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Review Article

Physiogenesis and Psychogenesis in the 'Post-Concussional Syndrome'

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The aetiological factors relevant to the development of post-concussional symptoms are reviewed. From the numerous studies carried out to date, it would appear that both physiogenic and psychogenic influences are important in their genesis. However, where mild-to-moderate injuries are concerned, organic factors are chiefly relevant in the earlier stages, whereas long-continued symptoms are perpetuated by secondary neurotic developments, often of a complex nature.

Sir Aubrey Lewis (1942), referring to the post-concussional syndrome, described it as "that common, dubious, psychopathic condition – the bugbear of the clear-minded doctor and lawyer". In this forceful declaration, he encapsulated the strong divisions of opinion that have surrounded the nosological status, and especially the genesis, of the syndrome since before the turn of the century. Such uncertainties persist today.

Central to most descriptions are headache and dizziness, but to these may be added abnormal fatiguability, insomnia, sensitivity to noise, irritability, and emotional instability. Anxiety and depression are often prominent. Difficulties with concentration and memory may feature strongly among the complaints, and some degree of overt intellectual impairment may on occasion be detected. With this mixture of quasi-organic and subjective symptoms, variously reported, it is scarcely surprising that the concept lacks clarity and that its aetiology has remained in doubt. Nevertheless its ubiquity following even minor blows to the head, and the regularity with which it features among claims for compensation, have ensured that it persists as an important subject for medical interest and debate.

A striking feature in the literature is the disparity between those who argue for physiological or for psychological causation. Some see it as founded in subtle cerebral pathology, while others adduce evidence that its roots may lie in conflict and anxiety. Psychiatric disability following head injury is often ripe for such divisions of opinion, since undoubtedly the injury imparts at a blow both physical and emotional trauma. Before focusing on the 'post-concussional syndrome' ('post-traumatic syndrome') directly, some of the principal aetiological forces at work when the head has been injured are reviewed. They include organic, psychological, social, even cultural factors, all of which deserve close consideration.

TABLE I
Factors relevant to psychiatric disability

1. Pre-traumatic
Age
Cerebral arteriosclerosis
Alcoholism
Mental constitution
Genetic vulnerability
Previous psychiatric illness
Personality (including being prone to accidents)
Pre-existing psychosocial difficulties
Domestic
Financial
Occupational
Recent life events
2. Peri-traumatic
Brain damage
Transient (contusion, oedema, hypoxia, raised intracranial pressure, circulation)
Permanent (amount, location)
Other physical damage (skull, scalp, vestibular apparatus)
Emotional impact and meaning
Fear of accident
Fear of early symptoms
Circumstances of accident
Setting
Significance
Type (road traffic accident, industrial, domestic, sport)
Iatrogenic (early information, management, investigations)
3. Post-traumatic
Intellectual impairment
Other impairments (physical disabilities, deformity, scars)
Epilepsy
Emotional repercussions of accident (including *depression*)
Ensuing psychosocial difficulties
Domestic
Financial
Occupational
Compensation and litigation

Pre-traumatic factors

First, there are aspects already present before the injury occurred (Table I). Age alone will be important, in more than one respect. The ageing brain is less resilient to organic insult, and the ageing person less adaptable to its effects. Among adults, rising age has proved to be associated with diminished chance of returning to work (Heiskanen & Sipponen, 1970), increased memory difficulty (Russell, 1932), an increased incidence of anxiety and fears (Adler, 1945), and increased mortality (Kerr *et al*, 1971). Studies of children have shown a different spectrum of disabilities compared with those occurring in adults, certainly with the classical 'post-concussional symptoms' figuring less frequently (Black *et al*, 1969).

Pre-existing problems such as arteriosclerosis or alcoholism will add further to the ageing factor. Remarkably little research attention appears to have been given to the influence of alcoholism despite its high prevalence in head-injured populations. Thus Waller (1968), investigating road deaths in California, found that 58% of drivers, 47% of passengers, and 36% of pedestrians had alcohol in the blood, most with levels exceeding the legal maximum for drivers. The effects of a chronic high alcohol intake in delaying reparative processes within the central nervous system are now appreciated, from laboratory experimental studies (West *et al*, 1982).

With regard to mental constitution, we must consider genetic propensity to neuroses, depression, and even the major psychoses. Such liability may be obvious when there is already a history of psychiatric illness. Both the physical and the psychological traumas of the injury may thus be operating on a particularly vulnerable person. The personality, or in Symonds' (1937) phrase the "kind of head that is injured", can be crucially important to the processes of rehabilitation.

