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THE POSTCONCUSSION SYNDROME AND THE SEQUELAE OF MILD HEAD INJURY

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Although neurologists frequently evaluate and treat patients with mild head injury, the sequelae continue to be controversial. The following two cases illustrate some elements of this conflict, including the credibility of the patient with subjective complaints and the effect of secondary gain and litigation on persistent symptoms.

Case 1. Neurosurgeon as Victim

This 42-year-old, moderately coordinated, neurologic surgeon was in Vail, Colorado, in 1984, for a meeting of the National Traumatic Coma Data Bank. Following the morning meeting, he spent the afternoon touring the mountains of Vail, and while descending one that had only a modest incline, he lost his balance and fell, striking his head. He was rendered immediately unconscious for a period of a few seconds, certainly no more than 10 to 15 seconds. On awakening, the world appeared upside down, with the sky below and terra firma above. This condition cleared, but a very modest vertigo persisted. This did not interfere in any way with descent from the mountain and in fact did not interfere with further skiing activities. Neuropsychologic testing was soon recommended by the neuropsychologists at the meeting but was respectfully declined.

On returning home, the neurosurgeon noted that he was a bit more distractable than was his norm and that he had a great deal of difficulty remembering recent events, including particularly the location of objects necessary for work, such as a dictaphone, briefcase, and keys. List making to

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recall meetings scheduled and tasks to be performed became necessary; such lists were not necessary before. Referencing articles from memory storage was difficult; authors were frequently transposed and dates incorrectly recalled. Information processing did not appear to be affected, but the ability to attend to a task required a higher level of energy expenditure than previously. These symptoms persisted, but they improved gradually over a period of approximately 18 months, and by the fall of 1985, they appeared to have reached their asymptote. Modest improvement in information storage retrieval has continued, indicating that neither Alzheimer's nor a presenile dementia was revealed by the head injury. Function as judged by others remains good but is not optimal.[106]

Case 2. Unforeseen Misfortune

Penny Pellito, age 52, was a customer in a Home Depot store in Fort Lauderdale, Florida, in April, 1987. Vertically stacked lumber fell, striking her on the top of her head (Keller L: Personal communication, 1991). She had a small scalp laceration but there was no loss of consciousness. She sued Home Depot, Inc., for at least $100,000 for alleged brain damage and an unspecified amount for paranormal injury. She claimed that the mild head injury robbed her of a supernatural power: her ability to go on "automatic" to undergo pain-free surgery without anesthesia.

On February 8, 1991, the Broward Circuit Court jury of three men and three women awarded Penny Pellito $5000 for physical injuries but found that she was 80% negligent. They also awarded her husband, James, $1000 for loss of her services.

"The jurors are 'in the majority of the way people feel,' " Pellito said after the verdict. "Welcome to the real world. They don't look beyond, to what can be." Her husband was less sanguine. "They are still in the caveman stage," he said. Home Depot's attorney, James Zloch, was elated with the verdict.

Pellito also says she is a psychic, but had she foreseen the outcome of her lawsuit, she may not have proceeded to trial. She and her husband rejected a pretrial settlement offer of $17,000 from Home Depot, insisting on more than $1 million, Zloch said.

Judge Paul M. Marko III agonized for 2 days before letting the jury even consider awarding Pellito money for paranormal damages. "There's no legal *precedent for either allowing it or denying it," he said after the verdict.*[80]

DEFINITIONS OF POSTCONCUSSION SYNDROME AND MILD HEAD INJURY

The postconcussion syndrome follows usually mild head injury and comprises one or more of the following symptoms and signs: headaches, dizziness, vertigo, tinnitus, hearing loss, blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, diminished taste and smell, irritability, anxiety, depression, personality change, fatigue, sleep disturbance, decreased libido, decreased appetite, memory dysfunction, impaired concentration and attention, slowing of reaction time, and slowing of information processing speed. Rare sequelae of mild head injury include subdural and epidural hematomas, seizures, transient global amnesia, tremor, and dystonia (Table 1). The most common complaints are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity.[38, 41, 42, 117, 137] Loss of consciousness does not have to occur for the postconcussion

Table 1. SEQUELAE OF MILD HEAD INJURY

Headaches
Muscle contraction type
Migraine
Cluster
Occipital neuralgia
Supraorbital and infraorbital neuralgia
Secondary to neck injury
Secondary to temporomandibular joint syndrome
Owing to scalp laceration or local trauma
Mixed
Cranial nerve symptoms and signs
Dizziness
Vertigo
Tinnitus
Hearing loss
Blurred vision
Diplopia
Convergence insufficiency
Light and noise sensitivity
Diminished taste and smell
Psychologic and somatic complaints
Irritability
Anxiety
Depression
Personality change
Fatigue
Sleep disturbance
Decreased libido
Decreased appetite
Cognitive impairment
Memory dysfunction
Impaired concentration and attention
Slowing of reaction time
Slowing of information processing speed
Rare sequelae
Subdural and epidural hematomas
Seizures
Transient global amnesia
Tremor
Dystonia

From Evans RW: Post-concussion syndrome. *In* Evans RW, Baskin DS, Yatsu FM (eds): Prognosis of Neurological Disorders. New York, Oxford University Press, 1992; with permission.

syndrome to develop. Perhaps 50% of patients with mild head injury will develop the postconcussion syndrome.[105]

The terms *mild* and *minor head injury* are frequently used. The term *mild head injury*, however, is preferred to delineate the continuum of mild, moderate, and severe.[73] In addition, *minor* can denote an injury of little consequence, which can be misleading. Varying criteria to define mild or minor head injury have been used, including varying durations of loss of consciousness and posttraumatic amnesia. In more recent years, the following criteria have been used: an initial Glasgow Coma Scale score of 13 to 15, inclusion in some studies of patients with intoxication and a history of prior alcohol or drug abuse, prior head injury, or prior psychiatric history; and at times the inclusion of patients with skull fractures or cerebral contusions. Strict criteria used in recent

studies[38, 98] are essential to ensure studying similar types of injuries without confounding variables.[175] For future studies, reasonable criteria for the definition of mild closed head injury would include a duration of loss of consciousness of 30 minutes or less or being dazed without loss of consciousness; an initial Glasgow Coma Scale score of 13 to 15 without subsequent deterioration; and absence of focal neurologic deficits without evidence of depressed skull fractures, intracranial hematomas, or other neurosurgical pathology.

EPIDEMIOLOGY

Head trauma of all degrees is one of the most important public health problems. Mild head injury accounts for 75% or more of all brain injuries.[88] The annual incidence of mild head injury per 100,000 population has been estimated to be 131 for San Diego County, California,[87] 149 for Olmsted County, Minnesota,[5] and 511 for Auckland, New Zealand.[176] For an industrialized country such as the United States, estimates of the relative causes of head trauma are as follows: motor vehicle accidents, 45%; falls, 30%; occupational accidents, 10%; recreational accidents, 10%; and assaults, 5%.[77] By comparison, in a 1983 survey in the People's Republic of China, traffic-related accidents accounted for 31.7% of head trauma, but most of these were bicycle accidents.[172] In the elderly, falls are more likely the cause, and motor vehicle accidents are more common in the young.[135] Males are more commonly injured with a 2:1 ratio. About one half of all patients are between the ages of 15 and 34. It has been estimated that 20% to 40% of all patients with mild head injuries in the United States do not seek medical care.[51]

HISTORICAL ASPECTS

The postconcussion syndrome has been recognized for at least the last few hundred years.[152, 163] One interesting historical case involved a 26-year-old maidservant who had been hit over the head with a stick and complained of retrograde amnesia. Six months later, she was still complaining of headaches, dizziness, tinnitus, and tiredness. A judge requested the opinion of Swiss physician J. J. Wepfer and two other surgeons, who stated: "We can't say anything definite, but it is certain that there has been a grave contusion of the head and that this will leave its mark in the form of an impediment." Although similar prognostic opinions are still given, this particular statement was made in 1694.[33] Boyer in 1822, Astley Cooper in 1827, and Duputren in 1839 all described the clinical picture of cerebral concussion with persistent symptoms.

