Minor Traumatic Brain Injury handbook

Diagnosis and Treatment

Gary W. Jay

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Dedication

This book is dedicated to many people, especially the patients who have survived and even prospered after an MTBI, as well as to the physicians, other clinicians, and the attorneys who fought for them.

As always, this book could not have been written without the loving support of my wife Suzanne and my daughter Samantha, who has begun to write her own books.

Finally, this book is also dedicated to Alvin Arakaki, who learned early and taught the right thing to do. He is missed.

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Foreword

Did you know that there is no such thing as mild (acquired) traumatic brain injury (MATBI)? For two of the largest automobile liability insurance companies, this is their reality.

Don't you love it?

And if you don't believe them, there are a host of clinicians, many of the neuropsychological persuasion as well as neurologists and other physicians who are, apparently, charter members of the "If I Don't Want to Believe It, It Ain't True School of Medicine."

So, is MATBI (in this book, synonymous with MTBI) a form of mass hallucination? Is it a form of mass hysteria?

Neither explanation can answer the question: Why do patients from all over the country who experience similar types of trauma have such similar if not identical complaints? Do they all read the same Cliff Notes®? Are they all determined to "rip off the insurance companies?"

Of course not! The facts of the matter are simple: MATBI does exist. It is real. It can devastate a patient and his or her family. It is not a wastebasket diagnosis that is used by unprofessional clinicians when they don't know what is going on. It is not a different way of saying, "depressed."

Over the last eight years, since the advent of more mangled — uh — managed care, I have had to hire a full time person just to get approvals for treatment from the automobile medical liability companies. I have watched my patients undergo Independent Medical Examinations (IMEs), one after the other, demanded by the insurance companies for the sole purpose of stating that there is absolutely nothing wrong with a patient. This, while the patients lose their jobs, their families, even undergo bankruptcy, all because the insurance company or their trusty henchman, the third-party administrators, refuse to accept or acknowledge the diagnosis.

I have undergone deposition after deposition at the request of the insurance company, typically, where their pet attorney does his or her best to try to disprove what I have stated and written about a patient's medical problems and diagnosis.

My patients have never lost a case, with the exception of a 72-year-old gentleman, who for other reasons did his own case in, on his own.

For the most part, the purpose of this book is to present the facts in a coherent manner which is relatively easy for physicians to read and understand and to help them sharpen their diagnostic skills regarding MTBI; to give the attorneys who care about their clients another source of medical information they can use to help them; and to be useful to all of the above when some microcephalic minion of an insurance company states that there is no such thing as a mild traumatic brain injury.

That being said, I want to explain this textbook. It has two parts. Part One is a medical overview of most of the important aspects of MTBI. I wrote it in the same

style I used in the *Headache Handbook*, as I have received many wonderfully complimentary comments about that book. The second half of this text is written by a number of different professionals, and details the clinical aspects of diagnosis and treatment by specific specialty. I was even lucky enough to find an attorney, who I respect greatly, to write a chapter on the legal aspects of MTBI.

I have included patient case studies in Part One, along with the now infamous "Ah-Has" from the headache book, which are now the *Bottom Line*, important points that really need to be considered.

In my clinical experience, patients with MTBI are remarkably underserved, underdiagnosed, and generally, misunderstood. Because of the seriousness of the consequences of those two "uns" and one "mis," I hope that the reader will take the time to learn the facts and use them to help the patients who experience the trauma of a minor acquired traumatic brain injury.

Because of the breadth of the topic, I have included information about, but not dwelt on, the moderate to severe traumatic brain injury patients. This book is for the clinicians and others who deal or need to deal with the patients with minor or minor to moderate traumatic brain injury.

I have tried to maintain the "just like I lecture" style, but I have greatly supplemented that with very pertinent information from hundreds of authors.

Other clinicians have been good enough to read though this manuscript in draft form. Both were unsure about including my comments on the insurance problems faced by the MTBI patient. After several discussions, they agreed that this information, like the clinical information, is fact, and should therefore be fair game for a textbook.

Facts are facts, whether you like them or not.

To all of you clinicians and readers who help to care for these patients, as well as for all of you patients who may access this book, "God Bless You." This one's for you!

I hope you will find this text useful.

Part One

1 What is Mild Traumatic Brain Injury?

The problem of mild acquired traumatic brain injury is an old one. It was described in the 1860s by Erichson and Trimble[1,2] and called the "postconcussion syndrome," as well as the "posttraumatic syndrome." Dr. Page, in the mid 1880s, began a good bit of controversy by expressing doubts that the closed "spinal concussions" seen in the railway accidents described by Erichson were real.[3] He went so far as to indicate that patients suffering from closed brain or spinal nondefinable injury were malingerers.

The workers' compensation laws were introduced in the late 1880s and made much more worker friendly in 1906. This had a significant effect on the growing debate, as more complaints of similar work-related injuries were made.

The medical investigations into the pathophysiology of closed head trauma and closed head acceleration/deceleration injuries began in the 1940s.[4] Over the following two decades several medical papers notably concluded that minor closed head injury and/or simple concussion could cause significant neuronal loss and profound clinical changes.[5,6] While the pathophysiological mechanisms were not known, MTBI was seen as a real clinical entity.

A major problem was noted. It appeared that minimal or minor closed head trauma frequently induced emotional or "neurotic" changes in patients. The early difficulties delineating cerebral function and/or dysfunction which had resulting characterological changes were problematic.[7-11] As the neurological examination was frequently found to be essentially normal, such posttraumatic sequelae were felt to be fallacious and the patient a slacker.

These problems were exacerbated by the members of the medical-legal field, with attorneys in workers' compensation and personal injury law trying to prove or disprove real clinical dysfunction, while physician experts had very little in the way of objective clinical or radiological evidence to make their point.