> This was strikingly illustrated in the case of a youth who sustained minor but definite frontal-lobe damage. He had had a stormy adolescence with problems of addiction and irresponsible trends. The injury occurred when he was emerging from such difficulties and showing clear evidence of maturing towards a stable lifestyle. Following the injury he became grossly psychopathic. Here one could argue that more than most he had stood in need of intact frontal lobe functions. His premorbid constitution had had a powerful influence in shaping his response to the injury.

Pre-existing psychosocial difficulties are notoriously prone to be overlooked. The patient's domestic and financial stability, his occupational satisfactions and dissatisfactions, may powerfully determine his response to injury and to recovery therefrom. Moreover, we may see the 'scapegoat motive' in action, whereby the injury serves as a pivotal experience on which to pin the blame for pre-existing worries and unhappiness. This shifting of the blame may be seen to operate both in the patient's mind and sometimes in that of his attendants. Yet the pre-existing problems may quite often be more intrusive and directly relevant to his disability than any aspect of the injury itself.

It is also important to recognise that disturbing life events may be operating surreptitiously in an entirely similar manner. Selzer *et al* (1968) were able to show an excess of personal crises and conflicts in the lives of drivers causing fatal accidents during the months before these occurred. No less than 20% had had an acutely disturbing experience during the preceding 6 hours. Whitlock *et al* (1977) similarly found a significant excess of such items as moving house, marital separations, serious discord, or changes of work in the recent lives of victims of accidental injury. Thus, far from occurring in an emotional vacuum, accidents are prone to occur just when the person already has to cope with other sources of conflict and upheaval.

Peri-traumatic factors

All of the above occurs as the prelude. The accident then imparts both physical and psychological damage (Table I). The brain damage itself is partly transient, partly permanent, in part demonstrable and in part forever elusive. It is now essential, for example, to think in terms not only of structural disruption, but of changes in circulation, changes in brain metabolism, and perhaps far-reaching changes in neurotransmitter function. The old 'molecular disturbance of neuronal function', postulated by early writers (Oppenheim, 1889), has a fresh scientific respectability. For example, considering stroke, it may be noted that Robinson *et al* (1984) now suspect widespread noradrenergic brain dysfunction as playing an important role in the genesis of post-stroke depression.

With new techniques, moreover, there is more awareness of subclinical structural damage immediately after injury. Jenkins *et al* (1986), for example, scanned 50 patients with magnetic resonance imaging (MRI) during the first few days, showing twice the yield of lesions that could be detected with computerised axial tomography (CT). Cortical contusions could sometimes be identified when there had been no loss of consciousness. Deep white-matter

lesions (seen in 15 patients with MRI, compared with only 1 on CT) were sometimes present when concussion had last for only 5 min.

The more readily definable contribution of coarse brain lesions has been extensively researched. Both the severity of such damage and its principal locations within the brain are known to affect both cognitive and broader behavioural sequelae, as reviewed, for example, by Lishman (1973) and in detail by Newcombe (1983). Other physical damage, particularly to the vestibular apparatus, can dictate important symptoms without being immediately obvious.

Considering peri-traumatic factors of a psychological nature, the purely emotional shock can be immense, some accidents occurring in terrifying circumstances that remain alive in the patient's memory. The ordeal of coming close to death, the destruction of the 'myth of personal invulnerability', can itself dictate prolonged invalidity. This has been well documented in studies of persons surviving floods, fires, and other disasters that have not injured the head at all.

> A 50-year-old man remained severely disabled for six years following an accident in which his lorry had jack-knifed on the motorway, causing a pile-up of casualties. His degree of depression and withdrawal was such that he was considered by some observers to be demented. He had regressed to total dependence on his wife who had to wash and shave him. Yet close enquiries about the nature of the accident showed that he had not been rendered unconscious, and that the likelihood of any head trauma had been very slight indeed. He had responded catastrophically to the circumstances of the ordeal (Lishman, 1973).

Added to this, head injury may have a special meaning to the patient – he may fear the consequences of 'concussion', and worry about the long-term import of early symptoms. The post-traumatic amnesic gap sometimes serves a paradoxical protective function in saving the patient from awareness of the more disturbing details of his injury.

