The postconcussional syndrome revisited

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The nature and origin of the postconcussional syndrome

The postconcussional syndrome refers to the emergence and variable persistence of a cluster of symptoms following a mild head injury. Common to most descriptions are somatic symptoms (headache, dizziness, fatigueability) accompanied by psychological symptoms (memory and concentration difficulties, irritability, emotional lability, depression and anxiety). Between one-third and half of patients experience the syndrome over the first few weeks following a mild head injury[1-4]. While most become asymptomatic in the months following, a substantial minority experience persistent and troublesome symptoms even 6 months to 1 year later[3,5].

We have come some considerable distance since Miller[6], in his Milroy Lectures, coined the term accident neurosis and attributed the postconcussional syndrome to the compensation situation (a manifestation of financial gain). A large body of authoritative research has challenged such an extreme position. For example, while Miller contended that the syndrome is not observed in severe head injury McKinley[7] found the symptoms in over half of such patients at 6 months post injury. Miller stated that the syndrome was only seen in compensation situations, yet in studies by Wrightson and Gronwall[8] and Rimel *et al.*[9] compensation was relatively uncommon. Miller suggested that the syndrome disappears on compensation settlement yet in the follow-up study by Rutherford and Fee[10] 39% had symptoms at settlement and 34% still had symptoms 1 year later. Present evidence clearly indicates that compensation is neither necessary nor sufficient for the great majority of patients who have persistent symptoms following a minor head injury.

The evidence of early brain changes, both functional and structural, following a mild head injury is now overwhelming. The earliest studies of cerebral circulation by Taylor and Bell[11] and postmortem pathological changes by Oppenheimer[12] have been complemented by a number of physiological studies[3,13,14] and neuroimaging investigations[15]. These provide incontrovertible evidence for either transient or permanent functional and structural changes throughout many areas of the brainstem and cortex following a relatively mild closed head injury.

However, Lishman[16] has cautioned against the error of confusing a simple association between independent observations and evidence of aetiological causation and stressed the need for systematic prospective investigation.

The evidence from two recent prospective studies in Belfast is reviewed in an attempt to shed further light on those factors responsible for the emergence and persistence of the post-concussion syndrome.

Methodology

Details of the recent Belfast studies have been given elsewhere[3,17,18]. The inclusion criteria were a post-traumatic amnesia of less than 24 h. All patients with alcohol consumption on the day of injury, previous history of head injury or brain disease, history of alcohol or drug abuse or any significant injury to other parts of the body at the time of the accident were excluded. The first study consisted of 45 consecutive admissions in the age ranges 14-65 years. Notably, 46% of the group had post-traumatic amnesias (PTA) of less than 1 h. The second study consisted of a high risk group - 28 males in the age range 14 to 30 years and in whom 76% had PTAs of less than 1 h.

A range of clinical [including the Present State Examination (PSE)[19]], social [the Brown Life Event Questionnaire (LEQ)][20], and laboratory investigations were carried out during recovery and follow up period. The first follow-up evaluation was at 6 weeks post injury (early follow-up). Late follow-up evaluations were made of 6 and 12 months, respectively, for the two studies.

In both studies, electroencephalogram (EEG) spectra from temporal and parietal regions of each hemisphere (T4-T6, P4-02, T3-T5, P3-02) were separately examined within 24 h of at the injury event and at 6 weeks follow-up. Total power at the following frequency bands were calculated; delta (0.5-3 Hz), theta (3.5-7.5 Hz), alpha (8-12 Hz).

Brainstem auditory evoked potentials were recorded again within 24 h of injury and at 6 weeks follow-up. These were elicited using 80 μs pulses from an AMPLAID Mark 3 auditomer delivered to each ear via PDH 39 earphones at an intensity of 70 dB above normal hearing level (NHL). Averages from 1024 stimuli were formed and the right and left ears were stimulated separately. Responses were recorded from vertex and ipsilateral mastoid positions, the first (I) and fifth (V) negative peaks of the brainstem auditory evoked potential (BAEP) were identified from plots of the responses and the I-V intervals measured in milliseconds.

Results

The evidence for physiogenesis

From the computerized cortical EEG activity recorded in the immediate postconcussional period, a large amount of diffusely distributed abnormal slow wave activity was a universal finding. A marked reduction in this activity was observed over all scalp sites at

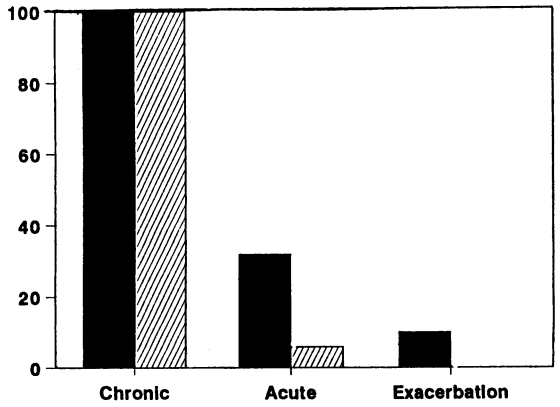

Figure 1. The percentage of subjects with abnormal central conduction times detected in the I-V interval of the brainstem auditory evoked potentials for the three outcome groups. ■ =Day 0; ▨ =6 weeks

6 weeks follow-up. All EEG channels showed significant correlations with symptom count. That is, the greater the EEG improvement score, the fewer the symptoms at early follow-up. However, no association was found between cortical EEG measures and measures of post-traumatic amnesia. The left temporal region showed the least recovery and while there was no overall relationship between chronicity and EEG abnormality, a trend was evident between this slow left temporal recovery and chronicity of symptoms at late follow-up evaluation.