Starting in the 1960s, more research began to support Erichson's original contentions that minor head trauma could induce severe disturbances of cerebral function.[12-14] Evidence of true dysfunction was identified by more sophisticated neurological and vestibular testing.[14] Clinicians only infrequently used neuropsychological testing early on, but it added more data showing cognitive dysfunction after minor head or soft tissue trauma, including the medical problem legally known as "whiplash", or cervical extension/flexion or acceleration/deceleration injury.

The so-called posttraumatic syndrome, which was called the postconcussion syndrome if there was an attendant loss of consciousness, was found to produce, in

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some patients, a host of varied symptoms with or without accompanying objective clinical neurological findings. These symptoms were found to vary, in many cases secondary to, it was initially felt, the age and emotional or psychological predispositions of those injured.

Mild (acquired) traumatic brain injury (MATBI or MTBI — used interchangeably in this book) is the clinical entity in which the brain has sustained a pathological injury. The pathology can be secondary to a direct contusion, or neurochemical, axonal, or circulatory injury.

The nosology of the term "minor TBI" must be called into question. It is noted that MATBI may induce neuronal dysfunction which may produce persistent symptoms, indicating that such "mild" injuries to the brain may produce effects which are not "minor" at all, and which may last for indeterminate periods of time.[15]

At this time there is, thankfully, a consensus definition of mild traumatic brain injury which 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.[16] This 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 of 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. Posttraumatic amnesia is not greater than 24 hours.

It is extremely important to note that the definition includes patients with direct head trauma *as well as* those who suffer an acceleration/deceleration injury ("whiplash") without specific direct head trauma. Loss of consciousness *is not a clinical requisite for a classification of MATBI*, in spite of the pronouncements of multiple pseudoexperts, including those who do know better, those who should know better, and those who get paid to skirt the truth. (I am trying to be nice here!) These are the good folk who should know the difference between "lies, damn lies, and statistics."

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

Zasler 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."[17]

To that, I add, "It depends on who is looking, and *why*: if they are patient-oriented, or working for an insurance company or defense attorney, their findings may be very different from those of someone who cares for the truth and medical accuracy as it pertains to a specific patient."

MATBI is a contentious issue in very litigious times. The diagnosis, and especially the treatment, of MTBI can be rather expensive, and in the managed care environment, no one wants to be responsible for fulfilling an insurance contract. It may injure the insurance companies' bottom line. It would also cause a loss of income for attorneys who work for insurance companies, who get paid to "prove" that no one ever suffers an MATBI.

Now, if a person is injured and his brain is literally oozing out of his ear, it is extremely difficult to declare that such a patient does not have a brain injury. Fortunately or unfortunately, the patient who suffers an MATBI may look normal. That makes it easier to sell the lie to a jury.

The purpose of the rest of this book is to make the pathophysiology, diagnosis, and treatment issues of an MATBI perfectly clear. To anyone and everyone.

Personally, I am tired of seeing patients who have suffered an MATBI be made worse by the deadly combination of iatrogenic and nomogenic factors. This will be dealt with later in this text.

For now, we'll move on to the epidemiology of the problem. If there is no such thing, how come it is found in multiple societies by multiple people who can't even read about, never mind practice, the symptoms of the disorder that isn't there?

2 Epidemiology and Causation

One of the major problems clinicians face when attempting to obtain any idea of the true epidemiological aspects of MATBI is that the literature is rife with studies which utilized different criteria for the diagnosis of this entity.

INCIDENCE

Studies performed during the 1980s and early 1990s that attempted to quantify the incidence of MATBI were methodologically different and may not necessarily be considered equivalent. Inclusion criteria were different in most of these studies. A large number relied on subjective patient information, with both urban and rural patients being given the same written or verbal questionnaires.[18-27] The incidence of MATBI ranged from 152 to 367 per 100,000 people. Again, significant differences were found in the methodologies of these studies.

Krause,[28] in 1993, felt that the general incidence was 200/100,000 population. More recently, Krause again stated that there were approximately 2,000,000 brain injuries occurring each year, an incidence of 175–200 per 100,000 population, with an associated 56,000 deaths per year.[29] For now, these figures appear to be the most commonly cited.

In these figures, from the way Kraus evaluated the numbers, there may be a significant number of individuals who experience an MATBI and who do not go to an emergency room or immediately to their primary care provider, an MD, or a chiropractor. The actual number of these patients has not been established.

Another problem is the clinical acumen of primary care physicians, as well as specialists, who may not make the diagnosis when a patient presents to them. Some counties that have regional centers appear to do better at diagnosis, as will be discussed below.

Stewart and his associates[30] tried to determine the frequency of cognitive deficits in emergency room patients with MATBI, and to identify the factors in the initial history and physical examination that would be predictive of cognitive deficits. Seventy patients were admitted into their study, all having a history of blunt trauma or deceleration injury to the head, and a Glasgow coma scale (See Chapter 9) of 14–15. Only 36 completed the follow-up, and 42% of those patients completing the study had either mild or moderate cognitive deficits one week post injury. The authors concluded that history and initial physical examination were poor predictors of these deficits. They also reported that the patients who completed the study were more

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commonly employed and less likely to have used alcohol or "sensorium-altering" drugs. Of interest was that the finding of abnormal cerebellar function noted in the initial evaluation was associated with cognitive deficits at one week. Only 4 of 15 patients with initial cognitive deficits had abnormal cerebellar examinations at follow-up.