In 1879, Rigler[129] raised the important issue of compensation neurosis when he described the increased incidence of posttraumatic invalidism after a system for financial compensation was established for accidental injuries on the Prussian railways in 1871. Countering this view, Erichsen[43] was a London surgeon who believed that minor injuries to the head and spine could result in severe disability owing to "molecular disarrangement" or anemia of the spinal cord. His 1882 book[43] was the medicolegal authority of the time and was frequently sighted in court cases.

In 1888, Strumpell discussed how the desire for compensation could lead to exaggeration. In 1889, Oppenheim popularized the concept of traumatic neurosis, in which a strong afferent stimulus resulted in impairment of function of the central nervous system. Charcot countered Oppenheim's work and suggested that the impairment described was actually due to hysteria and neurasthenia.

The Boston Medical and Surgical Journal[21] published several articles covering these different points of view. The leading article drew the following conclusions:

> In this iconoclastic age when we are not allowed to believe in a personal devil, or good honest ghosts, or even to coddle our own pet superstitions and hobbies without a suspicion of mental degeneration, it is natural that the medical "bugaboo" raised by Mr. Erichsen some years ago, and christened spinal concussion, should meet with little quarter at the hands of the modern scientific observer. It is possible, however, that in this, as in other things, the skeptic may have gone too far, and that although it was no ghost that has alarmed us there may actually have been some phosphorescent light which we do not understand, and the nature of which we cannot fully explain. . . . A rose, however, under any other name, will remain as fragrant to the sufferer, and whether the ailment be termed railway spine or traumatic neurasthenia, the condition is equally distressing.

Another landmark work was the 1934 paper by Strauss and Savitsky.[152] They argued that concussion can occur without loss of consciousness and cited examples of significant intracranial trauma such as subdural hematomas, which can be caused by injuries not resulting in loss of consciousness. Strauss and Savitsky discussed the interrelationships of the head injury, premorbid personality, and the stress resulting from dealing with the aftermaths of the injury:

> There can be no denying that the present mode of handling these unfortunate persons in compensation bureaus multiplies the psychic stresses and strains and complicates an already almost intolerable situation of life. The harshness, injustice and brutal disregard of complaints shown by the physicians and representatives of the insurance companies and their ready assumption of intent to swindle do not foster wholesome patterns of reaction in injured persons. The frequent expression of unjustifiable skepticism on the part of examiners engenders resentment, discouragement and hopelessness and too often forces these people to resort to more primitive modes of response (hysterical). The repeated psychic traumas bring out the worst that there is in them and makes manifest all their frailties and constitutional insufficiencies. This is especially true in view of the fact that the blow itself is known to give rise to defects in personality integration. In addition, the premorbid make-up of the injured persons varies considerably and undoubtedly contributes much to the manner in which they handle their problems. The trauma, moreover, lowers resistances and thresholds and brings prominently into consciousness repressed conflicts and difficulties.

They concluded:

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

In contemporary courtrooms, defense attorneys are still quoting from the writings of Miller.[115] In 1961, Miller summarized the viewpoint of those who believe that the postconcussion syndrome is really a compensation neurosis: "The most consistent clinical feature is the subject's unshakable conviction of unfitness for work. . . ." Symonds[157] took an equally strong opposing position in 1962 when he wrote, "It is questionable whether the effects of concussion, however slight, are ever completely reversible."

Hollywood Head Injury Myth

Although extensive data of the last 2 decades strongly support an organic basis for the postconcussion syndrome, much doubt still exists among some physicians[81] as well as lay persons,[7] defense attorneys, and agents of insurance companies. In a survey of neurosurgeons in the United States, 45% gave an opinion that emotional and compensation factors were the chief mechanism underlying the postconcussion symptoms.[112] One explanation is that for many people, their knowledge of the sequelae of head injuries is entirely the product of "movie magic." This Hollywood head injury myth, which Robertson[132] has called the "Three Stooges Model," has been an extreme source of misinformation.

The reader can easily reference any of the different movie and television genres and bring to mind examples. In western movie barroom brawls, the cowboy may be punched in the face and hit repeatedly over the head with chairs without much effect before one of the showgirls comes up from behind, striking the cowboy over the head with a liquor bottle. The cowboy collapses unconscious, only to be fully recovered in the next scene. In detective, boxing, kung fu, and other action stories, kicks, punches, and blows (which in reality would be fatal or near-fatal) delivered to the face and head in rapid repetition are brushed off by the combatants after eliciting only a grimace and a grunt. The infliction of head trauma is one of the funniest routines in cartoons and slapstick movies of any vintage. Our actual experience is miniscule compared with the thousands of simulated head injuries the average person witnesses in the movies and on television. Therefore the neurologist has a difficult job of educating a public reared on this type of mythology.

To help make the opposite point, the neurologist can conjure up the image of the havoc wreaked by the powerful fists of professional boxers, even with the ample padding of 8 or 10 ounce gloves. Most people are familiar with the meaning of the abbreviations TKO and KO. The punch drunk syndrome of cumulative head trauma in boxers[131] (well-described by Martland in 1928[107]) and the examples of two of the most successful boxers, Joe Louis and Muhammad Ali, are also quite familiar to most. Starting with this example from sports, the public may then be more receptive to the presentation of general factual information.

Gene Tunney's Concussion

Gene Tunney[164] described the effects of a concussion sustained in 1927 while training for the second fight with Jack Dempsey.

> I established my training headquarters at Speculator, New York, where I remained until September 1. During the early part of this training period I had a very curious experience. One day while boxing with a sparring partner, Frank Muskie, we bumped heads. The part of my skull which is the thinnest, near the temple, struck the toughest part of his, the top. I was terribly dazed. As I straightened up a long, hard right swing landed on my jaw. Without going down or staggering, I lost all consciousness of what I was doing, and instinctively proceeded to knock Muskie out.
>
> Another sparring partner, Eddie Eagan, entered the ring; we boxed three rounds. I have no recollection of this, nor have I any recollection of anything that occurred until the next morning when I awakened in my little cabin by the water's edge, wondering who I was and what I was doing there.
>
> As I lay in this awful state of returning consciousness I became greatly frightened. Gradually my name came to me. That I was a pugilist soon followed, then the thought of being champion—impossible—unbelievable. I must have had a long dream. Gradually came the realization that I had not been dreaming. I rose and asked guarded questions. I wanted to know all about the events of the day before. For three days I could not recall the names of my most intimate acquaintances. I had to stop training. I did not leave my cabin, except to eat or take a short walk. On these occasions all seemed queer. I was unable to orient myself. The sensation I had was as though hot water had been poured through a hole in my skull and flowed down over the brain to my eyes, leaving a hot film. There were three newspaper men at camp reporting my activities. They had to be deceived. This story was too sensational to permit it to get out.
>
> I confided my condition to no one but Eddie Egan. He was keeping a diary. It is amusing now to read his notes of those few goofy days.
>
> After returning to normal, I decided that any sport in which such accidents could occur was dangerous. I realized I had had a concussion. The first seed of retirement was sown then. The possibility of becoming "punch-drunk" haunted me for weeks.

Tunney wisely retired as world heavyweight champion in 1928 after successfully defending his title twice.

NEUROPATHOLOGY

In 1835, Gama[52] wrote, "Fibers as delicate as those of which the organ of mind is composed are liable to break as a result of violence to the head." For 120 years following, neuropathologists were more concerned with describing more obvious focal contusions owing to coup and contrecoup injuries and hematomas.

In 1956 and with more cases in 1961, Strich[153, 154] observed diffuse axonal injury resulting from sheer and tensile strain damage. Microscopically these injuries have the appearance of retraction balls, which were first described and named by Cajal.[25] Strich[154] explained that "when a nerve-fibre in the peripheral or central nervous system is cut, axoplasm flows out of both cut ends and is visible as a large blob."

