

## Post concussion syndrome

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### Summary

Individuals sustaining mild traumatic brain injuries often report a constellation of physical, cognitive, and emotional/behavioral symptoms referred to as post concussion symptoms (PCS). The most commonly reported post concussion symptoms are headache, dizziness, decreased concentration, memory problems, irritability, fatigue, visual disturbances, sensitivity to noise, judgment problems, depression, and anxiety. Although these PCS often resolve within one month, in some individuals PCS can persist from months to years following injury and may even be permanent and cause disability. When this cluster of PCS is persistent in nature, it is often called the post concussion syndrome or persistent PCS. Both physiological and psychological etiologies have been suggested as causes for persistent post concussion symptoms and this has led to much controversy and debate in the literature. Most investigators now believe that a variety of pre-morbid, injury-related, and post-morbid neuropathological and psychological factors contribute to the development and continuation of these symptoms in those sustaining mild traumatic brain injury (MTBI).

### Introduction

Concussion or mild traumatic brain injury (MTBI) is the most common form of traumatic brain injury and the majority of individuals who sustain such injuries are young adults. Mild traumatic brain injury was once thought to be trivial in terms of consequences, however, there is now substantial evidence that neuropathological, neurophysiological, and neurocognitive changes occur even in these so-called mild injuries (Dikmen & Levin, 1993). Diffuse axonal injury has been identified as a common feature of MTBI in both animal and human studies (Povlishock *et al.*, 1992). Individuals sustaining mild head injuries often complain of a number of physical, cognitive, and emotional/behavioral symptoms referred to as post concussion symptoms (PCS). These symptoms can persist from months to years following injury and may even be permanent and cause disability (Brown *et al.*, 1994; Gouvier *et al.*, 1992).

We begin this review with an introduction on PCS briefly describing the phenomenology, prevalence, and etiology. We then discuss the neurocognitive functioning, neuroimaging/electrophysiological measures, and psychosocial/injury related factors associated with PCS and persistent PCS/post concussion syndrome. Finally we outline the treatment and prevention of persistent PCS.

### Post concussion symptoms

#### Phenomenology

Post concussion symptoms (PCS) are a cluster of symptoms that frequently occur following MTBI. It should be noted, however, that such symptoms occur after moderate to severe injuries as well. As depicted in Table 1, PCS consist of a number of commonly self-reported physical, cognitive, and emotional/behavioral symptoms including: headache, dizziness, irritability, difficulty concentrating, memory problems, fatigue, visual disturbances, sensitivity to noise, judgment problems, depression and anxiety (Alexander, 1995; Gouvier *et al.*, 1992). This cluster of symptoms has also been called 'late symptoms' because they are often reported a few days and weeks following the injury. However, it is important to note that two of the symptoms in this cluster, headache and dizziness, are actually the exceptions and they typically occur immediately as well as later in the course of recovery. The other early symptoms that individuals complain of immediately following their injury include nausea, vomiting, and drowsiness and are generally short-lived (Bohnen & Jolles, 1992; Rutherford, 1989).

While PCS frequently resolve within one month, numerous studies have now demonstrated that PCS are reported for months and even years post-injury in a significant minority of individuals after MTBI.

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**Table 1.** Post concussion symptoms (PCS)

| Physical            | Cognitive                        | Emotional    |
|---------------------|----------------------------------|--------------|
| Headaches           | Memory deficits                  | Depression   |
| Dizziness           | Attention/concentration deficits | Anxiety      |
| Fatigue             | Executive function deficits      | Irritability |
| Visual disturbances |                                  |              |
| Noise sensitivity   |                                  |              |
| Light sensitivity   |                                  |              |

In fact, symptoms have been reported to persist for 15 years or more after the injury (Binder, 1986; Bohnen & Jolles, 1992; Rutherford, 1989). When this cluster of PCS is persistent in nature, it is often called the post concussion syndrome or persistent PCS. As an initial attempt to standardize the diagnosis of post concussion symptoms, the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994) fourth edition (DSM-IV) has research criteria for the diagnosis of 'post-concussional disorder'.