Another study found the importance of immediate expert care for traumatic brain injury. Gabella et al.[31] compared urban to rural traumatic brain injuries in Colorado for 1991 and 1992. Annual, average traumatic brain injury varied from 97.8 per 100,000 population for residents of the most urban group, to 172.1 per 100,000 population for the residents of rural, remote counties. Mortality rates ranged from 18.1 per 100,000 in the urban setting to 33.8 per 100,000 people in the remote rural populations.

Another report from the same year, 1997, indicated that the data from Colorado, Missouri, Oklahoma, and Utah, when evaluated from l990–1993, included a decreased annual rate of TBI, and that the rates of TBI were highest in association with motor vehicle accidents and falls.[32] In 1992 a report in the *Oklahoma State Medical Association Journal* stated that 4000 people were disabled or killed after head injury each year in Oklahoma.[33]

The Virginia Brain Injury Registry analyzed statistics for 1988–1993. They found age-adjusted incidence rates of TBI were greatest for children under 6 years of age at 237/100,000, and least for persons aged 40–69 years of age, at 56/100,000. They noted that TBI occurred more frequently in males (1.4:1), and mortality rates were also higher in males as compared to females (1.6:1).[34]

Surveillance of TBI cases requiring hospitalization or that were fatalities in Utah during 1990-1992 found an annual incidence rate of 108.8 per 100,000 population. This rate was significantly lower than previous identified rates of TBI.[35] The Alaskan Trauma Registry looked at hospital trauma admissions in 1991–1992 and found an incidence rate of 129.5 per 100,000 population.[36]

Note that the differences between the incidence rates appear to depend on a number of factors, including hospitalization and mortality. Very little comment is made regarding mild traumatic or mild to moderate traumatic brain injury, as the ability to diagnose this problem is dependent on follow up not typically performed by a hospital emergency room.

The epidemiology of TBI has been looked at in various countries, but specific incidence data has not always been given.[37-42]

A national survey conducted by Statistics Canada in 1986–1987 found the overall household prevalence rate of TBI was 62.3/100,000 adults, with a male predominance. They found that the TBI rates were highest in the 45–64 age group, which was three times greater than the 15–24 age group.[43] They also determined that 84% of adults with TBI have co-occurring disabilities, particularly limited mobility and agility. Again, no specific information regarding MTBI was garnered.

Two Australian studies looked at the incidence of TBI in north versus south Australia. The incidence of hospital-treated TBI in the North Coast region found an annual incidence of approximately 100/100,000 population. They noted that most of the injuries were mild (62.2%).[44] South Australia had a much higher incidence of TBI, 322/100,000 population, which exceeded studies with comparable

methodologies in areas of the United States and Europe. Young males had the highest incidence of TBI, typically secondary to a motor vehicle accident. Hospitals in the area surveyed accepted more than 4000 new cases of TBI each year. At discharge, over 1000 of these cases had some degree of residual impairment and required postinjury services.[45]

A New Zealand study attempted to identify the incidence of MTBI.[46] This study defined MTBI by the acute management needed, including care out of the hospital or hospital admission of not more than 48 hours, including the presence of posttraumatic amnesia. The incidence seen at the four Auckland hospitals was 437/100,000 population for ages 15 and over, and 252/100,000 population for ages under 15. The major causes were motor vehicle accidents and falls. Persistent symptoms occurred in 5% of patients 15 years or older.

The epidemiology of TBI in Johannesburg, South Africa, was evaluated.[47] The overall incidence was 316/100,000. The incidence in whites was 109/100,000 overall, with a male-female ratio of 40.1. The data for blacks showed an incidence of 355/100,000 population, with a male-female ratio of 4.4:1. There was an incidence in black males aged 25–44 of 763/100,000 population. Whites had a 419/100,000 incidence in the same age group. The overall incidence of fatal TBI was 80/100,000. The nature of the injuries showed interpersonal violence accounting for 51% of nonfatal TBI among blacks, and only 10% among whites. Motor vehicle accidents caused 27% of black nonfatal TBI and 63% for whites. In spite of the large incidence, no data on the diagnosis of MTBI was given.

In northern Sweden a retrospective study found the incidence of TBI to be 24.9/10,000 population for the age range of 16–60. It was found that many of the patients with an early diagnosis of brain concussion, who were hospitalized for one day, experienced losses in preinjury functions and abilities.[48]

Northern Norway's incidence of TBI was found to be 229/100,000 population during 1993, with a male preponderance of 1.7:1. In this retrospective population-based survey, the most common causes were found to be falls in 62%, motor vehicle accidents in 21%, and assaults in 7%.[49]

A study from Taiwan found 58,563 TBI cases over a six-year period (July 1, 1988 – June 30, 1994). The major etiology of trauma was traffic accidents (69.4%), followed by falls and assaults. Motorcyclists accounted for the majority of TBI cases among traffic accidents. Using the Glasgow coma scale, 79.5% of cases were considered mild, 8.9% moderate, and 11.6% severe. Outcomes were determined by the Glasgow Outcome Scale (GOS), and good recovery was found in 87.2% of cases.[50] Note that the percentage of "good outcomes" was higher than the number of "mild" TBI patients. Using the GOS, a "good outcome" may be associated with moderate disability.

BOTTOM LINE

The epidemiological studies rarely looked at MTBI. Those that did specifically, and those that did not (the vast majority), had different diagnostic criteria and methodologies. The outcome determinations were different.

It appears that the lack of specific studies belies the true incidence of MTBI. Part of this is most probably specific diagnostic criteria. A good bit of it is the lack of emergency room follow-up. That is, ER folks don't know, usually, what happened to a patient after discharge from the ER. This is not a criticism. Follow-up is not their job.

