Persistent post-traumatic headache, postconcussion syndrome, and whiplash injuries: the evidence for a non-traumatic basis with an historical review

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Abstract

There has been intense controversy about postconcussion syndrome since Erichsen's publication in 1866 on railway brain and railway spine. The fascinating history of this debate will be reviewed and then the non-organic explanations for postconcussion syndrome, headaches after head injury, and chronic whiplash injuries and headaches will be explored including the following: psychogenic, psychosocial, sociocultural, base rate misattribution, chronic pain, compensation and litigation, and malingering

There are approximately 1.4 million reported incidents of traumatic brain injury in the United States every year [1] with mild injuries accounting for 70-90%.[2] However, the incidence of mild head injury is probably in excess of 600 persons per 100,000 population as many cases go unreported.[2] Headaches are variably estimated as occurring in 25 to 78 percent of persons following mild traumatic brain injury. [3],[4] For an industrialized country such as the United States, estimates of the relative causes of TBI are as follows: motor vehicle accidents (45 percent), falls (30 percent), occupational accidents (10 percent), recreational accidents (10 percent), and assaults (5 percent).[5]

Headache is a cardinal feature and the most common symptom of the postconcussion syndrome (PCS), a symptom complex, which also commonly includes dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity.[3,4] As concussion is a trauma-induced alteration in mental status that may or may not involve loss of consciousness,[6] loss of consciousness does not have to occur for PCS to develop.

Thirty to 80 percent of patients with mild to moderate brain injury will experience some symptoms of PCS. This wide range of reported incidence reflects variabilities in the patient population studied and the criteria by which a diagnosis of PCS is made, either using individual symptoms or defined clinical criteria. Two clinical criteria, the International Classification of Diseases, ICD-10 and the DSM-IV, are commonly used and give widely different results, even within the same patient population.[7] Neuropsychological testing is often the only test to demonstrate “objective” findings in some patients with persistent cognitive symptoms after mild head injuries but the findings may be inconsistent when performed or interpreted by different psychologists and there are many reasons why the results may be invalid (including psychiatric co-morbidity, cooperation and motivation of the patient, tests administered, and the skill and clinical sensitivity of the examiner).[8]

The fascinating history of this debate will be reviewed and then the non-organic explanations for PCS, headaches after head injury, and chronic whiplash injuries and headaches will be explored.

Historical aspects

The earliest uses of the term,"PCS," that I can find are two publications from 1934, Grinker's neurology textbook[9] and an article by Strauss and Savitsky reflecting controversy over the topic going back at least to the 1860's[10]:

> "In our opinion, the subjective posttraumatic syndrome, characterized by headache, dizziness, inordinate fatigue on effort, intolerance to intoxicants and vasomotor instability, is organic and is dependent on a disturbance in intracranial equilibrium due directly to the blow on the head. We suggest the term "postconcussion syndrome" for this symptom complex."[11]

One interesting historical case involved a 26-year old maid servant who had been hit over the head with a stick and complained of retrograde amnesia. Six months later, she was still complaining of headaches, dizziness, tinnitus, and tiredness. A judge requested the opinion of Swiss physician J.J. Wepfer (he was a "father of neurology" and described the "Circulus arteriosus" before Willis[12]) and two other surgeons, who stated," We can't say anything definite, but it is certain that this will leave its mark in the form of an impediment." Although similar prognostic opinions are still given, this particular statement was made in 1694.[13] Boyer in 1822, Astley Cooper in 1827, and Duputren in 1839 all described the clinical picture of cerebral concussion with persistent symptoms.

John Erichsen (figure) was Professor of Surgery at University College Hospital, London, England, surgeon extraordinaire to the queen, and one of the best known surgeons in the world.[14] His famous textbook, "Science and Art of Surgery," was published in 10 editions, translated into 15 languages, and was the standard medical book issued to every Union medical officer in the American Civil War. In 1866, he published a series of six lectures, "Certain obscure injuries of the nervous system commonly met with as the results of shocks of the body received in collisions on railways." An additional treatise with eight new lectures was published in 1875. In 1882, a new and revised edition was published which is available online without charge.[15] These types of injuries became known as railway spine or railway brain, since many occurred in railway accidents. However, of the 53 patients Erichsen describes in his 1882 book, only 17 were injured on railways. The others sustained blunt trauma from falls or blows in other circumstances. Erichsen believed that minor injuries to the head and spine could result in severe disability due to "molecular disarrangement" or anemia of the spinal cord. He commented on the controversy of the time:

> "There is indeed no class of cases in which medical men are now so frequently called upon to give evidence in the courts of law, as those which involve the many intricate questions that arise in actions for damages against railway companies for injuries of the nervous system, alleged to have been sustained by passengers in collisions; and there is no class of cases in which more discrepancy of surgical opinion may be elicited."