Not surprisingly, the circumstances of the accident can also be important. The setting may have been peculiarly conducive to fear, anger, or resentment. The circumstances may have special significance, as when a reckless driver has injured his family or a workman has been forced against his will to use faulty equipment. Exploring the details surrounding the accident can be rewarding in revealing such circumstances (Pilowsky, 1985). And even ignoring highly individual determinants of this nature, it is possible to show that disability tends to differ from one broad category of accident to another – whether on the roads, at work, or in the home (Brain, 1942; Adler, 1945; Miller, 1966).

Next, there is the iatrogenic factor. In the early post-injury phase, the patient is vulnerable and often highly suggestible to the treatment he or she receives and how much is explained to him or her.

> A policewoman of 22 sustained a minor blow on the head when travelling on duty in a car. Continuing headache led to skull X-ray which showed some increased convolutional markings. A CT scan was therefore undertaken in case a pre-existing hydrocephalus had been exacerbated. This was normal but the foramen magnum seemed enlarged. A myelogram was therefore in turn carried out to exclude any possibility of abnormality at the cranio-cervical junction and was also normal. Throughout the six weeks of investigation her headaches steadily worsened – and were still troublesome some three years later. Her attention had indeed been firmly focused on her early symptoms.

Post-traumatic factors

When the acute phase of the injury is over, the patient has to adjust to what has occurrred (Table I). Intellectual impairments may be marked in degree or of a very subtle nature, sometimes so subtle that they are easily overlooked. The "changed organism", in Goldstein's (1942, 1952) terms, may find it difficult to cope. Much will depend on how far the disability is overt and what allowances accordingly are made. Physical disability will have an impact of its own, with deformities or scars possibly having a disproportionate influence on the patient's mental state. Epilepsy can represent a serious added burden.

The emotional repercussions may be closely tied to the above or follow more directly. Depression is an ever-present risk. Guilt may be highlighted, or important losses sustained. Sometimes the repercussions will build as chains of causal sequences over a considerable period of time:

> A man of 30, for example, was injured by a falling ladder, which struck him behind the ear. A period of intense vertigo and vomiting was followed by postural instability. He became phobic of walking far or of travelling, lost his job, and took to working in a public house nearby. He was depressed and began drinking heavily.
>
> Examination 18 months later still showed evidence of labyrinthine damage and his phobias were easily demonstrable. By then, however, he had fallen into debt, and had quarrelled with his wife who had left him (Lishman, 1978).

In examples such as this we see the gradual build-up of ensuing psychosocial difficulties, which make their own clear contributions to the patient's mental state.

Interruption of education or threats to a career can likewise have profound consequences on the patient's life:

> A boy of 18 was injured just prior to taking up his place at art college. He had been an excellent student and had won the place against competition. Attempts to cope, however, led to repeated failures, quarrels within the family, and increasing loss of self-esteem. He took numerous jobs of a simple labouring nature, usually losing them quickly through boredom. He developed a pattern of absconding from home on drinking sprees and often came into conflict with the law. Four years after the injury he had made an excellent physical recovery but was still completely adrift.

Tarsh & Royston (1985) have documented the powerful influences of changes within the family structure in contributing to prolongation of disability – overprotection from family members resulting in childlike dependence, and upheavals in the normal family hierarchies and roles.

The patient often becomes deeply involved in processes of litigation. Litigation is not an easy path to follow; hopes are aroused, doubts engendered, and conflicting advice is received from lawyers and doctors. The whole process conspires towards a state of chronic conflict and often long-drawn-out frustration. The repeated rehearsal of symptoms before a variety of audiences, some encouraging, some sceptical, does not help the patient to be clear about what he is truly experiencing. When symptoms are of a predominantly subjective nature it is hardly surprising that he will often fail to keep them in perspective. There is now evidence that the so-called 'compensation neurosis' cannot be expected to resolve, in all cases, once the litigation is ended (Steadman & Graham, 1970; Kelly & Smith, 1981). Many patients, indeed, remain chronically disabled in the long-term.

The above influences generate, reinforce, or perpetuate, post-concussional psychiatric disability, including the post-concussional syndrome. Examination of such a number of contributory factors comprehensively is especially difficult with a syndrome containing several well-recognised components, all of which may not necessarily share the same determining forces. The syndrome, moreover, is not a static entity, but prone to change with time – over the days, weeks, even very many months succeeding the injury. Different factors may come into operation in relation to different components during rehabilitation and recovery.

