

## MINOR TRAUMATIC BRAIN INJURY: REVIEW OF CLINICAL DATA AND APPROPRIATE EVALUATION AND TREATMENT

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### Abstract

The clinical entity of minor traumatic brain injury (MTBI) is secondary to signs and symptoms encompassing neuropathological, neurochemical, neurobehavioral, neuropsychological and behavioral deficits. The patients who suffer this disorder are often given little help, medically, secondary to issues regarding the perceived reality of the disorder. A few individuals deny the existence of MTBI. Some believe the symptom complex to be strictly functional, while others believe that spontaneous recovery will occur and no treatment is necessary.

When discussing traumatic brain injury the descriptors, "mild, moderate, and severe," are used to describe the severity of the acute injury. These labels do not describe the severity of the sequelae nor are they indicative of the intensity of specific treatment. A clear understanding of MTBI, its sequelae and necessary treatment is imperative to insure timely intervention. Delay or lack of early intervention appears to be responsible for "persistent sequelae" in MTBI.

This paper will describe various aspects of the etiology of MTBI, with recommended evaluation and treatment guidelines. A functional assessment scale specifically for persons with MTBI is also presented. Several case histories are included for illustration purposes.

### Introduction

Mild traumatic brain injury (MTBI) is the clinical entity in which the brain has sustained pathological injury. The pathology can be a direct contusion, neurochemical, axonal or circulatory.

The terminology Minor TBI is called into question. It is noted that MTBI may induce neuronal dysfunction which produces persistent symptoms, indicating that mild injuries to the brain may produce effects which are not "minor" and which may last for indeterminate periods of time.<sup>1</sup>

A consensus definition of mild traumatic brain injury has been published by the members of the mild traumatic brain injury committee of the brain injury interdisciplinary special interest group (BISIG) of the American Congress of Rehabilitation Medicine.<sup>2</sup> The definition states:

"A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by at least one of the following:

1. any period of loss of consciousness
2. any loss of memory for events immediately before or after the accident
3. any alteration in mental state at the time of the accident (e.g. feeling dazed, disoriented or confused)
4. focal neurological deficit(s) that may or may not be transient

The severity of injury does not exceed:

1. loss of consciousness of approximately 30 minutes or less
2. after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13 to 15 is found
3. post-traumatic amnesia is not greater than 24 hours"

Of great importance is that the definition includes patients with direct head trauma as well as those who suffer an acceleration/ deceleration injury "whiplash," without direct head trauma. Loss of consciousness is not a clinical requisite for a classification of MTBI.

The members of the BISIG note that symptoms of MTBI may last for varying lengths of time and can consist of persistent physical, emotional, cognitive, and behavioral symptoms which may produce a functional disability.

Zasler<sup>3</sup> stated, "Clinicians should remember that gross absence of proof is not necessarily proof of absence. In unsophisticated hands there may be no evidence whatsoever that someone has had a significant injury, whereas in different hands and to other eyes, the patient may indeed have objective examination findings clinically as well as neurodiagnostically."

These issues prompt the need for guidelines in the diagnosis, treatment and management of the patient with MTBI. It is hoped that the guidelines presented in this article for outpatient treatment of post-acute MTBI patients may be used as a starting point.

Guidelines, in and of themselves are meaningless, unless one has a good understanding of what the guidelines are meant to do. No group of patients is totally homogeneous in terms of their needs, without a clear understanding of MTBI, the guidelines are virtually worthless. Therefore, a mini "state of the art review" of MTBI will be presented. The authors have attempted to be succinct in reporting the facts as they are known.

### Clinical aspects of MTBI

There are an estimated 400,000 to 500,000 individuals hospitalized in the United States each year for head trauma.<sup>4,5</sup> Patients with MTBI who are hospitalized number approximately 131 to 150 per 100,000 in this country. Between 60 to 82 percent of all admissions to the hospital are for head trauma.<sup>6,7,8</sup> These numbers do not include those patients who visit an emergency room secondary to trauma and have suffered an injury to their brain. The number of people who experience a minor concussion and/or head trauma who never make it into an emergency room is thought to be significantly larger, possibly as high as 2,000,000 per year.<sup>9</sup> Economically, the cost is felt to exceed \$3.9 billion per year.<sup>10</sup> This number does not account for the non-economic costs of vocational, familial and social morbidity.

Risk factors for persistent deficits, appear to include patients over 40 years of age, lower educational, socioeconomic status, alcohol and drug abuse, and multiple minor head injuries, even without previous sequelae.<sup>11</sup> Multiple trauma to the head can be cumulative, resulting in the sequelae of brain injury. These facts account for the term categorizing patients with MTBI as the "silent epidemic."<sup>12</sup>

Postconcussive syndrome (PCS) refers to a large number of signs, symptoms which typically, in combination follow a mild traumatic brain injury. Many of these problems, medical, behavioral, cognitive and emotional, are noted below:

The most common medical problems found in the patient with MTBI include:

- Post-traumatic headache
- Post-traumatic musculoskeletal pain syndromes
- Vestibular disturbance
- Visual disturbance
- Fatigue

The most common cognitive, emotional and behavioral deficits include:

- Memory impairment
- Depression
- Irritability
- Anxiety
- Loss of self-esteem
- Job loss/disruption
- Denial
- Difficulties with social interactions and family relationships
- Lack of initiative
- Word finding problems
- Decreased ability to concentrate
- Poor impulse control
- Slowed information processing
- Behavioral/personality changes
- Perseveration

It is possible to discern some of these deficits on a basic neurological or psychological examination. However, many are not found unless specific testing is performed. Therefore, an appropriate evaluation must be performed to identify them.

Table One lists more of the typical cognitive, behavioral and emotional problems/deficits frequently found in patients who have suffered a MTBI.

It is important to note that many, if not most of the problems listed will not be found in the typical MTBI patient unless the correct questions are asked, and appropriate testing is initiated. The MTBI patient may not necessarily even be aware of many of these deficits. It is therefore important that the patient's family, friends, significant others, etc., be consulted for additional history. Many times a patient will deny a problem simply because denial is one of the symptoms which may be inherent in a MTBI, as well as being a specific trait of certain personality structures: therefore, it is not appropriate to state that a patient has no sequelae secondary to a minor traumatic brain injury, solely on the word of a patient suffering from the problem. This is rarely an issue.

This, again, underscores the need for a complete and appropriate evaluation in patients suspected to have a minor traumatic brain injury. The MTBI evaluation is discussed in detail below.

For years the debate of the very existence of sequelae of MTBI has leaned toward the perception of psychogenic problems or symptom magnification. As the onset of symptoms of MTBI may occur several days or more after an injury, the terms malingering, along with psychogenic, have been used in reference to such patients.

The existence of actual neuropathological changes, some of which may evolve gradually,<sup>13</sup> have placed many of these symptoms into proper perspective, in spite of the fact that, typically, cerebral MRIs and CAT scans are negative, at least initially.

Many MTBI patients show a great deal of distress over their symptoms which, if they are cognitive or behavioral, may be considered to be psychogenic. It is frequently not appreciated that these patients are aware of their dysfunction yet may lack a concrete manner to express it. These patients often hold much higher expectations for themselves when they are told that their injury was "mild," and become frustrated at the lack of attention shown to them. The same type of situation may be seen in the unfortunate patient with early Alzheimer's disease, when they know that their abilities are not the same as they were. They become frightened as they realize their deficits, which often go unnoticed by others, nevertheless remain significant to them, and impact their ability to function.

A significant problem is posed by those who believe that patients who have sustained a mild traumatic brain injury will spontaneously return to normal in a three month period. They reason that it is a waste of time and money to evaluate and treat them.

Some even feel that MTBI is self limited and treatment is essentially frivolous.

Current literature indicates that there are long-term complications for MTBI patients, in the cognitive, behavioral, emotional and vocational realms. Examination of MTBI patients at three months after injury shows that many have the same deficits in task performance and memory as did patients with moderate to severe injuries.<sup>12</sup> Studies have shown that while many patients with mild MTBI do exhibit spontaneous recovery in any number of aspects within three months of the injury, sequelae, including neuropsychological, may persist for up to three to five years after injury with resulting disability.<sup>14</sup> Some sequelae may be permanent.

Failure to correctly diagnose the deficits following MTBI, some of which may be subtle and difficult to detect, is most probably secondary to lack of expertise on the part of the evaluating physician, not secondary to fallacious complaints on the part of the MTBI patient.

### Pathophysiology of MTBI

Mild traumatic brain injury (MTBI) appears to be secondary to three major pathological features: 1) Acceleration/deceleration of the head 2) skull distortion with resultant pressure gradients intracranially 3) cervical spine stretching which induces myofascial problems along with autonomic nervous system difficulties in some patients.<sup>15</sup> Direct head trauma is not necessary in the etiology of MTBI. The speed, magnitude and direction of the acceleration/deceleration force is the determining factor in regards to extent and permanence of axonal dysfunction, as it is the inertial force transmitted from sudden deceleration which induces the diffuse axonal injury leading to MTBI. (Axons are the parts of the nerve cells which connect nerve cell bodies from the various parts of the brain to other areas. Axonal injury makes these cerebral connections faulty or impossible, physiologically, to maintain).