In 1968, Oppenheimer[122] extended this observation to five cases of mild head injury in which the patients died from fat embolism or pneumonia. He described one such case:

> A man of 66 was admitted to hospital with multiple rib fractures, having been knocked down by a motor scooter. He had been stunned, and had a retrograde amnesia of 10–15 minutes and a post-traumatic amnesia of about 20 minutes. There was a parietal bruise, but no skull fracture, and no neurological signs. He was a bronchitic, and died of chest complications 13 days after the injury. At necropsy there was bronchopneumonia with small abscesses. The brain looked entirely normal except for a tiny softening in the lateral sulcus on one side of the midbrain. There was no vascular disease, and no sign of brain swelling. Histologically, there was some myelin destruction and numerous axonal retraction bulbs in the midbrain lesion. Nine blocks, from various parts of the brain, were stained for microglia. In every block, at least one microglial cluster was found.

The observation of diffuse axonal injury in cases of mild head injury has been further confirmed.[1, 18, 95, 125] A neurochemical substrate for mild head injury with release of the putative excitatory neurotransmitters acetylcholine, glutamate, and aspartate has been suggested.[67] Axonal rupture can occur at the time of injury. A gradient of axonal damage can occur, however, ranging from disruption of axoplasmic transport to delayed rupture owing to release of putative neurotransmitters and other mechanisms.[125] Animal studies, including studies of monkeys and cats subjected to mild head injury, have demonstrated similar diffuse axonal injury.[74, 125] Finally, abnormalities in cerebral hemodynam-

ics have been demonstrated in animals and humans after mild head injury.[39, 126, 158, 161]

HEADACHE TYPES

Headaches have been estimated to occur in about 30% to 90% of patients who are symptomatic after mild head injury (Table 2). Paradoxically headaches may occur more often with longer duration in patients with mild head injury as compared with more severe degrees of trauma.[174] The different types of posttraumatic headaches are discussed: muscle contraction, occipital neuralgia, secondary to neck and temporomandibular joint injury, migraine, cluster, supraorbital and infraorbital neuralgia, and owing to scalp lacerations or local trauma. Muscle contraction headaches often associated with occipital neuralgia account for perhaps 85% of all posttraumatic headaches.[105] The headaches may be daily or episodic. Many patients have more than one distinct type of headache. Not infrequently, patients have headaches with both migraine and muscle contraction features.

Muscle Contraction Type and Greater Occipital Neuralgia

Muscle contraction type headaches occur in a variety of distributions, including generalized, nuchal-occipital, bifrontal, *cap-like*, or in a headband distribution. The headaches are typically described as a pressure or aching, although there may be a minor throbbing component, and may be constant or intermittent with variable duration. Because head injuries are commonly associated with neck trauma, the headaches may be perpetuated by neck pathology such as myofascial injury,[57] intervertebral disk disease, or exacerbation of preexisting spondylosis. Temporomandibular joint injury can be caused either by direct trauma or the jarring effect associated with the head trauma. Patients may complain of associated hemicranial or ipsilateral frontotemporal muscle contraction type headaches.

Occipital neuralgia may occur from direct trauma to the nerve with a blow to the back of the head or more commonly is associated with muscle spasm of the superior trapezius and semispinalis capitis muscles. Patients describe an aching, shooting, or burning type pain, which may radiate over the ipsilateral occipital scalp and sometimes may be referred to the temporoparietal, frontal, or retro-orbital area. The pain may be a dull aching lasting hours or days at a time or less commonly brief paroxysms of an electrical or lightning-like sensation lasting seconds to minutes. Occipital neuralgia can also occur bilaterally. Although occurring with great frequency, this diagnosis is often overlooked by many physicians. Headaches can also be referred from trigger points in other neck muscles, including semispinalis cervicis, digastric, and the sternocleidomastoid.[161]

Migraine

Migraine headaches are typically throbbing and may be worse with movement or coughing. The headaches are frequently associated with nausea, vomiting, light and noise sensitivity, and dizziness. In a minority of cases, migraines occur with an aura that is usually visual, although other deficits can

Table 2. PERCENTAGE OF PATIENTS WITH PERSISTENCE OF SYMPTOMS AFTER MILD HEAD INJURY

	1 Week	1 Month	6 Weeks	2 Months	3 Months	6 Months	1 Year	2 Years	3 Years	4 Years	5 Years
Headache	71[88*]	90[35]	24.8[133]	31.5[36]	78[130]	21.6[117]	35[35]	22[35]	20[35]	24[42]	
	36[27]	31.3[117]			47[88]	27[27]	8.4[138]	24[27]			
		56[98]					18[27]				12[150] (headaches and/or dizziness)
Dizziness	53[68]	12[35]	14.5[138]	23[36]	22[68]	13.1[117]	26[35]	18[35]	16[35]	18[42]	
	19[27]	21.9[117]				22[27]	4.6[138]	18[27]			
		35[98]					14[27]				
Memory problems		18.8[117]	8.3[138]		59[130]	15.3[117]	3.8[138]			19[42]	
Irritability		24.7[117]	9[138]			19.6[117]	5.3[138]				

*Superscript numbers refer to references.

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certainly be associated. The headache can occur in just about any distribution of the head or face, unilateral, bilateral, or generalized.

Mild head injury can cause acute migraine episodes. Matthews[110] called this "footballer's migraine" to describe young men playing soccer who had multiple classic migraine attacks triggered only by mild head injury. Similar headaches have been triggered by mild head injury in boxing,[110] American football,[12] and rugby[6] often in athletes with a positive family history of migraine. Following mild head injury, children, adolescents, and young adults can develop a variety of transient neurologic sequelae not always associated with headache perhaps owing to cerebral vasospasm.[66] Four clinical types have been described, which can cause hemiparesis; somnolence, irritability, and vomiting; transient blindness (often precipitated by occipital impacts); and brain stem signs.[64]

Recurring attacks of classic and common migraine can develop after mild head injury with or without loss of consciousness as well as after whiplash type neck injuries in patients from their teens to old age often with a positive family history of migraine.[11, 173] The time interval from the injury to the first onset of migraine can vary from hours to weeks. The migraines can occur as often as almost daily.[173]

Orgasmic Cephalgia

To my knowledge, the new onset of orgasmic cephalgia following mild head injury has not been described.[2] I have treated two such patients. The case history of one follows.

This 25-year-old man was at work on 4/18/91 when an 8-inch pipe, some 9 feet long, fell approximately 8 floors, striking him across his asbestos mask, chin, and right forearm. He was dazed without loss of consciousness. A laceration of the chin was sutured. He sustained multiple fractures of the right forearm and hand requiring four subsequent operations.

Following the injury, the patient had complaints of headaches on a daily basis described as a nuchal occipital and bifrontal aching, pressure, or throbbing, without associated symptoms. He also had complaints of mild memory problems, difficulty sleeping, blurred vision, and daily posterior neck pain. Starting 3 to 4 weeks after the injury, he developed bifrontal throbbing headaches that started with orgasm and lasted 2 to 3 hours. The headaches were associated with photophobia but not with nausea, vomiting, scotoma, or sensorimotor complaints. This type of headache occurred after about 90% of orgasms and did not occur at other times. His family history was negative for migraine, and he had no history of migraine.

Four months after the injury, he was placed on nadolol, 40 mg daily, and amitriptyline, 25 mg orally at bedtime. Over the next 5 months, he had multiple orgasms but only one additional headache triggered by orgasm.

Cluster Headaches

Rarely cluster headaches can develop after mild head injury. Reik[127] described four adults who developed cluster headaches within weeks of the injury. The headaches were recurrent in all four patients, with one having episodes over 46 years. Mathew and Rueveni[109] reported additional cases. Mathew has now seen a total of 13 patients who are more resistant to treatment than idiopathic cluster patients (Mathew N: Personal communication, 1991).