On the other hand, many patients may not immediately go for follow-up with their primary care physicians. If they do, and I've seen this far too often, their symptoms are discounted and the diagnosis of MATBI is usually not made, or the patient is bluntly told that they should be all better in three to six months, that any problems that persist are either illusionary or secondary-gain related.

Then there is the issue of patients who experience head trauma, with alterations in mental state at the time of the accident, possibly with a short period of loss of consciousness or minimal memory loss, who do not seek medical attention. This may be secondary to lack of insurance, or lack of close medical facilities for rural patients. It may also be secondary to the immediate feeling that, "I'm all right." These patients then experience the full brunt of medical and legal antagonism if indeed they are not "all right" and see a physician weeks or months later after being unable to deal with any persistent symptoms.

So, it appears that the real incidence of MATBI may not as yet be known.

Two recent studies give one pause. A look at the incidence of TBI in a New Zealand prison population found that 86.4% of 118 respondents had sustained a TBI, with 56.7% reporting more than one. All reported problems with general memory and socialization.[51]

An attempt to evaluate the prevalence of TBI in a psychiatric population was done.[52] It was found that a greater percentage of psychiatric patients reported TBI than control groups of medical patients and students. The TBI was typically considered mild, according to the Traumatic Brain Injury Questionnaire used to assess TBI. The authors note that the role of TBI in the emergence, expression, and treatment outcome of psychiatric patients needs to be further examined.

Yes, it does.

CAUSE OF INJURY

As noted in the brief review above, motor vehicle accidents (MVAs) are the most common cause of TBI, followed by falls, violence, and recreation.[18,21,23,25,53] MVAs appear to account for approximately 50% of the TBIs.

It has always been fascinating for me to observe the sophistic machinations of the (typically) insurance defense industries which utilize nonmedical criteria to state that a patient could not have been injured in a low velocity (or a high velocity) motor vehicle accident. They claim that it just isn't possible. While I don't feel that this textbook is the place to evaluate the physics and biomechanics that these people cite, it is very much worth mentioning.

The things which are important in the evaluation of an MVA include: the physical attributes of the driver and passengers (size, age, strength, immediate knowledge that the accident will occur, use of alcohol or drugs, physiologic impairment, and experience), vehicle design, and environmental factors. At the time of the actual

accident or crash, important factors include physical attributes of the occupants of the vehicle, use of restraint systems, and safety modifications to the vehicle.

At the time of the accident, did the patients have the time to brace themselves, or were they taken totally by surprise and unprepared? Was the patient's head turned at the time of the vehicle crash? What were the physical characteristics of the patient?

Clinically, I see fewer physical problems in patients who were prepared and had braced themselves immediately prior to the crash. Fewer of these patients appear to develop significant cognitive deficits, particularly if their heads are braced sufficiently to prevent an acceleration/deceleration injury (whiplash), but, the physics must be considered.

When a two-ton vehicle strikes another massive piece of metal, the force of the impact is imparted "down-line," to the part of the vehicle (or its occupant(s)) which is least connected to the frame. That is typically the occupant(s). The least massive part of the occupant is the neck, which is connected to the body at one end and to the head at the other. The head is heavier than the neck, so the physical force of the crash is most commonly directed to the most moveable part — the head. A forward then backward (acceleration/deceleration) movement occurs, many times more than once, and is frequently associated with the back of the head striking the headrest or, in a small truck, the rear window. If the head is well-braced, the forces from the crash may still effect the lesser-braced entity — the brain, which floats in fluid and cannot be tethered down. The brain itself can undergo the brunt of the physical forces by being forced forward and backward onto the bony cage (skull) that encases it. If the head itself is not braced and undergoes the resultant acceleration/deceleration, these movements may amplify the injurious effects on the soft, essentially gelatinous brain tissue.

If the occupant's head is rotated at the time of the crash, these rotational forces, which accompany the forward/backward acceleration/deceleration forces on the brain, make the patient far more likely to sustain a cerebral injury.

There are many variables, starting with the ability of the patient to brace prior to the impact forces. The size of the automobile's occupant is important. A low velocity impact would possibly have much less physical damage associated with it if the driver was the phenomenal Denver Broncos quarterback John Elway, as compared to someone who is not in good physical condition strengthwise, does not have an 18-inch neck, is less than 5 foot 6 inches tall, is female, and so on.

I've never heard these types of facts dealt with by the so-called defense experts whose stories depend on who is paying their bills. This is disgraceful, nonmedical, and sophistic, but the courts of law tolerate it, much to the detriment of truly injured patients.

Gennarelli et al.[54] noted that pedestrians who are struck by motor vehicles are most likely to sustain head injuries. In motor vehicle versus bicycle accidents, between 1984-1988, there were an estimated 2,985 TBI deaths and 905,752 bicycle-associated head injuries.[55,56] Motorcycle riders (or donor-cycle riders, if you prefer) have a 5- to 6-time higher risk of TBI than people in other fatal MVAs.[57] Between 1979–1986 there were 15,194 motorcycle deaths associated with head injury in the United States.[57] In 43% of motorcycle fatalities, the drivers did not use helmets.[58]

And still, people fight for their right to forego wearing helmets when they ride motorcycles or bicycles. We pay for their freedom to donate their lives and their organs.

The second leading cause of TBI is falls, which account for 20%–30% of injuries. The majority of falls involve in children under 5 years of age and people over 75 years of age.[18,21,23,28,59]

Recreational injuries, particularly sports-related injuries, are routinely underestimated.[60] TBI may occur in 5% of football injuries.[61] Football also has the greatest percentage of concussive injuries of all contact sports.[62]

Boxing is the only sport whose sole purpose is to render an opponent unconscious. The "punch-drunk" syndrome was first identified in 1928. Subsequent studies have identified pathognomonic, neuroanatomical changes associated with this syndrome, including fenestrated *cavum septi pellucidi* and neurofibrillary tangles without senile plaques.[63,64] Neurocognitive changes are also seen.

