

Vol. 8, No. 2 May 2001

## Masquerades of Brain Injury

### Part I: Chronic Pain and Traumatic Brain Injury

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## INTRODUCTION

Traumatic brain injury (TBI) is a prevalent but poorly understood health problem. Mild TBI (MTBI), often referred to as concussion, accounts for approximately 80 percent of the reported 373,000 cases of TBI that occur each year in the United States<sup>1</sup>. The most frequent cause of MTBI is motor vehicle accidents and its victims are typically young males 15 to 24 years of age. Post-concussion sequelae may impede physical, emotional, social and vocational functioning.

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MTBI has been defined as a traumatically induced physiological disruption of cerebral function, as manifested by **at least** one of the following: 1) loss of consciousness of no longer than 20 minutes; 2) any loss of memory, either retrograde or anterograde; 3) any alteration in mental status at the time of the accident, even in the absence of loss of consciousness or amnesia; 4) physical symptoms which are potentially related to brain dysfunction (e.g. nausea, headache, dizziness, tinnitus, visual aberrations, olfactory deficits, or extended periods of fatigue); and/or 5) development of post-traumatic cognitive deficits which cannot be completely accounted for by emotional factors. By definition, severity of injury must not exceed the following parameters in order to qualify as "mild": 1) Glasgow Coma Scale score of 13 to 15 without worsening; 2) post-traumatic amnesia of  $\leq$  24 hours; and 3) loss of consciousness  $\leq$  30 minutes<sup>3</sup>. Generally, individuals with intracranial lesions and those with lower GCS scores (e.g. 13 or 14) are at risk for poorer outcomes<sup>2,4</sup>.

Importantly, the diagnosis of MTBI should take into account the following information: clinical history from self report and medical records, temporal relationship between symptoms and injury, nature of post-concussive complaints vis-a-vis expected symptomatology, corroboration by rescue squad, hospital and other "non-invested" collateral sources, degree to which symptom improvement corresponds to natural history of neurologic recovery, and neurodiagnostic results including neuropsychological testing<sup>1</sup>. Within this context, lawyers and clinicians should understand that the preponderance of evidence to support a working diagnosis of MTBI rests initially on the subjective history elicited by the examining clinician. Subsequently, neuropsychological assessment may offer the primary and presumptively most sensitive evidence in the diagnosis of MTBI.

A diagnosis of MTBI can have far reaching implications in terms of financial, vocational, treatment and disability status. Given that neuropsychological tests and interview data on post-concussive symptoms generally serve as the primary sources of evidence for diagnosing MTBI, the validity and utility of neuropsychological assessment procedures must be assured. However, there is increasing recognition that diagnosis of brain injury and attribution of symptoms to a concussion are complicated by the following:

1. Many post concussive symptoms have a relatively high base rate in individuals without brain injury and symptom reports may be unreliable<sup>5,6</sup>;
2. A meta-analytic review of studies suggest that persistent cognitive sequelae following MTBI have a low incidence<sup>7</sup>;
3. Multiple factors, aside from brain injury, can negatively affect neuropsychological test performance and produce symptoms similar or identical to those seen with post concussive disorders (PCDs);

4. Misattribution of symptoms to brain injury can reinforce negative expectancies and have a self-perpetuating effect.

In the Binder, Rohling & Larrabee study<sup>7</sup>, meta analytic statistical procedures were computed for 11 independent samples across eight studies to examine the long term neuropsychological effects of MTBI. Depending upon the method used to estimate variance, MTBI associated impairment at least three months post injury was found to be in the range of three to eight percent. These findings offer strong evidence that persistent sequelae following MTBI are relatively uncommon.

Further, numerous factors have been identified which can interfere with or even impair cognitive functioning and performance on neuropsychological tests, independent of recent brain injury. For example, Sbordone and Purisch<sup>8</sup> identified the following factors: prior brain injury or insult; congenital or pre-existing neurological conditions; partial complex (and other) seizures; pain and/or symptoms secondary to physical injuries; peripheral sensory or motor deficits; current and chronic medical illnesses; sleep deprivation and/or excessive fatigue; alcohol/drug abuse; medications; psychiatric illness; significant recent psychosocial stress; suboptimal motivation and/or malingering; negative interactions with the examiner, cultural/linguistic factors; vocational/avocational background; sophistication with test procedures and practice effects. In addition, a number of other factors associated with premorbid or preinjury ability structure (e.g., normal variability in cognitive strengths or weaknesses) might be cited. Such factors represent potential confounds in the interpretation of neuropsychological test results or the diagnosis of brain injury and require a differential diagnosis.