Other Headaches

Supraorbital neuralgia can result when there is injury to the supraorbital branch of the first trigeminal division as it passes through the supraorbital foramen just inferior to the medial eyebrow. Similarly infraorbital neuralgia can result from trauma of the inferior orbit. The patients have diminished sensation and sometimes loss of sweating in the distribution of the nerve and may complain of a shooting, tingling, aching, or burning pain in the appropriate distribution. A dull aching or throbbing pain may also occur around the area of injury.[169]

Vijayan[168] described a rare type of posttraumatic headache, dysautonomic cephalgia, following injury to the anterior triangle of the neck or carotid sheath. Acute local pain and tenderness in the anterior triangle of the neck can be followed weeks or months later by severe unilateral frontotemporal headache, ipsilateral increased sweating of the face, dilatation of the ipsilateral pupil, blurred vision, ipsilateral photophobia, and nausea. The frequency of the headaches can occur a few times per month lasting hours to days.

Dysesthesias over scalp lacerations frequently occur. In the presence or absence of a laceration, an aching, soreness, tingling, or shooting pain over the site of the original trauma can develop. The symptoms may persist for weeks or months but rarely for more than 1 year.

CRANIAL NERVE SYMPTOMS AND SIGNS

Dizziness and Hearing Loss

Dizziness is a common complaint following mild head injury occurring in 53% of patients within 1 week of the injury[98] and persisting in 18% after 2 years.[27] Various types of peripheral and central pathology can cause the dizziness. Mild head injury without a temporal bone fracture can result in a labyrinthine concussion with vertigo, hearing loss, and tinnitus. Spontaneous or positional nystagmus and occasionally hypoactive calorics can be recorded with electronystagmography (ENG). Peripheral vestibular disturbances with canal paresis have been described in 17.1% of patients after mild head injury.[165] Positional nystagmus has been noted in 30% of patients with dizziness on discharge from the hospital after mild head injury.[27]

Mild head injury can also cause benign positional vertigo owing to dislodged otoconia from the utricular macule settling onto the cupula of the posterior semicircular canal.[8] The resulting dizziness can recur over a period of 1 to 10 years.[8] Occasionally mild head injury without a skull fracture can result in a perilymph fistula, with the sudden onset of vertigo or hearing loss or both. Prolonged I-V latencies on auditory brain stem evoked potential recordings are consistent with brain stem injury as the cause of dizziness in some cases.[119] Postural imbalance caused by disturbances of postural tonic activity can be demonstrated by abnormal statokinesimetry studies.[9]

Conductive type hearing loss can occur after mild head injury owing to blood in the middle ear or disruption of the ossicular chain. Occasionally sensorineural hearing loss can occur owing to an unsuspected temporal bone

fracture with normal skull radiographs.[23] Routine otoscopic examination is essential to detect hemotympanum. Bilateral sensorineural hearing loss without a fracture can also occur.[165]

Visual Symptoms

Blurred vision following mild head injury is reported by 14% of patients.[117] Convergence insufficiency is the most common cause. Although the anatomic localization is not known, lesions of the occipital lobe and upper midbrain are possibilities.[89] The diagnosis can be made by measurements of convergence fusional reserves. The near point of convergence alone is an unreliable measure.[89] Diplopia owing to III, IV, and VI cranial nerve palsies can be caused by mild head injury. Trauma is the most common cause of IV nerve palsy and may follow apparently trivial head trauma.[90] Optic nerve contusions can result in diminished visual acuity and hue discrimination.

Other Symptoms

Head trauma is the most common cause of anosmia.[34, 68] Decreased smell and taste are reported by more than 5% of patients after mild head injury.[117] Damage to the olfactory filaments can be caused by mild head trauma. Light and noise sensitivity were reported in 7.2% and 15% of patients 14 days after mild head injury.[60] This sensitivity has been documented in two studies comparing patients after mild head injury with controls.[19a, 171]

PSYCHOLOGIC AND SOMATIC COMPLAINTS

Nonspecific psychologic symptoms are common after mild head injury and include personality change, irritability, anxiety, and depression.[147] Within 3 months of injury, up to 51% to 84% of patients have posttraumatic symptoms.[130, 137] The symptoms can be quite persistent, with an incidence estimated at 15% to 33% at 1 year[35, 138] and 15% after 3 years.[35] Lishman[102] has suggested interpreting these complaints in the context of pretraumatic, peritraumatic, and posttraumatic factors (Table 3). There is an increased risk of developing depression in patients after mild head injury as compared with controls.[141] Posttraumatic stress disorder may also occur following mild closed head injury and has some symptoms similar to the postconcussion syndrome (see the article by Merskey in this issue).

Fatigue is a common complaint reported by 29% of patients at 4 weeks after the trauma and by 23% at 6 months.[117] Possible explanations for the fatigue include sleep deprivation; frustration with persisting symptoms such as headaches, dizziness, and blurred vision; increased effort necessary to compensate for cognitive deficits;[59] and stress brought on by extrainjury factors, such as impaired school and work performance, financial and family problems, interaction with health care providers, and pending litigation. These factors can also contribute to the decreased appetite and libido reported by some patients.

A disruption of sleep patterns after mild head injury has been described.[123] Difficulty with falling asleep and arousals are the most common problems and are reported by 15% of patients 6 weeks postinjury.[137] Less often, increased sleep duration and daytime naps are reported. There has been a suggestion

Table 3. FACTORS RELEVANT TO PSYCHIATRIC DISABILITY

Pretraumatic
Age
Cerebral arteriosclerosis
Alcoholism
Mental constitution
Genetic vulnerability
Previous psychiatric illness
Personality (including being prone to accidents)
Preexisting psychosocial difficulties
Domestic
Financial
Occupational
Recent life events
Peritraumatic
Brain damage
Transient (contusion, edema, hypoxia, raised intracranial pressure, circulation)
Permanent (amount, location)
Other physical damage (skull, scalp, vestibular apparatus)
Emotional impact and meaning
Fear of accident
Fear of early symptoms
Circumstances of accident
Setting
Significance
Type (road traffic accident, industrial, domestic, sport)
Iatrogenic (early information, management, investigations)
Posttraumatic
Intellectual impairment
Other impairments (physical disabilities, deformity, scars)
Epilepsy
Emotional repercussions of accident (including depression)
Ensuing psychosocial difficulties
Domestic
Financial
Occupational
Compensation and litigation

From Lishman WA: Physiogenesis and psychogenesis in the "post-concussional syndrome." Br J Psych 153:460–469, 1988; with permission.

that chronic excessive daytime drowsiness can be triggered by mild head injury, but the data are inconclusive.[62]

Rarely mild, moderate, and severe head trauma can cause mania.[84, 128, 149] Starkstein et al[149] have suggested that ". . . the confluence of either anterior subcortical atrophy and a focal lesion of a limbic or limbic-connected region of the right hemisphere, or genetic loading and a limbic-connected right hemisphere lesion may account for the rare occurrence and specific factors necessary to produce secondary mania."

COGNITIVE IMPAIRMENT

In a study 4 weeks following mild head injury, 19% of patients complained of loss of memory, and 21% complained of difficulty with concentration.[117] Consistent with these complaints, deficits in cognitive functioning have been

documented, including a reduction in information processing speed,[60] attention,[53] reaction time,[119, 155] and memory for new information.[38, 98] Neuropsychologic testing needs to be tailored to detect these specific types of nonlocalizing deficits. Cognitive impairment is further discussed in the article by Levin in this issue and in the prognosis section of this article.

RARE SEQUELAE

Subdural and Epidural Hematomas

Jennett et al[78] estimate that a subdural hematoma occurs in less than 1 in 5000 patients who are seen in the hospital after mild head injury. The incidence of neurosurgical complications after minor head injury has been estimated to be between 1% and 3%.[31] Dacey et al[30] reported on the occurrence of neurosurgical complications after seemingly minor head injury in 610 patients with initial Glasgow Coma Scale scores of 13 to 15. Neurosurgical intervention was required for 18 patients (3%), including removal of subdural hematomas in three, removal of an extradural hematoma in one, and removal of an extradural and subdural hematoma in another patient.