Focal cerebral lesions from a direct blow to the head may include cerebral contusion, laceration and hemorrhage, the latter leading to hematoma (abnormal collections of blood) in extradural, subarachnoid, subdural and intracerebral compartments. These findings may be secondary to coup (the area of the brain beneath a blow to the head) and contra-coup (trauma to the cerebral areas opposite to the initial blow) lesions.<sup>16</sup> Brain stem dysfunction, in addition to cortical involvement, has also been noted.<sup>11,17</sup>

The existence of diffuse axonal injury (DAI) following an acceleration/deceleration injury (also called "whiplash") may be associated with a normal neurological examination. Pathological changes seen on post mortem examination have been found to include DAI (Diffuse axonal injury).<sup>11,18,20</sup> Animal experimentation has revealed similar findings.<sup>21-22</sup>

There has been no established direct correlation between the various aspects of cognitive impairment after mild to moderate TBI. Contusions of the frontal and anterior temporal lobes are

frequently seen<sup>23</sup> and appear to be related to impairments of attention, response regulation and memory. DAI in the upper cortex and frontal-diencephalic cerebral systems may induce slowed information processing and decreased selectivity and diminished allocation of cognitive work, consistent with system deficits after MTBI.<sup>24,25</sup>

Another major cause of MTBI are the changes in the post traumatic neurochemical milieu. While Gennarelli noted the possible axonal injury induced by changes in calcium ions,<sup>26</sup> others have also noted changes in the neuroexcitatory neurotransmitters<sup>31</sup> including acetylcholine and glutamate. The surges in these neurochemicals after injury contribute to neuronal "burn out" or cell death compounding the clinical effects of DAI.<sup>27-35</sup>

The neurochemical changes which induce neurophysiological damage to brain cells also give a possible handle on the treatment of MTBI using appropriate neuropharmacological agents, including vasopressin, choline and scopolamine.<sup>36-35</sup>

Another site of injury at time of MTBI is disruption of the blood-brain barrier, which allows further damage to the brain secondary to the influx of circulating excitatory neurotransmitters.<sup>36</sup>

### Neurological testing

Unfortunately, despite the wealth of existing data regarding the various etiologies of the pathological deficits found to be secondary to MTBI, they are not found on MRI, nor CAT scan, thus making them invisible.

Still, there is more to the usage of cerebral CAT scans and MRI than is initially apparent. CAT scans are very useful to "rule out" acute intracranial pathology, particularly skull fracture and hemorrhage. The vast majority of research indicates that patients with negative examinations and a Glasgow coma scale (GCS) of 15, along with a negative CAT scan can be sent home for observation by family or friends. The patients with a skull fracture and no neurological signs may be sent home. It has also been noted initially negative CAT scans can be positive in 12 to 24 hours, secondary to hemorrhage, associated with a patient's clinical deterioration.<sup>37-39</sup>

Cerebral MRI consistently finds more intracranial lesions than CAT scans.<sup>57-58</sup> The MRI can locate discrete areas of cerebral edema not seen on CAT scan, as well as determine white matter lesions not found on the CAT scan.<sup>59,60</sup> One must remember that cerebral MRIs and CAT scans are static views of the brain and can only image pathology greater than two mm. There is also some indication that the MRI can better identify lesions possibly compatible with neuropsychological findings than CAT scan.<sup>61</sup>

Studies indicate that the performance of skull films in the emergency room are not necessary, due to the fact that CAT scans are more clinically helpful and patients are being sent home for observation even with skull fractures.<sup>48,62,63</sup>

SPECT (single photon emission computer tomography) scans<sup>64</sup> and cognitive evoked potentials (the P300 wave)<sup>65</sup> were also found to be potentially useful in the diagnosis of MTBI.

### Neurosurgical risk

Another important variable in the initial evaluation of patients with MTBI who are seen in the emergency room is the possibility of neurosurgical intervention, which occurs in one to five percent of patients who are "alert" on evaluation in the emergency room, with GCS of 13 to 15.<sup>15,66,67</sup>

It is known that the incidence of an intracranial complication is increased in patients found to have a skull fracture, the presence of which increased the chance of neurosurgical intervention by a factor of 20.<sup>15</sup>

In one study of 610 patients with GCS scores of 13 to 15, three percent required neurosurgical intervention.<sup>68</sup> Another study<sup>69</sup> identified 183 patients over a ten year period with GCS scores of 13 to 15 who required neurosurgical intervention. The authors concluded that an acute intracerebral hematoma can never be totally discounted in patients with acute MTBI, even when there were no abnormal clinical signs on evaluation.

This reinforces the clinical finding of a "lucid interval" being seen in patients found to be neurologically clear on evaluation, who then deteriorate.<sup>69,70</sup> It was noted that small numbers of patients may clinically deteriorate 24 to 48 hours after initial assessment, due to delayed traumatic intracerebral hemorrhage (DTICH). This is most frequently seen in association with coup- and contra-coup lesions of the parieto-occipital cortex which secondarily effects the fronto-temporal lobes.<sup>71</sup> The conclusion is a small group of neurologically clear individuals who have sustained MTBI experience late clinical deterioration in spite of a normal CAT scan performed on initial evaluation.

### Common neurological sequelae to MTBI

Another fact that belies the belief that MTBI is not a "real" problem, and will go away by itself within one to three months is that organically based, neurological problems follow MTBI. These include, but are not limited to:

- 1) Post-traumatic epilepsy,<sup>72-74</sup> both generalized tonic-clonic (grand mal) and partial complex seizures, the latter possibly more common and secondary to a partial kindling effect after minor traumatic brain injury. Some of these seizures may only manifest as intermittent behavior change (temporal lobe seizures without gross motor movement).
- 2) Post-traumatic migraine of various types, totally different from the more common post-traumatic migraine ("typical" migraine with aura and neurologically complicated migraine)<sup>75</sup> and post traumatic tension-type headache.<sup>76</sup> These include acute confusional migraine;<sup>77</sup> transient global amnesia secondary to post traumatic migraine;<sup>78</sup> post traumatic migrainous hemiplegia;<sup>79</sup> and changes in mental status secondary to post traumatic migraine.<sup>80</sup>
- 3) Post-traumatic vertigo is common, second only to post traumatic headache in frequency following MTBI, which can be secondary to soft-tissue, or myofascial etiologies or more central (brain stem or cerebellar) or peripheral etiologies (end organ or nerve).<sup>81,82</sup> The concept of cervicogenic dizziness has also been discussed.<sup>83-86</sup> Difficulties with smell are frequent,<sup>87,88</sup> as are sleep disorders with associated difficulties with sleep-wake cycles.<sup>89-92</sup> Post traumatic tremor has also been seen.<sup>93</sup> Significant difficulties with light and sound intolerance are frequently seen.<sup>94-97</sup> The syndrome of inappropriate secretion of ADH (anti-diuretic hormone), also called diabetes insipidus, has also been well documented following a MTBI.<sup>98-101</sup> Post traumatic tinnitus is common,<sup>102-103</sup> while post traumatic delayed nonhemorrhagic encephalopathy following MTBI<sup>104</sup> is not.

### Post concussion syndrome

The post concussion syndrome (PCS) has been described as far back as the 1860s by Erichson and Trimble.<sup>105,106</sup> Two decades later, Dr. Page expressed that all patients suffering from closed head, non definable injury were malingering.<sup>107</sup> During the 1940s through the 1960s research concluded that closed head injury and/or simple concussion could cause real neuronal loss and clinically profound changes.<sup>18,108,109</sup>

The early difficulties delineating cerebral dysfunction and resulting characterological changes were quite problematic.<sup>110-114</sup> When the neurological examination was essentially normal, post traumatic sequelae were thought to be fallacious.

In the decade of the 1960s, more research began to support Erichson's contention that minor head trauma could induce severe disturbances of cerebral function, even after acute cervical acceleration/deceleration injuries, or "whiplash."<sup>115-117</sup>

The PCS appears to consist of symptoms consisting of neurophysiological, neuropathological, and neuropsychological and psychological/emotional aspects secondary to a mild traumatic brain injury.<sup>118</sup> The PCS can be chronic and disabling, as well as short lived, with a possible explanation being the interaction between the organic and psychological factors.<sup>119</sup> Unfortunately, it is very difficult to differentiate between the effects of primary neurological/neurophysiological/neuropathological injury and secondary psychosocial factors. Some feel that the typical PCS symptoms (most commonly, headache, dizziness, irritability) result from emotional stress associated with diminished cognitive performance secondary to MTBI.<sup>120</sup> Although the PCS has been thought of as a reflection of the psychological response to injury, there is considerable evidence to suggest that the PCS is primarily a physiological disturbance.<sup>121</sup> Reaction time testing has been used to support a structural, organic etiology for the PCS.<sup>122</sup>

A major difficulty is nosological (science of disease classification) to determine exactly what constitutes the PCS. Evans<sup>7</sup> states that the PCS refers to the large number of signs and symptoms found alone or in combination following MTBI, including headache, dizziness, fatigue, irritability, anxiety, insomnia, memory deficits, as well as light and noise sensitivity. He notes that studies have substantiated the existence of the PCS, that it is common, with resolution in three to six months, but with persistent symptoms and cognitive deficits persisting for more months or years.<sup>7</sup>

The signs and symptoms of the PCS following a MTBI have been well documented.<sup>5,110,123-133</sup> As indicated, headache, memory problems and dizziness are the most common combination of PCS symptoms.<sup>134</sup> However, no specific symptom complex has been found in the majority of patients with acute or chronic PCS.<sup>135</sup>

McLaurin and Titchener<sup>135</sup> have suggested that the PCS should include all of the consequences of head injury, regardless of its severity and the nature of the injury.

