Orthopaedic Surgeons, and published in the Academy’s course book, after passing the Academy’s review process. That publication includes the following passage which documents the history of the RSD construct having been discontinued, and reasons why it had been discontinued (see page 406):
"The concept of "complex regional pain syndrome" was created in 1994 specifically for the purpose of replacing "reflex sympathetic dystrophy" and "causalgia". Professional literature has clarified that this change was precipitated by the shortcomings of the concept of "reflex sympathetic dystrophy". Stanton-Hicks explained that: "The term RSD has lost usefulness as a clinical designation because it has been used so indiscriminately that it is no longer clear what it means". The misleading nature of "RSD" was highlighted by Landau: "That reflex sympathetic dystrophy is difficult to prove to be reflex, sympathetic, or dystrophy is increasingly evident". The International Association for the Study of Pain (IASP) has similarly documented the inappropriateness of the sympathetic and dystrophic implications of "RSD". The elimination of "RSD" has been formally endorsed in publications from medical associations such as the American Academy of Disability Evaluating Physicians. Similarly, "RSD" has been singled out by a state government as being unacceptable within workers' compensation."

Reference information:

- Differential Diagnosis for Complex Regional Pain Syndrome, Chapter 15 in: Melhorn JM and Shields NN. *9th Annual Occupational Orthopaedics and Workers Compensation: A Multidisciplinary Perspective*
  - Year of publication: 2007
  - Authors: Barth RJ and Haralson R
  - Publisher: American Academy of Orthopaedic Surgeons

When editors for the American Medical Association found out about the American Academy of Orthopaedic Surgeons curriculum/publication that was discussed in the preceding paragraph, they asked me to create a publication on the same subject, specifically for publication by the American Medical Association and for referencing in the AMA’s *Guides to the Evaluation of Permanent Impairment, Sixth Edition* (which was being created at that time). That project was published by the American Medical Association (after passing that Association’s unusually extensive review process), and is referenced twice in the *Guides to the Evaluation of Permanent Impairment, Sixth Edition*. That publication reiterates the information from the above (immediately preceding) paragraph, and adds: “Subsequently, any utilization of the concept of “RSD” would be misleading, meaningless, and would involve a departure from modern professional standards.”

Reference information:

- Differential Diagnosis for Complex Regional Pain Syndrome, *AMA Guides Newsletter*, pages 1-16.
  - Year of publication: 2007
  - Authors: Barth RJ and Haralson R
  - Publisher: American Medical Association

In 2008, the American Academy of Neurology produced a formal continuing medical education program which highlighted reasons why the RSD construct had been discontinued. Text from the curriculum material included the following passage:
Notes:

• From page 2AC.004-76: “Reflex Sympathetic Dystrophy: This vintage, pathophysiologically assuming label has been eliminated from the medical taxonomy. This became inevitable when it was shown, one decade ago, that self reported symptom relief, following what used to be assessed as "gold standard" diagnostic sympathetic blocks, amounted to an embarrassing placebo artifact. Thus, "sympathetically maintained pain," loosely defined as "any pain that is relieved by sympathetic blocks" went down the drain and carried RSD with it.”

Reference information:

• Title: PSEUDONEUROPATHY: CONVERSION VERSUS MALINGERING
  o Year: 2008
  o Faculty: Ochoa J.
  o Program: American Academy of Neurology 60th Annual Meeting

In 2009, editors for the American Medical Association asked me to create a summary of the entire history of the American Medical Association’s Guides Library discussions of complex regional pain syndrome (the modern construct that replaced reflex sympathetic dystrophy, and which was created for other purposes as well). After passing the American Medical Association’s unusually extensive review process, the project was published directly by the American Medical Association. The resulting publication documented all of the following (see page 2 of the publication):

• The RSD construct was formally terminated from diagnostic taxonomy as of 1994.
• Reasons for that formal termination included “the complete scientific failure of the concept of RSD”.

Reference information:

• A Historical Review of CRPS in The American Medical Association’s Guides Library. *The AMA Guides Newsletter*
  o Year of publication: 2009
  o Author: Barth RJ
  o Publisher: American Medical Association

For two years after the publication of the American Medical Association project referenced immediately above, I created and taught a formal continuing education program, based upon that publication, for the American Academy of Orthopaedic Surgeons. The curriculum material was accepted into the American Academy of Orthopedic Surgeons’ programming, and published in the Academy’s course books, after passing that Academy’s review process. The resulting continuing medical education programming and publications from the American Academy of Orthopedic Surgeons reiterated issues that have been listed above (see page 736 of the AAOS text referenced below), and also included the following passages (see page 756 of the AAOS text referenced below):

• “Reflex Sympathetic Dystrophy”
III.C. Complex regional pain syndrome is not synonymous with (not the same thing as) reflex sympathetic dystrophy

I have witnessed doctors and other making erroneous claims that complex regional pain syndrome and reflex sympathetic dystrophy are synonymous, or, the same thing. Such false claims might be based on the history of the RSD construct having been replaced by a construct which is called complex regional pain syndrome type one (Mersky and Bogduk 1994; Stanton-Hicks et al. 1995).

First, it must be noted that only type one of the complex regional pain syndrome construct was created to replace RSD. Therefore, it is not true to claim that RSD was replaced by CRPS in general (type two and the NOS type were not created for purposes of replacing RSD). (Mersky and Bogduk 1994 and 2012; Stanton-Hicks et al. 1995).

Secondly, and more specifically in regard to complex regional pain syndrome type one, the fact that RSD and complex regional pain syndrome type one do not mean the same thing is actually illustrated by the history of complex regional pain syndrome type one having been created for the specific purpose of replacing the failed concept of RSD. If these two constructs actually meant the same thing, then there would not have been any need for complex regional pain syndrome type one to have been created, and there would not have been any need for RSD to have been replaced by complex regional pain syndrome type one. The following discussion of relevant publications (and the history that they document) illustrates that the construct of complex regional pain syndrome type one was deliberately created in a fashion that causes it to be very different from the construct of RSD (so that it would not be vulnerable to the scientific failures that resulted in the discontinuation of the RSD construct).
This issue has been addressed in previous AAOS programming, in a highly summarized fashion. For example, in 2011, I created a continuing medical education program for AAOS which addressed the fact that RSD and complex regional pain syndrome type one are not synonymous. The following passage from that curriculum and associated course book summarizes the fact that RSD is not synonymous with complex regional pain syndrome type one (see page 756 of the source material):

- “Reflex Sympathetic Dystrophy
  - A complete scientific failure.
  - Formally discontinued by 1994.
  - Replaced by CRPS type 1
  - NOT synonymous with CRPS-1
    (CRPS-1 was intentionally created in a fashion that is drastically different from RSD, in order to avoid the scientific failures of RSD)”

Reference information:

  - Year of publication: 2011.
  - Author: Barth RJ
  - Publisher: American Academy of Orthopaedic Surgeons

Detailed examples of facts which clarify that RSD and complex regional pain syndrome type one are not synonymous can be highlighted by a simple comparison of texts from the time when RSD was a viable diagnostic issue, to modern versions of the same texts.