Figure. John Eric Erichsen, 1818-1896 (from Br Med J.1896;2:885)

However, doubt about the organicity of railway spine was raised by two other British physicians. In a summary of an 1881 paper, Wordsworth, “believed that all recovered on the settlement of their claims, and had resumed their wonted occupation. He remarked on the fact that medical men are seldom consulted in these cases except with a view to their assisting to obtain compensation; stated that in a long experience in hospital and private practice he had never seen any of these cases simply as patients either before or after the settlement of their claim for compensation.”[16] In an 1883 book, Page, another London surgeon, presented 234 of his own cases with information on legal settlement and follow-up, thought that Erichsen was scientifically inaccurate and challenged many of his cases, and proposed “general nervous shock” and “functional disorders” as explanations.[17]

In Europe, the organic concept of railway spine also became the focus of controversy. In 1871, a compensation law was passed in Prussia for passengers and railroad employees injured in railway accidents. The number of claims for alleged injuries greatly increased. Rigler doubted the organicity of these claims and proposed compensation neurosis as the cause.[18] In 1888, Strumpel, concurring, commented on the tendency to exaggerate because of the desire to be compensated. In 1889, Oppenheim disagreed with the compensation neurosis explanation and popularized the use of the term "traumatic neurosis." In 1888 and 1889, Charcot explained that the posttraumatic symptoms were actually due to hysteria and neurasthenia. In 1892, however, Friedmann proposed that posttraumatic cases characterized by headache, dizziness, vasomotor instability, and intolerance to alcohol be labeled the vasomotor symptom complex and that they were due to disordered intracranial circulation.[19]

There was similar controversy in the United States. In 1881, Hodges challenged the organicity of railway spine and separated the alleged injuries into different categories. These included the "so-called 'spine cases,' familiar to lawyers and courts of law as well as to physicians.... The fourth class includes those of functional disorder, presenting symptoms, the knowledge of which is obtained not by observation, but from the statements of patients...."[20] He further challenged the terminology:

> “The variety of influences giving rise to the symptoms under discussion attests the inexactness of the name by which they are designated, and justifies the use of the words 'so-called' as a prefix. The aetiological importance attached to them, however, is due, not to the specific peculiarities attached of the agency by which

they are provoked, but to the fact that annoying litigation and exorbitant claims for pecuniary damages are constantly the grave result of their existence."

In 1883, Putnam further questioned the etiology of "so-called concussion of the spine."[21] "For this state of affairs the retention in medicine of the term 'spinal concussion' is certainly in part responsible in that it satisfies in a measure the imagination, and excuses the rational explanation of the symptoms which are brought to his notice." He recommends considering brain dysfunction as contributory: "Thus it is probable that in the production of many of the hysteroid symptoms it is a disturbance of cerebral rather than spinal functions which is at fault ..." He further distinguished hysteria from malingering in medicolegal cases.

Also in 1883, Walton credibly suggests brain injury as the cause of symptoms in railway spine: "... these symptoms are rarely unattended by irritability, fretfulness, emotional tendency, and inability to confine the attention. These can only be the result of derangement in the higher cerebral centres."[22] He further describes how the brain is particularly susceptible to derangement from a jar as a result of a fall or collision compared to the spinal cord.

An Editorial in the *Boston Medical and Surgical Journal* in 1883 summarizes the controversy:

> In this iconoclastic age when we are not allowed to believe in a personal Devil or good honest ghosts, or even to coddle our own pet superstitions and hobbies without a suspicion of mental derangement, it is natural that the medical 'bugaboo' raised by Mr. Erichsen some years ago, and christened spinal concussion, should meet with little quarter at the hands of the modern scientific observer. It is possible, however, that in this, as in other things, the skeptic may have gone too far, and that although it was no ghost that has alarmed us there may actually have been some phosphorescent light which we do not understand, and the nature of which we cannot fully explain. The cases reported recently in the Journal by Dr. J. J. Putnam, and the paper in a late issue by Dr. Walton, point to the reality of a set of symptoms induced by traumatism which corresponds well with those hitherto termed spinal concussion, a name so misleading that many accurate observers through the influence of the name alone have been induced to deny the existence of what the name covers. A rose, however,under any other name will remain as fragrant to the sufferer, and whether the ailment be termed railway spine, or traumatic neurasthenia, or hysterical hemianaesthesia, the condition is equally distressing.[23]