Berrol<sup>136</sup> notes that of all the problems referred to as minor head injury, traumatic head syndrome, post concussive syndrome (PCS), post traumatic syndrome, post brain-injury syndrome and traumatic cephalgia, to name several, the term mild traumatic brain injury (MTBI) would be preferred, as it identifies the etiology of injury, its degree and the pathological substrate.

The term post concussive syndrome is still frequently seen in the literature. The nosological question to be determined is whether the PCS is secondary to the MTBI, or are the cognitive/neurological deficits found after MTBI a separate entity. The term PCS would thus encompass the non-neurological/neurocognitive and neurophysiological deficits leaving the term PCS to be used specifically for the other organ (non-cerebral) systems which display post-traumatic signs and symptoms.

Another question of great import, is whether or not the patients who have the PCS with neuro-cognitive deficits who do resolve within three to six months actually have the characteristic microscopic neuropathological changes found in acceleration/deceleration type injuries.<sup>137-139</sup> Or, do they have them but in lesser severity, or in areas of the brain which do not correspond to interpretation or identification by present testing procedures.

Immediately after MTBI, patients have complaints referable to several different organ systems. This has been identified by Alexander<sup>140</sup> as the PCS. He notes that the MTBI which can lead to brain injury, as the name implies, can also cause injury to the head, the neck (whiplash and soft tissue damage), the vestibular system, and psychological functioning. The initial complaints of deficits in cognition and sleep disorder are secondary to neuronal injury, while the headache may be secondary to cervical injury, neuronal injury or a combination; neck pain secondary to soft tissue problems; dizziness secondary to peripheral vestibular dysfunction or cervical injury; and the anxiety, moodiness

and irritability secondary to neurological injury, pain and/or psychological factors.

Chronologically, it is noted that neuronal recovery, to the extent plasticity allows, is certainly taking place at one month after injury.<sup>22,139,141-143</sup> Neurological recovery is thought to be "substantial" at three months.<sup>6</sup> At this time, patients with continued complaints range from 30 to 50 percent.<sup>144</sup> Over the next six to 12 months (greater than a year period from the initial injury) most patients will show continued improvement and "recovery."<sup>145</sup> It has been found, however, that even "well-recovered" patients, are still susceptible to periodic impairments secondary to physiological or psychological stress,<sup>141,146</sup> indicating that recovery is probably the wrong term; "compensated" for their injury may be more correct. That patients may have a permanent sense of decreased mental or cognitive efficiency<sup>147</sup> is also a function of the incorrect terminology, i.e. recovered vs. compensated.

### Does current nomenclature hinder treatment?

As noted above, questions arise regarding the definitions of MTBI and PCS. It has been stated that "minor" or "mild" traumatic brain injury is often not, clinically, "mild," as it may induce significant sequelae. Any injury to the most complex system in the human body, the brain, is rarely if ever truly "mild."

The descriptive terminology- mild or minor- by themselves indicates or expresses that the problem is relatively innocuous, unimportant or insignificant. A minor laceration is certainly not a cause for alarm, and possibly not even sutures are needed. Minor or mild abnormalities found in blood tests may mean nothing, clinically, and may in fact be a function of laboratory or test error.

When a patient is determined to have a "minor" or "mild" traumatic brain injury, the terminology itself may convey the wrong message to non-clinical personnel who are not experienced in dealing with brain injury. The classification emerges from the Glasgow coma scale scoring; minor or mild 13 to 15, moderate eight to 12, severe three to seven. The terms relate to the level of coma and not necessarily the severity of the sequelae.

### Persistence of symptoms

At one year, 85 to 90 percent of patients are felt to be "recovered" but are still symptomatic, with, therefore, 10 to 15 percent of patients<sup>133,148</sup> who are not only "not recovered," but also "not compensated" and still very symptomatic. The literature is replete with studies showing persistence of symptoms over the "magic" three month period. This literature indicates the symptoms and deficits following MTBI may indeed last for six to 12 months and even longer.<sup>7,11,60,136,147,149-153</sup>

In a survey of rehabilitation specialists who followed patients with MTBI for six to 18 months, they noted 21 percent of patients experience symptoms of the PCS two to six months after initial injury, and that 20 percent of these patients had the post-MTBI

syndrome.<sup>154</sup> In another survey of 51 patients, (23 responded) 25 percent reported continued sequelae from their injury. The patients with sequelae after one year were found to have reported more symptoms one week after the injury.<sup>155</sup>

Another report indicated that considerable evidence showed that PCS symptoms persisted in a significant proportion of patients after MTBI and were particularly prevalent in patients who indicated that they needed clinical intervention.<sup>156</sup> Memory, attention, information processing speed and efficiency were noted to be the typical cognitive domains effected by MTBI in these patients.<sup>157</sup>

It has been noted that symptoms with organic etiologies can mimic functional disorders.<sup>158</sup> Alves<sup>159</sup> noted that as recovery occurred, persistent symptoms can be an interaction between organic and psychosocial factors. These persistent symptoms are more than can be expected from the initial organic damage alone. Alves further states that a significant percentage of patients will exhibit persistent problems with symptoms 12 months post injury. He also stated that recovery from MTBI should also be considered in the social context in which it occurred. By recognizing the complexity of the recovery process, we should extend the concept of morbidity to include the specific socioeconomic, and emotional sequelae that the patient experienced.<sup>159</sup>

Mateer<sup>160</sup> described patients post MTBI as being more acutely aware of their cognitive deficits and problems with functional abilities. The patients go to a physician and are found to have a negative neurological examination. The patients are told that there is no organic reason for their problems. They should wait longer for recovery, learn to live with their problems, or to seek psychiatric help. These iatrogenically induced problems most likely lengthen the symptomatic period, as the patients begin to feel an ever increasing loss of control, fear of the unknown and concern that they may be "going crazy."

### **Persistent post concussive syndrome**

Alexander<sup>140,161</sup> has written extensively about the persistent postconcussive syndrome (PPCS), which might also be thought of as MTBI with Persistent Sequelae. It is worth reviewing the main aspects of his excellent work, while looking a bit more closely at some of his conclusions.

PPSC patients, after one year, continue to have symptoms commonly seen in acute PCS, such as headache, dizziness, sensory hyperesthesias and cognitive symptoms, including deficits in attention, memory and executive functioning. Also noted are prominent emotional symptoms of irritability, depression, nervousness, discouragement and anger.

Alexander<sup>140</sup> notes that there may be some "predictors" to the development of the PPCS, including the female sex, litigation, low socioeconomic status, prior MTBI and headache, along with serious associated systemic injury. These factors, he notes, are

implicated, but none accounts for more than a small percentage of cases of PPCS.

Several other authors also identify pain severity post injury as a predictor of the development of the PPCS post MTBI.<sup>161-163</sup> There is data to suggest a greater frequency of anxiety and depression months after initial injury.<sup>164</sup>

It is noted that peripheral vestibular injury with dizziness also has a close relationship with psychiatric disorders, particularly with affective disease and anxiety. Unfortunately, the significant aspects of dizziness secondary to myofascial difficulties, is often ignored. Zasler<sup>98</sup> discusses cervicogenic dizziness (noted above). Alexander<sup>140</sup> does not seem to anticipate the psychological aspects secondary to this problem, making it seem to be more of a primary psychological problem versus being secondary to a true organic problem.

In discussions of the PPCS, it is noted that chronic pain and headache are fairly universal accompaniments of the PPCS. It has also been noted that patients with chronic headache, not caused by MTBI, have many of the same complaints, including fatigue, sleep disorder, depression, and dizziness as well as difficulties with concentration and memory. Finally, it is noted that psychological factors may aggravate these headaches.

The literature notes that anxiety can decrease concentration and complex mental processes,<sup>118,165</sup> while depression can cause decreased cognitive functioning, particularly in concentration, memory and executive functions.<sup>156,166,167</sup> This latter problem has also been called "depressive pseudodementia."<sup>168</sup>

Alexander's<sup>140</sup> "logical fallacy" is therefore only partially true. One cannot consider that if everyone with a TBI has impaired concentration, then everyone with impaired concentration (after MTBI) has a neurological etiology. The problem is that patients with MTBI with associated pain and affective difficulties may have impaired concentration for multiple reasons, including post MTBI neuropathological changes.

Furthermore, this does not take into consideration other aspects of a patient's clinical situation. Alexander<sup>140</sup> asks the question: when does the physiogenesis of a clinical problem become psychogenesis? It is noted that this is certainly difficult to determine, and may have an iatrogenic contribution. Alexander does indicate while the major issue is physiogenesis transforming to psychogenesis, he does note that physiogenesis can be very underestimated. He also notes that there is no single psychological factor, physiological factor or demographic factor leading to PPCS.<sup>140</sup>

The factors of pain and headache, particularly chronic post-traumatic tension-type headache, have been extensively described.<sup>169-172</sup> The pathophysiology of chronic tension-type headache itself is a combination of myofascial nociception and affective changes (which develop over time secondary to the headache pain) inducing significant changes in the neurochemistry of the central

nervous system. These changes, consisting of neuronal exhaustion and the "empty neuron syndrome" are associated with significant changes in the serotonergic, noradrenergic and endogenous opiate neurotransmitter systems. These central nervous system neurochemical changes become the primary etiology of the continued headache. See Figure One.