The following lists provide such a comparison of texts. The first list is based on relevant editions of the International Association for the Study of Pain’s Classification of Chronic Pain. The information regarding the RSD construct is from the first edition of that text (from 1986) (Merskey 1986). This was the only edition of that Classification that was published prior to the formal discontinuation of the RSD construct. Consequently, it is the only edition of that Classification which provides a discussion of the RSD construct. The information regarding the complex regional pain syndrome type one construct is from the current edition (Merskey and Bogduk 2012), which was written long after the formal discontinuation of the RSD construct (and which consequently discusses complex regional pain syndrome type one, the construct which replaced the RSD construct). This Classification is an especially prominent text for this subject matter, because the 1994 edition was the formalization of the discontinuation of the RSD construct, and the formal introduction for the complex regional pain syndrome construct (Merskey & Bogduk 1994).
Notes:

These editions of the *Classification* are only being offered as evidence that RSD and complex regional pain syndrome type one are not the same thing, or synonymous. These texts cannot be relied upon for all other potential issues.

- **"Definition":**
  - The definition of RSD specified “pain in a portion of an extremity”
    - In contrast, the definition of complex regional pain syndrome type one makes no mention of “an extremity”.
  - The definition of RSD specified “associated with sympathetic hyperactivity”.
    - In contrast, the definition of complex regional pain syndrome type one makes no mention of “sympathetic hyperactivity”.
  - The definition of RSD did not involve any mention of the disproportionality that is definitional for complex regional pain syndrome type one.
    - In contrast, the definition of complex regional pain syndrome type one specifies “pain that is seemingly disproportionate in time or degree to the usual course of pain after trauma or other lesion”
  - The definition of RSD did not make any mention of the lack of association with a specific nerve territory/dermatome that is definitional for complex regional pain syndrome type one.
    - In contrast, the definition of complex regional pain syndrome type one specifies “not in a specific nerve territory or dermatome”.

- **"Site":**
  - The site of RSD was specified as “adjacent to a traumatized area”.
    - In contrast, the discussion of the site of complex regional pain syndrome type one makes no mention of “a traumatized area”.

- **"System":**
  - The system discussion for RSD specified that the central nervous system was only “possibly” involved.
    - In contrast, the system discussion for complex regional pain syndrome type one specifies the involvement of the central nervous system.
  - The system discussion for RSD did not involve any mention of involvement of the musculoskeletal system.
    - In contrast, the system discussion for complex regional pain syndrome type one specifies the involvement of the musculoskeletal system.

- **"Main features":**
For RSD, visceral disease or central neurological disease were not listed among the main features of the construct. In contrast, the “main features” discussion for complex regional pain syndrome type one specifies “may be related to visceral disease, e.g. angina or central neurological disease such as stroke”.

For RSD, fluctuation over time in the intensity of the pain was not listed among the main features of the construct. In contrast, the “main features” discussion for complex regional pain syndrome type one specifies “the intensity of pain may fluctuate over time”.

For RSD, allodynia was not listed among the main features of the construct. In fact, the entire discussion of RSD failed to mention allodynia. In contrast, the “main features” discussion for complex regional pain syndrome type one specifies that allodynia is a main feature that “may” manifest.

For RSD, hyperalgesia was not listed among the main features of the construct. In fact, the entire discussion of RSD failed to mention hyperalgesia. In contrast, the “main features” discussion for complex regional pain syndrome type one specifies that hyperalgesia is a main feature that “may” manifest.

For RSD, edema or oedema was not listed among the main features of the construct. The entire discussion of RSD only mentioned “oedema” as an “associated symptom” which “initially” manifests. In contrast, the “main features” discussion for complex regional pain syndrome type one specifies that “edema is usually present” (rather than being an associated symptom that is only present “initially”, as had been the case for the RSD construct).

For RSD, hyperhidrosis/excessive sweating/increased sweating was not listed among the main features of the construct. The entire discussion of RSD only mentioned “hyperhidrosis” (excessive sweating) as an “associated symptom”. In contrast, for complex regional pain syndrome type one, “increased sweating” is specified as a “main feature” which may appear.

For RSD, decreased sweating was not listed among the main features of the construct. In fact, the entire discussion of RSD failed to mention decreased sweating. In contrast, for complex regional pain syndrome type one, “decreased sweating” is specified as a “main feature” which may appear.
For RSD, impairment of motor functioning was not listed among the main features of the construct. In fact, the entire discussion of RSD failed to mention “impairment of motor functioning”.

- In contrast, for complex regional pain syndrome type one, “impairment in motor functioning” is specified as a “main feature” which is “frequently seen”.

For RSD, spreading to other parts of the body was not listed among the main features of the construct.

- In contrast, for complex regional pain syndrome type one, spreading is specified as a “main feature” which “may” occur.

For RSD, the entire discussion of RSD only mentioned potential spreading as a possibility that is limited to the “contralateral limb”.

- In contrast, for complex regional pain syndrome type one, spreading is specified as a “main feature” which “may” occur in a proximal fashion (a very different direction of spread than the “contralateral limb” spread that is specified for RSD), “or, rarely, spread to involve other extremities” (rather than being limited to the “contralateral limb”, as was the case for RSD).

- “Associated symptoms” / “Signs” / “Associated symptoms and signs”;

Note: This section has a hybrid title because “Associated symptoms” and “Signs” were separate discussions for RSD (in that edition of the Classification), but have been combined into a single discussion for complex regional pain syndrome type one (in the modern edition of the Classification).

For RSD, “aggravated by use of body part” was specified as an associated symptom.

- In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

For RSD, “relieved by immobilization” was specified as an associated symptom.

