British Journal of Psychiatry (1988), **153**, 141–146

Annotation

Psychosocial Sequelae of Head Injury – Anatomy of a Relationship*

R. J. McCLELLAND

The nature and extent of psychological and social disability following head injury remain issues of considerable practical and theoretical interest. Practical because of the size of the problem and the resulting burden of care; theoretical because head injury should provide a powerful model for an improved understanding of the relationships between cerebral disorder and psychological impairment. (Lishman, 1973)

Head injury has been described as the 'silent epidemic' of our time, attributed in major part to the fast pace of modern lifestyle and compounded by the advances in technology which increase the probability of survival following injury (Adamovich *et al*, 1984). It is a major health problem for young adults and accounts for approximately 15% of all deaths among young adults in the UK (Jennett & MacMillan, 1981). Similar findings from the United States indicate that head injury is the major cause of death in adults under 35 years of age (Adamovich *et al*, 1984). Probably the most reliable guide to incidence is attendance rates at accident and emergency departments. In Scotland, 18 attendances for every 1000 of the population each year result from head injury (Strong *et al*, 1978).

The incidence rates belie the enormity of the problem, since the survivors from severe closed head injury are predominantly adolescents and young adults with relatively normal life expectancies and constitute a major burden on families and on services. While psychosocial adjustment is only one aspect of outcome, quality of life becomes dominated increasingly by such issues as time since the injury increases.

Pathophysiology

In times of peace, head injuries are predominantly 'closed', and hence the main focus of interest is less on the 'head injury' than on brain injury. The major aetiological factors are road traffic accidents and assault. In a recent Scottish study, these two accounted for over half of all attendances of young adults at accident and emergency units (Strong *et al*, 1978). Less frequent causes are sporting injury, especially boxing and horse-riding, industrial injury, and perinatal trauma. The late psychological and social sequelae do not develop in a vacuum, but are the end result of a series of processes in which physiological impairment and structural damage to the brain are key elements. An understanding of these early processes is crucial to an understanding of the severity and diversity of psychological deficits that may follow (Fig. 1).

In closed head injury there is often widespread damage. Axonal lesions, particularly in the brain stem and cerebral hemispheres, result from shearing and rotational forces causing tearing of the axons (Strich, 1956). Grey matter damage, especially within the frontal and temporal lobes, results from compression and tearing by dural edges and hard bony prominences (Ommaya *et al*, 1971; Graham *et al*, 1978; Adams *et al*, 1982). Several haemodynamic changes may result in further damage. Intracerebral haemorrhage and oedema may cause severe local damage and a secondary rise in intracranial pressure. Ischaemic damage from impaired perfusion may arise in the territory of a major intracerebral vessel or in the boundary zone between two vessels. Impaired cerebral perfusion may arise from three interactive processes: reduced blood pressure from associated peripheral bleeding or chest injuries; cerebral oedema and raised intracranial pressure; and impairment of the central autonomic control of cerebral perfusion, the result of brain stem injury (Crockard, 1982).

Late complications may add further serious damage (chiefly subdural haematoma, hydrocephalus, and epilepsy). Epilepsy is much more common following an open head injury, particularly where infection has occurred. The risk of recurrent seizures is greatest in the period immediately after head injury, and is a relatively uncommon development by one year after the injury (Jennett, 1975).

The diversity and severity of residual impairments is a balance between the foregoing trauma events and complications on the one hand and reparative

*First Roche Lecture, Annual Meeting of Irish Division of Royal College of Psychiatrists, October 1986.

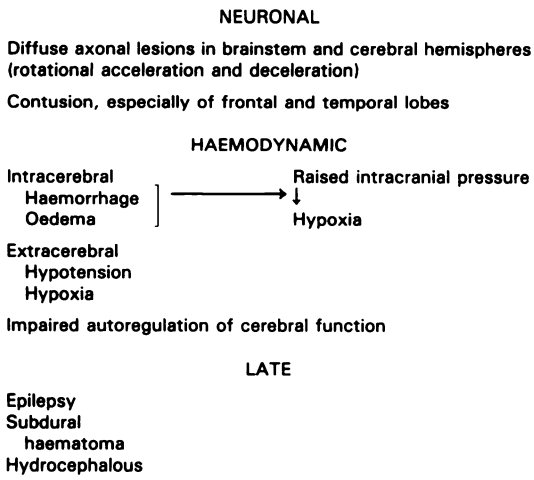

FIG. 1.

processes on the other. Neuronal repair and recovery is generally more successful in younger people. The plasticity of the nervous system and the capacity of residual intact brain tissue to subsume the functions of damaged tissue make significant contributions to good functional recovery, particularly in very young children.