TABLE II
Relative incidence of post-traumatic complaints

	Rutherford et al (1977)	*Lidvall et al (1974)*	*Keshavan et al (1981)*	*Total*
Follow-up period	*6 weeks*	*0–3 months*	*3 months*	
Total patients	*145*	*83*	*60*	*288*
Percentage showing				
Headache	25	58	47	39
Dizziness	15	47	30	27
Fatigue	9	37	37	23
Anxiety	19	23	28	22
Insomnia	15	NK	37	21
Sensitivity to noise	NK[1]	7	30	17
Difficulty with concentration	8	30	8	15
Irritability	9	13	17	12
Subjective memory impairment	8	16	8	10
Depression	6	NK	NK	6
Any of above	51	46	65	52

1. NK = not known.

The nature of the post-concussional syndrome

Table II shows the relative incidence of complaints in three series of head-injured patients, all carefully studied during the early weeks or months following injury. Most of the injuries had been of a relatively mild nature. Rutherford *et al*'s (1977) patients had been admitted overnight to an observation ward, and the symptoms were determined from a checklist administered at 6-weeks follow-up examination. Lidvall *et al*'s (1974) patients had been admitted to hospital with relatively mild injuries and co-operated fully by the second day; symptoms described at any time during the next 3 months were noted. Keshavan *et al*'s (1981) series consisted of patients admitted consecutively to an emergency service, and followed up at 3 months. In addition to the 10 main symptoms listed in the table, others that are not infrequently mentioned include sensitivity to light as well as to sound, decreased alcohol tolerance, and impairment of sexual interest and performance.

A close concordance for most symptoms is observed among these three series – headache heading the list, then dizziness, fatigue, anxiety, etc. To a striking extent, these symptoms are subjective, and not directly accessible to observers. A high

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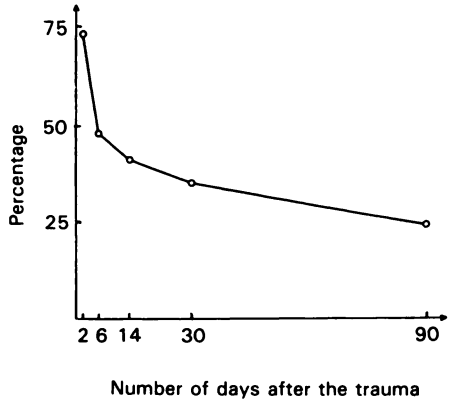

FIG. 1 Percentage of patients reporting one or more post-concussional symptoms on various occasions during the observation period (reprinted with permission from Lidvall *et al*, 1974).

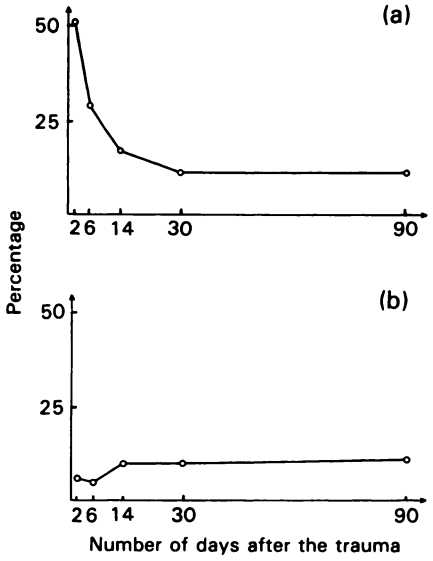

FIG. 2 Differing time courses of headache and anxiety, as reported on various occasions during the observation period. (a) headache; (b) anxiety (reprinted with permission from Lidvall *et al*, 1974).

proportion of patients are affected during this early period, barely half, even of those with quite mild injuries, having no symptoms.

Lidvall *et al*'s (1974) study has particular importance to consideration of the symptoms' time course. All patients suffering concussive head injury from the Stockholm area were included, provided they were able and willing to co-operate with questionnaires by the second day after injury. These were then followed up prospectively with careful documentation of symptoms at regular intervals until 3 months had elapsed. Eighty-three patients were studied in this fashion (Fig. 1). There was a marked and continuing decline in the percentage of patients displaying one or more post-concussional symptoms, from 73% at 2 days to 24% at 3 months, the fall being particularly marked during the first post-injury week. Thus, there is a fundamental problem in understanding the aetiology – different groups of patients will often be studied at different points along this course. Different symptoms, moreover, may follow different time gradients. Headache (and dizziness also), for example, tended to be gradually less frequently reported, the fall being quite precipitous after starting at 50% (Fig. 2, Lidvall *et al*, 1974). Anxiety, by contrast, tended to increase from the 6th to the 14th day (the week after the majority of the patients were discharged from hospital), and irritability followed a similar course. It may safely be concluded that different processes are likely to underlie such differing time courses.

