How Neuroplasticity (NP) can win your case, part 7 of a series on NP:

-Again, realize that bad NP changes in your patients provides the scientific basis that refutes and disproves many of the reports and testimonies of biased treating and defense IME doctors.

-When the patient continues with pain complaints/delayed recovery, the patient is under-treated, or not treated at all; and when their complaints persist or grow, they are not believed, and are either placed at MMI or their entire case is just denied. The treating Dr or later the IME Dr says the pt is at MMI and all possible treatments have been exhausted and the diagnoses/exam includes: pain disorder, Waddell's signs, ROM differences, psychological associated or caused symptoms, symptom magnification, differing diagnoses, pre-existing disease, aging, osteoarthritis, poor claimant credibility/being an unreliable historian, poor provider credibility, lack of understanding of causality, increasing signs and symptoms that are seemingly unrelated to the current claim, and more.

-The following is from an biased defense IME Dr's report (very well written, just wrong!): This patient has pain. I do not doubt that she feels pain. However, there is no actual medical diagnosis, as pain is not a diagnosis. The multiple diagnostics study showed no pathology. The treating physicians are unable to identify the pain generators. It is not plausible that the current, chronic severe pain this patient feels is medically reasonably related to the mechanism of action that caused this workers compensation claim to be opened.This all supports the fact that there its no logical, medically probably diagnosis for continuing pain in this patient. As her current pain is not proximately related to the date of the injury, there is no impairment rating as pain complaints are not ratable. Her impairment rating is 0!

-Provider misdiagnoses is frequently caused by the lack of recognition of bad NP and central sensitization.

-Note that pain is now a ratable diagnosis in the AMA Guides, 6th Edition. On page 32 of the 6th Edition, it notes the biopsychosocial approach to chronic pain (pain that persists beyond 3 months or after healing is expected) has replaced the outdated biomedical reductionist perspective used previously (a perspective that tries to explain a patient's continuing pain in psychologic, aging, or cultural terms, negating the possibility of an interrelation of causal phenomena-including NP). It notes that each person experiences chronic pain uniquely, and a range of psychosocial and economic factors can interact with pathophysiology. Moreover, even as healing occurs, all patients experience some degree of physical deconditioning associated with stiffness and possible muscle atrophy, frequently associated with periods of inactivity, inhibition of functions of pain, fear of pain avoidance. It further notes that in striking contrast, the traditional outdated biomedical approach assumes that all pain symptoms have specific physical causes, and attempts to eradicate the cause directly by identifying and rectifying the presumed pathophysiology. On page 34 of the 6th Edition, Section 3.1 E, it discusses persistent pain as a disease entity. It notes that there is accumulating

evidence that persistent pain should be considered a disease entity in its own right. Indeed, permanent changes in the responsiveness of both the peripheral and central nervous systems can persist even after all tissue healing has ensued; thus, persistent pain can become a self-perpetuating condition. The individual is often left with ongoing pain without identifiable signs of the original silent disease process that initiated the pain. This results in a multitude of consequences that can lead to a significant impairment for the individual infected, including physical impairment, mood dysfunction, and social disruption.

- From the online edition: State of Colorado, Department of Labor and Employment, DIVISION OF WORKERS' COMPENSATION
  RULE 17, EXHIBIT 9, Chronic Pain Disorder
  Medical Treatment Guidelines Effective: February 14, 2012 on p. 8:

CENTRAL SENSITIZATION The experience of pain evoked by the excitation of nerve fibers that normally relay non-painful sensations to the spinal cord. This results when non-nociceptive afferent neurons act on a sensitized central nervous system (CNS). Experimental data suggest that pathways normally carrying pain signals themselves become overstimulated and/or fail to respond to inhibitory influences causing increased pain. An example is 'wind-up' which occurs when cells in the dorsal horn of the spinal cord increase their rate of action potential discharge in response to repeated stimulation by nociceptors.