Mistaking the effects of such conditions as mood disorders, sleep disturbance and chronic pain for those of brain injury sequelae results in inadequate treatment of generally reversible conditions. Furthermore, they may be interacting with or potentiating the effects of brain injury symptoms to increase functional impairment. Methodical neuromedical and neuropsychological assessment can help differentiate sequelae of brain injury from confounding clinical conditions<sup>9</sup>. The identification of confounding clinical conditions such as chronic pain sequelae can minimize unnecessary medical costs, prolongation of inappropriate treatment, and eventual failure at intervention that produces helplessness and unnecessary chronic disability. The negative expectancies and symptom reinforcement arising from misdiagnosis (i.e., "nocebo effect"<sup>10</sup>) violate the primary ethical responsibility of health care providers to first "do no harm"<sup>9</sup>.

This article is the first in a series that reviews empirical evidence addressing the multiple factors that must be ruled out as part of differential diagnosis of brain injury. We will examine the effect of chronic pain on cognitive functioning and symptom presentation. Future articles will exam-

ine the effect of Psychiatric Disorders (Depression, Somatoform Disorders, Post Traumatic Stress Disorder), Response Bias and Malingering, Sleep Disturbance and Fatigue, Medication/ Drug Effects and Hormonal/ Metabolic Abnormalities.

## CHRONIC PAIN AND MILD BRAIN INJURY: IMPLICATIONS FOR ASSESSMENT

Pain, as defined by the International Association for the Study of Pain (IASP), is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."<sup>11</sup> Chronic pain is generally defined as pain that persists for more than six months. The literature regarding the effect of chronic pain on neuropsychological test performance has recently produced three reviews. These reviews reinforce the necessity of addressing issues of chronic pain in the context of MTBI. Each review employs a somewhat different focus and each is worthy of examination.

### REVIEW #1

Martelli, Grayson & Zasler<sup>12</sup> published a review on the effect of post traumatic headache (PTH), as well as chronic pain more generally, on neuropsychological test performance. PTH is the most common sequela following trauma to the head, neck or upper back and the most common post concussive symptom. Such trauma can be accompanied by a constellation of cognitive, emotional, and physical symptoms, including maladaptive responses to injury and to headache pain more specifically. PTH, which can occur with or without evidence of brain injury, has an incidence estimated as high as 90 percent<sup>13</sup>, with persistence longer than six months (i.e., chronic PTH) estimated as high as 44 percent<sup>14</sup>.

Martelli et al concluded that when specific, sensitive neuropsychological measures are employed, PTH is often found to exert a significant negative effect on neuropsychological test performance, at least for persons presenting with persistent subjective complaints. Decrements in information processing speed and complex attention are most frequently observed, while reductions in cognitive flexibility and verbal associative fluency, as well as learning and memory appear to represent secondary deficits which may be mediated by decreased information processing and attention.

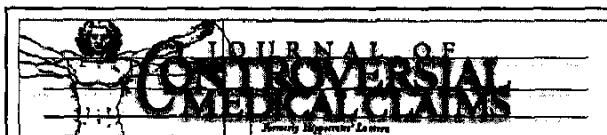
The authors also reviewed more general investigations of the effect of chronic pain on cognition independent of PTH. The studies reviewed showed that more chronic pain and more pain related symptomatology typically produce impaired performances on select neuropsychological tests and that abnormal SPECT findings are present in persons with many chronic pain syndromes. The pattern of neuropsy-

chological impairments appeared similar in many respects to that produced by MTBI. The authors cautioned that given the overlap in neuropsychological impairments associated with chronic pain and MTBI, chronic pain and especially PTH pose a special challenge to differential diagnostic formulation.

Given the high incidence of PTH, the validity and utility of neuropsychological test based inferences regarding brain injury necessarily depend on assurances that the effects of chronic headache and other chronic pain symptoms are taken into consideration.

### REVIEW #2

Nicholson<sup>15</sup>, in an effort to examine the controversial nature of persistent post concussive symptoms following MTBI, surveyed the literature concerning the relationship of pain, cognition, and TBI. Again, it was noted that headache is the primary complaint in virtually all surveys of post-concussive disorders. It was concluded that cognitive difficulties are common in patients with either acute or chronic pain, with or without indication of brain injury.