Finally, in a textbook of the nervous system in 1893, the New York neurologist Landon Carter Gray comments on the effect of litigation on the patient with railway or other traumatic injuries:

> Certain it is that the psychical condition of these patients is a very unfortunate one. However uneducated they may be, newspapers and the talk of everyday life has filled their minds with dread of the mysterious and baleful consequences that may happen to those who receive injuries, particularly in railway accidents.

> They have also heard for years of the damages, often enormous, which corporations have been obliged to pay. When the accident occurs, the nervous system undoubtedly receives a shock, ... and this shock should receive immediate and judicious treatment by rest, isolation, and medicaments. But instead of this, a lawyer or his agent the so-called 'runner' of this country--quickly appears upon the scene, and spurs the patient on to a suit for damages by exaggerating the injury and its consequences, so as to make the too-willing sufferer believe that the company can be readily forced to pay damages. Then come the long years of weary suffering, anxiety, waiting, and disappointment, unrelieved by proper treatment, for although the patient and the lawyer may not consciously discourage treatment, yet too many hopes and interests would be blasted by a cure to ever permit of treatment being properly carried on, even if any self-respecting physician could be found to undertake it. Months, perhaps a year or more, are passed in waiting for the suit to be tried.... Finally, the case being at last successfully ended, it may turn out, ... that the costs of the action and the lawyer's fee will leave but a pitiable sum of money at the disposal of the patient.... All this disturbance that follows the accident is oftentimes, I am firmly convinced, a more potent cause of the neurasthenia than the accident itself ...
>
> The element of simulation in all these diseases produced by injury should always be carefully considered whenever there is a question of a suit for damages. Physicians make a great mistake, however, in entering upon the examination in a mental state of bias against the alleged sufferer, and justice will be much more equally done if all the symptoms are carefully and impartially gathered before any conclusion is reached. But a sharp distinction should always be made between the symptoms that are *objective,* and those that are *subjective . ..* [24]

Many physicians are surprised to discover that physicians were extensively involved in medical jurisprudence including tort cases during the nineteenth century in the United States.[25]

By the 1960's, two British neurologists famously joined the centennial era debate. In 1961, Miller summarized the viewpoint of those who believe that PCS is really a compensation neurosis: "The most consistent clinical feature is the subject's unshakable conviction of unfitness for work...."[26] Symonds[27] took an equally strong opposing position in 1962 when he wrote, "It is questionable whether the effects of concussion, however slight, are ever completely reversible."

Current controversy over PCS

There are a number of non-organic explanations for PCS which suggest an origin for their subjective symptoms other than traumatic brain injury in some people.

Psychogenic -- A psychogenic origin to PCS is suggested by a number of empiric and clinical observations. The symptom complex of PCS (headache, dizziness, and sleep impairment) is similar to the somatization seen in psychiatric disorders including

depression, anxiety, and post-traumatic stress disorder. Anxiety and depression can also produce subjective and objective cognitive deficits that are similar to those seen in PCS and that improve with antidepressant treatment.[28] In some studies of patients with PCS, premorbid depression is quite prevalent, as high as 46 percent.[29] Patients with mild TBI with and without PCS symptoms have high levels of psychiatric symptomatology on structured psychiatric interviews.[30],[31],[32] In some series, patients with mild TBI have a higher rate (10 to 20 percent) of incident psychiatric disease, major depression, anxiety and panic, and acute and post-traumatic stress disorder (PTSD), compared with controls or the general population.[33],[34]

A prospective study in Oslo, Norwary of PCS among 115 patients with mild, moderate, and severe traumatic brain injuries also supports the psychogenic origin.[35] PCS symptoms were reported to a greater degree in persons with mild TBI at 3 months post-injury than the more severely injured. One year after injury, there were no differences between groups in the presence of PCS symptoms. Greater levels of somatic, cognitive and anxiety symptoms which were present at 3 months and shorter post-traumatic amnesia duration were important predictors for the severity of PCS at 12 months.