Lidvall *et al* (1974) were also able to examine the clusters which the symptoms formed with one another, i.e. the basis on which syndromes are designated. Just after the injury, a large and almost exclusive cluster was formed from headache, dizziness, fatigue, and difficulty in concentration. At 2 weeks, this cluster persisted, but a second had appeared, consisting of difficulty with concentration and anxiety. At 1 month, anxiety became coupled with headache and fatigue. At 3 months, the prominent cluster consisted of anxiety and headache. It seems, therefore, that, at first, the syndrome is dominated by headache and dizziness, but that over quite a short period of time, it becomes more polymorphous. Anxiety becomes prominent in the later clusters.

In the longer term, such changes doubtless continue. Jones (1974) traced 80% of a large group of over 4000 patients discharged from an emergency department after minor head injuries not requiring admission, and tested the time course of their symptoms. Forty-two per cent had become asymptomatic within 3 weeks of their injuries; 57% continued with symptoms, mostly headache and dizziness, for at least 2 months. But at 1-year follow-up investigation, only 1% were still symptomatic and continuing to seek medical advice. Of these 36 patients, a few had had complications, but the great majority had not. Twenty were re-examined in hospital, and no physical causes for symptoms discovered.

It is therefore important to note that very different populations of patients with post-concussional symptoms are encountered, according to whether they are studied weeks, months, or years after the head injury has occurred. A study of Jones' (1974) patients at 3 weeks would have given vastly different results from any study conducted at 1 year – those still complaining would represent a tiny percentage only of those who would have been encountered earlier. Any causative factors unearthed could not be expected to apply equivalently.

Aetiological investigations

Some of the investigations undertaken are now reviewed, to see the ways in which the various aetiological factors have been explored. It will be apparent how markedly the studies differ on a range of significant parameters – the type of injury studied, its severity, the aetiological factors chosen for scrutiny, and certainly the time elapsed since injury. Ideally, all aetiological factors should be measured simultaneously and at repeated intervals during recovery, to gain a just appreciation of their relative values, and, most importantly, of how they might sometimes operate in conjunction with one another. No such large-scale survey seems to have been undertaken, but several smaller investigations are nonetheless informative.

1. Lidvall et al *(1974)*

A group of 83 head-injured patients were followed prospectively for 3 months, and the 38 who developed 'post-concussional symptoms' compared with those without. The females and the unskilled workers were particularly at risk. No relationship was found with length of post-traumatic amnesia or the presence of intellectual impairment, neither when the post-concussional group was considered as a whole, nor when any individual symptom was considered in isolation. Care was taken to measure early attitudes and stresses, with a series of questionnaires, and significant relationships emerged. Mental stress at work and in the home was commoner in the post-concussional group. From the earliest stages, these patients had had more anxiety about the accident, more worries about other ailments, and more fears that they had sustained serious and possibly permanent brain damage. All such factors suggested peri-traumatic or secondary psychogenesis. Nevertheless, an organic factor could be discerned – a strong relationship persisted between early evidence of otological dysfunction and the ultimate development of dizziness.

2. Rutherford et al *(1977)*

A consecutive group of 145 mild head injuries in Belfast were followed, and the symptoms experienced 4 and 6 weeks after the accidents charted. Seventy-four patients complained of one or more post-concussional symptoms. The number of symptoms encountered was significantly higher in females than males, and tended to rise, although non significantly, with age and with length of post-traumatic amnesia. A strong influence was seen in relation to type of accident – significantly more symptoms emerging when blame could be attached to an employer or a large impersonal organisation, than in patients who blamed themselves or 'Acts of God'. However, evidence of a possible organic factor was again found. When examination at 24 hours had disclosed headache, diplopia, anosmia, or other abnormality on neurological examination, the symptom rate proved to be significantly higher 6 weeks later. Evidence for both organic and non-organic components in the genesis of the symptoms was thus adduced.