#### Prevalence

There is much variation in the reported prevalence of PCS and persistent PCS. Prevalence rates at three months post-injury have been found to range from 24–84% (Rutherford, 1989). Such variability may be related to methodological differences between studies but may also reflect a lack of homogeneity among the population of individuals with persistent PCS (Bohnen & Jolles, 1992).

#### Etiology

The etiology of persistent PCS or the post concussion syndrome has generated much controversy concerning whether such symptoms are due to alterations in neurophysiology and neuropathology secondary to the injury, or due to pre- or post-injury psychological factors (Szymanski & Linn, 1992). The subjective and rather nonspecific nature of PCS has helped fuel this controversy.

Rutherford (1989) has suggested that while the underlying brain dysfunction is present from the moment of injury, it takes time and the everyday stressors of life to elicit these PCS. Alternatively, there is now good evidence that axonal injury may come from a delayed pathophysiological reaction occurring over several hours (Dixon *et al.*, 1993) and such a mechanism may be responsible for the onset of PCS following MTBI.

The frequency of post concussion type symptoms among non-brain-injured normal controls, non-brain-injured personal injury claimants, medical patients, psychiatric patients, and chronic pain patients is high (Gouvier *et al.*, 1988; Lees-Haley &

Brown, 1993; Fox *et al.*, 1995a; Fox *et al.*, 1995b; Smith-Seemiller *et al.*, 2003). However, in the studies of medical and psychiatric patients, greater symptom endorsement occurred in patients who reported being knocked unconscious, particularly for some of the more somatic and cognitive PCS (headache, memory and concentration problems, sensitivity to noise, etc.). Further regression analyses demonstrated that loss of consciousness was a strong predictor of more PCS. Moreover, in a recent study of chronic pain and MTBI patients, while both groups endorsed PCS, a greater proportion of MTBI patients endorsed memory problems, slowed cognition, and light/noise sensitivity compared to the chronic pain patients (Smith-Seemiller *et al.*, 2003). These results suggest that some PCS, particularly the somatic and cognitive symptoms, may be more likely associated with the head trauma while others may be related to situational factors or general psychological distress (Fox *et al.*, 1995a; 1995b).

Psychological and motivational factors such as involvement in litigation and pre-morbid/post-morbid psychological problems have also been identified as causes of persistent PCS and the terms post-traumatic or 'compensation neurosis' have been used. Compensation neurosis dates back to the 1960s with Miller being the most outspoken supporter that PCS directly resulted from involvement in litigation, i.e., that patients exaggerate/malinger these symptoms in order to gain monetary compensation (Gouvier *et al.*, 1992; Miller, 1961). However, in contrast to Miller, numerous other researchers have documented the presence of PCS in patients not involved in litigation and failed to show a relationship between PCS and litigation (Bohnen & Jolles, 1992; Rimel *et al.*, 1981; Wrightson & Gronwall, 1981). The influence of psychological disturbance is still an issue, however. Pre-morbid emotional problems and post-injury level of psychological distress have been implicated in persistent PCS but not consistently demonstrated.

It is clear that persistent PCS occur in a number of individuals following MTBI but not in all. It appears that both purely psychogenic and purely physiogenic views are limited. Most researchers now believe persistent PCS to be the result of multiple pre-morbid, injury-related, and post-morbid neuropathological and psychological factors (Alexander, 1995; Bohnen & Jolles, 1992).

#### PCS and neurocognitive functioning following MTBI

There is evidence to indicate that the presence and chronicity of PCS experienced by individuals after MTBI corresponds to demonstrable impairment in neurocognitive functioning. Rimel *et al.* (1981) published one of the first studies to show a high rate of PCS and neuropsychological impairment in