PTSD is common among United States soldiers who have sustained blast trauma and can also produce symptoms similar to PCS resulting in misdiagnosis of traumatic brain injury in patients who have recovered from concussions.[36] In one study of US soldiers, mild TBI was a risk factor for postconcussion and other somatic symptoms before, but not after, adjusting for incident depression and PTSD, suggesting that these conditions are important mediators of PCS symptoms.[37] Another survey of military personnel also found that PTSD was the strongest factor associated with PCS symptoms.[38] In this population, there may be links between post-traumatic stress disorder, impaired sleep, and chronic headaches.[39]

Psychosocial--Some studies have found poor social support and increased social adversity among patients who suffered prolonged symptoms than among those whose symptoms had remitted.[28,40]

Sociocultural--The very low, even absent, rates of postconcussion symptomatology, in some countries and in children, that are sometimes reported suggests a prominent role for sociocultural factors in the pathogenesis of PCS, perhaps because of misattribution or litigation.[41],[42]

Base rate misattribution-- A high base rate level of PCS symptoms in the general population can lead to misattribution of symptoms to PCS.[43] In a study of 104 healthy university community adults (61% female) with a mean age of 23 years, the following percentages endorsed the following symptoms from the ICD-10 criteria for PCS as present in the prior 2 weeks: fatigue, 76%; irritable, 72%; nervous or tense, 63%; poor sleep, 62%; poor concentration, 61%; sad, 61%; temper problems, 53%; headaches, 52%; memory problems, 51%; dizziness, 42%; extra sensitive to noises, 40%; nausea, 38%; and difficulty reading, 36%.[44] Similarly, in a study of 85 adults (63 females) without head injury, other identifiable neurological diseases or psychiatric diseases with a mean age of 33.9 years, the following percentages endorsed these symptoms from

the Rivermead Post Concussion Symptoms Questionnaire as present during the past 24 hours: poor concentration, 59%; forgetfulness, 59%; fatigue, 54%; sleep disturbance, 51%; irritable, 44%; blurred vision, 41%; headaches, 40%; light sensitivity, 35%; dizziness, 32%; and depressed and tearful, 32%.[45] As another example in an older population, in women aged 45–54 years, 60% reported memory problems over the past 6 months, 60% reported headaches and 72% reported irritability.[46]

In support of this theory, a number of studies have compared patients with mild TBI to non-head-injured controls finding a high prevalence of the same symptoms in both groups, indicating a high base rate of symptoms in the general population.[47],[48],[49]

A prospective United States emergency room study of 339 consecutive patients followed over 12 months found that the strongest predictors of symptoms after mild head trauma are baseline mental and physical health status but postconcussion symptoms are not significantly influenced by the presence or absence of head trauma.[50] The link between persistent symptoms and minor trauma may be entirely related to attribution bias (see next section) although it is possible that minor trauma could increase symptoms in vulnerable individuals.

Expectation as etiology--Because patients expect PCS symptomatology after TBI, they and their physicians may mistakenly attribute their common base rate complaints to the head injury, when they are actually unrelated. At the same time, surveys of individuals with no history of head injury find that most people identify symptoms of PCS as expected after head injury.[51]

Chronic pain — Patients with chronic pain have symptoms of PCS at a rate similar to a comparison group of patients after head injury.[52],[53] Similar patterns of cognitive deficits may be seen in patients with chronic pain and PCS.[28] It is not clear whether this reflects a shared prevalence of psychiatric disorders among sufferers of PCS and chronic pain syndromes, suggests that PCS is a manifestation of a chronic pain syndrome, or reflects the ubiquitous nature of these symptoms.

Compensation and litigation — Studies demonstrate a relationship between persistent PCS and potential financial compensation.[54],[55],[56] Personal injury claimants without head trauma reported high rates of complaints consistent with PCS as compared to controls.[57] On neuropsychological testing, there is a dose-response relationship between an increasing amount of potential compensation and an increasing rate of failure on malingering indicators particularly in those who have suffered only a mild traumatic brain injury.[58] On the other hand, failure of patients to recover after claims are settled does not necessarily invalidate this theory, as a financial settlement may in fact reinforce illness behavior.