Clinically dealing with these problems necessitates the utilization of an interdisciplinary approach to the treatment of the myofascial problems, the affective difficulties (depression, anxiety, etc.), the secondary sleep disorder and the primary problem, the changes in nociceptive system functioning.

These factors lead to several important considerations in the evaluation and treatment of these clinically complex patients. First, in the patient with a MTBI, post-traumatic headache and depression requires a comprehensive evaluation. This evaluation should be performed by a physician knowledgeable in MTBI, a clinical psychologist or psychiatrist, and include physical and other therapy evaluations as indicated.

Second, upon completion of the above evaluations, the team should have a discussion about the best approach to treat the patient. Granted, some of the symptoms can have multiple etiologies and therefore must be treated simultaneously to insure the best outcome. Generally, one cannot treat one entity and omit the other to be successful. Delay or avoidance of concurrent treatment will only become more costly.

### **Iatrogenic and nomogenic induced exacerbation**

To quote Lishman,<sup>173</sup> "The repeated rehearsal of symptoms before a variety of audiences, some encouraging, some skeptical, does not help the patient to be clear about what he is truly experiencing." The patient who has experienced a MTBI with subtle or even less than subtle cognitive deficits, with or without pain and other clinical problems noted above, may see physicians who essentially negate their deficits, telling them to wait longer for healing; that there is nothing wrong with them; or that they are "neurotic" and should seek psychiatric counseling.<sup>169</sup> The patient is left in a quandary, in that their neurological examination may be normal; yet, they are still experiencing difficulties. This may lead to the patient further doubting themselves, increasing their affective difficulties and feelings of loss of control, even questioning their sanity. They may also decide that they did not do a good job of explaining their symptoms to the physician. They may feel that they need to be even more explicit, subconsciously magnifying their problems the next time, to get the attention and the hoped for recognition of their problems and then treatment.

The roots of MTBI with persistent sequelae, or the persistent post-concussion Syndrome appear to be extensive and in many clinical cases, have nothing to do with the actual clinical problems which were initially in evidence. As Alves<sup>159</sup> noted, concurrent to recovery is an interaction between organic damage and

social factors which may produce more persistent symptoms than may have been expected on the basis of organic damage alone. Malingering has been noted to be only rarely present,<sup>15,159</sup> and the myth that the end of litigation will cure the MTBI deficits has also been found to be rarely true.<sup>7,156,171</sup>

The lack or delay of treatment with this symptom complex can increase the psychosocial factors within the impairment. The lack of support by the professionals, the restrictions imposed by some reimbursement sources, and skeptical loved ones (since the professionals say there is nothing wrong) leads to anger, frustration and depression. The symptom complex may exacerbate or increase. Some patients may become so dysfunctional that they are terminated from their jobs. This pattern can continue, leading to greater dysfunction.

### **Patient evaluation**

Typically, though not always, the patient goes to an emergency room after their injury. The appropriate neuro-imaging studies, if warranted, are performed. Patients with higher GCS scores for mild TBI<sup>15-15</sup> may be kept overnight for observation or sent home. The patient may also go first to their family physician or directly to a chiropractor, hours or days after an injury, where initial evaluation is performed, and not be seen in the emergency room.

These patients are given instructions to be awakened every hour or so, and to return if vomiting or decreased sensorium is seen. They are typically given pain medication. When the symptoms do not seem to clear in a short period of time, the patient is usually sent by their family physicians for a neurological evaluation.

The neurological findings are usually normal, including the verbal mental status examination. Physicians with MTBI experience will check for abnormal frontal lobe reflexes, such as the palmomental, or snout reflexes. If the patient has a history of loss of consciousness or altered sensorium, proclaims cognitive problems with memory, concentration, multitasking, information processing or changes in behavior, including emotional lability, increased irritability, or demonstrates to the examining physician difficulties with pragmatics, the patient should be sent for further workup to determine the presence or absence of the cognitive sequelae of a MTBI. Unfortunately, this evaluation is usually performed weeks or even months after the initial insult.

Along with the patient history of cognitive problems, there are also frequent complaints of post-traumatic myofascial (or soft tissue) pain, and sleep disorder. As the problems become multifactorial and complex, the appropriate consultation should be obtained.

In the patient who complains of pain and/or headache and is seen early after injury, physical therapy may be enough to stop the pain problem before chronicity, with its attendant affective and neurochemical alterations, occurs. The use of narcotic analgesics should be strongly discouraged, as they may further enhance cognitive difficulties. If, after six to 12 weeks, there is no

significant diminution in the pain and headache, along with depression, consideration should be given to referral of the patient to a specialty pain program.

In the majority of patients with MTBI and pain, an interesting dichotomy presents during treatment for pain. Patients in an interdisciplinary pain program are typically taught to grade their pain, on a momentary basis, on a zero to 10 scale, or a zero to 100 scale. When they begin treatment, the numbers are typically high, corresponding with physical findings of muscle spasm, trigger points and loss of function such as range of motion. As treatment progresses, typically in a four to six week program, the patients' pain complaints may not change. That is, their identification of their pain level (i.e. seven over 10) may not change or change only minimally, while functional evaluation will reveal a return to normal range of motion, absent palpable spasm and/or trigger points. It is important to realize that this dichotomy is not a manifestation of malingering, but appears to be more of a learned, or even a perseverative response. On observation, pain behaviors are diminished, the patients affect is improved, but they may still claim to endure what appears to be an artificially high pain level.

It is therefore imperative that constant patient reevaluation is performed during treatment. Once the patient has reached the appropriate functional improvement, if cognitive complaints persist, it is appropriate to send them for a neuropsychological evaluation, preferably done by a psychologist with special training in evaluating cognition after a MTBI.

All treatment should be functionally based, particularly the rehabilitation of a MTBI. The functionality of the treatment is what will decrease a patients fears and prepare them to continue their lives in the real world.

### Neuropsychological

The neuropsychological evaluation is utilized to delineate cognitive and behavioral symptoms. It is used to help explain, "What is wrong" when all other clinical tests have been normal.

The need for and value of neuropsychological evaluation is frequently raised as some individuals still do not understand this relatively new medical specialty. Another reason for this might be what some would consider, the lack of definitive evidence, when the testing is done. Raw data from a single neuropsychological evaluation is frequently subject to more than one interpretation.

Cognitive impairment is often diffuse, with more prominent deficits seen in the areas of information processing, attention, memory, cognitive flexibility and problem solving. The ability of neuropsychological testing to determine these factors in patients from one month post injury to years after, is well documented.<sup>9,60,153,157,161,175-181</sup> These tests are difficult to incorporate into a clinical office examination and this, among other factors, prob-

ably leads to the failure of many clinicians to recognize these deficits, and thus these patients are pronounced "normal."<sup>16,138,143,182</sup>

Research indicates that if complaints of memory or other cognitive changes, irritability, or fatigue persists for one month or more post injury, a comprehensive neuropsychological evaluation should be performed.<sup>60</sup> Determining conclusively the existing deficits is of primary importance to the rehabilitation process.

Process observations are an integral part of neuropsychological testing. Every effort should be made to have the treating neuropsychologist administer the testing so these observations can be made, rather than just having the raw test data evaluated. It should also be noted that, a fixed battery of tests, e.g. Halsted-Raytan, or Luria will not necessarily be sensitive to all the cognitive deficits sustained after a MTBI. A good neuropsychological test is flexible. Research, as well as clinical experience, has shown that measures of attention, concentration speed and efficiency of information processing are the most sensitive to neurological based, organic deficits post MTBI.<sup>90</sup> Attention and information processing are not by themselves specific to MTBI.

A specific hallmark of MTBI, appears to be the breakdown of information processing or the number of operations the brain can simultaneously perform. Slow thought processes, memory deficits, easy distractibility and lack of attention after MTBI are thought to be due to attentional deficits secondary to decreased ability to process information.<sup>110,183</sup> Gronwall developed the paced auditory serial attention test (PASAT),<sup>110</sup> which has been found to be extremely sensitive to MTBI.

The formal neuropsychological testing procedures involve the neuropsychologist going over a patients history and past records, a flexible test battery, process observations (how the patient takes the test, along with their reactions as they take it), input from outside observers, and tests of mood and personality. Once the tests are evaluated, patient feedback is given.

There are a number of variables which can influence a patient's neuropsychological performance, including age, socioeconomic status, family dynamics, anxiety and depression (which must be diminished, optimally, prior to evaluation), the unconscious process of symptom magnification, litigation, malingering, drugs, alcohol and pain.<sup>181,184,185</sup>

The risk of dysfunctional response to MTBI secondary to different personality aspects is mentioned by Kay.<sup>80</sup> Kay notes five personality styles, including those who are highly driven, possibly obsessive-compulsive, overachievers, whose sense of self is tightly tied to intellectual achievement, and persons with tendencies toward grandiosity, with elements of narcissistic personality style, who tend to minimize and deny as well as hide their difficulties until their lives crumble around them before they will acknowledge their difficulties to others. He also mentions persons who have suffered emotional deprivation as chil-



dren, persons with strong tendencies toward dependency and patients with the "borderline" personality.