- In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text. Instead, immobilization is specified as an issue which is followed by complex regional pain syndrome – this indicates that immobilization is a potential source of the problem (rather than a source of relief from the problem, as was specified for RSD).
Notes:

- For RSD, “sometimes follows a herniated intervertebral disc” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

- For RSD, “sometimes follows …spinal anaesthesia” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

- For RSD, “sometimes follows …poliomyelitis” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

- For RSD, “sometimes follows …severe iliofemoral thrombosis” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom. For the entire text, thrombosis is only mentioned as a differential diagnostic issue (an issue that would actually exclude complex regional pain syndrome from diagnostic consideration, rather than being an associated symptom as was specified for RSD).

- For RSD, “sometimes follows …cardiac infarction” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

- For RSD, “this may appear as the shoulder-hand syndrome” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom, or anywhere else within the text.

- For RSD, “Raynaud’s phenomenon” was specified as an associated symptom.
  - In contrast, for complex regional pain syndrome type one, this issue is not listed as an associated symptom. Instead, Raynaud’s disease is specified as a differential diagnostic issue. This means that it is actually an issue which excludes complex regional pain syndrome from diagnostic consideration (instead of being an associated feature, as Raynaud’s phenomenon was specified for RSD).
Notes:

- For RSD, “guarding of the affected part” was not mentioned as an associated symptom, a sign, or anywhere else in the text.
  - In contrast, for complex regional pain syndrome type one, this issue is specified under “Associated Symptoms and Signs”, and is specified as “usually observed”.

- “Laboratory findings”:
  - For RSD, the discussion of laboratory findings was limited to “roentgenograms”, which were only of relevance to “advanced cases”.
    - In contrast, for complex regional pain syndrome type one, the list of laboratory findings includes “noncontact skin temperature measurement”, “measurements of skin blood flow”, “testing of sudomotor function”, “three-phase bone scan”, and “radiographic examination”. There is no mention of “advanced cases” (the context to which the discussion of laboratory findings for RSD was limited).

- “Usual Course”:
  - For RSD, the entire text for the “Usual Course” discussion read: “Persists indefinitely if untreated; small incidence of spontaneous remission.”
    - In contrast, for complex regional pain syndrome type one, the entire text for the “Usual Course” discussion reads: “Variable.”

- “Relief”:
  - For RSD, the discussion of “relief” does not include any mention of the “comprehensive and interdisciplinary approach” that is emphasized for complex regional pain syndrome. Instead, the discussion is limited to sympathetic block, physical therapy, sympathectomy, and corticosteroids.
    - In contrast, for complex regional pain syndrome type one, the text specifies: “most authorities recommend a comprehensive and interdisciplinary approach”. The discussion does not include any specific mention of the items that were specified in the RSD discussion (e.g. sympathetic block, physical therapy, sympathectomy, or corticosteroids).

- “Complications”:
  - For RSD, phlebitis was not listed as a complication (or otherwise mentioned anywhere within the text).
Notes:

- In contrast, for complex regional pain syndrome type one, phlebitis is specified as a complication.

  - For RSD, cellulitis was not listed as a complication (or otherwise mentioned anywhere within the text).
    - In contrast, for complex regional pain syndrome type one, cellulitis is specified as a complication.

  - For RSD, “disuse atrophy” was listed as a complication.
    - In contrast, for complex regional pain syndrome type one, “atrophy” is specified as a complication, but there is no mention of any relationship between such atrophy and “disuse” (either in the discussion of complications or any other portion of the text).

  - For RSD, the discussion of such “disuse atrophy” as a complication was not accompanied by any discussion of duration or prognosis. (NOTE: disuse atrophy is usually reversible via discontinuation of the disuse).
    - In contrast, for complex regional pain syndrome type one, “trophic changes” are specified as being a permanent complication which “can be seen”.

  - For RSD, weakness was not listed as a complication (or otherwise mentioned anywhere within the text).
    - In contrast, for complex regional pain syndrome type one, weakness is specified as a complication.

  - For RSD, suicide was listed as a complication that was limited to cases which were “untreated”.
    - In contrast, for complex regional pain syndrome type one, there is no limitation (such as the RSD limitation to untreated cases) for suicide as a complication.

  - For RSD, “drug abuse” was listed as a complication that was limited to cases which were “untreated”.
    - In contrast, for complex regional pain syndrome type one, there is no limitation (such as the RSD limitation to untreated cases) for “inappropriate drug use” as a complication.

  - For RSD, “permanent functional disability” is not mentioned as a complication, and the discussion of disability in another portion of the text makes no mention of duration or prognosis (e.g. permanence).
Notes:

- In contrast, for complex regional pain syndrome type one, “permanent functional disability” is specified as being a complication which “can be seen”.

- “Social and Physical Disability”/ “Social and Physical Impairment”:

  Note: This section has a hybrid title because the first phrase was used for RSD (in the corresponding edition of the Classification), but the second phrase was used for complex regional pain syndrome type one (in the modern edition of the Classification). 

  o For RSD, “inability to perform...occupational and recreational activities” was not listed as an issue for this section (or otherwise mentioned anywhere within the text).
  
  o In contrast, for complex regional pain syndrome type one, “inability to perform...occupational and recreational activities” is specified for this section.

- “Pathology”:

  o For RSD, the entire text of this section simply says “Unknown.” The “abnormal inflammatory responses” that are “likely” for complex regional pain syndrome type one are not mentioned in this section or anywhere else in the text.
  
  o In contrast, for complex regional pain syndrome type one, the pathology is specified as “Unknown”, but it is also specified that “Abnormal inflammatory responses are likely to play a role”.

- “Essential features”:

  o For RSD, “Burning pain in distal extremity usually after minor injury without nerve damage” is specified as “essential features”.
  
  o In contrast, for complex regional pain syndrome type one, there are no “essential features”.

- “Differential diagnosis”:

  o For RSD, none of the issues listed in the following bullet point regarding differential diagnosis for complex regional pain syndrome type one are listed.
  
  o In contrast, for complex regional pain syndrome type one, all of the following are specified as differential diagnostic issues: regional vascular disease, cellulitis, other regional infection, Raynaud’s disease, specified neuropathy, erythromelalgia, specified regional motor disease, regional autoimmune process.
Another book (Bonica’s Management of Pain) similarly has multiple editions that allow for such independent verification that the RSD construct is not the same thing as, or synonymous with, complex regional pain syndrome type one (e.g. editions which preceded the formal discontinuation of the RSD construct provide information regarding that construct, which can then be contrasted against the modern edition which provides information regarding the complex regional pain syndrome type one construct). This is an especially prominent book for this subject matter, because the first edition of this book has been specified as the trigger for the widespread adoption of the RSD construct in the 1950’s (Bonica et al. 1990).