Shah et al[145] reported on the small subgroup of patients with neurosurgical pathology after mild head injury. Thirty adult patients were evaluated in the emergency room with a Glasgow Coma Scale score of 15, a nonfocal neurologic examination, no seizures, and no skull fractures. Computed tomography (CT) scans showed 15 patients with intraparenchymal hemorrhage, 13 subdural hematomas, three epidural hematomas, and seven subarachnoid hemorrhages. At the time of injury, 37% had no loss of consciousness, and superficial cranial trauma was observed in only 63%.

Bollinger[20] first introduced the concept of delayed traumatic intracranial hematoma in 1891. Both subdural and epidural hematomas can later appear after an initial CT scan is normal.[37, 94, 116] After an apparently mild head injury, an epidural hematoma can have a delayed evolution of symptoms and mimic postconcussion syndrome.[13] The clinician needs to be vigilant to recognize these rare complications.[37]

Seizures

Seizure disorders can be a sequela of all degrees of head trauma (see the article by Willmore in this issue). The risk within 5 years of a posttraumatic seizure after mild head injury without a skull fracture in one population study was 0.8%, which is similar to the general baseline incidence.[4] The unusual occurrence, however, of a seizure within a short time period after mild head injury without a depressed skull fracture or contusion certainly raises the possibility of a causal link.

Transient Global Amnesia

Transient global amnesia has been described after mild head injury. Haas and Ross[65] described nine patients, ages 11 to 28, who had episodes of transient global amnesia lasting 2 to 24 hours following mild head injury probably without loss of consciousness. Five of the nine patients had a headache associated with the amnestic episode. During the episode, almost all of patients voiced repetitive queries or comments. A 12-year-old boy had a sec similar episode subsequently triggered by a second mild head injury at the of 14. He had a paternal grandmother with an episode of transient glo amnesia at the age of 73. A 16-year-old subject of the study had a 15-year- brother who had two similar episodes of transient global amnesia after m head injury. Based on the generally young age of the patients, associa headaches in some, and positive family history in others, Haas and R suggested that these episodes were due to posttraumatic confusional migrai Additional patients with similar episodes have been described.[50, 92]

Movement Disorders

Movement disorders have been described as a sequela of mild head injur (see the article by Goetz in this issue). Biary et al[16] described a posttrauma tremor similar to essential tremor, although myoclonic-like jerking was fi quently present. Six of the seven patients had a postural and kinetic trem and the seventh had a resting, postural, and kinetic tremor. The tremor cou involve the head, hands, legs, trunk, and tongue. The onset of the tremor w anywhere from immediately after the head injury to 4 weeks later. All of t patients had normal CT or magnetic resonance imaging (MRI) scans of t brain. Clonazepam reduced the tremor in three patients, and propanol decreased the tremor in another. Primidone was not of benefit. The tremor d not resolve in any of the patients studied. I have treated two similar patient one with nadolol and the other with alprazolam, with modest improvemen Additional similar cases of posttraumatic tremor have also been encountere by Jankovic (Jankovic J: Personal communication, 1991).

Parkinson's disease can follow more severe head trauma or multip episodes of mild head injury.[121] Stern et al,[151] however, have suggested th head trauma should be reassessed as a potential risk factor for Parkinson disease based on their study of various environmental exposures, early li experiences, and head injuries in 149 patients. Goetz and Stebbins[54] reporte that head injury or stress caused by motor vehicle accidents could transientl increase the dysfunction of Parkinson's disease without altering the long-ter prognosis.

Mild head injury associated with a whiplash injury has been reported a causing torticollis.[75, 144] Mild head injury has also been associated with hem dystonia.[86]

TESTING

The judicious use of testing needs to be individualized for each patien (Table 4). Imaging studies available include skull radiographs, CT scan, an MRI studies of the brain. Neurophysiologic studies available include electroe mephalography (EEG) and auditory brain stem responses (ABRs). For som patients with prominent complaints of dizziness, hearing loss, or tinnitus, referral to an otorhinolaryngologist and ENG and audiometric testing may b worthwhile to assess for the presence of peripheral and central pathology, a discussed in the previous section. Patients with persisting blurred vision, diplopia, acuity changes, or field cuts may require referral to an ophthalmologis and formal assessment of visual fields. Finally, neuropsychologic evaluation

Table 4. TESTING

Imaging	
	Skull radiograph
	Computed tomography scan
	Magnetic resonance imaging
Neurophysiologic	
	Electroencephalogram
	Auditory brain stem response*
	Brain mapping*
	Electronystagmography
	Audiogram
Ophthalmologic examination	
	Visual fields
Neuropsychologic testing	

*Clinical utility has not been demonstrated.

can be quite helpful for patients with prominent cognitive complaints or psychologic problems. Neuropsychologic testing is discussed further in the article by Levin in this issue.

Imaging Studies

Skull Radiographs

The indications for skull radiographs after mild head injury continue to be controversial. Because of concerns about overuse, attempts have been made to develop criteria for obtaining this study.[31]

The United States Food and Drug Administration panel analyzed 7035 patients with head injuries.[108] Skull radiographs were not recommended for the low-risk group with the following possible findings: asymptomatic, headache, dizziness, scalp hematoma, scalp laceration, scalp contusion/abrasion, absence of moderate or high-risk criteria. For the moderate-risk group, recommendations included extended close observation, consideration for plain skull radiographs or CT scan, and possible neurosurgical consultation. Skull radiographs may be helpful if abnormal but do not exclude intracranial injury if normal. The possible findings in the moderate-risk group are history of change of consciousness at time of injury or subsequently, history of progressive headache, alcohol or drug intoxication, unreliable or inadequate history of injury, age less than 2 years (unless injury very trivial), posttraumatic seizure, vomiting, posttraumatic amnesia, multiple trauma, serious facial injury, signs of basilar fracture, possible skull penetration or depressed fracture, and suspected physical child abuse. The high-risk group patient is a candidate for neurosurgical consultation and emergency CT scan examination. Possible findings in the high-risk group are depressed level of consciousness not clearly due to alcohol, drugs, or other cause (e.g., metabolic and seizure disorders); focal neurologic signs; decreasing level of consciousness; and penetrating skull injury or palpable depressed fracture. Using these criteria, the chance that an occult intracranial injury would be missed would be no higher than 8.5 in 10,000 cases. The chance of missing an intracranial injury in the low-risk group because of failure to diagnose an occult skull fracture would be about 3 in 10,000.

Gorman,[55] in England, reported on a series of 12,395 patients who presented to the accident and emergency department and recommended the following more liberal criteria for obtaining skull radiographs: all children with a history of or external evidence of injury to the head; any patient with neurologic signs, whether attributed to alcohol or not; those patients with clinical signs of a skull fracture; all patients with lacerated wounds of the scalp, in whom penetration is possible; any patient initially knocked out or amnestic; and patients with significant symptoms following a head injury. Skull radiographs should be considered in patients not included in the above-mentioned categories but who have other injuries warranting admission in their own right or other conditions by themselves dictating admission. Jennett[76] contends that these more liberal criteria for obtaining skull radiographs may be more appropriate in Great Britain than in the United States, where neurosurgeons and CT scanners are much more widely available.

Feuerman et al,[49] in a Southern California study, found no benefit from skull radiographs for evaluating mild head injury. ". . . [W]e emphasize the importance of careful clinical evaluation of these patients. The physician should not allow a management algorithm to replace the exercise of clinical judgement." Rosenborn et al,[133] in a study in Denmark, similarly concluded, "The incidence of intracranial complications in patients without and with skull x-ray with or without fracture does not differ significantly. In these circumstances we do not find any justification for routine skull x-ray after milder head trauma."

Computed Tomography Scan

As suggested in the previous discussion, because neurosurgical complications of mild head injury are uncommon, the yield of CT scans of the brain following mild head trauma is quite small. Feuerman et al[49] concluded:

> A Glasgow Coma Scale score of 13 or 14 places the patient at risk either of having a hematoma requiring surgery or of deteriorating. We recommend that a head CT scan be obtained on all patients with Glasgow Coma Scale score of less than 15, abnormal mental status, or hemispheric neurological deficits. If no operative lesion is found on the CT scan, the patient should be admitted for observation because there is still a risk of deterioration. Those with a Glasgow Coma Scale score of 15, a normal mental status, and no hemispheric neurological deficit may be discharged to be observed at home by a competent observer despite basilar or calvarial skull fracture, loss of consciousness, or cranial nerve deficit.