Brain stem conduction times were significantly delayed in 46% of subjects in the first study group and in 27% of the young males in the second study. It will be recalled that the second group consisted of young males, the great majority of whom had PTAs of less than one hour. A significant correlation was found between prolonged brain stem conduction time and PSE ID severity scores at early follow-up ($R=0.04$, $P<0.01$). Conduction times were also highly correlated with PTA scores ($R=0.5$, $P<0.004$).

This is the first reported study examining the relationship between impaired brainstem functioning and longer-term outcome. Three outcome profiles were identified and the results from the two studies have been combined. The largest group (acute group - 31 (66%)) were relatively symptom free by 6 weeks. Six patients had persistent symptoms throughout the period of follow-up (chronic group). A further 10 patients showed symptom exacerbation at final follow-up (exacerbation group). In spite of the small sample size a clear relationship was observed between brainstem findings and outcome (Figure 1). For the acute group one-third had delayed central conduction time reducing to 6% only at 6 weeks. By contrast all 6 of the chronic group had abnormal conduction times at day 0 and again at 6 weeks. In patients with symptom exacerbation, only one showed abnormal central conduction time at day 0 and all were normal by 6 weeks post-injury.

The evidence for psychogenesis

This is the first systematic assessment of the mental health of patients following mild head injury. In the first study the PSE carried out at 6 weeks post-injury gave a case rate of 36%, mostly consisting of anxiety and depression. A further 29% were borderline cases. This compared with 4% of cases among matched controls.

Cases were on average 10 years older than non-cases. Over half of cases were females compared with only 18% of non-cases. Taking age and gender together, among adults under 25 years only three of 17 males (18%) were cases compared with five of six (83%) women over 25 years of age. The level of chronic social adversity revealed by the LEQ was four times higher for cases (3.3) compared with non-cases (0.8; $P<0.001$). However, no significant differences were observed in the rates of life events between cases and non-cases.

The relationship between chronicity and psychosocial variables was also examined. Thirty per cent of the group had three or more symptoms at late follow-up. Patients with persistent symptoms were significantly older (mean age 39 years) than those whose symptoms had remitted (mean age 28 years; $P<0.05$). Caseness at early follow-up also predicted chronicity. Forty-six per cent of cases at 6 weeks had become chronic cases at late follow-up compared with only 8% of non-cases. Chronic social difficulties were on average twice as common among those whose symptoms had become chronic compared with those whose symptoms had remitted.

No differences in premorbid personality or social adjustment were observed between patients whose symptoms had remitted and those who had become chronic cases.

Discussion

The postconcussional syndrome refers to the emergence and variable persistence of a well recognized symptom cluster. While it has been recognized that the postconcussional syndrome is a common early sequel of minor head injury, the Belfast studies have also demonstrated a high level of psychiatric caseness in this period. Depressive and anxiety syndromes were the most prominent.

In these two studies several strands of evidence point to the contribution of both physiological and psychological factors in the emergence of the syndrome. While no association was observed between post-traumatic amnesia and cortical EEG measures at early follow-up, a clear positive correlation was obtained between PTA and brainstem conduction time. This suggests a greater dependency of the amnesia found in this clinical setting on brainstem functioning than cortical functioning. There is incontrovertible evidence of both cortical and brainstem dysfunction. While the cortical changes are mostly transient, brainstem changes were largely unaltered at 6 weeks follow-up. Relationships were also observed between symptom measures and cortical and brainstem functioning. Chronic social adversity, older age and female gender were associated with caseness even at 6 weeks post-injury. However, there was no evidence of premorbid personality vulnerability.

Lishman's[16] careful review of available literature concluded that physiological factors contributed mainly to the emergence of the postconcussional syndrome while psychological factors contributed more to the chronicity of the syndrome. The findings of the Belfast studies suggest that psychosocial factors, particularly social adversity contributes to its emergence. While age and gender also appear to be relevant it is unclear from the present evidence what mechanisms underlying or associated with age and gender may be contributing (biological and/or psychosocial).

It is the chronicity of the post-concussional syndrome that has generated much of the controversy surrounding the post-concussion syndrome. While compensation as the major mechanism has been clearly challenged, there can be little doubt that psychological and social issues contribute. However, a clear association between measures of brainstem dysfunction and persistence of symptoms has also been demonstrated. These findings are supported by a recent study of whiplash by Radanov and colleagues[21] in which initial neck pain intensity and injury-related cognitive impairment predicted symptoms at 6 months post-injury. Probably one of the clearest strands of evidence for organic contributions to chronicity is the cumulative effect of repeated minor, subconcussive injury in boxers[22-24].

In seeking to understand the contribution of different aetiological factors to the emergence and persistence of the postconcussional syndrome it is well to remember the strong tendency to dichotomize medical problems into either mind problems or body problems. Much of the debate surrounding the postconcussional syndrome are caught in this dualism. The hypothesis which best fits available evidence is damaged brains and troubled minds. First, psychological and physiological vulnerability contribute to the emergence of the syndrome. Second, head injury is both an emotional as well as a physical event. The meaning of the event as well as the direct physical assault on brain processes are of aetiological importance in the emergence of the syndrome. Third persistence of the syndrome is a final common path in which psychosocial and biological factors contribute in varying degrees in individual patients. A holistic approach which takes account of a person's physical nature and in which meaning and relationships matter is essential for a full understanding of the sequelae of minor and indeed major head injury. The task for clinicians is a careful assessment of the relevant contribution of each factor in individual cases and the management of those aspects which are most responsive to intervention.

One of the most important interventions is the mobilization of each patient's personal resources, a process which must be commenced in the early rehabilitative phase. One simple measure is the provision of a full explanation of the natural history of this often quite chronic condition.

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