These two editions of this book are only being offered as evidence that RSD and complex regional pain syndrome type one are not the same thing, or synonymous. These editions cannot be relied upon for all other potential issues.

For RSD, the following comparison is based on the last edition of the book to be published before the formal discontinuation of the RSD construct (the Second Edition; Bonica et al 1990). For complex regional pain syndrome type one, the following comparison is based on the current edition (Fishman et al. 2010). Many of the differences between RSD and complex regional pain syndrome type one which were listed above are again highlighted by a comparison of the relevant editions of Bonica’s Management of Pain. For the sake of simplicity, I am avoiding another detailed listing of such differences. This discussion of Bonica’s Management of Pain will simply list some issues that were not mentioned above, such as:

- “Reflex sympathetic dystrophy is an all inclusive term applied to a great variety of seemingly unrelated disorders” (see page 230 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - In contrast, complex regional pain syndrome is characterized as a single syndrome (e.g., “a distinct syndrome”, a “disease”), rather than being “an all inclusive term applied to a great variety of seemingly unrelated disorders” (for example, see page 314 of the modern edition, the Fourth Edition, from 2010).

- The RSD construct involved a course over time that included three distinct stages (see pages 232-233 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - In contrast, the complex regional pain syndrome construct does not involve such sequential stages (see page 320 of the modern edition, the Fourth Edition, from 2010).

- For the RSD construct, “sympathetic interruption early in the course of this syndrome provides effective relief of pain and disappearance of the rest of the symptoms”; “sympathetic interruption produces prompt relief of pain and disappearance of pathophysiology”; “prompt recognition and effective treatment result in rapid remission
Notes:

- of symptoms and complete recovery”; “can be cured if recognized and properly treated” (see pages 220, 230, and 241 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - In contrast, the discussion of complex regional pain syndrome does not involve any such promise of cure by “sympathetic interruption” (for example, see page 325 of the modern edition, the Fourth Edition, from 2010).

- For the RSD construct, diagnostic criteria included “history of recent or remote accidental or iatrogenic trauma or disease” (see page 234 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - No such diagnostic criterion is included in the modern conceptualization of complex regional pain syndrome type one (for example, see page 319 of the modern edition, the Fourth Edition, from 2010).

- For the RSD construct, diagnostic criteria included “pain that is burning, aching, and/or throbbing in character” (see page 234 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - No such diagnostic criterion is included in the modern conceptualization of complex regional pain syndrome type one (for example, see page 319 of the modern edition, the Fourth Edition, from 2010).

- For the RSD construct, diagnostic criteria included “relief of pain and modification of signs after regional sympathetic blockade”. NOTE: The text states further that this criterion “is considered by most writers on the subject as one of the most important diagnostic features of reflex sympathetic dystrophy” (see page 234 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - No such diagnostic criterion is included in the modern conceptualization of complex regional pain syndrome type one (for example, see page 319 of the modern edition, the Fourth Edition, from 2010).

- For the RSD construct, indexing for transcutaneous nerve stimulation leads to text which specifies that such treatment has been “reported as effective” (see pages 1xxi and 235 of the Second Edition of this text, from 1990 – prior to the formal discontinuation of the RSD construct).
  - No such support for that treatment is indexed in regard to complex regional pain syndrome (see page 1658 of the modern edition, the Fourth Edition, from 2010).

The above discussion has already identified the definitional role of the sympathetic nervous system for RSD (and lack thereof for complex regional pain syndrome), and the diagnostic role of sympathetic block for RSD (and lack thereof for complex regional pain syndrome) as examples of independent verification that the RSD and complex regional pain syndrome...
Notes:

constructs are not the same thing, or synonymous. These differences between the two constructs are reiterated by a variety of other publications from the International Association for the Study of Pain and the American Medical Association, including all of the following:

- “The most important difference between CRPS (complex regional pain syndrome) and earlier views of RSD is that sympathetic dysfunction is not assumed to be the basic pathophysiologic mechanism.”
  Reference information:
  - *The Guides Casebook*
    - See page 15 for the specific passage that is quoted above.
    - Year of publication: 1999
    - Editor: Brigham CR
    - Publisher: American Medical Association

- “while a positive response to sympatholysis was previously necessary to satisfy a diagnosis of RSD, it has no place in the current criteria” (for CRPS).
  Reference information:
  - *Complex regional pain syndrome*
    - See page 293 for the specific passage that is quoted above.
    - Year of publication: 2001
    - Editors: Harden RN, Baron R, and Janig W
    - Publisher: International Association for the Study of Pain

- “regional sympathetic blockade has no role in the diagnosis of CRPS” (complex regional pain syndrome)
  Reference information:
  - *Guides to the Evaluation of Permanent Impairment, Fifth Edition*
    - See page 495 for the specific passage that is quoted above.
    - Year of publication: 2001
    - Editors: Cocchiarella L and Andersson GBJ
    - Publisher: American Medical Association

IV. A fact-based definition of complex regional pain syndrome, created with a focus on the pervasiveness of legal claims in cases which involve this diagnosis

This subsection also provides an organized summary of the information that was provided in previous sections of this chapter, and other information from the history of the complex regional pain syndrome construct.

The definitional information which is provided below is fact-based, and referenced. This distinguishes it from many other publications which supposedly provide definitions of complex regional pain syndrome (e.g. Merskey & Bogduk 1994 and 2012; Stanton-Hicks et.al.
Notes:

1995; Fishman et al. 2010), in that those other publications appear to be based on what someone thinks complex regional pain syndrome should mean, rather than being based on the actual characteristics of the construct, and rather than describing what can be expected of a patient when he/she comes into a doctor’s office with a complex regional pain syndrome diagnosis already in place.

This definitional discussion of complex regional pain syndrome was created specifically with legal contexts in mind, because of two primary considerations: 1) This text is being prepared for an AAOS course which is specifically focused on the legal intrusions that are inherent to workers compensation and other cases which involve legal claims; 2) The construct of complex regional pain syndrome is almost inseparable from legal claims (referencing provided below – see point #14) – consequently, a thorough discussion of complex regional pain syndrome necessitates careful consideration of the pervasive legal context of cases which involve this construct.

Early in a process in which a doctor is asked to interact with the legal system, that doctor will typically be asked (by lawyers) to provide definitions. This subsection is intended to provide doctors with a handy reference that they can offer in response to such requests. Doctors should consider avoiding pretending that they can adequately serve as dictionaries (by immediately providing a definition verbally, based on nothing other than the doctors’ memory), and should consider instead referring the requesting attorney to this document.