Dacey et al[30] have proposed additional management schemes.

Magnetic Resonance Imaging

MRI is more sensitive than CT scan in evaluating mild head injury. Levin et al[96] performed a comparative study of CT scan and MRI on 20 consecutive patients admitted with mild to moderate nonmissile head injuries. MRI had no additional influence on surgical management over CT scan. MRI detected parenchymal lesions primarily in the frontal and temporal regions in 85% of patients, which were not detected by CT scan. Follow-up scans at 1 and 3 months showed marked reduction of the size of lesions.

Hesselink et al,[70] in another comparative study of MRI and CT scans, reported that MRI detected 98% of brain contusions compared with only 56% by CT scan. MRI was superior in demonstrating brain contusions during the subacute and chronic stages after head trauma. Yokota et al[179] performed another comparative study of 177 patients with mild to severe head injury. MRI was superior to CT scan in diagnosing acute nonhemorrhagic contusions.

In addition, MRI provided some information to evaluate the severity of diffuse axonal injury and to predict delayed traumatic intracerebral hematoma.

Multiple attempts have been made to develop the most cost-effective method of evaluating patients with mild head injury. Foregoing skull radiographs and liberally obtaining CT scans of the head in the acute situation increase the chance of finding the rare patient with neurosurgical pathology. The cost of treatment might be less if these patients are then discharged with appropriate observation at home rather than admitted to the hospital.[31] MRI is more sensitive than CT scan in all stages of evaluation but entails perhaps 30% extra expense, while rarely affecting outcome.

Neurophysiologic Assessment

Electroencephalography

During the last 50 years, EEG studies have frequently been done to evaluate mild head injury. Dow et al[40] reported an interesting patient series in 1944. They equipped a room of the first aid station at Kaiser's Oregon Ship Building Corporation with an EEG machine. During the study at this site, where 33,000 people were employed, 197 employees underwent EEG studies shortly after sustaining mild head injuries and were compared with 211 coworker controls. Sixty-two percent of the control group and 57% of the head-injured group had normal EEGs. Thirty percent of the control group and 33% of the head-injured group had borderline records with increased θ activity. Eight percent of the control group and 10% of the head-injured group had abnormal EEGs. The records performed within 30 minutes of the injury showed a greater percentage of abnormalities than those taken after 30 minutes. Clinical judgment was more predictive of time lost from work than the EEG results.

As the shipyard study demonstrates, abnormal studies in patients after mild head injury are not specific because of the high frequency of abnormal EEG studies in controls. Lorenzoni[103] further confirmed this poor specificity in an EEG study of 72 patients with head injuries in whom an EEG had been obtained before the injury. The pretraumatic EEG studies were obtained between 2 and 8 years before the accident, with 33 normal and 39 abnormal results. The posttraumatic EEG studies were obtained within 4 weeks of the injury. Fifty-seven percent of the pretraumatic normal records were still normal, and 43% showed focal or generalized slow wave activity after the injury. Thirty-one percent of the studies that were abnormal before the injury were normal after the injury, 23% were unchanged, and 46% were worse. Spike and wave complexes were not present after the head injury unless they had been present before.

Although it is difficult to determine with certainty in the individual whether abnormalities were caused by the mild head injury, as a group, athletes with prior mild head injuries do have an increased incidence of abnormal EEG recordings. Tysvaer et al[167] studied 37 former soccer players of the Norwegian National Team. Sixteen of the 37 had chronic symptoms such as headache, irritability, dizziness, lack of concentration, and loss of memory. A significantly increased incidence of EEG abnormalities was found in former players compared with matched controls. The number of cumulative mild head injuries may be reflected in an EEG study of a group. In a study of 40 ex-boxers, Ross et al[134] found a significant correlation between EEG abnormalities and the number of bouts fought.

EEG studies after mild head injury may be abnormal in only a minority of patients, showing diffuse or asynchronous θ and δ activity and a decrease of α amplitude and frequency. These abnormalities may resolve with time. An EEG study may be helpful when a posttraumatic seizure disorder is suspected. The utility of EEG studies after mild head injuries is restricted owing to limited sensitivity and specificity.

Brain Mapping

Abnormalities in EEG power spectral analyses have been reported after mild head injury.[119, 160] These two studies are intriguing additional evidence of cerebral cortical dysfunction following mild head injury and certainly warrant additional confirmation in a research setting. The Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology, however, does not recommend routine use of EEG brain mapping for evaluation of mild head injury.[3] The assessment paper concludes, "On the basis of the present medical literature, the sensitivity and specificity fail to substantiate a role for these tests in the clinical diagnostic evaluation of individual patients. . . ."

Auditory Brain Stem Response

ABRs have been reported as being abnormal following mild head injury. Montgomery et al[118, 119] reported an abnormal I-V interval in half a group of patients after mild head injury. The abnormality persisted in most for at least 6 weeks. In a similar study, Schoenhuber et al[140] found abnormal ABR results in about 10% of patients after mild head injury. There was no specific pattern of abnormality detected, although interpeak latency I-III was most often affected.[140]

Schoenhuber et al[142] reported a prospective study of 103 patients in whom ABRs were recorded within 48 hours of mild head injury. At 1-year follow-up, 80% of the patients had at least one persisting complaint, with irritability reported by 54%, memory loss by 47%, and depression by 39%. ABR abnormalities, however, were found with the same prevalence in patients with and without a postconcussion syndrome. They concluded:

> This study confirms the disturbance of brainstem function in some head-injured patients. However, the lack of correlation with a postconcussion syndrome limits the prognostic value of BAER [brain stem auditory evoked response] recordings for postconcussion syndrome. The data suggest that BAERs not be used for medicolegal evaluation of patients with a postconcussion syndrome.

PROGNOSIS

During the last 50 years, many prognostic studies have been performed. Comparison among the studies is difficult, however, owing to significant differences, including the definition of mild head injury, testing employed, study design, and subject characteristics (Table 5).[45, 97]

Loss of Consciousness and Posttraumatic Amnesia

The probability of having persistent symptoms and neuropsychologic deficits is the same whether a patient is dazed only or if loss of consciousness of varying duration of less than 1 hour occurs.[35, 93] The duration of posttraumatic

Table 5. VARIABLES IN PROGNOSTIC STUDIES

Definition of mild head injury
Loss of consciousness and duration
Duration, if present, of posttraumatic amnesia
Glasgow Coma Scale score
Presence of skull fracture, cerebral contusion
Use of testing
Radiologic
Neurophysiologic
Neuropsychologic
Study design
Prospective vs. retrospective
Length of follow-up
Spontaneous volunteering of symptoms vs. responding to a checklist
Interview vs. mailing
Use of matched controls
Number of subjects
Symptoms assessed
Subject characteristics
Socioeconomic and educational level
Age and sex
Prior head trauma
Use of alcohol and drugs
Pending or completed litigation

From Evans RW: The post-concussion syndrome. *In* Evans RW, Baskin DS, Yatsu FM (eds): Prognosis of Neurological Disorders. New York, Oxford University Press; 1992; with permission.

amnesia has been reported as being predictive[117] and not predictive[42] of postconcussion sequelae. There is no correlation between duration of posttraumatic amnesia and time off from work after the injury.[177]

Test Results

EEG and ABR results are not prognostically useful, although an abnormal MRI scan of the brain may be helpful. An abnormal EEG is less predictive than clinical judgment in predicting time lost from work.[40] In addition, there is no correlation between an abnormal ABR study and postconcussion symptoms.[142] Levin et al, however, found that frontal and temporal lesions seen on MRI had prognostic value for deficits of frontal lobe functioning and memory.[96] Follow-up scans at 1 and 3 months revealed a marked reduction of lesion size with improvement in cognition and memory.