- From the online edition: State of Colorado, Department of Labor and Employment, DIVISION OF WORKERS' COMPENSATION
  RULE 17, EXHIBIT 9, Chronic Pain Disorder
  Medical Treatment Guidelines Effective: February 14, 2012, page 6:

> Chronic pain is a phenomenon not specifically relegated to anatomical or physiologic parameters. The prevailing biomedical model (which focuses on identified disease pathology as the sole cause of pain) cannot capture all of the important variables in pain behavior. While diagnostic labels may pinpoint contributory physical and/or psychological factors and lead to specific treatment interventions that are helpful, a large number of patients defy precise taxonomic classification. Furthermore, such diagnostic labeling often overlooks important social contributions to the chronic pain experience. Failure to address these operational parameters of the chronic pain experience may lead to incomplete or faulty treatment plans. The term "pain disorder" is perhaps the most useful term in the medical literature today, in that it captures the multi-factorial nature of the chronic pain experience.

Some examples from the literature in support of bad NP are:
1. Pain. 2014 Jun 27: S0304-3959(14)00308-X. doi: 10.1016/j.pain.2014.06.019.
Early Afferent Activity from the Facet Joint after Painful Trauma to its Capsule Potentiates Neuronal Excitability and Glutamate Signaling in the Spinal Cord.
Crosby ND1, Gilliland TM1, Winkelstein BA2.

Cervical facet joint injury induces persistent pain and central sensitization. This study suggests that early afferent activity from the injured facet induces development of spinal sensitization..

2. Clin J Pain. 2010 May;26(4):339-47. Different activation of brain areas in patients with complex regional pain syndrome (CRPS I) compared with healthy controls during perception of electrically induced pain: a functional MRI study. Department of Diagnostic and Interventional Radiology, University Hospitals, Ulm, Germany.
Many arguments favor central maladaptive changes in pain processing as an important causative factor. These results show that changed cerebral pain processing in CRPS patients. These changes are not limited to the diseased side but show generalized alterations of cerebral pain processing in chronic pain patients.

3. Arch Phys Med Rehabil. 2012 Jul 9.
A study on Central Hypersensitivity in Patients with Subacromial (part of the shoulder) Impingement Syndrome. Case Western Reserve University School of Medicine, Cleveland, Ohio.

CONCLUSION: This study provides further evidence that chronic subacromial impingement syndrome patients have significantly lower Pressure Pain Thresholds than controls in both local and distal areas from their affected arm consistent with primary and secondary hyperalgesia, respectively. Data suggest the presence of central sensitization among subjects with chronic subacromial impingement syndrome.

4. Fibromyalgia Brain Misreads Pleasure/Pain Signals, November 5 in Arthritis & Rheumatism by Marco L. Loggia, PhD, and colleagues, related to chronic pain is:
The widespread pain and lack of response to opioids in patients with fibromyalgia (FM) might be partly a result of mistakes in brain signals. "We show that FM patients exhibit reduced brain activity in response to visual cues informing them of impending pain onset (pain anticipation) and pain offset (relief anticipation). This reduced responsiveness was seen in various regions, including regions involved in the processing of rewards/punishment. This is an interesting observation, given that several studies are suggesting a relationship between chronic pain and alterations in reward neurocircuitry." Dr. Loggia is instructor in radiology at Harvard Medical School and assistant in neuroscience at Massachusetts General Hospital, Boston.

Activation responses in patients with FM were also abnormal in several areas of the brain. Dr. Loggia said the researchers were quite surprised by the extent of the changes in brain function in FM. "While I did expect to observe reduced responses to the anticipatory cues in some regions (eg, the regions involved in the descending pain modulatory system, which is known to be altered in these patients), I did not expect to see so many regions exhibit this behavior. The new findings also help explain another aspect of FM: lack of response to opioids. It suggests that altered neurotransmission in

this and similar reward/punishment processing brain regions might support the lack of therapeutic efficacy of opioids in treating FM pain.

"I think the main message clinically is that there are prominent central changes in pain processing and that the preponderance of evidence is that this is a central nervous system disease," Daniel J. Clauw, MD (professor of anesthesiology, hematology, and psychiatry and is also director of the Chronic Pain and Fatigue Research Center at the University of Michigan in Ann Arbor).

The next and final article in this series discusses using this info to specifically refute common denials.