Measures of severity and recovery

In order to understand better the natural history of head injury and what contributions have been made by different therapeutic interventions, reliable estimates of severity are essential. The two most useful clinical measures to date are depth of coma and duration of post-traumatic amnesia. The Glasgow Coma Scale, based on a standardised cumulative score of eye-opening response, motor responsiveness, and verbal responsiveness, has considerably reduced the amount of semantic confusion and subjectivity which surrounds the assessment of unconsciousness (Jennett, 1976). The measurement of post-traumatic amnesia (PTA) introduced by Russell & Nathan (1946) is a retrospective assessment of the period from time of injury to full awareness and the ability to retain a stable record of events. It therefore consists of the duration of coma and anterograde amnesia. PTA following closed head injury, ranging from minutes to several months, is generally considered to be the best available behavioural measure of severity.

However, problems still exist in making a precise estimate of severity. The wide variety of injuries do not produce homogeneous damage to the brain, and some measures of severity such as depth of coma depend more on the functional integrity of specific neural substrates than others. For example, closed head injury with resultant major damage to frontal lobe structures may not be associated with major damage to the brain stem reticular formation, and therefore may not be reflected in either deep or prolonged coma.

Clinical measures of severity have been successfully complemented by a variety of laboratory measures. While imaging techniques are especially valuable for assessing penetrating injuries, measures of cerebral function are more sensitive to the wide variety of subtle neural impairments. The responsiveness of the ongoing EEG to brain injury is dependent on many variables, particularly level of consciousness and the nature of the trauma. The most severe post-traumatic EEG disturbance is electrocerebral silence. The prognosis following such silence is usually poor, particularly where silence is prolonged. Slowing of the EEG is one of the most common findings in head injury and the degree of slowing is generally correlated with the severity of trauma (Stockard & Bickford, 1975). An alternative to measures of spontaneous EEG are measures based on evoked potentials, in which detection of the resulting neural impulses as they traverse the central neural pathways provides a measure of conduction velocity in the CNS. Central conduction velocity is extremely sensitive to trauma, and several recent studies have shown the value of both somatosensory and auditory evoked potentials in the early evaluation of closed head injury. Narayan *et al* (1981), in a study of head injury, compared evoked potentials with CT scanning, intracranial pressure monitoring and clinical examination. Information from evoked potentials provided the most accurate single prognostic indicator. In a follow-up study, Hume & Cant (1981) observed exponential recoveries in central conduction time over many months, the degree of recovery correlating with clinical improvement recovery. Tsubokawa *et al* (1980), in a recent study of patients with severe head injury (Glasgow Coma Scale 7), showed that the auditory brain stem potentials recorded immediately after admission provided more reliable information about brain stem functioning than either neurological signs or CT findings. In our own head injury studies we have found that the brain stem auditory evoked potentials are extremely sensitive to the occurrence of a mild closed head injury (Montgomery *et al*, 1984; McClelland, 1985).

To understand the relationship between head injury and outcome it is also important to provide measures of outcome itself, embracing the nature and severity of the impairments, the disabilities, and the handicaps. The Glasgow Outcome Scale (GOS), with

five levels of disability, has provided a simple and reproducible method of assessing outcome (Jennett & Bond, 1975). Although this has been refined to allow more subtle classification, the GOS provides only a global assessment of each patient's level of dependency. Livingston & Livingston (1985) have recently developed a new scale which allows a more detailed psychosocial evaluation, the Glasgow Assessment Scale. The separate subscales cover different aspects of the patient's life, enabling areas of special difficulty to be identified. In its present form the Glasgow Assessment Scale has good interrater reliability and concurrent validity with the global ratings from the GOS.