Another major factor in the pathogenesis of TBI is alcohol and drug abuse. Alcohol use is a predisposing factor in 35%–72% of all TBIs, particularly in relationship to MVAs, assaults, falls, firearm accidents, and other causes.[65-66] While illegal drugs were found in significant numbers of tested patients in an urban trauma center, the role of prescription drugs is less well defined.[67]

BOTTOM LINE

The majority of causes of TBI are controllable or, at the very least, amenable to change. New technology in motor vehicles is attempting to decrease the overall morbidity and mortality of MVAs. Injuries secondary to bicycle and motorcycle accidents can be lessened by the use of appropriate protective equipment, such as motorcycle helmets. The use of alcohol and drugs is a very difficult societal problem, but laws are striving to make the use of alcohol or drugs while driving more serious offenses.

It is obvious that we can work to decrease the incidence of TBI, but it will be slow going, to say the least.

Next, we go into a gray area to some. The postconcussion syndrome has been thought to be another term for TBI. But, is it?

3 The Post-Concussion Syndrome

The post-concussion syndrome (PCS) appears to include multiple signs and symptoms consisting of neuropathological, neurophysiological, and neuropsychological, as well as physical and psychological or emotional aspects, secondary to a mild traumatic brain injury.[68]

The most common medical problems found in the patient with PCS (and MTBI) include:

- Posttraumatic headache
- Posttraumatic musculoskeletal pain syndromes
- Vestibular disturbance
- Visual disturbance
- Fatigue
- Posttraumatic seizure disorder

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
* Work finding problems
* Decreased ability to concentrate
* Poor impulse control
* Slowed behavioral processing
* Behavioral/personality changes
* Perseveration

The PCS can be both chronic and disabling, or short-lived and benign. A possible explanation for this may be the interaction between organic and psychological factors.[69] It is very difficult to differentiate between the effects of primary neurological, neurophysiological, and neuropathological injury and secondary psychosocial factors. It is felt by some that the typical PCS symptoms, including headache, dizziness, and irritability, result from emotional stress associated with diminished cognitive performance secondary to MATBI.[70]

The influence of accident mechanisms associated with more severe symptoms was studied and it was found that patients with more severe deficits had, at the time of a motor vehicle accident: been an unprepared occupant; been in a rear-end

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collision, with or without subsequent frontal impact; and had a rotated or inclined head position at the moment of impact.[71]

The "postconcussional disorder" (PCD) has been recently accepted and is found in an appendix of the DSM-IV. A major criterion is loss of consciousness. It is felt that it would be better to utilize the BISIG definition (see chapter 2).[72]

Many researchers have looked for a primary psychological/emotional etiology for the PCS.[73]

Gasquoine[74] felt that symptom persistence was associated with increased emotional distress. He notes that this fact is also true in patients with severe head injury as well as back injury, and relates more to the patient's interpretation of the effect of the trauma than to objective "indicators of brain injury severity."

Landy[75] looked at the more objective symptoms of headache and cervical pain and found that 70% of patients "get better" within a few weeks post MVA, while about 30% continued to complain of headaches and/or cervical pain. He felt that prolonged management and slow court settlement lead to extensive introspection by the patient and, thus, prolongation of symptoms. His results also repeat the long held knowledge that patients with more severe head or neck injuries had a lesser incidence of chronic post-traumatic headaches or cervical symptoms.

Barrett et al.[76] compared two groups of PCS patients, one of which was hospitalized for observation following a brief loss of consciousness, while the others went to the emergency department, and then home. It was found during follow up at two and twelve weeks that the type and frequency of complaints were similar in both groups. However, at twelve weeks, the number of complaints/symptoms were significantly less in the group of hospitalized patients.

Several groups noted that the PCS was more frequently found after blunt head trauma and other trauma than would have been predicted.[77,78]

Using a questionnaire, Bohnen et al.[79] evaluated the longevity of long-term PCS complaints. Their results indicated that in a percentage of patients, MTBI might not ever resolve.

In an attempt to evaluate the importance of psychological factors in the outcome of whiplash injuries, Mayou and Bryant[80] utilized interviews at 3 and 12 months postinjury. The majority of the patients in their study continued to complain of persistent cervical symptoms, while a "sizeable minority" reported specific posttraumatic psychological symptoms such as intrusive memories as well as phobic travel anxiety, which was felt to be "similar to those described by patients suffering multiple injuries". They concluded that travel, social and psychological morbidity was more prevalent than previously recognized. They did not deal with the issue of the recognized posttraumatic stress disorder (PTSD).

Cicerone and Kalmar[81] urged clinicians to use a great deal of caution before attributing PCS symptoms or neuropsychological deficits to a preexisting affective disorder. Leininger et al.[82] looked into the idea that MTBI patients do not develop persistent neuropsychological deficits. They found that patients with the PCS/MTBI had measurable neuropsychological deficits, and the severity of those deficits was independent of gross neurological status immediately post injury.

Looking at symptomatic patients two years post whiplash injury, Di Stefano and Radanov[83] evaluated complaints of memory and attentional difficulties with

neuropsychological testing. They found that memory problems were minimal, while problems in selective aspects of attentional functioning after whiplash were present. These could explain the patients' cognitive complaints, and could induce adaptational problems in daily life.

