Pergamon

Journal of Psychosomatic Research, Vol. 39, No. 6, pp. 675–693, 1995
Elsevier Science Ltd
Printed in Great Britain.
0022-3999/95 $9.50 + .00

0022-3999(95)00006-2

THE POST-CONCUSSIONAL SYNDROME: PHYSIOGENESIS, PSYCHOGENESIS AND MALINGERING. AN INTEGRATIVE MODEL

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Abstract—Current models of post-concussional symptoms after mild head injury rest on the dichotomy between organic and psychogenic factors, which underpins Lishman's formulation; organic genesis and psychologically-driven persistence (*Br J Psychiatry* 1988; **153**: 460–469). Recent prospective neuropsychological and bio-social studies of mild head injury, and perspectives from cognitive behavioural and health psychology, are reviewed. It is argued that the organic–psychogenic conceptualization inadequately explains chronic post-concussional symptoms. Psychosocial and cognitive–behavioural factors and the coping process may influence post-concussional symptoms over their entire time course, in particular the late phase.

A multifactorial model of chronic post-concussional symptoms is proposed which integrates biological processes with these factors. It is through the recognition and identification of separate processes that questions about outcome, the limits of the impact of organic and psychosocial factors, the nature of exaggeration, and appropriate therapy, may be resolved.

Keywords: Head injury; Post concussional syndrome; Medico-legal; Malingering; Neuropsychology; Health behaviour

INTRODUCTION

The post-concussional syndrome refers to the emergence and variable persistence of a group of symptoms following head injury, particularly mild head injury. Most descriptions include somatic symptoms (headache, dizziness, fatiguability) accompanied by psychological symptoms both cognitive (poor memory and concentration) and affective (irritability, emotional lability, depression and anxiety). A third to a half of patients experience the syndrome over the first few weeks after mild head injury [1–4]. Whilst most become asymptomatic in the ensuing months, a substantial minority experiences persistent symptoms 6 months to a year later [4, 5].

The simultaneous occurrence of physical and emotional trauma means that conventional models of the post-concussional syndrome tend to be dichotomous with symptoms viewed as of physiological [1] or psychological aetiology [6]. This division of opinion is most polarised after mild head injury, the focus of this review. The aim of this paper is to provide a framework within which current research can be understood.

Lishman's [1] careful review of the literature up to 1987 concluded that physiological factors contributed mainly to the onset of the post-concussional syndrome, while

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psychological factors contributed more to its long-term course. Recent research, in particular the Belfast studies [4,7,8] and perspectives from cognitive–behavioural and health psychology will be reviewed to examine this model. It is argued that both in terms of the genesis and maintenance of post-concussional symptoms, the traditional distinction between physiological disorder and psychological disturbance, is outmoded, and that:

(1) psychosocial and cognitive–behavioural factors (including social support and stresses, personal resources and the coping process) may influence the entire spectrum of the post-concussional syndrome, including its onset, but more often its course and severity, to an extent which varies between patients;
(2) organic factors, whilst important in the genesis of post-concussional symptoms, may also influence their persistence [7];
(3) psychophysiological and cognitive processes mediate the effects of stress on the patient at all stages [9].

It is suggested that current multifactorial concepts of chronic pain which emphasize the integration of biomedical with cognitive, affective and behavioural factors [10], may provide a helpful model for post-concussional symptoms. The problem of individual differences in susceptibility or persistence of post-concussional symptoms is a central concern, but can be best discussed when the various aetiological factors have been reviewed.

POST-CONCUSSIONAL SYMPTOMS AFTER MILD HEAD INJURY

The terms 'minor' or 'mild' are used to define a head injury in which the period of unconsciousness is short (under 15 min [11]) or that of post-traumatic amnesia (PTA) is under 1 hr [12], there is no skull fracture or known intracranial mass lesion, and the Glasgow Coma Scale (GCS) scores are 13 or more at admission. Severity of head injury is not defined in terms of outcome [13].

Although the post-concussional syndrome is contentious, symptoms show remarkable uniformity across most series worldwide. Early post-concussional symptoms include headache and dizziness, and vomiting, nausea, drowsiness and blurred vision, which are short-lived. Symptoms evident after 1–3 months include headache (44%), dizziness (28%), fatigue (26%), anxiety (22%), insomnia (21%), noise sensitivity (17%), poor concentration (15%) and memory (10%), irritability (12%), depression (6%), light sensitivity, alcohol intolerance, or any of these (52%)—figures pooled from four series [1, 3].

Controversy persists concerning the chronicity of these subjective symptoms after mild head injury, with considerable variation reported in their prevalence at various times after injury. At 3 months the point prevalence varies between 24 [2] and 84% [14], at 6 months is under 30% and at 1 year under 20% (Table I).

Studies vary considerably in terms of measures of head injury severity such as length of post-traumatic amnesia (PTA), and how it is assessed, incidence of skull fractures, and Glasgow Coma Scale (GCS) scores [15, 16]. Studies also differ widely in their use of controls; inclusion of patients with past head injury, psychiatric history or substance abuse [14]; timing of assessment; method of eliciting symptoms; presence of compensation claims; and attrition rate on follow-up.