Psychological sequelae

Physical impairments such as spasticity or isolated cranial nerve lesions usually improve over a period of months and seldom result in significant handicap. By contrast, mental impairments, embracing personality change, chronic affective disturbance, intellectual deterioration, memory impairment, and impaired concentration, are frequently enduring and contribute most to chronic disability and handicap (Jennett & Bond, 1975). Mental and physical impairments often coexist, compounding total disability. The impact of disability on the family must also be considered, and mental impairments are once again the most stressful and disruptive (Brooks, 1978; Oddy *et al*, 1978). As Lishman (1973) has commented, it is most unlikely that the large range of mental and physical impairments which occur after a head injury share a common aetiology, even though all appear to have taken origin from a common event.

Following a severe closed head injury, intellectual impairment and memory impairment are found in a substantial proportion, ranging from 10% to 50% of patients in different studies (Miller & Sterne, 1965; Fahy *et al*, 1967). There is general agreement that the more severe the initial closed head injury, the more severe are the final cognitive impairments. In a two-year follow-up study of patients with severe head injury, characterised by PTA of greater than 7 days, Brooks (1972) reported marked and often prolonged deficits, especially in performance IQ. In subsequent studies, patients with PTAs greater than 4 weeks had severe IQ deficits. Impaired memory performance and general slowing following severe closed head injury are among the commonest neuropsychological sequelae (Brooks, 1972).

However, several studies have shown that the magnitude of the correlation between PTA and outcome is in fact quite small, with significant correlations confined to the first few months after injury (Lishman, 1968; Mandleberg, 1976). More detailed follow-up studies of IQ have shown that positive correlations between severity of impairment and duration of PTA are confined to some subtests only (Brooks, 1976; Gronwall & Wrightson, 1981). Severity of coma has been found to be a poor predictor of outcome. Any relation between Glasgow Outcome Score and cognitive impairment is confined to the first few months after injury (Brooks *et al*, 1986). Thus the relationship between more detailed quantitative measures of cognition and the present coarse measures of severity is relatively weak (Newcome & Fortuny, 1979).

Other factors besides the severity of the injury affect outcome. Brain damage is rarely homogeneous, and localised lesions can have very different effects. Damage of the left hemispheres gives rise to more severe deficits than corresponding damage of the right (Lishman, 1968). Time factors are important, and post-traumatic impairments show substantial improvements over a period of months. Neuronal repair and recovery is the dominant factor over the early post-recovery period, whereas reacquisition of lost functions by intact neural networks, improvement in motivation and adjustment account for significant improvements over the late post-injury period (Lishman, 1973).

Personality change in the form of altered and idiosyncratic reactions to persons and situations is undoubtedly one of the most distressing problems for patients, for their families and for those involved in continuing care and rehabilitation services. In one five-year follow-up study of severe head injury, most patients were found to have evidence of severe personality change (Brooks *et al*, 1986). There is substantial evidence for a neurological basis for such changes, with damage to the frontal and temporal lobes being major contributory factors (Table I). The frontal lobe syndrome, characterised by disinhibition, euphoria, lack of tact, and childishness, is now well recognised as a late sequel to frontal lobe damage. Following injury to the temporal lobe, aspects of the frontal lobe syndrome may be observed, together with irritability and increased aggressiveness. Damage to central and basal frontal areas is typically accompanied by loss of spontaneity and lack of vitality. Accompanying this wide range of personality changes there is usually some loss of insight and awareness.

Depression, anxiety, irritability, and obsessional traits are frequently observed following head injury of all types. They can best be regarded as part of an emotional reaction and are among the commonest psychological sequelae of head injury (Lishman, 1973). While there is a commonly held view of a simple disassociation between severity of brain damage and neurotic reaction, patients with severe organic damage frequently exhibit neurotic symptoms.