An interesting study was performed by Parker and Rosenblum,[84] who looked at intelligence and personality difficulties after whiplash or MTBI in adults, an average of 20 months post MVA. They found a mean loss of 14 points of Full Scale IQ from the estimated preinjury baseline (using WAIS-R) with no evidence of recovery. They also found a number of personality dysfunctions including organic or cerebral personality disorder. Thirty of 33 patients had psychiatric diagnoses including post-traumatic stress disorder, psychodynamic reactions to impairment, and persistent altered consciousness. They concluded that cognitive loss was induced by the interaction of brain injury with distractions including pain and emotional distress. The report also repeated the fact that the presence of MTBI after MVAs was probably consistently underestimated.

While the PCS has been thought of as a reflection of the psychological response to injury, there is considerable evidence suggesting that the PCS is primarily a physiological disturbance.[77] Reaction time testing, for example, has been used to support a structural, organic etiology for the PCS.[85]

It has been found likely that cervical injury contributes to the symptomatology post PCS/MTBI, and vice-versa.[86] Testing has shown that cervical injuries secondary to whiplash can induce a distortion of the posture control system as a result of disorganized cervical proprioceptive activity.[87] Others note that restricted cervical movements and changes in the quality of proprioceptive information from the cervical spine region affect voluntary eye movements. Acceleration/deceleration (flexion/extension) injury to the neck secondary to whiplash may result in a dysfunction of the proprioceptive system. Oculomotor dysfunction after cervical trauma may therefore be related to disturbances in cervical afferent input.[88] Patients who have sustained head or cervical trauma appear to exhibit an increased reliance on accurate visual input, and are unable to utilize vestibular orienting information to resolve conflicting information from the visual and somatosensory systems.[89]

Soustiel et al.[90] evaluated 40 patients post mild head trauma using brainstem trigeminal and brainstem auditory-evoked potentials (BTEP, BAEP) and middle-latency auditory-evoked potentials (MLAEP) within 48 hours of injury and again at 3 months. They defined PCS as the presence of at least four of the following: failure to resume previous professional activity, memory deficits, headache, dizziness and vertigo, behavioral and emotional disturbances, and other neurological symptoms. Initially, all three evoked potentials were abnormal, showing prolonged latencies indicative of disseminated axonal damage. Only the MLEAPs correlated to outcome at three months, particularly in its psychocognitive aspects, suggesting that organic diencephalic-paraventricular primary damage may account for the presence of the PCS.

PET, SPECT, and MRI studies have been done to attempt to correlate cerebral dysfunction to PCS symptoms. (See Chapter 4 for further information on this technology.) PET looks at glucose metabolism (in these studies), while single photon emission computed tomography (SPECT) looks at cerebral perfusion.

Six patients with PCS and 12 normal controls were tested. The patient group had significant hypometabolism and hypoperfusion in the bilateral parieto-occipital regions, as compared to the controls. In some patients there was also hypometabolism found in other regions. It was hypothesized that parieto-occipital hypometabolism can be caused by activation of nociceptive afferent nerves from the upper cervical spine.[91]

Another study examined 13 patients with a late whiplash syndrome, using PET and SPECT. The authors did not find hypometabolism in the parietotemporo-occipital regions. They did find hypometabolism in the frontopolar and lateral temporal cortex, and in the putamen. They did not recommend that PET or SPECT be used as diagnostic tools for routine examination of patients with a late whiplash syndrome.[92]

SPECT was compared to MRI/CAT scans in 43 patients. The SPECT was found to be abnormal in 53% of patients, MRI was abnormal in 9%, and CAT scan was abnormal in 4.6% of patients post MTBI/PCS. The SPECT scan appeared to be more sensitive to post MTBI changes, especially in patients with persistent PCS (see below), than MRI or CAT scan. No statistical relationships were found between the SPECT scan results and age, previous psychiatric history, history of substance abuse, history of multiple MTBI, or concurrent neuropsychological symptoms.[93]

BOTTOM LINE

"The truth is out there," but we don't seem to have determined the best method of identifying it. The tests noted above were given to patients with PCS, by author statement. The relationship between PCS and MTBI is discussed below, as well as in the next chapter.

Nosologically, it is difficult to determine exactly what constitutes PCS. Evans[94] states that PCS refers to the large number of signs and symptoms found alone or in combination following MATBI, including headache, memory problems, dizziness, fatigue, irritability, anxiety, insomnia, and sensitivity to light and sound. He further indicates that studies have substantiated the existence of PCS, that it is common, with resolution in three to six months, but with persistent symptoms and cognitive deficits persisting for months or years.

Headache, dizziness, and memory deficits are the most common combination of PCS symptoms.[95] There is no specific symptom complex found in the majority of patients with acute or chronic PCS.[96] The multiplicity of signs and symptoms of PCS have been well documented.[69,95,97-105]

One group has suggested that PCS should include all of the consequences of head injury, regardless of its severity and the nature of the injury.[106]

Berrol[107] states that the term mild traumatic brain injury (MTBI) is preferable, as it identifies the etiology of the injury, its degree, and the pathological substrate much better than other past terms: minor head injury, traumatic head syndrome, postconcussive syndrome, posttraumatic syndrome, postbrain-injury syndrome, and traumatic cephalgia.

The term postconcussive syndrome (PCS) continues to be frequently used in the literature. The important nosological question is whether PCS is secondary to the MTBI, or are the cognitive/neurological deficits found after MTBI separate entities.

The term PCS would then encompass the nonneurological, neurocognitive, and neurophysiological deficits, leaving the term PCS to be used specifically for the other organ (noncerebral) systems that display posttraumatic signs and symptoms.

BOTTOM LINE

Teleologically, it appears to make more sense to separate the etiologies of the problems encountered post MATBI. A patient with physical findings such as post-traumatic headache may indeed, post trauma, have a postconcussive syndrome. Patients with neurocognitive deficits and other neurological difficulties have direct evidence of a (mild) traumatic brain injury. The author feels it more appropriate to differentiate the two disorders. This would mean that a patient may indeed have both an MTBI and a PCS. Both entities must be treated, and, as will be discussed later, the PCS should be treated first.