3. Keshavan et al *(1981)*

An analogous investigation in India was reported, involving 60 consecutive head-injury admissions to an emergency service. The number of post-concussional symptoms experienced by patients at 3-months follow-up examination was noted. It may be significant that the injuries here were probably somewhat more severe, in that the symptom rate was found to correlate significantly with the duration of post-traumatic amnesia, and with the presence of intellectual deficits. But it was also strongly associated with scores on premorbid neuroticism, derived from relatives' accounts. By contrast it was striking to observe how clearly physical and social disability correlated with the organic factors measured (PTA and intellectual deficits), but not with the index of pre-traumatic neuroticism. Nevertheless, both organic and non-organic influences were again upheld in relation to the genesis of the post-concussional symptoms.

4. Kay et al *(1971)*

A retrospective case-note review of 474 patients admitted to a neurosurgical unit was carried out. Brain damaged ($n = 61$), 'post-concussional' ($n = 94$), and recovered groups ($n = 268$) could be discerned 3–6 months later. The remaining 51 patients had died. The post-concussional group had shown less severe injuries than the brain-damaged group, and indeed on a composite score of severity were

essentially indistinguishable from those who had made complete recoveries. Psychosocial aspects were the factors that seemed influential in distinguishing between the post-concussional and recovered patients – marital status, social class, type of accident (industrial, etc.), and any previous history of psychiatric illness. However, an organic contribution could still be pinpointed – visual symptoms (diplopia and blurring) and anosmia, persisting after the acute stage had passed, were commoner in the post-concussional than in the recovered group.

5. Lishman (1968)

The penetrating head injuries from World War II that were analysed illustrate yet another approach. These were evaluated retrospectively and in a much less discriminating manner. No attempt was made to evaluate psychosocial factors, but symptoms were viewed in relation to several indices of brain damage. Of 670 soldiers with penetrating injuries, 144 showed marked psychiatric disability 1–5 years later. In 71 of these, there were persisting complaints of headache, dizziness, fatigue, or sensitivity to noise. This 'somatic complaints' group could be compared with the remainder. On several indices of brain damage (depth of penetration of injury, amount of brain tissue destroyed, length of post-traumatic amnesia), such patients consistently emerged as having the milder injuries. They had shown less intellectual impairment. And such symptoms clustered repeatedly with other symptoms that appeared to owe little if anything to any organic aetiology (e.g. difficulty with concentration, depression, and anxiety).

6. Dencker (1958, 1960)

This remarkable study adopted a strict case control approach. Dencker collected 36 monozygotic twin pairs and 81 dyzygotic pairs in which only one of the twins had had a head injury. They were all examined 3–25 years later (mean 10 years) and symptom profiles compared. The head-injured twins were inferior on a variety of tests of intellectual function, usually with rather subtle defects, as might have been expected. But no differences were found between members of individual monozygotic pairs in classical post-concussional symptoms. The monozygotic pairs proved to be more concordant than the dyzygotic for an impressive range of symptoms, including headache, dizziness, sensitivity to noise and light, even subjective memory impairment. These, then, seemed to owe more to constitutional factors, by way of genetic make-up, than to any brain damage that had occurred.

This survey of several research approaches has thus revealed a number of contradictions. Associations which emerge in one study may fail to appear in another. There is sometimes evidence of organic influences, sometimes mainly of psychosocial variables. When both are looked for together, both are sometimes observed. Altogether, we can summarise the types of evidence brought forward as follows, dealing first with the organic then the non-organic contributions. It is assumed that grossly obvious organic causes such as subdural haematoma or hydrocephalus have already been excluded by suitable screening procedures.

Organic evidence

Evidence was provided first of all for the 'clustered symptom complex' itself. The very pervasiveness of headache, dizziness, and fatigue from the early stages (Table II) suggests an organic process at work. Some 50% of patients can be expected to experience such symptoms, and, in the great majority, they can be expected to die away with time. There is evidence, moreover, that several of these complaints may be commoner after head injury than injury to other body parts. McMillan & Glucksman (1987) found that headache, dizziness, fatigue, irritability, and sensitivity to noise and light were all significantly commoner after minor head injury than after orthopaedic injuries, mostly to the upper limbs.

Certain relationships between early evidence of brain damage, particularly to brain stem and basal brain regions, and the development of post-concussional symptoms were observed. Lidvall *et al* (1974) found that otological dysfunction predicted the later development of dizziness; Rutherford *et al* (1977) showed an association between frequency of symptoms and early headache, diplopia, or anosmia. Some, although certainly not all, investigations have found a positive association with indices of severity of injury, such as duration of post-traumatic amnesia, or evidence of intellectual impairment (e.g. Keshavan *et al*, 1981). In these last respects, different studies have, however, given remarkably different results.