Hoge et al discuss misattribution of PTSD and depression as traumatic brain injury among U.S. solidiers as follows, “Lacking an accepted medical definition for postconcussive symptoms or impairment, the VA created a disability category called“residuals of TBI.” The 2008 federal regulation creating the category assigns a

40% disability to persons who have three or more subjective symptoms that "moderately" interfere with functioning or who have "objective evidence" of "mild impairment of memory, attention, concentration, or executive functioning resulting in mild functional impairment." The regulation ignores extensive literature demonstrating the strong association between compensation and persistence of symptoms after concussion."[36]

Malingering--Some patient with persistent PCS may be malingering. Potential indicators of malingering include premorbid antisocial and borderline personality traits, poor work record, and prior claims for injury; uncooperative, evasive, or suspicious behavior; inconsistencies in neuropsychological test performance; or engaging in activities inconsistent with reported deficits, having significant financial stressors, and lack of reasonable follow-through on treatments.[54,59]

Current controversy over post-traumatic headaches after head trauma

The percentage of patients with persistent headaches after the head injury is as high as 78%[60] after 3 months, 35% after 1 year,[61] 24% after 2 years,[62] and 24% after 4 years.[63] For those with complaints of persistent headaches, there is evidence to suggest that the trauma may not be responsible.

Inverse dose-response--Paradoxically, headache prevalence and duration is greater in those with mild head injury compared with those with more severe trauma.[64,65] similar to the finding of more PCS symptoms with mild trauma.[35] In addition, a systematic review found that the prevalence of all types of chronic pain is higher in those with mild traumatic brain injury (75.3%) than those with severe traumatic brain injury (32.1%).[66] A prospective United States emergency room study of 339 consecutive patients followed over 12 months found that the strongest predictors of symptoms after mild head trauma are baseline mental and physical health status but postconcussion symptoms are not significantly influenced by the presence or absence of head trauma.

Increased prevalence of migraine in US soldiers-- U.S. soldiers deployed to Iraq for one year have been reported to have a much greater prevalence of migraine (17.4% of males and 34.9% of females in the prior year) than civilians which may be due to psychological and physical stresses of a combat environment triggering migraine in susceptible individuals. [67] According to the International Classification of Headache Disorders second edition post-traumatic criteria, the onset of chronic posttraumatic headache attributed to mild head injury should develop within 7 days after the trauma. [68] If the onset of the de novo migraine is greater than 7 days, then the migraine should not be diagnosed as post-traumatic. When patients are questioned some time after the injury about the exact time of onset, there is the possibility of recall bias as well in military and civilian patients.

Base rate levels, litigation, and stress--Persistent PTH has been challenged as due to pre-existing headaches or litigation. Lithuania was selected to evaluate post-concussion syndrome and post-traumatic headache outside the medico-legal context because there

are minimal possibilities for economic gain as fledgling insurance companies do not recognize post-concussion syndrome and because there seem to be less expectations of persisting symptoms than in a western society.[41] Mickevičiene retrospectively sent questionnaires 22-35 months after presentation to 200 emergency department patients in Kaunas, Lithuania, who had mild headache injuries with loss of consciousness for less than 15 minutes and received 131 survey responses.[69] All of the mildly head injured had headaches acutely which had disappeared in 96% within one month. Compared to controls, there was no significant difference of subjective cognitive symptoms. There was no specific effect of the head injury when various definitions and different constellations of core symptoms of PCS were used. This study questions the validity of PCS as a useful construct and is consistent with a non-traumatic etiology for those with persistent headaches.

Mickevičiene et al then prospectively evaluated 300 subjects presenting to the emergency department with a mild head injury for 1 year in Kaunas, Lithuania with questionnaires and obtained an initial response rate of 72%. [70] The prevalence, frequency, and visual analogue scale scores of headaches both after 3 months and after 1 year did not differ significantly between the injured and the controls. After 1 year, most symptoms did not differ between the injured and the controls with the exceptions of slightly significant differences of more sporadic memory problems, concentration problems, and dizziness in the injured. The authors conclude that, “our results cast doubts on the validity of postconcussion syndrome as a useful clinical entity, at least for head injuries with loss of consciousness for <15 minutes.” Thus, no litigation, no expectation of symptoms, no post-concussion syndrome.