Definitional aspects of complex regional pain syndrome include:

1. Complex regional pain syndrome is an unreliable construct (e.g., Borchers & Gershwin 2014) that was created by a “special consensus conference” which was reportedly organized by two people in 1993 (Stanton-Hicks et al 1995).

   NOTE: At least one prominent, but late-published, report (from people who reportedly were not actually involved in the conference) claims that this 1993 conference was an activity of the International Association for the Study of Pain (IASP) (Galer et al., 2001). However, I have not found any confirmation of that report in earlier discussions of the conference that were published directly by the IASP, and which are attributed to people who were reportedly involved in the conference (e.g. Janig 1996; Stanton-Hicks 1995), in spite of reading all of the relevant publications that I can find.

2. This concept was intentionally created in a fashion which the IASP’s publications characterize as “purely descriptive” (e.g., Janig 1996) (rather than being explanatory, etiological, definitional, etc.).

3. This concept was initially formalized in the 1994 edition of the IASP’s
Notes:

Classification of Chronic Pain (Merskey and Bogduk 1994).

NOTE: That initial formalization substantially strays from the later published reports of the results of the 1993 conference (e.g., whether a history of a “noxious event” is necessary for purposes of making such a diagnosis) (Stanton-Hicks et al. 1995). This initial discrepancy highlights the unreliability which has plagued the complex regional pain syndrome construct from its beginning.

NOTE: The details of the IASP’s initial formalization is not being included in this list of definitional information, because that initial formalization was discontinued in 2012 (Merskey & Bogduk 2012), due to its demonstrated tendency to mistakenly apply the complex regional pain syndrome construct to clinical presentations for which the construct was known to be irrelevant (e.g., Galer 1998; Harden & Bruehl 2005).

4. Complex regional pain syndrome is a pervasively non-scientific, even anti-scientific, construct.

NOTES:

- The primary published report regarding the creation of the construct specifies that the associated efforts involved “acknowledging a lack of scientific understanding” (Stanton-Hicks et al 1995).

- In fact, an imperviousness to scientific advancement was reportedly built into the construct. For example, the primary published report regarding the creation of the construct indicates that the construct was intentionally created in a fashion that was so ambiguous that it would be able to “stand despite any scientific findings” (Stanton-Hicks et al 1995).

- Readers should note that the immediately preceding bullet point indicates that the creators of the construct were deliberately taking an anti-scientific approach, by creating a construct that could not be scientifically scrutinized, tested, etc. As is explained in the Foster & Huber text that is fully referenced at the end of this chapter, science is ideally a process of attempting to disprove concepts. This is an aspect of science that has been especially strongly embraced by American legal systems (Foster & Huber; Sinclair). Consequently, when this construct was intentionally created in a fashion that does not allow for
scientific scrutiny/testing, so that it could withstand any scientific efforts, discoveries, etc., such creation was anti-scientific. This aspect of the construct has previously been emphasized in literature from the American Academy of Neurology (Biller), which noted that complex regional pain syndrome is “evasive of the refutability principle”.

- This anti-scientific history would justify a conclusion that the complex regional pain syndrome construct is not a matter of health science, and a conclusion that it is instead (borrowing words from the US Supreme Court, as reported in the Foster & Huber text that is fully referenced at the end of this chapter) simply a matter of “subjective belief or unsupported speculation”.

- As is discussed further below, the construct was actually created due to scientific failures for the previous construct of reflex sympathetic dystrophy, rather than having been created due to any scientific findings which were supportive of the concept of complex regional pain syndrome.

5. A sub-construct of complex regional pain syndrome, referred to as type one, was created (by the 1993 “special consensus conference”) for the purpose of replacing the failed concept of reflex sympathetic dystrophy (Merskey & Bogduk 1994; Stanton-Hicks et al. 1995).

NOTES:

- By the time that the 1993 “special consensus conference” took place, the scientific failures of the reflex sympathetic dystrophy concept were so comprehensive, that it had become apparent that the concept needed to be abandoned (removed from diagnostic taxonomy) (e.g., Biller et al. 2001; Janig 1991 & 1996; Merskey & Bogduk 1994; Stanton-Hicks et al 1995).

- The 1993 “special consensus conference” apparently decided that, instead of simply allowing the failed concept of reflex sympathetic dystrophy to be terminated in the absence of any replacement, they wanted to create a new concept to replace reflex sympathetic dystrophy (Stanton-Hicks et al 1995). I have not found an adequate justification for that creation a new construct to replace the failed reflex sympathetic dystrophy.
construct, in spite of reading everything I can find on the subject over the past 20 years.

6. The 1993 "special consensus conference" intended the complex regional pain syndrome construct to be an ambiguous "umbrella term", rather than to represent a reliable entity (Stanton-Hicks et al. 1995; Janig 1996).

7. As part of the development of that ambiguous "umbrella term", the construct of complex regional pain syndrome was applied, by the 1993 "special consensus conference" (Stanton-Hicks et al 1995), and subsequently by the IASP (Merskey & Bogduk 1994) to the historical concept of causalgia (as well as reflex sympathetic dystrophy). Specifically, a sub-construct of complex regional pain syndrome, referred to as type two, was created reportedly for the purpose of replacing causalgia.

NOTES:
- I have never found an adequate justification for that application of the ambiguous umbrella construct to causalgia, in spite of reading everything I can find on the subject over the past 20 years.
- This issue increased the unreliability of the complex regional pain syndrome construct. For example, causalgia was not actually removed from the IASP’s Classification of Chronic Pain (Merskey & Bogduk 1994 & 2012). In other words, complex regional pain syndrome type two was reportedly created to replace the causalgia concept, but the causalgia concept was not actually replaced. I have never found an explanation for the IASP’s self-contradiction in this regard, in spite of reading everything that I can find on the subject over the past 20 years.

8. The complex regional pain syndrome construct was deliberately created in a fashion that is extremely ambiguous, so that it would not be subject to the same types of scientific failures that doomed the concept of reflex sympathetic dystrophy.

NOTE: The intentional nature of this planned ambiguity has been specified in the primary published report regarding the creation of the construct, which noted that the associated efforts involved a focus on creating sufficient ambiguity to ensure that the construct would "stand despite any scientific findings" (Stanton-Hicks et al 1995).