TABLE I

Frontal syndrome
Disinhibition
Euphoria
Blunting
Temporal syndrome
Aggressiveness
Basal syndrome
Spontaneity
Loss of vitality
Other
Anxiety
Depression
Tension
Fatigue
Irritability
Obsessional traits
Hypochondriasis

Such symptoms may, however, be more difficult to disentangle from the other features of severe head injury where the more florid aspects of behavioural and personality change dominate the clinical picture. The importance of recognising such neurotic symptoms resides in the need for a full understanding of the contribution made to head injury sequelae by psychological as well as organic factors. In the clinical evaluation of individual patients, the contributions of premorbid personality vulnerability and the stressfulness and the meaning of the head injury event all need to be carefully evaluated. Consideration also needs to be given to the full social context of the event and its consequences. The high incidence of depressive and anxiety states following spinal cord injuries and burn injuries clearly demonstrates the importance of non-organic factors in symptom formation (Noyes *et al*, 1979; Fullerton *et al*, 1981).

No discussion of head injury would be complete without reference to the psychiatric sequelae of minor head injury. As part of the continuum, minor head injury is usually defined as injury in which consciousness is lost only briefly, if at all, or in which post-traumatic amnesia is brief. In most studies the boundary between minor and major injury is a PTA of 24 hours. Minor head injury generates the greatest controversies regarding the chronicity of symptoms and the relevance of psychological and physical factors in their causation.

A wide range of symptoms are frequently observed following minor head injury, the more common physical symptoms being headache, dizziness, hearing difficulty, tinnitus, and fatigue. Psychological symptoms include impaired concentration, memory difficulty, irritability, anxiety and depression (Rutherford *et al*, 1977; Wrightson & Gronwall, 1981). Patients usually complain of several symptoms in the period immediately after head injury, and as many as half still have symptoms six weeks after injury (McClelland, 1985). Evidence for neural impairment in patients with mild and minor head injury is improved by neurophysiological, blood-flow and post-mortem studies. Symptoms in the early post-traumatic period can reasonably be attributed to the direct effects of injury on brain function (Taylor & Bell, 1966; Oppenheimer, 1968; Lewin, 1970; Montgomery *et al*, 1984).

Greater controversy surrounds the persistence of symptoms and the relation between chronic symptoms and severity of injury. In some patients it is possible that subtle neurological changes persist, leading to chronic residual symptoms. In support of a chronic neural deficit is the evidence provided by the cumulative effects of repeated minor head injuries such as occur in boxers (Roberts, 1969). A polar opposite view was given by Miller (1961), who argued that most of the chronic symptoms following minor head injury can be considered 'accident neurosis' and 'invariably resolve' once compensation is obtained. However, Kelly (1975) and others have presented strong evidence against this view, showing that a majority of patients with symptoms return to work before compensation is settled. Similar symptoms are common in patients not receiving compensation. Nevertheless, it seems likely that psychological mechanisms are important in the development of chronic symptomatology. Pilowsky (1985) has demonstrated that accidents are often more traumatic psychologically and emotionally than appears at first sight, and it is paradoxical that the accident experience is often least discussed and worked through by those responsible for patient management.

The balance of organic and psychological factors in the aetiology of chronic head injury sequelae in individual cases can only be established by a careful analysis of the facts. The persistence of several symptoms from the time of injury, with coexistent neurophysiological dysfunction, is strong evidence in favour of a physical basis for symptoms. The absence of neurophysiological impairment and the late development of new symptoms point to a likely psychological origin.

Social impairment and handicap

The full impact of brain injury and resulting impairments is manifested in social adjustment. Livingston (1986), in a study of patients with severe closed head injury, reported high levels of dependency. Almost half were considered by relatives incapable of being left in charge of the home and over 20% required someone to look after them. The studies of both Oddy *et al* (1978) and Thomsen (1974)

highlight the social isolation and social dislocation resulting from severe closed head injury.

While the majority of patients with head injury ultimately return to work, this depends greatly on the severity of the injury. Many patients with prolonged periods of PTA or unconsciousness fail to return to work (Lewin, 1970; Lecuire *et al*, 1971; Oddy *et al*, 1978). In addition, those who do return frequently have to take jobs with reduced responsibility and reduced satisfaction.

