Research Update on Whiplash Diagnosis and Treatment

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Part One of Two

Who are we talking about today?

This article is about patients who have no obvious MRI findings of nerve root encroachment and no fractures on x-rays or CT scan. Despite all this they continue to have serious disabling neck, upper back, low back pain and/or dizziness, numbness, burning or tingling that all started wth a car crash.

"Whiplash" Defined

"Whiplash" is not a whip or a lash. Early models of whiplash focused on sprain-strain injuries. Literarily this is an injury to the ligament (sprain) and to the muscle (strain). As a result, it was thought "Whiplash" occurred when the neck was hyper-extended (moved too far back). This was one of the reasons for adding headrests to cars. However, more recent research has shown that this doesn't happen.[i] In fact, the mechanism that causes "Whiplash" is now known to be a differential acceleration or deceleration. What does this mean? To understand, let's take a rear end crash. The torso is pushed forward while the head lags behind. This movement produces injury causing shear forces on the neck and likely a pressure build-up of cerebral spinal fluid (the fluid that surrounds the spinal cord and nerve roots). These forces can injure the ligaments and joints as well as the dorsal root ganglion (nerves in the neck that transmit information about feeling) .[ii] [iii]

How do we really know that people get hurt by this new differential acceleration/deceleration model?

Studies by the Insurance Institute for Highway Safety show that seats designed to reduce these forces in rear end crashes result in as much as a 49% reduction in real world claims for neck injuries (Volvo WHIPS seat versus older Volvo seats).

If "Whiplash" isn't a Whip or a Lash, why are we still using this term?

Good question. The answer is that we shouldn't use this term, other than to refer to the historical context or to communicate a subject. While many terms have been bantered about, none has stuck. I prefer the more accurate term, Serious Neck Injury or SNR. This term will be used in this text.

Why is the sprain-strain model only one quarter right?

Sprain-strain means that a ligament and muscle have been injured. However, recent research shows that CPK levels (a blood enzyme that is elevated when muscles are damaged) are not increased in Serious Neck Injury patients. [iv] We do have evidence that ligaments are injured There is clinical evidence that cervical facet joints are injured too. We know this from the following data:

- Live volunteer crash testing shows spearing of these joints in simulated rear end crashes
- Autopsy studies from patients who died of other causes such as blunt abdominal trauma. These studies show bleeding of the joints, micro fractures, and ligament damage. [v] In addition, other studies by the same authors also show that while these injuries can be seen on a histology slide (a slice of tissue under a low power microscope), they can't be seen on MRI, Cat Sans, or x-ray.
- Real world treatment studies are showing that when you perform procedures to provide long term relief from facet joint pain, patients not only report severe neck pain goes away, but over-reaction and psychological disturbances resolve as well [vi] [vii]

If Serious Neck Injury patients don't injure their muscles, why do Serious Neck Injury patients have chronically tight muscles?

Medical providers who see SNI patients learn patients report massage and hands on muscle work helpful for temporary relief and to prevent extra injury caused by chronic muscle spasms. Muscle tightness in this population is likely related to abnormal regulation of muscle tone by the nervous system.

Here are several examples that demonstrate how muscle dysfunction may occur in whiplash:

- Animal studies demonstrate neck joints help modulate muscle tone. Injury to these structures may explain one source of abnormal muscle regulation. [viii]
- SNI patients demonstrate lower thresholds for both sensory and muscular stimulation. Central nervous system damage is observable by their over-reaction to stimuli. [ix] [x]
- Disturbances of the visual coordination and balance system may change the coordination of muscles. [xi] [xii] [xiii]

The research in this area has exploded in the past few years. Here's a list of structures that are likely damaged in these patients along with an explanation of how these damaged structures can wreck havoc on the patient:

- Facet joints (neck joints). Neck pain, headaches, upper back pain. [xiv] There is recent evidence that injured neck joints can cause numbness, tingling, or burning in the arms and hands. Muscle tightness and decreased neck and upper back motion (see reference above).
- Ligaments. Cervical instability can contribute to neck pain. [xv] This means that the "duct tape" that holds the neck bones together (the ligaments) is torn or stretched beyond repair. This causes nerves and joints to become irritated and the neck and upper back muscles to get tight. Ligamentous injury can cause numbness, tingling, and weakness in upper and lower extremities.
- Dorsal Root Ganglia. These nerves in the neck help to route pain signals and other signals involved with normal sensation to the brain. Sensory allodynia (painful sensation with non-painful stimuli) is common in SNI patients. [xvi] [xvii] [xviii] [xix] [xx] This means that many patients find that they feel things much more intensely after the crash. Small things that wouldn't have bothered them before now cause pain. In addition, damaged DRG's can cause nerves to fire abnormally provoking pain. Hence, normal movement which should be non-painful becomes painful because of malfunctioning nerves. [xxi] [xxii]
- Brain. It is believed that disinhibition may play a role in reduced modulation of pain. What the heck does that mean? In essence, the patient's brain can no longer block incoming pain messages, so signals fire unheeded, magnifying the pain of once harmless sensations.

Is SNI a self-limited condition?

Self-limited conditions might cause us temporary discomfort, but ultimately they go away and have a limited impact on our lives. For the vast majority of patients with simple whiplash the recovery is quick. However, between 3-5% of patients in a crash will develop SNI. The peer-reviewed research on this injury doesn't support that it is self-limited. Here are a few citations:

- Berglund performed a large epidemiologic study that looked at thousands of people not exposed to a rear end crash versus hundreds who were exposed and later saw a specialist. The patients involved in rear end crashes had a 160%-370% increase in headache complaints, upper and lower back pain, as well as fatigue, sleep disturbances and ill health. [xxiii]
- Bunketorp reported on a series of patients followed from the time of intake into the Emergency Room and followed them forward for 17 years. One group reported car crash injuries in the ER that later required specialist care; the second group reported no injuries in the ER and did not seek specialist care. The most surprising finding of this study was that the injury group at 17 years had a disability rate of 30-35%, while the non-injury group had a disability rate of only 6%. [xxiv]
- Squires (an orthopedic surgeon in Bristol England) observed the same group of SNI patients for 15.5 years. After more than fifteen years, 70% continued to complain of symptoms referable to the original crash. Neck pain was most common, but low-back pain was present in half of the subjects. Women and older patients had the worse outcomes. Radiating pain was common in those with severe symptoms. Perhaps the most important finding of this study is that between 10 and 15 years after the accident 18% of the patients showed improvement, whereas 28% deteriorated and 54% remained the same. .[xxv]

We welcome investigation of the searchable links available in the complete article available online at http://www.spinalinjuryfoundation.org/profpublication.htm

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