9. In 2012, the IASP changed its conceptualization of complex regional pain syndrome. One of the many changes involved the introduction of a third sub-construct, referred to as complex regional pain syndrome not otherwise specified (Merskey &
Notes:

Bogduk 2012). The 2012 revision of the IASP’s *Classification of Chronic Pain* states that this sub-construct was created for clinical presentations which only partially match up to IASP diagnostic requirements for complex regional pain syndrome type one or type two. Consequently, there is no longer any expectation that two clinical presentations which are both labeled with the complex regional pain syndrome construct will have anything in common (e.g., one can involve pain in the absence of swelling or any other characteristics of the construct, the other can involve only swelling in the absence of any pain or any other characteristics of the construct). This development drastically increased the unreliability and ambiguity that has plagued the construct since its creation.

10. The construct is devoid of any pathophysiological considerations.

NOTES:

- This is clearly specified in the original formalization (Merskey & Bogduk 1994), in that the “physiology” is stated as “unknown” for both type one and type two.

- Similarly, the 2012 revision of that conceptualization (Merskey & Bogduk 2012) states: “Pathology / Unknown. In CRPS II, the pain syndrome follows a major nerve injury, but that does not explain its pathological basis.”

11. The construct of complex regional pain syndrome has not been reliable from one published source of information to another, and even over time in regard to single sources of information.

NOTES:

- The IASP has provided multiple conceptualizations over time (e.g., Merskey & Bogduk 1994; Merskey & Bogduk 2012).

- The American Medical Association has provided multiple conceptualizations over time (e.g., Brigham 1999; Cocchiarella & Andersson 2001; Rondinelli et al. 1999).

- At all points in history, the IASP and AMA conceptualizations of the moment have always been substantially discrepant from one another (the publications that are referenced in the above two bullet points can be reviewed in order to see the discrepancies).

- A wide of variety of conceptualizations have been published (e.g., American College of Occupational and Environmental
Medicine 2008; Cocchiarella & Anderson 2001; Harden & Bruehl 2010b; Merskey & Bogduk 1994; Merskey & Bogduk 2012; Mosby’s Medical Dictionary 9th Edition 2013; ODG Treatment in Workers Comp; Rondinelli et al. 2009; Stanton-Hicks et al. 1995). Such published conceptualizations extensively contradict one another (e.g. whether the construct involves a single health condition or is instead an umbrella concept, whether the construct definitionally involves the sympathetic nervous system, whether the construct definitionally involves a history of trauma/noxious event, whether relevant clinical presentations are most often associated with a history of myocardial infarction, whether the construct applies to parts of the body other than the upper extremities, how many sub-constructs are recognized, etc.). The conflicting nature of such conceptualizations further highlights the unreliability of the construct.

A 2014 “comprehensive and critical review” emphasized the unreliability of the construct (Borchers and Gershwin 2014).

The ambiguous and unreliable nature of the complex regional pain syndrome construct (as has been discussed above) creates additional obstacles to scientific scrutiny being applied to it.

- Any scientific findings that were based on the original 1994 IASP conceptualization (Merskey & Bogduk 1994) will not be relevant to any of the modern conceptualizations (because all of the modern conceptualizations are substantially different than the 1994 IASP conceptualization).

- Any scientific findings based on the 2012 IASP conceptualization (Merskey & Bogduk 2012) will not be relevant to any of the other modern conceptualizations (as discussed Section II of this chapter), because the 2012 IASP conceptualization is substantially different from the other modern conceptualizations.

- The creation of the “not otherwise specified” sub-construct (Merskey & Bogduk 2012) eliminates any hope of reliability for the complex regional pain syndrome construct (as was discussed above, and will be further discussed below).
12. The concept of complex regional pain syndrome was specifically created in a fashion that causes it to be an inherently non-injury-related issue, and later published reports have similarly highlighted its non-injury-related nature.

NOTES:

- The construct has apparently always been defined as involving a clinical presentation which is disproportionate to any claimed inciting event, such as injury (e.g., Merskey & Bogduk 1994 and 2012). The primary published report regarding the creation of the complex regional pain syndrome construct specified this disproportionality as a "distinguishing characteristic" of the construct (Stanton-Hicks 1995). Such disproportionality is the opposite of the dose-response gradient which is characteristic of causative relationships (Melhorn et al. 2014). Consequently, the construct actually means that the clinical presentation is inconsistent with any claimed injury (or any other claimed inciting event).

- Similarly, a 2009 review of previously published discussions (published by the entity which initially formalized the construct – the IASP) concluded that there is also a lack of reliable association between the type of claimed injury and the pattern of the clinical presentation (Janig 2009). This means that there is not a qualitative association between a relevant clinical presentation and any claimed injury (as well as there not being an association in terms of proportionality/quantitative issues).

- Consistent with the constructs inherent lack of injury-relatedness, relevant clinical presentations have been documented as developing in the absence of any claimed injury (e.g., Harden 1999; Van Leare & Claessens 1992; Veldman 1993; Verdugo & Ochoa 2000). Such reports are of relevance to the prevention/elimination standard of causation analysis (Melhorn et al. 2014) which specifies that within a causative relationship, "if the exposure can be prevented or eliminated from a population, the development of the disease or condition does not occur". Given the multiple published reports that relevant presentations occur in the absence of even a claimed injury, attempts to claim injury-relatedness for complex regional pain syndrome are inconsistent with the prevention/elimination standard of causation analysis.
Notes:

- A wide variety of diagnostic protocols have been published (e.g., American College of Occupational and Environmental Medicine 2008; Cocchiarella & Anderson 2001; Harden & Bruehl 2005; Harden et al. 2007; Merskey & Bogduk 1994; Merskey & Bogduk 2012; ODG Treatment in Workers Comp; Rondinelli et al. 2009; Stanton-Hicks 1995). The various protocols substantially contradict one another (e.g. differential diagnosis considerations, number of types of the construct, requirements in regard to diagnostic criteria, etc.), thereby further highlighting the unreliability of the construct.

- The inherently non-injury-related nature of the complex regional pain syndrome construct is also highlighted by all of the following:
  - The original IASP conceptualization specified that a history of injury (or any other type of “noxious event”) was not a required part of the construct (Merskey and Bogduk 1994).
  - Efforts to apply some minimal scientific scrutiny to that original IASP conceptualization resulted in a recommendation for the elimination of even the optional criterion which involved consideration of a history of injury (or any other type of “noxious event”) (Harden et al. 1999).
  - That optional 1994 IASP criterion which involved consideration of a history of injury/ “noxious event” is completely absent from modern conceptualizations (American College of Occupational and Environmental Medicine 2008; Merskey & Bogduk 2012; ODG Treatment in Workers Comp; Rondinelli et al. 2009).