Subject Characteristics

There are many subject characteristics that may affect prognosis, including age, sex, occupation, socioeconomic status, personality, intelligence, history of prior head injuries, prior use of alcohol or illicit drugs, and multiple trauma. Age over 40 years is a risk factor for increased duration and number of postconcussion symptoms[35, 42, 69] and slower recovery from cognitive deficits.[10, 59] Late symptoms occur more often in women than in men.[42, 138]

Rimel et al[130] reported that the following variables were significant predictors for return to work by 3 months: older age; higher level of education, employment, and socioeconomic status; and greater income. By 3 months, 100% of executives and business managers had returned to work compared with 68% of skilled laborers and 57% of unskilled laborers.

Symonds[156] observed, "The symptom picture depends not only upon the kind of injury, but upon the kind of brain." Preexisting psychopathology and premorbid personality are important factors.[83] Patients with high IQ recover more rapidly than low IQ patients, even though the degree of initial impairment of information processing speed is not related to intelligence.[58] Greater motivation of high achievers may be responsible.

Cumulative diffuse axonal injuries and contusions may explain why prior head injury is a risk factor for persistence and number of postconcussion symptoms.[26, 61] An increased number of posttraumatic sequelae and additional slowing of reaction time are associated with a history of prior alcohol abuse.[26] Alcohol intoxication makes the initial assessment of patients with head injury more difficult[22] and is a risk factor for neurosurgical sequelae. Finally, multiple trauma with associated orthopedic or soft tissue injuries contributes to the persistence and frequency of postconcussion symptoms[38] and can cause additional functional impairment, depression, anxiety, and stress.[14, 26]

Postconcussion Symptoms

The percentage of patients reported with various symptoms after mild head injury varies because of the many differences among the prognostic studies (see Table 2). The percentage of patients with headaches at 1 month is variably reported as 31.3%[117] and 90%[35] and at 3 months as 47%[98] and 78%.[130] At 4 years, 24% of the patients had persisting headaches.[42] The frequency of dizziness at 1 week varies from 19%[27] to 53%.[98] Dizziness is reported in 18% of patients at 2 years[27, 35] and at 4 years.[42] Memory problems are reported in 18.8% of patients at 1 month,[117] 59% at 3 months,[130] 15.3% at 6 months,[117] and 19% at 4 years.[42] At one month, 24.7% of patients reported irritability,[117] and at 1 year, 5.3% reported irritability.[138]

Comparison to controls is important because the symptoms of postconcussion syndrome are so common in the general population. Dikmen et al[38] compared 20 patients with mild head injuries with carefully matched controls 1 month after the injury. The subjects and controls reported the following symptoms: headaches, 51%, 38%; memory difficulties, 52%, 6%; difficulty concentrating, 42%, 21%; irritability, 68%, 42%; dizziness, 41%, 11%; fatigue, 68%, 41%; noise sensitivity, 52%, 10%; and light sensitivity, 32%, 28%.

Neuropsychologic Deficits

Deficits in cognitive functioning owing to mild head injury include a reduction in information processing speed, attention, reaction time, and memory for new information. Recovery of information processing speed occurs within 3 months in most patients.[60, 71, 98] Persisting impairment of attention deficits is still present at 3 months.[53] Reaction time is abnormal at 6 weeks[104] and 3 months,[71] with recovery occurring by about 6 months.[104] Memory for new information recovers over 1 to 3 months,[38, 98] although persisting impairment in visual memory and performance of digit span has been noted in one subgroup.[98]

Subjective symptoms may persist even after testing demonstrates resolution

of cognitive impairment. In the study of Levin et al,[98] although testing demonstrated almost complete cognitive recovery 3 months after the injury, 47% of subjects reported headaches, 22% dizziness, and 22% decreased energy.

Residual brain impairment may be present even after resolution of cognitive impairment and symptoms. As discussed earlier, the sequelae of mild head injury are cumulative even in persons clinically recovered. Physical, psychosocial, and environmental stress may reveal asymptomatic brain damage. Ewing et al[47] compared university students who had made a full recovery from mild head injury 1 to 3 years previously with matched controls. The two groups were tested at ground level and at a simulated altitude of 12,500 feet. The mild head injury group showed significant impairment as compared with the controls in tests of memory and vigilance performed with the mild hypoxia, although both groups performed similarly at ground level.

A significant minority of patients have persistent postconcussion difficulties and cognitive deficits after 3 months.[19b, 38, 93] Leininger et al[93] compared 53 patients with mild head injury who had persisting symptoms after 1 or more months with matched controls. Thirty-two percent of the patients were dazed without loss of consciousness, and 58% were unconscious for 20 minutes or less. Testing was performed from 1 to 22 months after injury. The most deficits were documented on tests of reasoning, information processing, and verbal learning. Test results were similar in patients assessed within 3 months of the injury as compared with the others tested after 3 months.

LITIGATION AND COMPENSATION CLAIMS

Physician as Expert Witness

As previously discussed in the historical aspects section, the possible effects of pending litigation or compensation claims has been controversial for the last century. In 1888, Everts[46] discussed the problems with using the physician as expert witness:

> What, then, is the real value of medical expert testimony? And who should be considered as medical experts? . . . Does that aggregration of knowledge known as "medicine" furnish the necessary principles for their qualifications as experts in all such cases? . . .
>
> . . . the more important qualification of the medical expert to determine questions of mental manifestations, and human actions, whether or not influenced by pathological conditions of brains, or other organs, remains to be accounted for. What has medicine in its widest range of instruction to offer on this subject? After all, how little! . . .
>
> Physiology, as taught in our schools, is indeed, still in doubt respecting the relation of mind to body. . . .
>
> . . . The natural tendency of experts, however, is to invalidate their opinions more or less, by the admission of color derived, imperceptibly, it may be, from the interest taken in behalf of the parties employing them. Instigated, also, by professional pride, experts, like detectives, are more zealous in finding what they are supposed to be peculiarly qualified to find, than otherwise. . . .

In another article in this issue, Beresford further considers the neurologist as expert witness.

Compensation Neurosis and Malingering

As a matter of routine, defense attorneys still cite Miller's 1961 study[115] of 200 consecutive cases of mild head injury seen for medicolegal examination in

Newcastle upon Tyne, England, and invariably raise questions of second gain and malingering. Forty-seven of the 200 patients were reported to ha gross and unequivocally psychoneurotic complaints. The patients exhibit characteristic behaviors during the consultation and displayed an attitude "martyred gloom." The patients frequently arrived late and were accompani by a family member who took an active part in the interview process. In mo than half of the patients, an obvious dramatization of symptoms was perceiv to be present.

> The most consistent feature is the subject's unshakable conviction of unfitness work, a conviction quite unrelated to overt disability, even if his symptomatology accepted at its face value. At a later stage, the patient will declare his fitness for lig work, which is often not available. . . . Another cardinal feature is an absolute refusal admit any degree of symptomatic improvement.

Miller makes behavioral observations of a biased sample, which may hav some validity, although they are by necessity quite subjective and judgmenta Symptoms are dismissed as being minor without substantiation, and informa tion is not provided on the percentage of patients with various complaints suc as headaches, dizziness, and memory problems. Investigations such as MR scans of the brain and current neuropsychologic testing techniques were c course not available. Miller did not totally reject the concept of a postconcussio syndrome. He stated in the same paper: "The consistency of the post-concus sional syndrome of headache, postural dizziness, irritability, failure of concen tration, and intolerance of noise, argues a structural or at least a pathophysio logical basis." Miller's study has stimulated many other investigators to explor issues of compensation further.

Guthkelch[63] reported 398 consecutive head injury patients he examined ir connection with a claim for compensation. Accident neurosis was defined by bizarre and inconsistent complaints, exaggeration of length of initial uncon sciousness, and attention-seeking behaviors. For example, two of the patients claimed to be unable to stand or take a single step without support and then were observed walking normally in the street within a few minutes after the examination. Three patients claimed compensation from disability while surreptitiously working full-time. Headaches were blinding or terrible and did not improve with time. In several cases, the patients gave an unsolicited and histrionic assertion of complete indifference to money, which was at variance with the patients' dealings with their attorney. Some patients claimed that they wanted to return to work but were forbidden by their attending physician, when in actuality this was not the case.