Factors other than the severity influence return to work. Successful return is more common in younger people. Specific impairments which frequently present major obstacles in rehabilitation include epilepsy, speech disorders, memory and intellectual failure, loss of initiative, labile mood, depression and irritability (Fahy *et al*, 1967; Lecuire *et al*, 1971; Roberts, 1976).

Another dimension of social outcome is family stress and burden. From the study by Brooks *et al* (1986) of patients with severe head injuries, a high burden of care was evident in the great majority. In the same series, Livingston (1986) reported high scores on the General Health Questionnaire, with many families complaining of being stressed. Health professionals must remember that they see the patient from a very different starting point to that of relatives and family. Good physical recovery after a prolonged period of unconsciousness may seem a very satisfactory outcome to the neurosurgical team, but the subtle and sometimes severe personality and behavioural changes result in a very different and often very difficult person being returned to the family. The personality changes which follow severe head injury and contribute most to the burden of care include loss of emotional control, irritability, reduced initiative, oddness and childishness (Fahy *et al*, 1967; Bond, 1975; Brooks & McKinlay, 1983; Brooks *et al*, 1986). Difficulties in interpersonal relationships are much more frequent following head injuries than other injuries, and high rates of marital breakdown have been reported (Thomsen, 1974; Rosenbaum & Najenson, 1976). There are often major life and role changes for spouses, and many report loneliness and isolation in addition to the relationship difficulties.

In evaluating the social distress for families, account must be taken of premorbid personal, interpersonal and marital adjustment. Several studies have demonstrated a higher than expected prevalence of pre-injury alcohol problems, violence and antisocial behaviour, and a higher rate of domestic and industrial accidents (Galasko & Edwards, 1974).

A third factor which will influence the family burden is service provision. There are major deficiencies of service provision in the face of great need for additional support (Panting & Merry, 1972; Thomsen, 1974; Oddy *et al*, 1978; Livingston, 1986).

Rehabilitation

Newson-Smith (1983) has pointed to the lack of services, lack of community and hospital provision and lack of clear planning for the young brain-damaged adult. Livingston (1986) has also stressed the priority needs for services for the families of the brain-damaged individual. The low uptake of existing services has been attributed to severe lack of facilities and lack of co-ordination of care. Present rehabilitation services focus on the disabilities resulting from physical impairments, while the growing weight of evidence points to the great burden of care arising from persistent psychological and behavioural problems. Patients frequently have multiple problems, leading to several specialisms being involved, usually with lack of co-ordination. Patients and relatives are frequently unable to cope with divergent views and approaches, resulting in defaulting at clinics.

As with any service or treatment programme, questions that might appropriately be asked are as follows: what types of client benefit most? what are the treatment methods? who can provide such treatment? how long should treatment last? what is the efficacy of present methods? At this time, however, only very partial answers to such questions are available.

Eames & Wood (1985) have shown the benefits of intensive behaviour modification programmes in managing difficult behaviour problems in adults with severe head injuries. Significant improvements were obtained, particularly in social acceptance and independence, and were sustained one year after completion of the treatment programme. To be effective, behaviour and rehabilitation programmes usually need to continue for at least 18 months, and in some cases several years. There is also a need for cognitive retraining and psychotherapy.

A team approach is essential for the rehabilitation of patients suffering from head injury, particularly for managing the late, predominantly psychosocial, sequelae. Psychiatrists have most of the training and clinical skills necessary to lead such a multidisciplinary team. The support and skills of neuropsychology, occupational therapy and social work are essential for an effective rehabilitation service. The family play a key part in rehabilitation and in most situations are the main providers for the injured individual. As the family require a great deal of support, their co-operation and involvement in any rehabilitation programme should be sought from the outset. From personal experience, the psychosocial rehabilitation of the head-injured patient and the care of his family require the skills of a specialist team. Specialist services, embracing long-term rehabilitation and continuing care facilities, are required in most health regions for this cinderella area of mental health.

References

ADAMOVICH, B. B., HENDERSON, J. A. & AUERBACH, S. (1984) *Cognitive Rehabilitation of Closed Head Injury Patients*. San Diego: College Hill Press.