patients after MTBI. These patients also showed mild impairment in neurocognitive functioning, primarily in the areas of attention/concentration, memory, and judgment/problem solving. A later study by Levin *et al.* (1987) found conflicting results. These researchers examined neurobehavioral changes in MTBI patients consecutively admitted to three medical centers at one week, one month, and three months post-injury. Neuropsychological test impairment was noted initially but generally resolved by the third month. Post concussion symptoms did not appear related to neurocognitive recovery. Subjective complaints were frequently still present at one and three months post-injury even in patients whose cognitive functioning improved to within normal limits. Another study evaluated patients with PCS six to 18 months after MTBI and did find deficits in attention/concentration and memory (Mariadas *et al.*, 1989). In addition, Leininger *et al.* (1990) demonstrated that patients with persistent PCS, one to 22 months after MTBI, performed significantly more poorly than controls on measures of information processing, reasoning, and verbal learning. Neurocognitive performance was not affected by time since injury, loss of consciousness versus alteration in mental status (i.e., disorientation/confusion but without loss of consciousness), or litigation status.

Bohnen and colleagues (1992a) attempted to clarify the issue of PCS and neurocognitive functioning further by directly comparing subjects with and without persistent PCS who had sustained uncomplicated MTBIs, i.e., MTBI without prior history of TBI, or psychiatric/neurologic illness. In one study, these investigators evaluated patients with and without PCS six months post-injury and a group of normal controls. Post concussion symptom patients showed deficits in selective and divided attention compared to patients without PCS and controls (Bohnen *et al.*, 1992a). In a later study by these researchers, MTBI patients were evaluated even longer post-injury, 12–34 months. No overall gross neurocognitive differences were found between patients with and without PCS or controls but an isolated deficit in sustained attention was noted for patients with PCS. Also among PCS patients, those with higher ratings on post concussive-cognitive symptoms performed less well on a sustained attention task than PCS patients with lower scores (Bohnen *et al.*, 1995). As these authors note, it may be that persistent neurocognitive deficits may be more prevalent among the subgroup of individuals experiencing PCS.

### **PCS and functional neuroimaging/ electrophysiological measures**

Structural neuroimaging, computed tomography (CT) and magnetic resonance imaging (MRI) have

had limited utility in the evaluation of MTBI and PCS. Abnormalities on CT scans are rarely found in patients with mild injuries. Abnormalities on MRI have been found more frequently but only in some patients. In a group of patients with mild-to-moderate injuries, Eisenberg and Levin (1989) found multifocal lesions primarily in frontotemporal regions. A study by Bigler and Synder (1995) involving a small group of patients with documented mild head injury who had prior neuroimaging evaluated pre- and post-injury scans. Despite persistent mild neurocognitive deficits and emotional sequelae, post-injury MRI did not show changes from pre-injury scans or differences from control subjects.

Functional measures such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and evoked potentials/event-related potentials (EPs/ERPs) appear to be more promising in identifying metabolic and electrophysiologic alterations in MTBI in general and those with persisting symptoms in particular. McAllister and colleagues (McAllister *et al.*, 1999; McAllister *et al.*, 2001) demonstrated brain activation pattern differences in fMRI studies of working memory after MTBI. This group of patients was significantly more symptomatic than controls, but had few measurable deficits on neuropsychological testing.

Ruff and colleagues (1994) examined glucose metabolism in MTBI patients with persistent neuropsychological deficits but few or no abnormalities on CT/MRI and in controls were found. Abnormalities, particularly hypometabolism, were found in the MTBI patients but not in controls. These abnormalities were seen primarily in frontal and anterior temporofrontal regions. A more recent PET study examined resting glucose metabolism, as well as activation cerebral blood flow in MTBI patients with persistent PCS and normal controls (Chen *et al.*, 2003). There was no difference between patients and controls for resting glucose metabolism. However, the persistent PCS patients demonstrated a smaller increase in right prefrontal blood flow compared to controls during a working memory task. The findings of frontal and temporal abnormalities in MTBI are not surprising. Orbitofrontal and anterior temporal regions are particularly vulnerable to contusions, lacerations, abrasions, hematomas, and intercerebral hemorrhages due to forceful contact with the rough bony surface of the skull in these areas during head injury (Mattson & Levin, 1987; Varney & Menefee, 1993). In addition, diffuse axonal damage may disrupt frontal pathways to other cortical and subcortical regions, including the limbic system. Damage to these regions have been linked to deficits in complex neurocognitive functioning including attention and memory as well as to emotional changes (Mattson & Levin, 1987).