Soon after injury, patients have complaints referable to several different organ systems. Alexander[108] identifies this as the PCS. He notes that the MTBI, which can lead to brain injury, can also cause injury to the head, neck (whiplash and soft tissue damage), the vestibular system, and psychological functioning. The initial complaints of deficits in cognition and sleep disorder are, he feels, secondary to neuronal injury, while the headache may be secondary to cervical injury, neuronal injury, or a combination; cervical 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.

BOTTOM LINE

The term PCS should not include central nervous system deficits. Vestibular dysfunction secondary to brainstem injury should be included in the MTBI while peripheral dysfunction should be a part of the PCS.

To the extent plasticity allows, neuronal recovery is certainly taking place at one month after injury.[109-113] Neurological recovery is thought to be "substantial," by some, at three months.[114] At this point, post injury, 30% to 50% of patients have continued complaints.[115] Over the next 6 to 12 months (longer than a year post injury) most patients will show continued improvement and "recovery."[116]

It has been found that even "well recovered" patients are still susceptible to periodic impairments secondary to physiological or psychological stress,[117,118] which indicates that recovery is most likely the wrong term. That these patients have "compensated" for their injury may be more correct. To say that patients may have a permanent sense of decreased mental or cognitive efficiency[119] would also be a function of incorrect terminology, i.e., recovered versus compensated.

Persistence of Symptoms

At one year, 85%–90% of patients are felt to be "recovered" but are still symptomatic,[106,120] leaving 10%–15% of patients who are not only "not recovered," but are also "not compensated" and still very symptomatic. The literature is replete with

studies showing persistence of symptoms after the magic, if not mythic, 3-month period. This literature indicates that the symptoms and deficits following MTBI and PCS may last for six to twelve months or even longer.[82,94,107,119,121-125]

Problem

Much of the literature equates MTBI and PCS, essentially using the terminology interchangeably. Therefore, breaking the literature reviews and thoughts into PCS and MTBI chapters by the author does not delineate both syndromes as "Bottom Lined" above. The majority of the literature includes cognitive and other neurological deficits in PCS.

A survey of rehabilitation specialists who followed patients with MTBI for 6 to 18 months found that 21% of the patients experienced symptoms of PCS 2 to 6 months after their initial injury, and that 20% of these patients had the "post-MTBI syndrome."[126] In another survey of 51 patients, where 23 responded, 25% of the respondents reported continued sequelae from their injuries. The patients with sequelae after one year were found to have reported more symptoms one week after injury.[127]

Cicerone[128] indicated that there was considerable evidence to show that PCS symptoms persisted in a significant proportion of patients after MTBI, and such symptoms were particularly prevalent in patients who indicated that they needed clinical attention.

Symptoms with organic etiologies, it has been noted, can mimic functional disorders.[129] Alves[130] indicated that as recovery occurred, persistent symptoms could be secondary to an interaction between organic and psychosocial factors. These persistent symptoms are more than would be expected from the initial organic damage alone. Alves further stated that a significant percentage of patients would exhibit persistent problems with symptoms 12 months post injury. He felt 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.

Mateer[131] found that patients post MTBI were more acutely aware of their cognitive deficits and difficulties with functional abilities. These patients would go to a physician and would have a negative neurological examination. They would be told that there is no organic reason for their problems, that they should wait longer for recovery, learn to live with their problems, or seek psychiatric help.

These iatrogenically-induced problems (cause and effect) most likely lengthen the patients' symptomatic period as they begin to feel an ever increasing loss of control, fear of the unknown, and concern that they must be "going crazy."

BOTTOM LINE

It doesn't matter what the medical problem is, particularly when, like most patients with MTBI, they look "normal." Physicians with little or no background in the diagnosis of MTBI or PCS, or bought and paid for consultants, do a great disservice

to MTBI patients. Constant repetition by physicians of the mantra, "There is nothing wrong with you. You look fine. There's no problem here," will demonstrably disrupt a patient's sense of self, their life, and their feelings that there are indeed people (specifically doctors and insurance companies) out to get them. This induces iatrogenic exacerbation of their symptoms as they strive, consciously or unconsciously, to prove to *someone* that they do have a problem. Then, to add insult to injury, this iatrogenically induced problem is used against them both by other physicians and the legal "warriors" who are bound and determined to prove that there is nothing wrong with them, thus saving their insurance company client's money.

PERSISTENT POSTCONCUSSIVE SYNDROME

Alexander[108,132] has written extensively about the "persistent post concussive syndrome" (PPCS). These patients, after one year, continue to have symptoms commonly seen in acute PCS, such as headache, sleep disorder, balance problems, dizziness, sensory hyperesthesias, and cognitive symptoms including deficits in attention, memory, and executive functioning. They are also frequently noted to have prominent emotional symptoms including irritability, depression, nervousness, discouragement, and anger.

Alexander[108] identifies some "predictors" of the development of PPCS, including the female sex, litigation, low socioeconomic status, prior MTBI, headache, and serious associated systemic injury. While these factors may be implicated, he states that none accounts for more than a small percentage of cases of PPCS.

Other authors identify pain severity post injury as a predictor of the development of the PPCS post MTBI.[133,134] Additional data suggests a greater frequency of anxiety and depression months after initial injury.[135]

Dizziness is a frequent symptom of the PCS. 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 problems are often ignored. Zasler[136] discusses cervicogenic dizziness. Dizziness secondary to myofascial trigger points in the sternocleidomastoid muscles, which is also frequently overlooked. In contradistinction, Alexander[108] does not appear to anticipate the psychological aspects secondary to this problem, making it seem more of a primary psychological problem than being secondary to a true organic problem.