ADAMS, J., GRAHAM, D., MURRAY, L. S. & SCOTT, G. (1982) Diffuse axonal injury due to non-missile injury in humans. *Annals of Neurology*, **12**, 557–563.

BOND, M. R. (1975) Assessment of the psychosocial outcome after severe head-injury. In *Outcome of Severe Damage to the Central Nervous System* (pp 141–157). Ciba Foundation Symposium No 34. Amsterdam: Elsevier – Excerpta Medica.

BROOKS, D. N. (1972) Memory and head injury. *Journal of Nervous and Mental Disease*, **155**, 350–355.

—— (1976) Wechsler memory scale performance and its relation to brain damage after severe closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 593–601.

—— (1978) Psychological sequelae of head injury. *Injury*, **10**, 74.

—— & MCKINLAY, W. (1983) Personality and behavioural change after severe blunt head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **46**, 336–344.

——, CAMPSIE, L., SYMINGTON, C., BEATTIE, A. & MCKINLEY, W. (1986) The five year outcome of severe blunt head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **49**, 764–770.

CROCKARD, A. (1982) Early management of head injuries. *British Journal of Hospital Medicine*, **27**, 635–644.

EAMES, P. & WOOD, R. (1985) Rehabilitation after severe brain-injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **48**, 613–619.

FAHY, T., IRVING, M. & MILLAR, P. (1967) Severe head injuries. *The Lancet*, *ii*, 475, 479.

FULLERTON, D., HARVEY, R., KLEIN, M. & HOWELL, T. (1981) Psychiatric disorders in patients with spinal cord injuries. *Archives of General Psychiatry*, **38**, 1369–1371.

GALASKO, C. S. B. & EDWARDS, D. H. (1974) The causes of injuries requiring admission to hospital in the 1970s. *Injury*, **6**, 107–112.

GRAHAM, D. I., ADAMS, J. H. & DOYLE, D. (1978) Ischaemic brain damage in fatal non-missile head injuries. *Journal of Neurological Science*, **39**, 213–234.

GRONWALL, D. & WRIGHTSON, P. (1981) Memory and information processing capacity after closed head-injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **44**, 889–895.

HUME, A. L. & CANT, B. R. (1981) Central somosensory conduction after head injury. *Annals of Neurology*, **10**, 411–419.

JENNETT, B. (1975) *Epilepsy After Non-Missile Head Injuries*. London: Heineman.

—— (1976) Assessment of severity of head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 647–655.

—— & BOND, M. R. (1975) Assessment of outcome after severe brain damage. *The Lancet*, *i*, 480–484.

—— & MACMILLAN, R. (1981) Epidemiology of head injury. *British Medical Journal*, *i*, 101–104.

KELLY, R. (1975) The post-traumatic syndrome. *Forensic Science*, **6**, 17–24.

LECUIRE, J., DECHAUME, J. P. & DERUTY, R. C. (1971) Long-term progress of the prolonged and serious traumatic comas. In *Head-injury: Proceeding of an International Symposium* (pp 161–162). Edinburgh: Churchill Livingstone.

LEWIN, W. (1970) Rehabilitation needs of the brain-injured patient. *Proceedings of the Royal Society of Medicine*, **63**, 8–10.

LISHMAN, W. A. (1968) Brain damage in relation to Psychiatric disability after head-injury. *British Journal of Psychiatry*, **114**, 373–410.

—— (1973) The psychiatric sequelae of head-injury: a review. *Psychological Medicine*, **3**, 304–318.

LIVINGSTON, M. G. (1986) Assessment of need for co-ordinated approach in families with victims of head injury. *British Medical Journal*, **293**, 742–744.

—— & LIVINGSTON, H. M. (1985) The Glasgow Assessment Schedule. *International Rehabilitation Medicine*, **7**, 145–149.

MENDLEBERG, I. (1976) Cognitive recovery after severe head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **39**, 1001–1007.

MCCLELLAND, R. J. (1985) A neurological investigation of minor head injury. In *Clinical and Experimental Neurophysiology* (eds D. Papakosostopoulos, S. Butler & I. Martin). London: Croom Helm.