All of the patients were employed at the time of the injury. About one half of the patients returned to work but left within a few days complaining of headaches and noise intolerance. About one half of the patients did not return to work until their compensation claim had been settled or they were turned down for disability. Accident neurosis was more common in manual workers sustaining accidents at work than in nonmanual workers. Psychiatric treatment was not found to be helpful. Guthkelch concluded, "Accident neurosis is not particularly common; even in this series, which was exclusively composed of patients with a compensation problem, it was identified in only 6.8% of patients."

Are Litigants Different than Nonlitigants?

Patients with litigation and compensation claims are quite similar to those without. Similar symptoms improving with time[93, 111, 114] and similar cognitive

test results[93, 111] are present in both groups. Patients applying for compensation do not have increased symptoms as compared with those without applications.[120] A study on posttraumatic migraine suggests that both groups respond similarly to appropriate treatment.[173] Patients usually become litigants or claimants because they genuinely have persistent problems.

Just having litigation pending, however, may increase the level of stress for some claimants and may result in an increased frequency of symptoms after settlement.[48, 113] The skepticism of treating physicians and insurance companies about persistent symptoms may accentuate this level of stress.

Not Cured by a Verdict

The end of litigation does not mean the end of symptoms or return to work for many claimants: They are not cured by a verdict.[113] Fee and Rutherford[48] reported that 39% of patients were symptomatic at the time of settlement, and 34% were still symptomatic 1 year after the settlement of claims. Patients who are older or employed in more dangerous occupations often do not return to work after settlement.[82]

Compensation neurosis or malingering in patients with litigation or claims pending is quite uncommon. In evaluating the sequelae of mild head injury, the physician should always consider a patient's motivation and to what degree secondary gain may be playing a part. Patients with premorbid neuroticism, inadequate or histrionic personalities, and psychosocial problems can certainly exaggerate or fabricate complaints.[17, 102] Subjective criteria for making a diagnosis of postconcussion syndrome should be clearly delineated from objective findings. Evaluation can be done in a nonthreatening manner; the physician does not have to be a district attorney. The diagnosis of accident neurosis, malingering, or conversion neurosis should be made with a great deal of caution because some patients with seemingly hysterical signs and symptoms may actually have underlying organic disease.[56]

TREATMENT

Treatment for the postconcussion syndrome is individualized after the patient's particular problems are diagnosed (Table 6). Simple reassurance is often the major treatment because most patients will improve after 3 months.

Headaches

Amitriptyline has been widely used for posttraumatic muscle contraction headaches[166] as well as for the nonspecific symptoms such as irritability, dizziness, depression, fatigue, and insomnia. A recent study by Label[91] showed equal efficacy for both amitriptyline and maprotiline with average doses of 25 to 50 mg. There were fewer side effects with maprotiline. The incidental use of nortriptyline and doxepin was also effective, whereas the use of fluoxetine and imipramine was less so. Muscle contraction type headaches may also respond to nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxant type medications.

Greater occipital neuralgia frequently responds to greater occipital nerve block with a local anesthetic,[146] which can also be combined with an injectable corticosteroid when the anesthetic alone does not produce lasting improvement.

Table 6. TREATMENTS FOR POSTCONCUSSION SYNDROME

Muscle contraction type headaches
Simple analgesics
NSAID
Antidepressants
Muscle relaxant
TENS unit
Barbiturates-narcotics (with caution)
Biofeedback
Migraine type headaches
Prophylactic drugs
β blockers
Antidepressants
NSAID
Calcium channel blockers
Valproic acid
Abortive drugs
Ergotamine
Dihydroergotamine
Sumatriptan
Isometheptene
Barbiturates-narcotics (with caution)
Occipital neuralgia
Greater occipital nerve block
NSAID
Muscle relaxants
Carbamazepine
TENS unit
Rarely surgical
Psychologic support
? Cognitive rehabilitation
Education for all involved

NSAID = Nonsteroidal anti-inflammatory drug; TENS = transcutaneous electrical nerve stimulator.

NSAIDs and muscle relaxant type medications, transcutaneous electrical nerve stimulator (TENS) units,[148] physical therapy, and manipulation[79] may also be beneficial. Paroxysms of shooting pain may respond to carbamazepine. Greater occipital nerve section or decompression is rarely indicated.[32, 57, 124]

Traumatically induced migraine headaches may respond to the usual migraine medications. A 70% response to propranolol or amitriptyline used alone or in combination has been reported.[175] Other antimigraine agents can be tried empirically, including calcium channel blockers, NSAIDs such as naproxen, and valproic acid. Abortive medications that may be helpful include ergotamine preparations, isometheptene-based medications, naproxen sodium, dihydroergotamine, and butalbital alone or in combination with codeine. Of course, barbiturates and narcotics should be used on a low-frequency basis for posttraumatic headaches to avoid habituation.

A short course of biofeedback training may be worthwhile for patients with persistent headaches of all types. Acupuncture[28] and naltrexone[159] have been reported as helpful for relieving headaches in single reports. Treatment with oxiracetam has been described as being helpful for postconcussion symptoms in another single report.[136] CDP-choline has been reported as useful for memory impairment in a single preliminary report.[101]

Psychologic Approaches and Education

When the psychologic symptoms are particularly prominent, supportive psychotherapy and use of antidepressant and antianxiety type medications may be helpful. The use of cognitive retraining for cognitive difficulties after mild head injury is controversial.[15, 100, 170] Because cognitive rehabilitation can be quite costly, prospective studies are needed demonstrating efficacy before widespread application can be recommended.

One of the most important roles for the physician is education of the patient and family member, other physicians, employers, attorneys, and representatives of insurance companies.[19] Many patients are greatly reassured to discover that their symptoms are not unique or crazy but are instead part of a well-described syndrome. The treatment program of education, short-term bed rest, and timely follow-up may hasten recovery in some patients.[127] Disbelieving family members may become more supportive with education. Perhaps third parties such as some employers and insurance companies could become less hostile to injured patients if provided with education also.

SUMMARY

The postconcussion syndrome refers to a large number of symptoms and signs that may occur alone or in combination following usually mild head injury. The most common complaints are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of consciousness and memory, and noise sensitivity. Mild head injury is a major public health concern because the annual incidence is about 150 per 100,000 population, accounting for 75% or more of all head injuries.

The postconcussion syndrome has been recognized for at least the last few hundred years and has been the subject of intense controversy for more than 100 years. The Hollywood head injury myth has been an important contributor to persisting skepticism and might be countered by educational efforts and counter-examples from boxing.

The organicity of the postconcussion syndrome has now become well documented. Abnormalities following mild head injury have been reported in neuropathologic, neurophysiologic, neuroimaging, and neuropsychologic studies.

There are multiple sequelae of mild head injury, including headaches of multiple types, cranial nerve symptoms and signs, psychologic and somatic complaints, and cognitive impairment. Rare sequelae include hematomas, seizures, transient global amnesia, tremor, and dystonia. Neuroimaging and physiologic and psychologic testing should be used judiciously based on the problems of the particular patient rather than in a cookbook fashion.

Prognostic studies clearly substantiate the existence of a postconcussion syndrome. Manifestations of the postconcussion syndrome are common, with resolution in most patients by 3 to 6 months after the injury. Persistent symptoms and cognitive deficits are present in a distinct minority of patients for additional months or years. Risk factors for persisting sequelae include age over 40 years; lower educational, intellectual, and socioeconomic level; female gender; alcohol abuse; prior head injury; and multiple trauma. Although a small minority are malingerers, frauds, or have compensation neurosis, most patients have genuine complaints. Contrary to a popular perception, most patients with litigation or compensation claims are not cured by a verdict.

Treatment is individualized depending on the specific complaints of the patient. Although a variety of medication and psychologic treatments are currently available, ongoing basic and clinical research of all aspects of mild head injury are crucial to provide more efficacious treatment in the future.

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