Kay<sup>181</sup> has described several other aspects to be taken into account when evaluating for MTBI, the first of which is: Individual Vulnerability." A significant number of variables will influence how a specific injury will effect a patient, and each person will have a specific level of vulnerability for each specific aspect. These include: neurological vulnerability, neurotransmitter balance, age, drug and alcohol abuse, family dynamics, previous central nervous system damage, personality structure, pre-existing psychological problems, current levels of stress, and litigation. Kay indicates that the interaction of the neurological and psychological-psychiatric variables determines the "Individual Vulnerability" for any single individual post MTBI. This could possibly also help to account for the different outcomes seen in patients after similar injuries.

Kay<sup>181</sup> also described the idea of the "shaken sense of self," which is seen after an undiagnosed MTBI, as a persons loss of confidence in their abilities increases, along with the decreased ability to even predict or anticipate their performance in any given situation. Failure to diagnose MTBI and at least discuss with a patient anticipated cognitive and behavioral deficits and problems can exacerbate psychological deterioration. If this occurs secondary to refusal to evaluate a patient for a potential MTBI, it becomes another form of iatrogenic exacerbation.

With neurologically based, organic "weak links" in the cerebrum post injury, a patient will be more vulnerable to anxiety and depression. The presence of significant emotional dysfunction years after a MTBI is not evidence that organically based neuropsychological problems do not exist. The primary or core organic deficits may be fueling and perpetuating the psychological overlay.<sup>181</sup>

An important topic is the measurement of neuropsychological deficit. Two neuropsychologists can come to different conclusions when looking at the same data. One reason for this is, usually, the fact that one of them administered the testing, and was therefore able to perform direct observations regarding the way the patient took the test.

An even more important reason is that there are several methods that can be used to "grade" or evaluate the test results. The first is by comparing the results to normative comparison standards, therefore comparing the patient to a large statistical cohort (number). This technique seems to typically find lack of statistical significance for many patients who would be found to demonstrate cognitive deficits if the second method, utilizing individual comparison standards, was performed.

The latter method determines specific measurement of deficit by comparing the patient's test results to his or her premorbid status, utilizing the Best Performance Method.<sup>186</sup> This technique utilizes test scores, other observations and historical data. During interpretation, the level of the best performance, be it the

highest score or group of scores, serves as the best estimate of premorbid achievement, and becomes the standard against which all other aspects of a patient's current performance is compared. Therefore, the methodology is more specific to the patient, and not a comparison of a single patient to large statistical cohorts. If a patient was cognitively impaired, their least depressed abilities found on testing, utilizing the best performance method, are felt to be the best representative of that person's original cognitive potential.<sup>186</sup> Lezak<sup>186</sup> also notes that a person's premorbid ability level can be reconstructed or estimated from many different kinds of behavioral observations as well as historical facts; if they are looked for by the clinician.

Behavioral problems, including psychiatric disinhibition, aggressive-violent behavior, and emotional lability are also frequently seen and associated with frontal-temporal lobe damage and/or limbic system damage. These difficulties must be dealt with.

### Speech-language pathology

If the neuropsychological evaluation is positive, speech-language pathology (SLP) and occupational therapy evaluations, functionally based, should be performed. When an individual sustains a MTBI, it may effect his or her language-cognitive skills. As cognitive abilities and language are intrinsically and reciprocally related functionally, an impairment of language may disrupt one or more cognitive processes. The reverse is also true, cognitive deficits may disrupt language skills and abilities.<sup>187</sup>

The comprehensive cognitive-communication skills assessment performed by the SLP includes four basic areas: 1) how a person codes and retrieves information; 2) how the person organizes information for processing and retrieval; 3) how the person reasons and problem solves; and 4) how language and cognitive deficits affect other areas of functioning.<sup>188,189</sup>

Areas frequently effected by a MTBI include short and long term memory, attention to tasks, ability to sequence information, problem solving, including deductive and abstract reasoning, reading comprehension, writing organization, grammar, and pragmatics, or social skills. These are more functionally based than the information gathered in the neuropsychological evaluation. The focus of speech-language treatment is to help the patient relearn lost skills or learn compensatory strategies to help that individual compensate for the areas of deficit.

The SLP will treat a patient's organization and problem solving abilities, visual and auditory attention, as well as areas of focused, selective, alternating and divided attention. Also involved is assessment of interaction skills, such as facial expression and tone of voice. As memory skills overlap into functional tasks such as reading and following directions, the SLP will also assess various memory types (visual, auditory, remote, delayed, etc.) and help design and implement appropriate treatment strategies.

Reading and writing skills are evaluated for functionality by the SLP. Compensatory strategies are then devised.

Pragmatic skills, the awareness of appropriate emotional, verbal and non-verbal behaviors are evaluated. Functional pragmatic skills may effect a person's ability to socialize with family and friends as well as job performance.

All of the cognitive aspects noted have a significant impact on a person's communication skills and his or her ability to function in the real world. The SLP works to teach patients how to relearn information or to incorporate compensatory strategies within their functional lives. It takes time and significant effort to generalize the use of compensatory strategies, including continued guidance and encouragement from the SLP, as patients go through trial and error periods to incorporate these strategies and new skills into their lives. The key to treatment success is practice in real life situations. For this reason, it is common for treatment to take place in the community, not just at the clinical site.

### Occupational therapy

The occupational therapist (OT), similar to the SLP, evaluates and treats functionality. The OT evaluation also involves identification of specific physical, cognitive or perceptual deficits which may interfere with a patient's ability to perform functionally oriented tasks. The treatment addresses the problems in individual or group treatments through the use of functional activities.<sup>190-192</sup>

Various types of attention (sustained, selective, alternating and divided) are evaluated. Deficits in divided attention, or multi-tasking activities, may make a patient unable to function at work, in a store or at home.

The other aspects of the OTs evaluation includes: memory storage and recall of both auditory and visual information. For example, the use of a day planner is an appropriate functional compensatory strategy to deal with memory deficits. Awareness of time and date may be impaired, as may be the ability to give and receive verbal and written directions. These aspects of cognition, along with financial management are addressed in treatment.

Executive functioning (the ability to plan, organize and carry out tasks) is an important aspect of OT treatment, as is problem solving and abstract reasoning. The evaluation of situational problem-solving, including safety and judgment issues is also important. Difficulties with visual perception, poor tolerance or endurance, and upper extremity functioning are also part of the OTs pervue. Activities of daily living are a key focus of OT treatment, including personal hygiene, cooking, eating, dressing parenting skills and responsibilities in the home environment.

Vocational status is evaluated. Work skills are practiced through actual work tasks, if available. Volunteer positions are utilized if actual work tasks cannot be obtained. Job interviewing skills are also addressed and practiced.

Community reentry and leisure skills are addressed in individual and group treatments; pragmatics are incorporated into these activities.

The OT treatment of the patient with a MTBI emphasizes the return to their previous roles and daily activities. This encompasses one's entire daily routine. The patient is part of the treatment team. Their input into the constantly evolving evaluation and treatment strategies is necessary and invaluable.

### Treatment paradigms

As noted above, a patient with post traumatic headache or pain, who has had these problems for weeks or months or longer, who is also depressed and anxious, should be evaluated as soon as possible. Once all the etiologies of the patient's problems can be determined, a quality treatment plan to deal with these issues can be devised. Pain and depression must be effectively dealt with early on.

There are a number of different types of treatment paradigms and designs. The purpose of this paper is to address the patient with mild to mild-moderate traumatic brain injury in the out-patient environment. Guidelines for treatment of the acute patient in the initial emergency room are currently being developed by the minor traumatic brain injury committee of the brain injury interdisciplinary special interest group of the American Congress of Rehabilitation Medicine. Other MTBI treatment paradigms, such as inpatient treatment, may have other considerations.

The single most important concept of treatment of a person with a MTBI is *functionality*. Computer oriented "cognitive rehabilitation" is not a functional treatment. The use of a computer enhances ones' abilities in only specific areas of cognition. It is not a functional form of treatment.

After a patient with MTBI has been thoroughly evaluated by an interdisciplinary team, including neurology or psychiatry, neuropsychology, speech-language pathology, and occupational therapy, an individualized treatment program is designed for the patient. For example, a 'limited services' program consisting of several SLP and OT treatment sessions a week, over the course of four to six weeks, may satisfy the patient's needs. Other patients with more severe deficits may need a full interdisciplinary day treatment program. In such a program, the patient is seen for treatment by the physician, the nurse rehabilitation specialist, SLP, OT, clinical or neuropsychologist, physical therapist (for continued pain and/or balance difficulties), and vocational specialist, to help in the patients return to work with appropriate accommodations, or the attainment of a new, more appropriate, vocation. Therapeutic recreation is also commonly used to help the SLP and OT in a more holistic approach to community re-orientation and re-integration.