MILLER, H. C. (1961) Accident neurosis. *British Medical Journal*, *i*, 919–925, 992–998.

—— & STERN, G. (1965) The long-term prognosis of severe head injury. *The Lancet*, *i*, 225–229.

MONTGOMERY, A., FENTON, G. W. & MCCLELLAND, R. J. (1984) Delayed brainstem conduction time in post-concussional syndrome. *The Lancet*, *i*, 1011.

NARAYAN, R., GREENBERG, R. & MILLAR, J. (1981) Improved confidence of outcome prediction in severe head injury. *Journal of Neurosurgery*, **54**, 751–762.

NEWCOME, F. & FORTUNY, L. (1979) Problems and perspectives in the evaluation of psychological deficits after cerebral lesions. *International Rehabilitation Medicine*, **1**, 182–192.

NEWSON-SMITH, J. (1983) Who cares for the adult brain damaged? *Bulletin of the Royal College of Psychiatrists*, **7**, 181–183.

NOYES, R., FRYE, S. J. & SLYMEN, B. J. (1979) Stressful life events and burn injuries. *Journal of Trauma*, **19**, 141–144.

ODDY, M., HUMPHREY, M. & UTTLEY, D. (1978) Subjective impairment and social recovery after closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **41**, 611–616.

OMMAYA, A. K., GRUBB, R. C. & NAUMANN, R. A. (1971) Coup and contracoup injury. *Journal of Neurosurgery*, **35**, 503–516.

OPPENHEIMER, D. R. (1968) Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **31**, 299–306.

PANTING, A. & MERRY, P. (1972) Long-term rehabilitation of severe head injuries. *Rehabilitation*, **38**, 33–37.

PILOWSKY, I. (1985) Cryptotrauma and accident neurosis. *British Journal of Psychiatry*, **147**, 310–311.

ROBERTS, A. H. (1969) *Braindamage in Boxers*. London: Pitman.

—— (1976) Long-term prognosis of severe accidental head-injury. *Proceedings of the Royal Society of Medicine*, **69**, 137–141.

ROSENBAUM, M. & NAJENSON, T. (1976) Changes in life patterns and symptoms of low mood as reported by wives of severely brain-injured soldiers. *Journal of Consulting and Clinical Psychology*, **44**, 881–888.

RUSSELL, W. & NATHAN, P. (1946) Traumatic amnesia. *Brain*, **69**, 280–300.

RUTHERFORD, W. H., MERRETT, J. D. & MCDONALD, J. R. (1977) Sequelae of concussion caused by minor head injuries. *The Lancet*, *i*, 1–4.

STOCKARD, J. & BICKFORD, R. (1975) The electroencephalogram in traumatic brain injury. In *Injuries of the brain and skull, part I* (eds P. Vinken & G. Bruyn). *Handbook of Clinical Neurology*, **23**, 317–367.

STRICH, S. C. (1956) Diffuse degeneration of the cerebral white matter in severe dementia following head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **19**, 163–185.

STRONG, I., MACMILLAN, R. & JENNETT, B. (1978) Head injuries in accident and emergency departments at Scottish hospitals. *Injury*, **10**, 154–159.

TAYLOR, A. R. & BELL, T. I. C. (1966) Slowing of the cerebral circulation after concussional injury. *The Lancet*, *ii*, 178–180.

THOMSEN, I. (1974) The patient with severe head injury and his family. *Scandinavian Journal of Rehabilitation Medicine*, 6180–6183.

TSUBOKAWA, T., NISHIMOTO, H. & YAMAMOTO, T. (1980) Assessment of brainstem damage by the auditory brainstem response in acute head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, **43**, 1005–1011.

WRIGHTSON, P. & GRONWALL, D. (1981) Time off work and symptoms after minor head injury. *Injury*, **12**, 445–454.

R. J. McClelland, MD, PhD, FRCPsych, DIC, *Professor of Mental Health, The Queen's University of Belfast, Department of Mental Health, The Whitla Medical Building, 97 Lisburn Road, Belfast, Northern Ireland BT9 7BL*