13. Scientific findings have indicated that most cases which involve a diagnosis of complex regional pain syndrome will be associated with a legal claim of some form (e.g., workers compensation claim, personal injury lawsuit) (reference: Allen 1999; Talmage et al. 2013; Verdugo & Ochoa 2000). This fact, combined with the definitional lack of association between the CRPS construct and any pathophysiology, provides a credible basis for concluding that a diagnosis of complex regional pain syndrome in an individual case which involves compensation incentives is best conceptualized as an artifact of those incentives, rather than being purely conceptualized as a health issue.

14. Because of the extreme overlap between diagnoses of complex regional pain syndrome and legal claims, it is important to note that it is necessary for any
Notes:

clinician/expert who provides information regarding such a claim to the legal system to explain to that legal system that a CRPS diagnosis...

- ...does not allow for a credible legal determination (or a credible scientific determination, or even a credible clinical determination) that an individual has a health abnormality.

- ...does not allow for a credible legal determination (or a credible scientific determination, or even a credible clinical determination) that a clinical presentation has been caused by an injury, an accident, occupational issues, or any other claimed causes that are common in legal/compensation claims.

- ...does not allow for a credible legal determination (or a credible scientific determination, or even a credible clinical determination) that an individual is vocationally disabled, has work limitations, or is in need of work restrictions.

- ...does not allow for a credible legal determination (or a credible scientific determination, or even a credible clinical determination) that a clinical presentation is permanent, involves permanent impairment, warrants an impairment rating greater than zero, etc.

- ...is an obstacle to credibility for any other legal determinations (and any other scientific determinations, and any other clinical determinations) that would be supportive of the legal/compensation claim.

NOTES:

- The diagnostic system for mental disorders (e.g., American Psychiatric Association 2000) has repeatedly been specified as a model for efforts that contributed to the creation of the modern diagnostic methods for complex regional pain syndrome (e.g., Bruehl et al. 1998; Galer et al. 1998; Harden et al. 1999; Harden et al. 2007; Harden 2012). Elements of the diagnostic system for mental disorders which have been replicated in the various diagnostic methods for complex regional pain syndrome include a lack of causative implications, a lack of pathophysiology, a descriptive focus rather than an explanatory or etiological focus, and a lack of scientific credibility (for relevant details in regard to the diagnostic system for mental disorders, see Barth 2014; for relevant details in regard to complex regional pain syndrome, see publications such as the following: Barth 2003; 2006a, 2006b, 2006c, 2009, 2011; Barth & Haralson 2007a & 2007b; Biller et al. 2001; Borchers 2014; Del Piñal 2013; Ring et al. 2010).

- The nature of the diagnostic systems for mental disorders and complex
regional pain syndrome is not only lacking in scientific credibility; this type of approach to diagnosis has also been specifically identified as an obstacle to scientific advancement (e.g., Insel 2013).

- Given the repeatedly documented history of the diagnostic system for mental disorders serving as a model for the conceptualizations of complex regional pain syndrome, the implications of that model should be fully manifested in regard to complex regional pain syndrome. For example, the diagnostic system for mental disorders has repeatedly been inextricably linked to published warnings that the system cannot be credibly used to support legal claims (such warnings have actually been placed in the various editions of the manuals for that system which have been published since the creation of complex regional pain syndrome) (e.g., American Psychiatric Association 2000, 2013). Therefore, such warnings are provided above (consistent with the repeatedly published reports that the modern conceptualizations of complex regional pain syndrome are modeled after the diagnostic system for mental disorders, these warnings are also modeled after the warnings from the various editions of the manual for that system).

15. Whenever this diagnosis is made, some form of mental illness is usually involved in the clinical presentation. Probabilities include depressive disorders, anxiety disorders, and personality disorders.

NOTES:
- Scientific findings have also revealed strong associations between relevant clinical presentations and pre-existing psychopathology, specifically including personality disorders, depressive disorders, vulnerability to anxiety, and vulnerability to somatization (Barth and Haralson 2007a and 2007b; Barth 2013a and 2013b).

- While the same is true for chronic benign pain in general (Barth 2013a and 2013b), scientific findings have indicated that people who develop complex regional pain syndrome-like presentations are even more psychologically dysfunctional than other chronic pain patients (Bruehl et al 1996).

- This set of circumstances provides a credible basis for concluding that a diagnosis of complex regional pain syndrome in an individual case is best conceptualized as a manifestation of psychopathology (in the extremely rare case which does not involve compensation incentives), or as a consequence of the
interaction of compensation incentives and psychopathology.

16. Scientific findings have indicated that all of the objectively verifiable clinical issues that have been written into various conceptualizations of complex regional pain syndrome (e.g., swelling, trophic changes) can be created through disuse (Butler 2001).

17. Scientific findings and several case reports have indicated that relevant clinical presentations are often self-inflicted (Buijs et al. 2000; Chevalier et al. 1996; Huyghe et al. 2002; Mailis-Gagnon 2008; Rodriguez-Moreno et al. 1990; Taskaynatan et al. 2005).

18. The concept of complex regional pain syndrome has not been scientifically validated as actually corresponding to any health condition (Biller et al. 2001; Borchers & Gershwin 2014; Del Piñal 2013; Ring 2010; Rondinelli et al. 2009).

19. Scientific findings have indicated that relevant clinical presentations have a very favorable prognosis, typically resolving within months (Sandroni et al. 2003).

20. Scientific findings have indicated that the majority of people who receive such a diagnosis will demonstrate invalid clinical presentations, when scientifically validated objective testing is administered (Greiffenstein et al. 2013).

21. A 2014 “comprehensive and critical review” concluded that: “There are no standards which can be applied to the diagnosis and would fulfill definitions of evidence-based medicine.” (Borchers & Gershwin 2014) In other words, a scientifically credible method for justifying such a diagnostic claim does not exist.

22. A variety of health science publications have called for the abandonment of the complex regional pain syndrome construct (Bass 2014; Biller et al. 2001; Borchers & Gershwin 2014; Del Piñal 2013; Ring 2010). In addition to the construct’s ambiguity, unreliability, and lack of scientific credibility, the reasons for such calls for abandonment of the construct include reports that the utilization of the construct deprives patients of adequate diagnosis, and consequently, deprives patients of adequate treatment.