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Pain was found to interfere with aspects of performance in six of six studies assessing the effect of an acute pain challenge with normal controls. In some cases, the effect was marked. Cognitive impairments were found in eight of nine studies of primary headache pain (i.e., not due to trauma or other specific causes) and without evidence of TBI. Cognitive difficulties were also evident in several other studies of chronic pain not involving headache and without indication of any TBI or other brain injury. Finally, several studies were reviewed that more directly assessed the relative importance of pain versus TBI in determining neuropsychological impairment. Many of these indicate that pain is as significant or more significant than TBI in determining neuropsychological impairment.

The detrimental effect of pain on neuropsychological test performance, acute or chronic, with or without any possible associated brain injury, appeared most evident on aspects of attention, memory, speed of processing, and executive control. It was stressed that such effects are similar to that which have been described for traumatic brain injury. Concerns were raised about the problem of differential diagnosis, especially in cases involving suspicion of TBI without clear supporting medical documentation and a concomitant pain problem. It was suggested that previous studies of TBI, particularly ones examining the persisting post-concussive syndrome, may have been confounded by pain related problems. It was further suggested that problems discriminating cognitive — behavioral effects of brain injury from other factors such as chronic pain, especially in MTBI, potentially limits the utility of neuropsychological assessment.

Numerous functional neuroimaging studies were cited indicating that pain, acute or chronic, may result in disruption of brain processes. Such disruption was assumed to resolve on resolution of the pain problems. Note was made of the considerable variability evident within or between studies and the possible confounding effect of associated problems such as fatigue, depression, anxiety, medication side effects, or other factors. It was stressed that not all pain patients complain of pain and that many appear to have no apparent cognitive problems. Need for detailed assessment of pain problems, especially psychological aspects, and further study of these issues, was emphasized. Finally, it was suggested that onset, maintenance, exacerbation or severity of pain problems may be related to a process of central sensitization associated with psychological factors or pre-existing vulnerability<sup>16-18</sup>.

### REVIEW #3

Hart, Martelli and Zasler<sup>19</sup> conducted a review of the effect of chronic pain on neuropsychological functioning that primarily emphasized on patient groups without a history of TBI or neurologic disorder, in order to help clarify the contribution of persistent pain and associated symptoms to observed impairments. Despite methodologies that gen-

erally limited the ability to differentiate the relative contribution of chronic pain and associated symptoms such as sleep disturbance, mood change and fatigue, the reviewed studies supported the conclusion that neuropsychological impairment is often apparent in patients with chronic pain. Tests of attentional capacity, processing speed and psychomotor speed appeared to be particularly sensitive to the disruptive effects of chronic pain.

The authors included a summary table of clinical studies that included information on clinical characteristics, comparison groups, medication status, litigation status, pain ratings, measures of emotional status, and tests which were found to be sensitive vs. insensitive to performance decrements in pain patients and/or to pain intensity level. Studies of patients with whiplash injuries (without head trauma) and studies comparing pain patients to a TBI sample were included.

The studies reviewed tended to show that cognitive impairment was associated with higher pain intensity and involvement of head and neck areas (e.g., symptoms referable to a "cervicoencephalic syndrome".) Studies tended to support an association between cognitive impairment and other concomitant symptoms of pain such as mood change, increased somatic awareness, sleep disturbance, and fatigue. However, they concluded that further studies are needed to clarify the variables that mediate the impact of pain on neuropsychological functioning and the unique role of various symptoms often associated with chronic pain.

### SUMMARY AND IMPLICATIONS

The three reviews presented in this paper support the conclusion that pain and pain related symptomatology can and often do produce impaired performances on neuropsychological tests, especially measures assessing attentional capacity, processing speed, psychomotor speed, and executive functions. Considered together, they demonstrate that the pattern of neuropsychological impairment appears quite similar to that in persons sustaining MTBI. Recent findings functional neuroimaging of regional cerebral blood flow abnormalities in persons with chronic pain are consistent with observed decrements in cognitive functioning. The implication for differential diagnosis, especially in cases of putative MTBI is that pain and its concomitants must be considered as a possible contributing or explanatory factor.

It should be emphasized that these reviews certainly do not indicate that chronic pain always causes cognitive impairments, nor that neurophysiologic changes are irreversible or anything more than pain reactive changes. Further, symptoms often associated with chronic pain, such as depression and sleep disturbance, as well as premorbid coping vulnerabilities, likely play a predominant role in mediating the impact of chronic pain on cognitive functioning.