Another important part of treatment is the neuropharmacological aspect. There are a number of medications which should not be utilized for a patient with a MTBI, including narcotics, barbi-

turates (in analgesics or anticonvulsants), and dilantin, which may increase cognitive dysfunction. Great care must be given to the use of any psychoactive medications, which may also be a necessary part of treatment, to deal with affective and behavioral problems which may hinder cognitive oriented functional treatment. The integration of therapeutic medication options in conjunction with a functionally oriented MTBI treatment program appears to enhance and becomes synergistic in achieving timely functional outcomes.<sup>193</sup> The use of such medications will, however, necessitate careful and continuous patient medical management by the prescribing physician.

In summary, treatment must be individualized and functional in nature. All appropriate areas of functionality must be evaluated and treated accordingly in individual and/or group treatment. Neuropharmacological treatment may be helpful.

### Functional assessment instruments

The most widely used assessment tool in rehabilitation is the functional independence measure (FIM). This tool has demonstrated its merits in many areas of physical rehabilitation including spinal cord injury and stroke. However, there is some controversy with regards to its merits when assessing TBI patients. There are questions of the reliability of the psychometric properties of the scale, which is scored as the sum of 18 activities, consisting primarily of motor and cognitive elements.<sup>194,195</sup>

Over the last five years a functional assessment scale (FAS) for patients with MTBI has been developed and used in the evaluation of over 250 patients with remarkable consistency at the Headache and Neurological Institute of Colorado (HNRIC). The HNRIC FAS™ measures 15 objective areas on a scale of one to five, with five being fully functional, giving an overall total score of 75 for “fully functional” patients.

Table Two shows the 15 objective areas utilized by the functional assessment scale. Figure Two shows an example of a patient's functional assessment scale. This figure demonstrates a 26 percent over all improvement in function over the patient's 12 week treatment program.

The HNRIC FAS™ has shown remarkable consistency, with the average patient achieving a 20 percent functional improvement (range 11 to 56 percent).

Table Three shows an example of one of the HNRIC FAS™ scales pertaining to money management. It is felt that this tool, and others like it, will help demonstrate the appreciable outcomes which can be obtained in treating MTBI.

### Case management

There exists a significant paucity in the literature regarding case management in cases of MTBI. This is not surprising given that case management is usually confined to “serious” or catastrophic

cases. However, in one of the few articles relating to MTBI and case management, Mattson<sup>196</sup> stated, “The expression ‘post-concussive rehabilitation’ is almost an oxymoron for a case manager. The label ‘post-concussive syndrome’ is often a catchword for a combination of seemingly unrelated psychologically and physiologically perceived symptoms leading to disability. The handicap seems insurmountable. By the time the person reaches a case manager, there is often a generous measure of despair, because current treatment is failing to ameliorate perceived problems or because the funder is crying ‘foul play’ and is threatening to cut off reimbursement.”

Mattson indicates the necessity of having a good clinical background and understanding of MTBI. Her article vividly illustrates the difficulties encountered when MTBI is not identified and treated appropriately early on.

The case manager should be a facilitator, working with the medical treatment team to help the patient receive appropriate treatment.

### Case histories

Case one: DM, a 34 year old right handed Caucasian female was involved in a head-on motor vehicle collision and sustained both a blow to her head as well as an acceleration/deceleration injury. She was seen initially at a local emergency room and released with pain medications for her complaints of headache. One of the authors was contacted six weeks after injury, via a conference call with her attorney, the patient and a newly appointed case manager. The case manager had a number of questions as to what an evaluation entailed, and how treatment would proceed. The patient was seen in consultation and found to have a significant cervical strain, post traumatic headaches, decreased balance, depression, and difficulties with word finding. She complained of emotional lability and poor memory and concentration.

She was first treated, over a period of three weeks, for her pain and depression in an interdisciplinary treatment program consisting of physical therapy, biofeedback therapy, psychotherapy and medical management. At that point, her pain was markedly diminished; her depression continued, but was not as deep. Her major pre-occupation was dealing with the changes in her cognitive abilities. MTBI evaluations consisting of neuropsychological, SLP and OT evaluations, were performed. The patient was found to have significant difficulties with information processing and speed; concentration; poor pragmatics; and significant difficulties with executive functioning.

She was treated in an outpatient interdisciplinary MTBI program for a twelve week period. Beginning in the sixth week, she returned to work and began working with the vocational specialist who, along with the OT, was able to determine an appropriate work situation with accommodations. The patient, at the end of 12 weeks reached maximum therapeutic benefit. She

returned to a new work situation on a full time basis, pain free, taking only an antidepressant.

Her functional assessment scale scores improved from 36 after initial evaluation, to 68 at the end of treatment, a 53 percent improvement. Eight month follow up has revealed no further need for medical intervention. She maintains the compensatory strategies developed for her by the treatment team and has fully incorporated them into her life.

Her nurse case manager attended the weekly team meetings, asked questions and was given whatever information she requested about MTBI. The nurse case manager worked effectively with the treatment team and was an important intermediary between the payer, the patient and staff.

Case two: WB, a 44 year old right handed woman was involved in a motor vehicle accident in January 1995. There was no loss of consciousness, although the patient described striking the back of her head on the headrest, as well as an acceleration/deceleration injury. She was seen in initial consultation for headache and cervical pain in March 1995. The patient had a significant cervical myofascial pain syndrome, her cervical range of motion being minimal on evaluation. She also complained of significant memory and concentration problems, depression, and described a post traumatic stress disorder (PTSD), with frequent flashbacks of her accident, nightmares, fear and anxiety when in a car, and an extreme fear of driving.

She was foreign born, but had been in the United States for 17 years. She had attended college in the United States. After four weeks of pain management, when her functional range of motion returned, she was eager to deal with her cognitive deficits.

A neuropsychological evaluation was requested; the request was denied. After numerous attempts and six weeks later, approval was finally obtained and the neuropsychological evaluation was performed. The evaluation found attention deficits, visual-spatial deficits, comprehension deficits and more. A request for evaluations by SLP and OT were made and denied. They were finally completed a month later.

The neuropsychological test data and report were sent for a paper independent medical evaluation (IME) by a psychologist. The IME psychologist state, "she had no cognitive problems since English was her second stated language."

The patients pain went into exacerbation, as she became very "stressed" (her word) by her inability to receive treatment. She complained of a different type of pain and numbness in her right arm. After an examination, an electromyogram and nerve conduction velocity (EMG/NCV) was ordered.

Her husband wanted her to return to driving. Because of her visual-spatial deficits and her fear of even being in an automobile, a driving evaluation was also requested. The EMG/NCV

and driving evaluation were denied. Another month went by and approval for a three week treatment program for SLP, OT and psychology was obtained. At the request of her husband the payer was informed that she had begun to drive. She continued to complain of significant anxiety, driving phobia, dizziness and right arm numbness, as well as continued cognitive problems. Requests were repeated for an EMG/NCV, a driving evaluation, and continued treatment, which had already been interrupted by two weeks, and were again denied. The physician wrote a letter stating that the patient should not be driving, and that a driving evaluation was needed to determine, legally, if she was capable of driving.

A week later, in August, the patient had another IME by a specialist in TBI who stated the original diagnosis made in April and May of MTBI was correct. He concurred with a driving evaluation and an EMG/NCV. In early September 1995, approval was given for three physical therapy visits, three SLP visits, three OT visits and two psychotherapy visits. On September 19, 1995, the recent IME, had been reviewed by the payer. An EMG and driving evaluation was approved, along with another one to two weeks of treatment.

The patient, feeling that she had been "jerked around" (again, her words) for five months decided not to proceed with the driving evaluation, as well as not to continue with treatment. She stated, "They won't let me get what I need, so I give up." In total, during the five month period, WB had been approved for five to six weeks of treatment, given in such a staggered fashion, as authorized by the payer, that the treatment team had been unable to carry out their specific treatment plan. While a great deal of non-authorized, free, treatment was given, it could in no way make up for the ravages of time and the very significant exacerbation of the patient's psychological, physical and cognitive complaints. The patient suffered increasing depression from this frustration. It is questionable as to whether the insurer's payer representative knew what had transpired.

Case three: TB, a 34 year old right handed male, was involved in a motor vehicle accident in September 1991. He was seen for initial consultation in January 1993. He had been treated for pain, prior to being evaluated for MTBI. The patient began treatment, but was fearful that his insurance company would not pay for treatment. A lien was taken. After three weeks, the patient, who fit the classical pattern described by Kay<sup>181</sup> of having a highly driven personality, a true overachiever, with elements of grandiosity, decided to leave treatment. He wanted to return to work and did not feel that there was anything terribly wrong with him.

His state of denial and minimization of problems would not allow him to admit to his difficulties, which had been identified on neuropsychological, SLP and OT evaluations. He had been a member of MENSA before his motor vehicle accident.

He was employed as a technical manager at a local television station before his accident. After the initial three weeks of treat-

ment, he returned to work doing construction. During his first week on the job, he drove a fork lift into a wall. He quit work and later returned, asking for treatment, which was given, though the patient still dealt with significant elements of denial. After treatment, he took a job at a computer store, where his physician ran into him. After the patient said hello and went back to work in another room, the man's co-workers, not knowing who the physician was, commented on how slow the man worked, and that he "couldn't even think of two things at a time."