Other investigations have sought directly for evidence of cerebral dysfunction among post-concussional patients, using a number of experimental techniques. Taylor & Bell (1966) observed slowing of cerebral circulation time in patients with post-concussional symptoms, most being studied at 4–8 weeks after injury. Brain-stem auditory-evoked responses have been found to be delayed, even in patients with very mild injuries who are free from other neurological abnormalities (Rowe & Carlson, 1980; Noseworthy *et al*, 1981; Montgomery *et al*, 1984).

Waddell & Gronwall (1984) were able to demonstrate lowered thresholds for tolerance to light, and possibly to sound also, using objective tests administered 1–3 weeks after injury.

Slowed information processing has been shown repeatedly. Gronwall & Wrightson (1974) used a serial addition test, revealing impairments at 1–2 months that progressively resolved thereafter. McMillan & Glucksman (1987) confirmed such a finding at 1 week, even though tests of intelligence and memory were unimpaired. MacFlynn *et al* (1984) found similar delays on a four-choice reaction-time test at 24 hours and 6 weeks after injury. Ewing *et al* (1980), in an interesting study, exposed patients to hypoxia a year or more after minor injuries, and found that after such a period they still performed less well than control subjects on memory and vigilance tasks.

Such observations confirm the presence of some subtle alteration of cerebral function shortly after injury, which is occasionally still detectable well afterwards. They have, however, tended to rely on normal uninjured control subjects for assessment, rather than directly comparing patients with and without post-concussional symptoms following head injury. We are left, in consequence, uncertain about the relationship between such demonstrated abnormalities and the genesis of the post-concussional symptoms. The most we can find are impressionistic statements; Taylor & Bell (1966) reported that symptoms of headache, dizziness, and poor effort tolerance could often be seen to subside in parallel with return of the circulation times to normal, and Gronwall & Wrightson (1974) observed that the slowing of mental function tended to be more severe and long drawn out in the presence of complaints of headache, fatigue, and difficulty with concentration. MacFlynn *et al*, (1984), however, were unable to detect any relationship between their reaction-time measures and the presence or number of post-concussional complaints.

It remains possible, therefore, that some at least of these experimental observations of post-concussional symptoms are revealing concomitants rather than aetiologically relevant aspects of cerebral dysfunction. It should also be noted that the vast majority of these investigations have been carried out during the early post-injury phase, most within several weeks of the occurrence of the injury.

Non-organic evidence

There is as much evidence for non-organic influences as for cerebral dysfunction in the syndrome. The studies already reviewed showed that factors such as stresses antedating and surrounding the injury are related to the chance of developing symptoms, as is the anxiety engendered by the accident and attaching to early complaints. Such matters are difficult to measure with precision, but nonetheless the associations have emerged fairly clearly. 'Neuroticism' in the personality, a history of psychiatric illness, and demographic variables such as sex and marital status have been shown to be influential. Even from shortly after the injury, Rutherford *et al* (1977) found that a feeling of blame towards an employer was related to the frequency of symptoms.

Other investigations reinforce these findings. Studies from the second World War, for instance, underlined the similarities between head-injured and non-head-injured soldiers seen in army neurosis units (Lewis, 1942; Guttmann, 1946). They seemed equivalent to a remarkable degree in terms of family and personal histories of neurotic disability, personality type, and even range of symptoms. Lewis (1942) concluded that the "long-lasting relatively intractable post-concussional syndrome is apt to occur in much the same person as develops a psychiatric syndrome anyway". Dencker's (1958, 1960) study of twins, as previously indicated, gives strong support for such a view.

Guttmann (1946) considered that environmental, social, and psychological factors were usually operative when post-concussional complaints were long maintained. Among 300 patients, he observed that headache persisting at 6 months had usually been precipitated by psychological causes. Ruesch & Bowman (1945) made several observations on a large group of civilian head-injured patients; those with post-concussional symptoms who lacked obvious signs of brain damage resembled non-head-injured neurotic patients very closely; the longer the symptoms persisted, the more multiple and diffuse they became; and whereas in the acute stages complaints were more frequent when there was collateral evidence of brain damage, in those with long-lasting symptoms this situation was reversed – the complaints were more prominent when brain damage appeared to be lacking.