23. Historically, there have been some issues which were almost definitional for the complex regional pain syndrome construct (almost common to the various conceptualizations). These issues are listed below, but it should first be noted that the near-definitional nature of these issues has now been compromised by the introduction of the “not otherwise specified” sub-construct. The issues which were almost definitional prior to the introduction of the “not otherwise specified” sub-construct included:
Notes:

- The concept involved a syndrome. Given a definition of “syndrome” such as “a set of symptoms that occur together” (Dorland’s Illustrated Medical Dictionary, 32nd Edition) this aspect of the construct has been clearly discontinued by the introduction of the “not otherwise specified” sub-construct which eliminates the premise that the construct involves any set of symptoms which reliably occur together. In fact, given the variability in clinical presentations which has been allowed by the various conceptualizations of the construct, it does not appear that the construct ever actually involved a syndrome.

- The construct involved a combination of subjective symptoms (e.g. pain) and signs which allow for objective verification (e.g. swelling, trophic changes). This formerly definitional issue is especially important to note, as I have repeatedly seen clinical presentations which involved nothing other than a complaint of pain, and yet the presentation was diagnosed as complex regional pain syndrome (such cases came to my attention regularly, even prior to the creation of the “not otherwise specified” sub-construct).

- Historically, the construct has been defined by the clinical presentation being inconsistent with any claimed cause (e.g., disproportionately severe in regard to any claimed cause, anatomically inconsistent with any claimed cause, etc.).

- The “type two” sub-construct involves a history of unambiguous injury (Rondinelli et al. 2009) to a large (Stanton-Hicks 1995) or major (Harden & Bruehl 2005) peripheral (Rondinelli et al. 2009) nerve (but the clinical presentation was inconsistent with any such injury).

- In contrast to type two, the “type one” sub-construct does not involve a history of nerve injury (e.g., Rondinelli et al. 2009; Gilron et al. 2006; Janig 2009). Consequently, the type one sub-construct has been specifically characterized, including in literature from the entity that initially formalized the construct – the IASP, as not involving neuropathic pain (e.g., Gilron et al. 2006; Janig 2009).

24. In spite of the potential for objective verification that was relevant to some aspects of the complex regional pain syndrome construct, the modern conceptualizations have moved in the direction of pure subjectivity.

NOTES:

- As has been explained by a writer who has been involved in most of the published discussions of the Budapest protocol
Notes:

(some version of the criteria set from which has been included in all of the modern protocols that have been discussed in this chapter) (Harden 2010c)...

- The modern criteria sets have been “deliberately based on bedside testing. Designing criteria that are accessible to any clinician, not requiring any special equipment or training”

- Consequently, the modern criteria sets involve “a very heavy reliance on the subjective (not only the subjective response of the patient, but the subjective impression of the clinician).

- “This is very problematic scientifically and statistically”

- This “very heavy reliance on the subjective” is an obstacle to any legal causation claim, because the first step of the normal scientific and professional method for evaluating a causation claim specifies that such claims are not credible unless they involve a definitive diagnosis that has been based primarily on subjective findings. In contrast to that requirement for justifying a causation claim, a diagnosis of complex regional pain syndrome will always be based primarily on subjectivity (by definition).

- The ACOEM protocol (American College of Occupational and Environmental Medicine 2008) that was discussed previously in this chapter is the only one which attempts to force some objectivity onto the diagnostic criteria (NOTE: The AMA protocols from 1997 until 2008 demanded such objectivity, but they have been formally discontinued, and the criteria sets for which they demanded objectively were different from the modern sets). However, the portion of the criteria set which is amenable to the objectification that the ACOEM protocol calls far is minimal, and subjectivity remains the dominant factor in all of the modern criteria sets.

25. Complex regional pain syndrome has an antonym: chronic regional pain syndrome (Wulle).

NOTES:
- The phrase "chronic regional pain syndrome" has been used specifically for the purpose of distinguishing clinical scenarios which definitely do not involve complex regional pain syndrome, from those which supposedly do involve complex regional pain syndrome.
Notes:

- One of the definitionally significant differences between these two issues is that conceptualizations of complex regional pain syndrome include trophic changes, while "chronic regional pain syndrome" refers to clinical presentations which specifically do not involve trophic changes.

- The scenarios which are categorized as "chronic regional pain syndromes" are distinct from the concept of complex regional pain syndrome in that the former does not involve objectively verifiable differences between one side of the body and the other (e.g., such as a difference in the circumference of soft tissues for one arm versus the other, a difference in the calluses of the palms, a difference in regard to bone density, etc.).

- Examples of clinical presentations which have been categorized as chronic regional pain syndromes in order to distinguish them from complex regional pain syndrome include: the effects of inappropriately extensive immobilization; movement restrictions caused by scar tissue or neuroma; pain which serves a socially-driven purpose for the patient.

NOTE: This final point is not being formally included in the above list of definitional issues, because it has not been studied or documented systematically (to my knowledge). However, if my non-systematic impression is accurate, then this may be the most important definitional issue. My impression, based on having collected and reviewed hundreds of healthcare records which apparently document the initial diagnosis of complex regional pain syndrome within an individual case, is: Most individual case diagnoses of complex regional pain syndrome are not based on any of the formal/published conceptualizations or diagnostic methods. Consequently, discussion of the formal/published conceptualizations and diagnostic methods appears to be an intellectual exercise that does not have any actual relevance for health care (or for science). The process of collecting and reviewing relevant healthcare records has left me with a strong impression that the only thing that a diagnosis of complex regional pain syndrome, within an individual case, reliably means is that the patient is complaining of pain, and the doctor who made the diagnosis is personally inclined to making such non-scientific diagnoses.

References (this reference list might not include materials that were fully referenced within the text of this chapter):
Notes:


Barth, RJ, and Bohr, TW. Challenges in the IASP’s Diagnostic Conceptualization for CRPS-1 (Formerly Conceptualized as RSD), Part 2. *The Guides Newsletter.* March/April, 2006b. American Medical Association.


Notes:


Notes:


Harden, RN, and Bruehl, SP. Diagnostic Criteria: The Statistical Derivation of the Four Criterion Factors. In: Wilson, PR, Stanton-Hicks, M, and Harden, RN. *CRPS: Current Diagnosis and Therapy.* International Association for the Study of Pain, Seattle: 2005.
Notes:


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
Notes:


ODG Treatment in Workers Comp: http://www.odg-twc.com/


Vermont Workers Comp Bulletin. CRPS Impairment Allowed Despite The Diagnosis' Failure to Meet AMA Guides Criteria: http://vtcompbulletin.blogspot.com/2013/06/crps-impairment-allowed-despite.html