The patient later went to work for a good friend, who wanted him to do computer work, as well as simple bookkeeping. After five months, his friend had to let him go. The patient returned to the computer store, at a lesser salary, with an accommodation in place. He did not have to answer phones when he was working on a computer.

The patient's insurer has refused to recognize the patient's injury. To date, they continue to pursue litigation, attempting to prove that the patient did not have a MTBI. In spite of major pressure, the patient continues to work. His home life has significantly deteriorated, with significant illnesses of immediate family members and poor financial stability, as the patient cannot perform his previous technical work, at a higher wage. In spite of all of these personal set backs, the patient continues to work and, in his words, "take everything one day at a time." He expresses significant regret for his cognitive losses.

To date, the patient's care has not been paid for. Litigation against the payer continues, four years post injury. It is one of the authors understanding that the payer and their legal counsel does not believe the patient has suffered a MTBI.

### **Recommended evaluation/treatment guidelines**

The recommended evaluation and guidelines below are for ambulatory patients who fit the diagnostic criteria given in the definition of MTBI by the mild traumatic brain injury committee of the brain injury interdisciplinary special interest group detailed above. These patients will have already received an initial evaluation. Many times, the chief complaint is post traumatic headache, pain or dizziness, or changes in emotional status, with lability, aggressive or violent behaviors. Changes in cognitive status may be volunteered by the patient. The comprehensive history should inquire into these areas. The patient should be asked about the neurovegetative signs of depression (sleep disturbance, appetite changes and loss of libido). Talking with a patient's spouse or significant other should be done at the time of the initial history, if possible. Inquiry about the patient's work status, including problems and changes should be a part of the history.

A neurological examination should be performed, with special attention paid to the cranial nerve examination, for anosmia (loss of smell), agusia (loss of taste), lateralization of hearing and poor eye convergence. Pathological frontal lobe reflexes

should be tested. Balance should be evaluated, as well as cerebellar functions. Sensory examination, including vibratory and positional sensation should be tested. As the patient describes his/her history watch for problems of word finding or variation in response to question. A thorough musculoskeletal evaluation should be performed, particularly if the patient presents with a history of headache or other post traumatic pain.

Particularly in the presence of pain, it may be difficult to make the diagnosis of a MTBI, although it may be suspected. Some patients have cognitive deficits so severe, they are very apparent. If the elements of pain and depression are primary, at least during the initial contact, the presumptive diagnosis of MTBI may be made a "rule out," and recommendations for its evaluation should be made.

As stated above, pain and depression should be treated as early as possible, as they may figure significantly in the development of MTBI with persistent sequelae, or persistent post concussive syndrome. Ideally, early intervention will decrease the chronic symptoms associated with pain and depression that can mask the cognitive sequelae associated with pain and depression.

If the patient is initially encountered within six weeks of their injury, their pain and depression may be relatively easily dealt with via appropriate therapy. If two or more months have elapsed, the patient may need to be treated in an interdisciplinary headache or pain treatment program, as myofascial and affective changes, along with sleep disorder and central neurochemical nociceptive changes will have to be dealt with. Inappropriate treatment, or no treatment, will create greater problems which will require more treatment in the future.

It must be remembered that functional gain or improvement in treatment should be closely monitored, as subjective pain complaints in a patient with MTBI may not change. Lack of improvement can be secondary to learned or preservative behaviors, or difficulty conceptualizing the pain rating scale.

Typically, after three to six weeks of pain treatment the pain and at least a major component of depression should be ameliorated. This is very important in the chronic patient before a concrete diagnosis of MTBI can be determined.

A neuropsychological evaluation should be done by a psychologist who has had specific specialty training in the neuropsychological assessment. As stated previously, it is recommended that a psychologist familiar with the best performance method of determining cognitive deficits be used, as the results obtained will be more specific for the patient and give the most treatment oriented information, if the presence of MTBI is determined. The neuropsychological evaluation should be performed as early as one month after injury if MTBI is clinically suspected.

If the neuropsychological evaluation is positive, speech-language pathology and occupational therapy evaluations should be performed, as these evaluations supplement the neuropsychological evaluation with more functionally related information.

After all evaluations are completed, the interdisciplinary team meets and determines a specific, individualized treatment program for the patient. Some patients with minimal difficulties may need minimal treatment, which could encompass only several weeks, while patients with more severe problems may require several months or more of intervention. If job performance is being affected, a job site visit and training in compensatory strategies may be required. Frequently, treatment, in whole or part, may involve re-training a patient to attend school. Many of the patients are students at the time of injury and have difficulty returning to school.

If a patient has a larger number or greater complexity of deficits, a full time residential treatment program should be considered. However, if the patient can be adequately supervised in their home setting, and can attend a very intensive outpatient program, then this alternative should also be considered.

Typically, a full interdisciplinary outpatient program will last from eight to 12 weeks, depending on deficits and individual needs. The patient will be treated by the physician; the psychologist or neuropsychologist; the speech-language pathologist and occupational therapists; biofeedback therapist, if desensitization is needed; physical therapy, for balance and continued pain problems; the therapeutic recreation specialist; and the vocational specialist are part of this interdisciplinary team.

Most functionally oriented programs will have a patient return to work by the sixth or seventh week of treatment, so that work related accommodations can be identified and implemented during the remainder of treatment. If a patient cannot return to their prior work, at that time, a volunteer position at a local facility may be obtained for the patient. Work related skills are re-taught.

Weekly or bi-weekly interdisciplinary team staff meetings should occur. This is the optimal time for the team to share information with the case manager, the patient and payer. These meetings should not only include patient specific information, but should also be a vehicle to inform other concerned individuals about the clinical and cognitive difficulties the patient is experiencing. It can explain why specific treatment protocols are being utilized. This is also a good time to present requests for compensatory equipment, etc..

Functionality is the key to the rehabilitation of a patient with a MTBI. The goal is to return a patient to his or her family, social systems, community and vocation.

The psychological aspects of treatment are ever changing. They encompass the patient as well as his or her family. Emotional lability, psychotic, aggressive or frightening behaviors must be

dealt with before they contribute to the dissolution of the family unit. The psychologist frequently must work with the patient as well as their family. Acute suicidal ideation must be dealt with immediately, as must psychotic, aggressive or violent behavior. Many times the psychological problems mentioned are relayed to the psychologist by another member of the treatment team, as the patient may be too frightened or embarrassed to tell the psychologist themselves.

The patient must be treated in the clinical situation, as well as allowed to journey into the community with a feeling of safety, by having their OT, SLP and/or therapeutic recreation specialist accompany them. Treatment staff visits to a patient's home and place of work are imperative, for the evaluation in these settings, to help the patient develop methods of generalizing specific skills.

If a patient has visual-spatial deficits or significant driving phobia, a specialist needs to perform a driving evaluation. If a patient can not drive safely, they should be taught how to use public transportation both in the clinic and field.

Group treatment is important and gives patients the assurance that they are not alone with their problems, as well as enabling them to learn from the experiences of other patients. The importance of these group experiences cannot be under stated. They are an important part of the therapeutic milieu which is developed by an experienced interdisciplinary MTBI treatment team. Just knowing that they are not alone, not the only one with their problems, is important to patients. Supervised group outings also give patients other ways to obtain feedback on their abilities and deficits. For example, in one group trip to a large computer company, it was instructive to the entire group of patients when one of the group members aggressively answered all of the questions asked by other patients of the company's tour

**Table One**

**Typical clinical/cognitive and behavioral problems associated with MTBI**

Planning	Information processing
Goal setting	Problem solving
Self-initiation	Social judgment
Self-monitoring	Emotional lability
Self-esteem	Frustration
Disinhibition	Procedural learning
Perseveration	Self-regulation
Flexibility	Endurance
Depression	Motor and sensory changes
Irritability/volatility	Impulse control
Attention	Pragmatics
Concentration	Aphasia
Memory	Denial

guide. This demonstration of the patient's poor pragmatics was important to the patient in question, the treatment staff and the other patients in the group.

Constant medical supervision is necessary, particularly when dealing with neuropharmacological interventions and treatment utilized for depression, aggressive behavior, extreme fatigue, emotional lability, psychosis, fast cycling bi-polar activity, and so on. Utilizing psychoactive medications in a physiological system which is disturbed or distorted secondary to injury demands constant vigilance. An appropriate functional outcome after treatment is return to work, accommodated as needed; compensatory strategies utilized at home for safety as needed (such as remembering to turn off the stove); good pragmatics; in fact, functional abilities, to the greatest degree possible, in all 15 aspects of the aforementioned HNRIC functional assessment scale.

When a patient can, for example safely, and by themselves, make a shopping list, drive safely to the store, obtain the needed items, pay the correct amount of money and recognize the correct change, safely drive home (remembering the directions unaided) and put the items in their proper places, when previously they could not functional rehabilitation has been achieved.

Figure Three is a short-hand version (algorithm) of the evaluation and treatment recommendations.

**Conclusions**

The diagnosis and treatment of a MTBI is never done in isolation. Now, more than ever, it involves the combined efforts of physicians and other clinical specialists expert in dealing with MTBI in conjunction with the payer and their representative(s).

All members of this "team" must work in concert with one single idea in mind: to maximize the patients' functional independence.