Electrophysiological indices of brain function, evoked potentials and event-related potentials

(EPs/ERPs) have been found to differentiate MTBI patients from uninjured controls in some studies (e.g., Freed & Fishman Hellerstein, 1997; Gaetz & Weinberg, 2000). A relatively recent study by Gaetz and Weinberg (2000) examined electrophysiologic indices of persistent PCS. The authors administered an EP/ERP assessment battery to both MTBI patients with persistent PCS and normal controls. Visual EPs and visual and auditory ERPs tended to be outside the normal range for the persistent PCS patients as opposed to the generally normal scores for the controls. In particular, the greatest differences between persistent PCS patients and normal controls were seen with primarily visual ERPs. The authors conclude that these results provide evidence of actual changes in brain function in MTBI individuals with persistent PCS and thus support a neuropathological basis for persistent PCS.

### PCS and psychosocial/injury-related factors

A number of psychosocial and injury-related factors have been implicated in the development of persistent PCS including prior history of head injury, neurological signs, female gender, older age, and psychological problems pre- and post-injury. Bohnen *et al.* (1992b) attempted to examine the influence of pre-morbid problems on PCS following mild head injury. Patients with complicated MTBI, that is those with prior history of TBI or pre-morbid emotional problems, and those without such pre-morbid histories, as well as normal controls completed a questionnaire including both traditional PCS and nonspecific emotional/functional symptoms. Factor analysis yielded two distinct groups of symptoms: post concussive-cognitive symptoms which consisted of typical PCS symptoms, as well as a second set of problems including decreased work capacity/efficiency, fatigue, etc., and emotional-vegetative symptoms such as heart palpitations, gastrointestinal problems, depression, and emotional lability. Patients were tested initially one to two weeks after injury and then at four to six weeks post-injury. There was no significant relationship between time since injury and the scores on the two scales. Patients with uncomplicated MTBI had significantly higher scores on the post concussive-cognitive scale than controls but not on the emotional-vegetative scale. Patients with complicated MTBI scored significantly higher on both scales than patients with uncomplicated mild head injury. These authors suggest that emotional/vegetative complaints appear to be secondary symptoms that may reflect a reduced ability to cope with environmental stressors in those with pre-existing complications.

The concept of PCS as reflecting a reduction in the ability to compensate for stressful conditions, secondary to residual damage, was examined by Gouvier and colleagues (1992). Post concussion

symptoms and the influence of stress were evaluated in college students with and without a self-reported history of MTBI. Their results failed to support this concept. The number of PCS was similar for both groups and was correlated with levels of daily reported stress for both injured and control subjects. Stress was related to higher symptom rates. In a conflicting report, investigators examined the relationship between PCS and stress after hospital discharge and found that at follow-up (six to 19 days post-injury), 71% of the MTBI subjects reported still experiencing PCS and 29% experienced a worsening of symptoms, but stress was not found to have a significant effect (Moss *et al.*, 1994).

Age and gender have been identified as factors related to the development of persistent PCS as well. Older age (over age 40) and female gender have been associated with higher rates of PCS in MTBI individuals. Early studies by Rutherford and colleagues found that at six months post-mild head injury, 51% reported PCS, and at one year post-injury, 14.5% still reported symptoms. Symptoms were more common in females, older patients and those with positive neurological signs at 24 hours following injury (Rutherford *et al.*, 1977; 1979). A much later study by Packard *et al.* (1993) found a high rate of PCS (65%) overall and noted that a higher percentage of females reported symptoms than did males. Fenton *et al.* (1993) similarly found that older age and female gender were significantly related to PCS and in addition found that mild head injury patients with persistent PCS had greater frequency of pre-morbid social difficulties as well as a higher frequency of problems with anxiety or depression at six weeks post-injury. Alves *et al.* (1993) found that female gender and Glasgow Coma Scale scores were correlated with PCS at 12 months post-mild head injury but failed to find a relationship with age or psychiatric/substance abuse history. A large study by Bohnen *et al.* (1994) investigated the intensity of vague everyday and post concussion-type symptom complaints including dysthymic complaints (depression, anxiety, tearfulness and so forth), vegetative/bodily complaints (headache, vertigo, lightheadedness, etc.), and performance complaints (decreased work performance, forgetfulness, etc.) in mild head injured patients one to five years post-injury and matched controls via a questionnaire mailing. Overall these complaints were more prevalent and more severe in mild head injured subjects although the pattern of complaints was similar for both patients and controls. They also found that a few factors increased the likelihood of these symptoms in patients: older age, female gender, pre-morbid emotional problems, co-morbid medical problems, neurological complication at time of injury, orthopedic fracture, hospitalization, lower education, and intoxication at the time of injury. Other factors were not related such as time since injury and insurance claim. The authors suggest that mild head injury