Stovner et al recently reported the pooled results from an analysis of unpublished information on headache features and diagnoses in both the concussed patients and in the control patients from these two Lithuanian retrospective and prospective studies.[71] Headache present 3 months and 1 year or more after head trauma with loss of consciousness for less than 15 minutes had the same prevalence and prognosis and was of the same type and severity as headache occurring after minor trauma not involving the head and neck. There was an inverse association between the duration of loss of consciousness and the headache severity. It is unlikely that a head or brain injury is the cause of a headache 3 months or more after injury. The headache may be an episode of a primary headache induced by the stress of the situation.

Current controversy over chronic whiplash injuries and headaches

Crowe, an orthopedist, is often cited as coining the term "whiplash" during a lecture in 1928;[72] however, the first use I have found in the medical literature appeared in an article by another orthopedist, Davis, in 1945.[73] In a 1956 article discussing

whiplash injuries and litigation, Gotten commented that some patients used the injury as a “convenient lever for personal gain.”[74]

Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck that may result from rear-end or side-impact motor vehicle collisions. The sequelae of whiplash injuries include neck and back injuries, headaches, dizziness, paresthesias, weakness, cognitive, somatic, and psychological symptoms, visual symptoms and rare sequelae.[75],[76] In 2007, there were 10,600,000 motor vehicle accidents, including 3,350,000 rear-end collisions, in the United States.[77] No reporting system exists and, thus, the actual number of whiplash injuries per year is unknown; however, if Dolinis' finding that 35% of Australian drivers in rear-end collision sustained whiplash injuries,[78] then more than 1 million persons in the United States may have whiplash injuries yearly.

In one prospective study, 82% of patients reported acute headaches from whiplash injuries.[79] In another prospective study, the following percentages of patients reported complaints of neck pain and headaches, respectively, at various times after the injury: 92% and 57%, 1 week; 38% and 35%, 3 months; 25% and 26%, 6 months; 19% and 21%, 1 year; and 16% and 15%, 2 years.[80],[81]

As controversial as PCS, there are non-organic explanations to account for some with persistent neck pain and headaches[82]including psychological factors[83],[84] and stressful life events,[85] social and peer copying,[86] culturally mediated early expectations of recovery which result in different outcomes,[87],[88] and secondary gain and malingering.[89],[90]

The medico-legal setting and claims are important risk factors for pain complaints. When the tort compensation system in Saskatchewan, Canada was changed to a no-fault system without payments for pain and suffering, the number of claims decreased by about 25%.[90] Persistent complaints of those involved in low-speed, rear-end collisions are not seen in volunteer subjects exposed to speed changes from 4 to 14 km/h.[91] Even a false or placebo rear-end collision without extension/flexion of the neck may give rise to head and neck pain.[92]

Lithuania is also an advantageous location for whiplash studies because there has been little awareness among the population of the idea that chronic symptoms may result from rear-end collisions, those with acute symptoms generally view this as a benign injury not requiring any medical attention, and there is no possibility of economic compensation as there is no insurance system.[93] Chronic pain and headaches were no more common in 202 accident victims than in controls in a retrospective study of chronic symptoms after rear-end motor vehicle accidents reported to police in Lithuania.[94],[95] Another prospective study of 210 persons involved in rear-end motor vehicle accidents in Lithuania was performed.[96] Headache in the whiplash group had the same prevalence, the same diagnoses and characteristic features, and the same prognosis as the uninjured controls indicating that the headaches are primary headaches probably elicited by the stress of the situation.

Conclusion

On the title page of his 1882 book,[15] Erichsen quotes Montaigne, “Je raconte, je ne juge pas.” [I tell, I do not judge.] However, if persistent symptoms including headache are uncritically accepted as causally related to mild head injuries and neck trauma, then many other possible explanations may be overlooked including base rate misattribution, psychogenic, psychosocial, sociocultural, expectation as etiology, and compensation and litigation.

By the end of the nineteenth century, most physicians had come to believe that railway spine was not organic.[97] In a letter to one of his defenders, Texas physician R.M. Swearingen, in 1896, Erichsen wrote, “At that time [1866], the pathology of the nervous system and injuries was very imperfectly understood, and even the nomenclature had not been invented. ‘Neurosis’ and ‘neurasthenia’ even, were unknown terms, and what I then, for want of a better name, called ‘concussion of the spine,’ is now universally recognized and described under the more modern appellation of ‘traumatic neurasthenia.’”[98] Do we need more modern appellation in the early twenty first century? As reviewed, PCS, whiplash injuries, and post-traumatic headaches have been controversial for some 150 years, and, this Texas physician believes, will continue to be so for many years to come.

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