There is no reason why this can not happen. The proverbial playing field must be leveled, with all players working towards the same goal. Good judgment, good clinical skills and the ability to be open to leaning about a difficult clinical problem should be prerequisites for being a member of the treatment team.

The goal of clinical medicine is to help: to do no harm. Everyone participating in the care of a patient with MTBI must believe the same thing. Not to do so is an abrogation of everything we all hold so dear, the willingness, when one has the ability and the responsibility, to help those in need.

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**Table Two**

**The functional assessment scales (FAS)**

1. Medical management
2. Vocational functioning
3. Home management
4. Money management
5. Transportation
6. Self care
7. Communication
8. Interpersonal/pragmatic ability
9. Family functioning/social network
10. Psychological functioning
11. Neuropsychological functioning
12. Neuromuscular functioning
13. Pain
14. Fitness
15. Leisure activities

**Table Three**

**Money management: the ability to plan and carry out budgeting and the exchange of money.**

- 1) Dependent for all aspects of personal finances (insurance, budgeting, paying bills) and money exchange
- 2) Consistently requires minimum to moderate structure/assistance for routine transactions; moderate assistance for incidental expenses (doctor, insurance, broken appliances). Able to complete simple cash transactions accurately; maximum assistance for other aspects of personal finance.
- 3) Able to independently complete routine transactions (monthly bills, budget, checks); requires minimal cues/structure for incidental expenses
- 4) Occasional cues/structure needed to complete all aspects of personal finances in a timely, accurate fashion.
- 5) Patient is independent, timely, accurate with all aspects of planning and carrying out personal finances.

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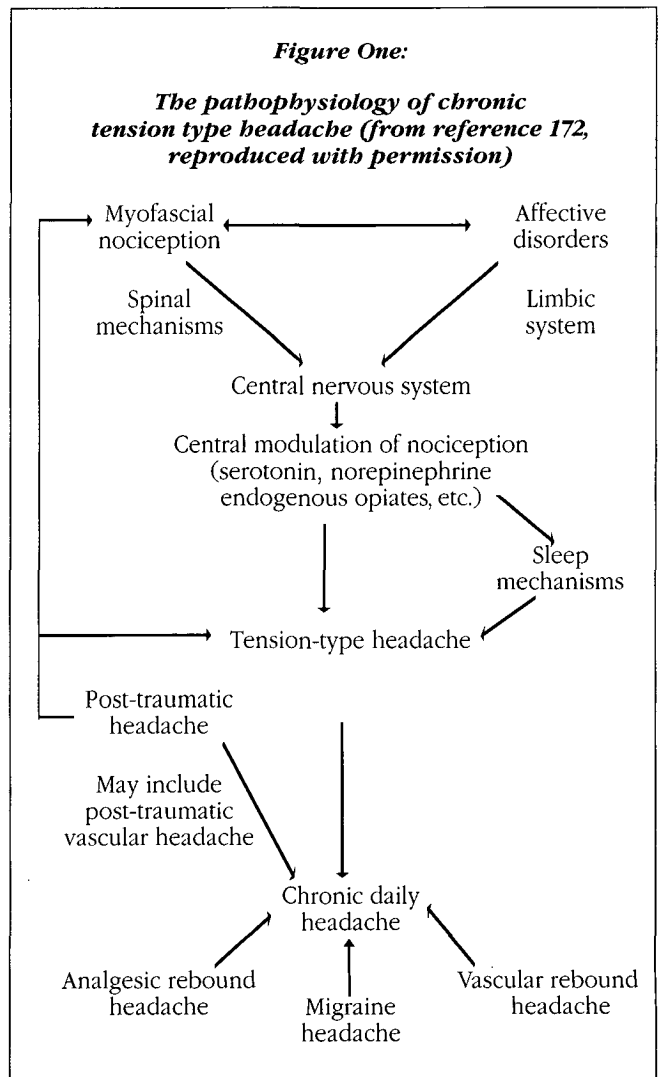




Figure Two

## HNRIC- Functional assessment scale— example

		Admission Date	Date	Date	Discharge Date	
1	Medical management	2	3.5	4.0	4.5	
	(work adjust.)					
2.	Vocational functioning	3	3.0	3.0	3.5	
	(OT)					
	(speech)	3	3	3	4.0	
	(psych)					
3.	Home managment	2	5	5		
4.	Money managment	2	4	5	5	
5.	Transportation	3	5	5	5	
6.	Self care	3	5	5	5	
7.	Communication	3	3	3.5	3.5	
8.	Interpersonal/ pragmatic ability	3.5	3.5	3.5	3.5	
	(speech)					
	(OT psych)					
9.	Family functioning/ Social network	2.5	3.5	3.5	3.5	
	(psych)					
	(social work)					
	(OT)					
10.	Psychological functioning	2.0	2.5	2.5	2.5	
11.	Neuropsychological functioning	2.0	2.0	2.5	2.5	
12.	Neuromuscular functioning	4.0	4.5	4.5	4.7	
13.	Pain					
	(PT)	4.0	4.5	5.0	4.5	
	(medical)	4.0	4.5	4.5	4.5	
14.	Fitness	1.5	1.5	2.0	3.0	
15.	Leisure activites	2.0	3.5	4.0	5.0	
		40	54	58	60.7	$\Delta = 297 = 26\%$
		2.7	3.6	3.9	4.0	$\Delta = 1.3 = 26\%$

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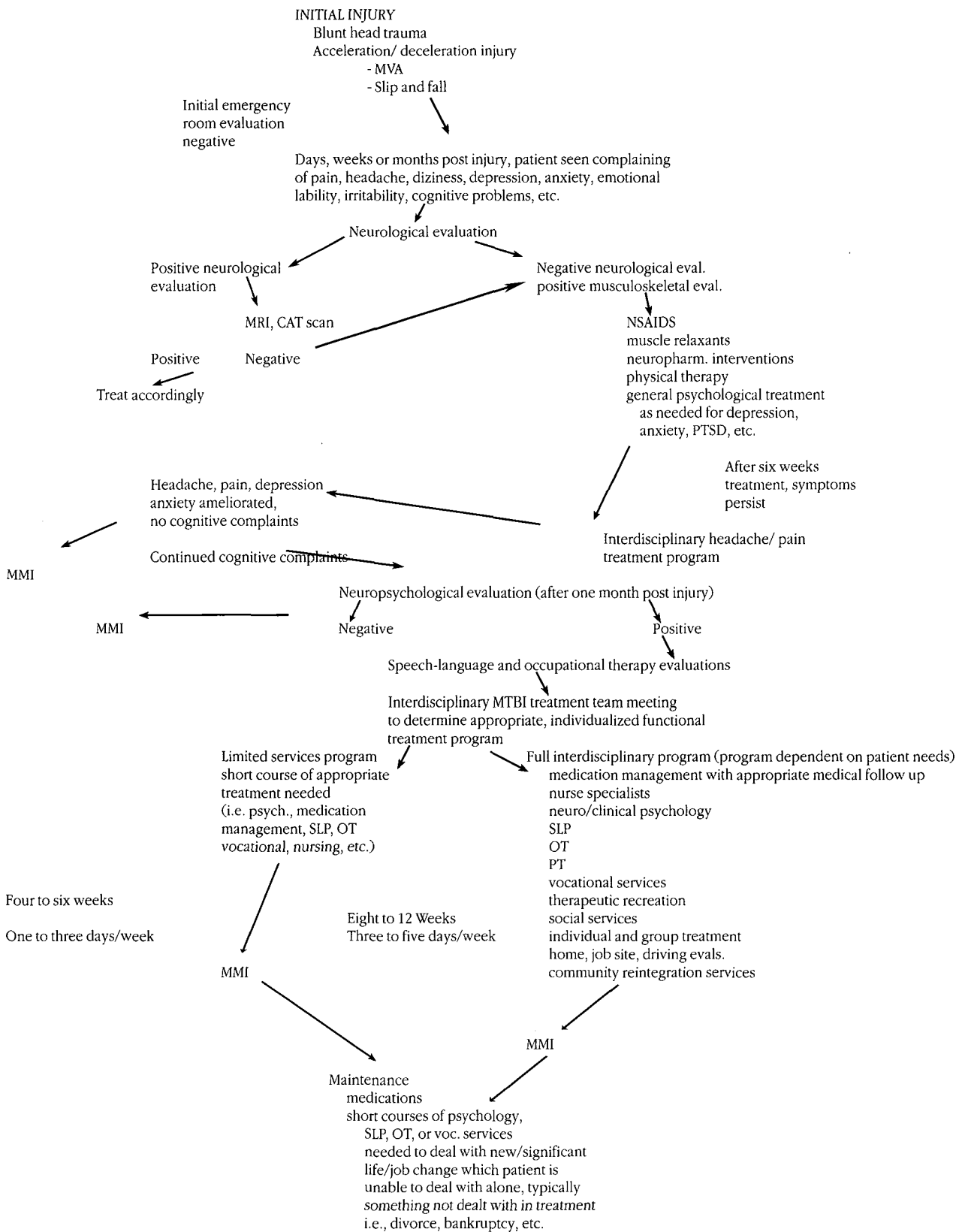
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**Figure Three**

**Post acute MTBI evaluation and treatment algorithm**



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