Synthesis

In the foregoing survey, both physiogenic and psychogenic causes have emerged for the 'post-concussional syndrome'. The evidence for each set of factors appears to be compelling, and we are led to think in terms of a complex interaction. Must it then be concluded that the search for greater clarity is a hopeless task? One possibility would be that both

sets of factors inevitably operate together and those aspects demonstrated are those that are measured most carefully. Another could be that the genesis of the syndrome differs from one individual to another, some patients responding to cerebral dysfunction and others to psychological influences.

In appraising the detailed evidence it seems possible to advance a short way further than this, while accepting that the situation is indeed very complex. An important aspect appears to hinge on the time course followed by the symptoms. As noted earlier, the post-concussional syndrome is not a static matter, but one which tends to decline markedly, and in some respects to alter, according to the time elapsed since injury. This applies both in the short-term and the long-term view. The investigations reviewed above have varied greatly along this time continuum. Thus those that have pointed to organic influences have in general been undertaken within a few weeks or months of injury – e.g. those showing altered cerebral circulation, delayed evoked responses, impaired information processing, or relationships to early neurological impairments. By contrast, those that have highlighted non-organic factors have mostly dealt with patients in the chronic later stages – those drawing comparisons with non-head-injured neurotic patients, uninjured twins, or those showing no trace of relationship with severity of injury or indices of brain damage. Many of these latter studies have been conducted on that very small proportion of patients who still complain of symptoms many months or even years after the injuries have occurred. Guttmann's (1946) patients, for example, had been injured 6 months earlier, Lishman's (1968) were followed up at 1–5 years and Dencker's (1958, 1960) at 3–25 years.

Such evidence is far from watertight, but in sum total it emerges as reasonably impressive. When the numerous studies concerning the post-concussional syndrome are carefully considered, the time factor after injury appears to be markedly influential in the associations we observe.

Thus a model may be tentatively formulated, which could be tested with a properly designed prospective investigation. The model proposes that the cerebral dysfunction engendered by head injury commonly yields a nuclear group of symptoms, headache, dizziness, and fatigue being the central group. At the outset, these are firmly organic in origin. In many respects, the less severe the injury, the more troublesome they may be to the patient – he will be aware of them more acutely when consciousness is regained rapidly, and will be attempting to cope while they are still severe. Also, more will be expected of him, as there will be less collateral damage to impose the invalid role. As the weeks go by, however, the symptoms are destined to recede, by a natural process of healing towards the status quo. If he is able to feel untroubled by them, and if left undistrubed by his environment, recuperation will in favourable cases be complete.

There are obstacles, however, to this natural process of resolution, and it would seem likely that, in patients with the milder forms of injury, these obstacles are mainly of a psychological nature. They may lie, for instance, in the patient's mental constitution, his tendency to worry unduly, to condition too rapidly, or to build anxiety around his symptoms. Or they may lie in the handling he receives in the early days and the attention that he is encouraged to focus on them. Obstacles may emerge in the form of other sources of distress – domestic difficulties, financial hardship, resentment about the origins of the accident itself. There may be the need to struggle to cope too early, or to face an uncongenial job. He may become significantly depressed. Later there may be conflict over compensation. If the obstacles are substantial, there may be secondary neurotic developments, founded in anxiety, and sometimes destined to be long-lasting. The symptoms of the post-concussional syndrome provide, indeed, the ideal nexus for neurotic elaboration – in contrast to physical disability, they are subjective, unverifiable, and irrefutable, and there is no way that the sufferer can reassure himself that they have disappeared.

Thus, the longer after the accident that post-concussional symptoms persist, the more evidence that non-organic factors play a part is gained. Complaining patients studied shortly after the accident will form a different population from those still complaining after several months or years. With time, those with an uncomplicated and purely organic contribution to their disability will be lost to the sample, and an increasing proportion will have secondary neurotic developments. Ultimately, indeed, in mildly injured patients, the organic cerebral contribution may give way almost completely, and we will be left with patients virtually all of whom are suffering from conflict, depression, and anxiety.

In conclusion, therefore, there is certainly an interplay of factors in the genesis of the post-concussional syndrome, with an intertwining of organic and non-organic contributions. But this interplay is time-dependent. Over many weeks and months there is a shifting balance, as the patient's innate proclivities, and his individual problems and conflicts, affect the initial symptoms of cerebral dysfunction. This model needs careful testing and exploration in properly designed prospective investigations. The identification of the obstacles – in general, and in the individual

patient – is the essential prelude to decisions about where treatment can most effectively be directed.

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