Post-concussion syndrome: 
clarity amid the controversy? 

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No head injury is too trivial to ignore’ (Hippocrates, 460–377 bc, in Ingebrigsten (1998))

Post-concussion syndrome has presented clinicians with controversy and intrigue for at least 130 years. Its symptoms, which often follow uncomplicated mild head injury (post-traumatic amnesia < 1 h, Glasgow Coma Scale score 13–15, loss of consciousness < 15 min) and moderate head injury (post-traumatic amnesia 1–24 h, Glasgow Coma Scale score 9–12, loss of consciousness 15 min to 6 h), commonly include headache, dizziness, fatigue, poor memory, poor concentration, irritability, depression, sleep disturbance, frustration, restlessness, sensitivity to noise, blurred vision, double vision, photophobia, nausea and tinnitus (King, 1997). Debate and argument have always plagued this syndrome, but research over the past decade or so has helped to clarify some of the areas of dispute and some interventions have been developed and evaluated. What follows is an attempt to illuminate the syndrome in the light of these developments.

EPIDEMIOLOGY AND NATURAL RECOVERY

The number of people who sustain a mild head injury and experience subsequent post-concussion symptoms is very high. In Great Britain each year, 250–300 hospital admissions per 100 000 of the population involve head injury, of which only 8% are severe injuries and at least 75% are mild (Jennett & MacMillan, 1981). The number of mild head injuries that pass through accident and emergency departments without admission is 4–8 times this number. Many, if not the majority of those with mild head injury experience no discernible symptoms whatsoever, but at least half experience some post-concussion symptoms. Most recover completely within 3 months of injury, but around a third have some persisting symptoms beyond this time. Around 8% have significant symptoms at 1 year and in some cases these symptoms are possibly permanent (Binder, 1997). Quite clearly, mild head injury is not always a mild experience.

NEUROPSYCHOLOGICAL IMPAIRMENT

A consistent feature of post-concussion syndrome is the correlation between the severity of symptoms and the severity of neuropsychological impairments in speed of information processing. These impairments tend to improve in line with symptomatic recovery over 3 months following injury, and where symptoms do persist at 6 months these cognitive deficits also tend to remain (Bohnen et al, 1992). Verbal and visuospatial short-term and long-term impairments are also a consistent feature of the syndrome, and these, too, typically recover over 3 months following injury. These impairments do not, however, correlate well with the severity of symptomatic presentation. Measures of other cognitive domains (e.g. reaction time) have so far yielded only equivocal results.

EVIDENCE FOR ORGANIC FEATURES

The extent to which the syndrome is primarily of organic or psychological origin, at any given time following injury, is a debate that is often heated, and sometimes polemical. Evidence of organic and quasi-organic processes involved in at least some presentations of post-concussion syndrome includes the following:

(a) post-mortem studies demonstrating diffuse microscopic axonal injury after mild head injury in humans and animals (Oppenheimer, 1968);

(b) macroscopic brain lesions being evident in 8–10% of individuals who have had routine computed tomographic or magnetic resonance imaging (MRI) brain scans performed within the first few weeks of injury (Sekino et al, 1981; Doezema et al, 1991); these lesions predominate in frontal, temporal and deep white-matter areas, and although their existence does not correlate well with post-concussion syndrome symptoms, they too show substantial resolution over 3 months (Levin et al, 1992);

(c) cellular damage and metabolic abnormalities being found in frontal white matter in patients with mild head injury and normal MRI scan within the first few weeks of injury (Garnett et al, 2000);

(d) abnormal intraparenchymal regional cerebral blood flow being found within the first few weeks of mild head injury (Nedd, 1993);

(e) subtle abnormalities on electroencephalographic and brain-stem evoked response measures being noted within 48 h of mild head injury (these, however, were unrelated to post-concussion symptoms or neuropsychological sequelae) (Schoenhuber & Gentilini, 1989);

(f) abnormal regional cerebral blood flow and reduced glucose metabolism in anterior and posterior temporal areas being found in subgroups of patients with persistent and disabling post-concussion syndrome up to 5 years after injury (Varney et al, 1995).

Other evidence suggestive of organic factors includes the following: slower recovery with increased age (cut-off at around 40 years) (Binder, 1986); poorer outcome and a cumulative effect of previous and successive mild head injuries (Gronwall & Wrightson, 1975); and poorer outcome with a history of alcohol or substance misuse (Lishman, 1988).

EVIDENCE FOR INVOLVEMENT OF PSYCHOLOGICAL PROCESSES

Evidence for psychological features includes the following:

(a) there is worse outcome where there is pre-existing psychopathological disorder (Lishman, 1988);

(b) the best early predictors of persisting post-concussion symptoms are psychological factors;
Time after injury | Possible emerging factors
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0–24 h (immediate symptoms) | Mainly organic factors
1 day–4 weeks (early symptoms) | Overdoing and failing tasks
| Increase in life demands following recuperation
| Misattribution of symptoms to malignant causes that have been missed
| Difficulties coping with cognitive impairments
| Initial concerns regarding longevity of symptoms/disabilities
| Dissonance regarding severity of head injury and severity of symptoms/disabilities
1–6 months (medium-term symptoms) | Unhelpful premorbid schemas and coping responses related to managing abnormal life events; inability to complete tasks; frustration and ‘mind over matter’ coping
| Concerns regarding potential permanence of symptoms
| Unhelpful strategies for coping with uncertainty (particularly uncertain aetiology of symptoms)
| Misperception of having suffered a severe brain injury
Over 6 months (long-term or possibly permanent symptoms) | Lack of understanding/belief from others
| Compensation-claim factors
| Issues relating to adjustment to long-term disability
| Unhelpful ‘secondary’ coping strategies (those employed following failure of initial strategies)
the relative contribution of these processes will have to be gauged from both positive and negative evidence of organic and psychological features. This has to be done without losing sight of the complex interactional effects between these factors. It is therefore essential that patients with persisting and disabling post-concussion symptoms receive individualised formulations of their particular problems.

DECLARATION OF INTEREST

None.

REFERENCES


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