can result in neurobehavioral sequelae in terms of sub-optimal physical and mental health that may not be completely reversible; furthermore, the results indicate that both physiological and psychological factors contribute to the persistence of symptoms. Ryan and colleagues (1998) found that a combination of psychosocial, pre-morbid, and neurocognitive factors were predictive of persistent PCS in a group of college students with a self-reported history of MTBI. Specifically, the most predictive variables were level of current post-injury psychological distress and female gender. Moreover, King (1996) examined predictors of persistent PCS among mild-to-moderately head-injured patients at seven to ten days and three months post-injury and found that the best predictors of persistent PCS were level of anxiety/depression and stress acutely following the injury.

In summary, both neuropathological and psychological etiologies have been suggested as the basis for persistent post concussion symptoms or post concussion syndrome. Most investigators now believe that multiple physiological and psychological factors contribute to the continuation of these symptoms in those sustaining MTBI.

### Treatment/prevention of persistent PCS

Given the potentially long-term morbidity associated with persistent PCS, the development of effective treatment and ultimately prevention strategies is critical. Post concussion symptoms are most frequently treated with medications (Mittenberg *et al.*, 2001). A survey of commonly prescribed medications by Evans and colleagues (1994) found that non-steroidal anti-inflammatory analgesics were most often prescribed, followed by antidepressant medications. Relatively recent studies by Fann *et al.* (2000; 2001) suggest that the serotonin reuptake inhibitor sertraline may be useful in reducing PCS after MTBI. However, it is important to note that large-scale, randomized control treatment trials are lacking.

Psychological treatments including education, reassurance, and cognitive restructuring or reattribution have also been found to reduce persistent PCS and improve outcome (Mittenberg *et al.*, 1996; Ponsford *et al.*, 2002; Wade *et al.*, 1998). A very recent intervention study by Ponsford and colleagues (2002) examined the impact of a PCS information and management booklet on outcome at three months post-MTBI. In addition to standard emergency room treatment, patients in the intervention group were assessed and received the information booklet within five to seven days post-injury. The control group received only standard emergency room treatment. Both groups were then assessed three months later. The intervention group reported fewer PCS and less stress overall compared to the

control group. In general, such psycho-educational techniques help MTBI patients to understand the nature of PCS and the expected course of recovery, as well as provide effective coping strategies. A recent meta-analysis of controlled psychological treatment studies suggests significantly reduced incidence of persistent PCS in MTBI patients who receive brief psychological intervention compared to those who receive standard acute care. Moreover, psychological treatment reduced symptoms by 0.32 standard deviations thus demonstrating a clinically as well as statistically significant effect size. In other words, 16% of untreated PCS patients would be symptom-free if they had undergone brief psychological treatment (Mittenberg *et al.*, 2001).

### Conclusion

Post concussion symptoms or PCS are a cluster of physical, cognitive, and emotional/behavioral symptoms that frequently occur following MTBI but can also be seen following more severe injuries. PCS often resolve within one month after injury, however, in some individuals PCS can persist from months to years. Both physiological and psychological factors have been suggested as etiologies for persistent PCS and/or post concussion syndrome. Based on the body of research to date, it is likely that multiple pre-morbid, injury-related, and post-morbid neuropathological and psychological factors contribute to the development and continuation of these symptoms.

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