Nonetheless, the potential impact of chronic pain presents a challenge to the validity and conclusions based solely on

neuropsychological data, especially in situations involving differential diagnosis in patients with presumptive MTBI. Research findings have several important implications for the neuropsychological evaluation of patients who have chronic pain as one of their presenting complaints. The available findings indicate that chronic pain and its concomitants represent a source of performance variance and that caution is warranted in interpreting decrements in neuropsychological test scores as signs of neurologic sequelae of brain disease or injury in patients with chronic pain.

A discussion of clinical approaches to pain assessment is beyond the scope of this paper. However, the following recommendations have been adapted from the Hart et al review<sup>20</sup> plus chapters and articles on pain assessment<sup>22-24</sup>. They are offered in order to increase clinician's awareness of this issue and possibly minimize the confounding effects of chronic pain on test performance.

- I. Clarifying the presence and intensity of momentary pain (i.e., at the time of an evaluation) is inadequate. The concomitants of chronic pain appear to play the more important role.
- II. Symptom checklists that include complaints often associated with chronic pain (e.g., fatigue, sleep disturbance, depression) may be helpful, and even indicated in many cases. Given limited validation of many of these measures, efforts to collect corroboratory data from family members or others is advised. Further, caution should be taken to limit potential sensitizing effects or encouragement of symptom focus, which could result in over reporting in patients who are already somatically focused. The repeated administration of a sustained, attention-demanding, timed test at the end of a session may help identify or corroborate possible fatigue-related deficits.
- III. Importantly, pain and its associated symptomatology are sometimes overlooked, not treated aggressively, or treated inappropriately. Consideration should be given to postponing cognitive assessment in cases where pain and related symptomatology have not yet received specific and/or appropriately aggressive treatment focus.
- IV. The effect of pain on sleep is an especially important consideration. Sleep is a potential moderator variable for the effects of chronic pain, as sleep deprivation has been found to impair cognitive performance [see <sup>27</sup> for a recent meta analytic review]. Improved sleep hygiene, and pharmacologic as well as non-pharmacologic treatments (e.g., cue controlled relaxation procedures) may be appropriate prerequisite interventions prior to completing neuropsychological evaluation<sup>20</sup>.
- V. In situations where pain persists, whether or not there has been appropriate treatment efforts, consideration should be given to adjusting test procedures to ensure optimal comfort and minimize emotional distress,

e.g., ensuring the most comfortable sitting position and optimized ergonomics, providing frequent breaks, allowing frequent standing or changing of position, modifying lighting conditions, instructing patients to bring any orthotics, cushions, heating or ice pads.

- VI. Measures to assess motivation and response bias seem indicated, not necessarily to identify malingering, but to help gauge the effects of chronic pain on the patient's ability to sustain optimal or near optimal effort. Given evidence that detrimental effects of chronic pain on cognitive performance may be related to increased somatic awareness and emotional factors<sup>15-21</sup>, standard measures of mood and emotional-personality functioning are important. Identifying emotional suffering, negative illness-related beliefs, and lifestyle interference that seem disproportionate to pain intensity should increase the level of caution in attributing performance decrements to brain dysfunction. For this reason, neuropsychological assessment of patients with chronic pain should sometimes include pain-specific evaluation techniques such as visual analogue scales to assess pain intensity, concomitant negative emotions and pain-related beliefs [e.g.,<sup>22-24</sup>]. As appropriate, pain behaviors including degree of lifestyle disruption and possible secondary gain can be assessed using self-report inventories and observation methods such as the Psychosocial Pain Inventory<sup>25</sup>, the Multidimensional Pain Inventory<sup>26</sup> and the Pain Assessment Battery — Research Edition<sup>27</sup>. Notably, the latter includes subscales for both "Extreme Beliefs" and "Symptom Magnification". Additionally, instruments such as the Kinesiophobia Scale and the Cogniophobia Scales [see <sup>22-24</sup> for tests and review] are useful for identifying pain related phobias and avoidance conditioning. These instruments have been designed to assess pain and anxiety based avoidant behavior in regards to physical and cognitive exertion, respectively; high scores can be expected to result in reduced effort on physically and/or cognitively demanding tasks, respectively. Anxiety and interfering "mind sets" may be ameliorated through therapeutic procedures such as psychoeducation, graduated exposure and cognitive reinterpretation.
- VII. Litigation is a variable that influences symptom report and test performance, and should always be considered in interpreting neuropsychological test performance<sup>22-24</sup>. Pain inventories that address issues of secondary gain and motivation should be employed in all evaluations. These should address cognition and pain, in addition to various other complaints, and tests of response bias should especially be employed when the patient is in litigation or seeking wage replacement benefits.
- VIII. In general, the clinician should be prepared to assess (or refer for appropriate assessment) chronic pain and its concomitants when the complaint is salient

and the limitations in everyday functioning and decrements in test performance seem atypical for the neurologic condition, or there is reason to suspect that successful adaptation is likely to depend upon coping with pain-related symptomatology.