Chronic pain and headache are fairly universal accompaniments of the PPCS. It is also known that patients who experience chronic headache not associated with a PCS have many of the same complaints, including fatigue, sleep disorder, depression, and occasionally, dizziness, as well as difficulties with concentration and memory. Psychological factors may aggravate these headaches.

It is also recognized that anxiety may decrease concentration and complex mental processes.[68,137] Depression can cause decreased cognitive functioning, particularly in concentration, memory, and executive functions.[128,138,139] The latter problem has also been called "depressive pseudodementia."[140]

Therefore, one cannot consider that if everyone with a PCS/MTBI has impaired concentration, then everyone with impaired concentration after PCS/MTBI has a

neurological etiology. The problem is that patients with PCS/MTBI associated with pain and affective difficulties may have impaired concentration for multiple reasons, including post MTBI neuropathological changes.

Alexander[108] asks the question, "When does the physiogenesis of a clinical problem become psychogenesis?" This may be difficult to determine and may have an iatrogenic component. Alexander does indicate that while the major issue is physiogenesis transforming to psychogenesis, physiogenesis can be very underestimated. He also indicates that there is no single psychological factor, physiological factor, or demographic factor leading to the PPCS.

STILL MORE

Fenton[141] attempted to reappraise the PCS. He reviewed data from two UK prospective studies of the initial aspects and course of postconcussive symptomatology using parallel psychosocial, neuropsychiatric, quantitative EEG (electroencephalogram, or QEEG), and brainstem-evoked potentials. Abnormal, prolonged brainstem-evoked potentials were seen in between 27% and 46% of patients. Prolonged symptomatology was noted in 13% of patients and was associated with a high percentage of brainstem dysfunction. The degree of QEEG recovery related to the intensity of early symptom reaction to trauma. Fenton felt that levels of perceived stress at the time of the injury or afterwards were not related to symptom formation, but chronic social difficulties were seen in 21% of patients who initially showed improvement but later, between 6 weeks and 6 months post trauma, experienced an exacerbation of symptomatology.

Taylor et al.[142] compared 15 whiplash patients to 10 patients with moderate to severe brain injury, and 24 chronic pain patients. They were assessed 4 years after initial injury via neuropsychiatric testing. It was concluded that the theory of neuronal degeneration in the etiology of whiplash-related cognitive complaints was not supported, nor was the specificity of neuropsychological tests in detecting the subtle effects of brain trauma.

Not to be outdone, Greiffenstein et al.[143] compared the motor skills "which are sensitive to central lesions, but…. also affected by peripheral injury and motivation" in a group of "proven brain injury" patients versus "healthy postconcussion patients." They concluded, "Motor skill deficiencies in postconcussion syndrome (PCS) are probably functional in nature."

I don't get it, either.

BOTTOM LINE

I think Bob Dylan said something like, "I don't know what it is, but there's something out there, Mr. Jones."

The PCS as well as the PPCS are not symptoms or syndromes looking for patients. As I indicated above, I believe that the PCS is different from an MTBI. Still, patients from around the country, around the globe, complain of the same symptoms after an acceleration/deceleration injury. There are tests and many studies

that show the presence of abnormalities. Again, we don't yet seem to know the best tests, the best window to perform them, or the best way to interpret them.

As clinicians, we also know that we have to listen to our patients. If something they say doesn't make sense, make like Sherlock Holmes (who was modeled after Dr. Bell, a neurologist) and investigate, actively, what the patient is telling you. It's your job.

To be antagonistic to a diagnosis, to not accept the presence of a diagnosis because of preconceived notions or thoughts of patient malingering — right off the bat, or because your opinion depends on who pays you — puts us back into the era of the Inquisition. That's not our job.

4 Pathophysiology

The primary mechanisms of traumatic brain injury (TBI) may include focal injury, as well as diffuse axonal shearing and neurochemical damage. Before we deal with these issues, let's take a brief tour of the normal physiology.

NORMAL COGNITIVE FUNCTIONING: THE VERY BASICS

The frontal and temporal lobes deal with complex, high-level behaviors including complex thought, memory, and language. The parietal and occipital lobes subserve sensation, vision, and perceptual information processing. All aspects of cerebral function are influenced by the others.

Normal frontal lobe activity includes executive functioning, or "Master Control." This includes integration of information from all other parts of the brain, including problem solving, planning, and emotional control.

After injury, abnormal frontal lobe activity includes: behavioral problems, inappropriate behaviors, poor problem solving, memory deficits, difficulties with routine activities, and poor insight into the presence of existing deficits.

The temporal lobes include the hippocampus regions, which are immensely important to memory. They also deal with speaking and understanding language, and correlation of sensory input including smell, taste, and hearing.

After injury to the temporal lobes, patients will develop memory problems, affective problems, and sensory and language problems. Also associated with temporal lobe injury is a higher incidence of seizure disorder.

The parietal lobes normally work to process various types of sensory information, including touch and position sense. After injury, patients will demonstrate problems with reading and writing, as well as spatial disorientation.

The occipital lobes normally enable effective processing and interpretation of visual information. When injured, they can induce difficulties in perception and interpretation of objects, words, and people.

When injured, the cerebellum can induce difficulties with coordinated movement of the extremities and the trunk, as well as difficulties with several other forms of information processing.

PRIMARY NEUROPATHOLOGY

Focal lesions are large enough to be visualized by the naked eye. They include cortical contusions, subdural hematomas, epidural hematomas, and intracerebral

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