According to Miller<sup>28</sup> chronic pain often represents the "weak link" in "postconcussion invalidism". Given the prevalence of chronic pain following TBI, especially MTBI, resolution of the post-concussion syndrome and successful adaptation may frequently depend on success in coping with PTH and other chronic pain symptomatology. As Devore<sup>29</sup> notes, even in the case of pain with comorbid psychological problems, management of symptoms is often successful provided that appropriate psychological consultation is provided.

In the paper by Martelli et al<sup>9</sup>, the most effective treatments currently employed in the treatment of headaches and post traumatic headaches are reviewed. Other sources more germane to other specific pain syndromes, as well as chronic pain generally, and its associated symptoms (e.g., sleep disturbance, depression), are available to assist with modulating symptoms that confound valid assessment of neurologic impairments. In almost all cases of chronic pain, multicomponent or combination treatments that also address contributing problems (e.g., reactive mood change, increased somatic focus, fatigue from deconditioning and sleep disturbance) offer the best hope for minimizing psychological and social dysfunction.

*Editor's note: This article is the first in a continuing series on brain injury.*

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This article may be the reader's first introduction to "nocebo". Nocebo is the converse of Placebo. Just doing anything may have beneficial results because of the patient's expectation, so for the same reasons interventions can also have a negative effect. This is why reassurance in one of the few things found to actually affect the outcome of acute low back pain, and consequently the absence of reassurance is likely to be associated with a poor outcome in low back pain. Most hospital emergency room discharge instructions to patients include language reassuring the patient that a good outcome is likely in muscle sprain/strain. This is even more important in potential TBI, where the symptoms are even more difficult to verify. Whether they realize it or not, lawyers can influence patients and their expectations, positively or more commonly negatively.

In any trauma situation the staff at an emergency room will screen for potential brain injury at the outset, and you should see that within the first or second line of the report. The EMS report will include a GCS. This information is important in assessing the likelihood of a brain injury. It is quite common to have the patient's report of symptoms increase after he or she has time to talk to friends and attorneys, but it is critical to the ER's mission to record this information. A notation of no loss of consciousness, or a GCS of 15 in the ER, is most probative of the degree of trauma, non-contemporaneous self reports.

The papers reviewed in this article demonstrate that the neuropsychological tests are not useful in MTBI. When CT and MRI were first available they were used on symptomatic patients and when changes in the spine were identified, it was thought that the finding explained the symptoms. Then as more people were imaged and the facilities were made available to image asymptomatic persons, the significance of the imaging finding significantly limited, to the point that the authorities recognized the nocebo effect of doing extensive imaging studies in the absence of clinical "red flags". Now Hart et al have performed neuropsychological studies on people without brain injury. Apparently the studies to person without brain injury show results that would be interpreted as TBI if the extremely low threshold for TBI had been met.

As a skeptic I would have to say, if the neuropsychological studies will show brain injury in person that are shown not to have had brain injury, then they are useless for establishing TBI. The neuropsychological batteries were really developed to quantify the extent of impairment in patients with documented brain injury. There are studies showing the Halstead-Reitan tests as highly accurate in identifying brain injury in cases of severe injury, but in studies with less severe injury, the H-R has been shown to have a much lower rate of accuracy. Faust, et al. *Brain Damage Claims: Coping with Neuropsychological Evidence*. Law and Psychology Press (1991), MTBI is exactly the kind of situation where the studies have been shown if be of low accuracy.

If you suspect on non-biological cause of the symptoms a simplified inquiry might go like this:

Q: Doctor, isn't stroke by definition a brain injury?

Q: Don't persons who survive a stroke begin to show improvement in symptoms beginning shortly after the stroke?

Q: Isn't it true that once the healing begins the symptoms rarely worsen without another stroke?

Q: In this case has the patient shown the recovery patterns expected of a actual brain injury?

Q: Isn't it true that functional condition show a wide fluctuation of symptoms over time?

Q: Doesn't the patient's record show a pattern consistent with a functional problem?