

22 OUTCOME AFTER SEVERE HEAD INJURY

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22.1 Outcome after severe head injury

Much more attention tends to be paid to describing and classifying the initial diagnosis and severity of injury than to defining the outcome after various types of trauma. Yet when it is the brain that is injured the need for accurate assessment of outcome is all the more pressing, because many survivors are left with a combination of mental and neurological deficits that markedly affect the quality of life. *Judging the medical efficacy and the cost-effectiveness of interventions both in the acute and rehabilitation phases depends on measuring and valuing the ultimate outcome.* Apart from this the patient and his/her family are clearly concerned to be kept informed about the prospects of recovery as time passes and to be advised about how to plan for the likely, and then to deal with the actual, outcome.

The combined effect of mental and physical disabilities results in a global disability that is often greater than the sum of these parts. This is because the mental impairments limit the capacity to cope with the physical disabilities, while for many patients the mental changes comprise the major and often the only persisting disability. For these reasons it is important not only to list the various components of medical and physical disability but also to make a global assessment of the patient's state in terms of overall social consequences of his/her brain damage.

Various terms previously used to describe outcome tended to reflect the viewpoint of different observers, some more optimistic than others. Optimistic assessments resulted from overemphasis on physical recovery and minimizing the mental impairments, and were apt to be taken by those who had dealt with the patient in the acute stage. For them the contrast with the previously comatose state is striking, while they naturally wish to justify their early therapeutic efforts by claiming a reasonable recovery. Terms such as practical, useful, reasonable, acceptable and worthwhile recovery usually prove to be euphemisms for

severe disability. On the other hand, assessments that list every detectable neurological deficit, many of which do not constitute any disability or handicap, can appear too pessimistic. Nor can social measures such as return to home or to work be relied on to indicate the degree of recovery. Exceptional family efforts may enable some very disabled patients to return home. Return to work is an unsatisfactory guide for the many patients who were not previously in work, while failure to return to work may indicate a decision to retire early or, in times of high unemployment, the patient being laid off for economic rather than health reasons. Occasionally, return to work is to a much lower level of job, provided by a sympathetic employer. For these various reasons it is much better to rely on some standard scale for assessing outcome. Many of these exist for patients with stroke, but they tend to focus in great detail on aspects of physical capacity and the ability to undertake activities of daily living. Moreover, most apply to elderly patients whose quality of life has different dimensions and expectations from that of the predominantly young patients who suffer head injury. It was for these reasons that we developed the Glasgow Outcome Scale.

22.2 Glasgow Outcome Scale

This scale is based on the overall social capability (or dependence) of the patient, which takes account of the combined effect of specific mental and neurological deficits, but without listing these as part of the definition (Jennett and Bond, 1975). It was devised for brain damage in general because it was required for studies both of head injury and of non-traumatic coma. Its successful use in collaborative international investigations has established that it can be reliably and readily used by different observers. When 150 Glasgow survivors after severe head injury were classified independently by a neurologist and by a neurosurgeon there was over 90% agreement, both for assessments at 6 months and at 12 months after injury (Jennett *et al.*, 1981).

The Glasgow Outcome Scale has five categories:

1. *Good recovery*
2. *Moderate disability*
3. *Severe disability*
4. *Vegetative state*
5. *Dead.*

Four categories of survival are recognized.

1. *Good recovery*

The patient is able to participate in normal social life and could return to work (although he or she may not have done so). Quite a number of those with good recovery do not, for a variety of reasons, return to work, while some moderately disabled patients do. Good recovery need not imply absence of sequelae, as many patients have minor cranial nerve deficits, residual mild limb weakness or some impairment on cognitive testing or personality change.

2. *Moderate disability (independent but disabled)*

These patients look after themselves, can travel by public transport, and some are capable of work. This may be of a sheltered kind but certain marked disabilities are compatible with a return to some occupations. Moderately disabled patients may have memory deficits or personality changes, varying degrees of hemiparesis, dysphasia or ataxia, post-traumatic epilepsy, or major cranial nerve deficits. The degree of independence required to reach this category is much higher than that commonly described by geriatricians as 'independent for activities of daily living', which may indicate no more than the ability to attend to personal needs in their own room but without independent mobility or the capacity to organize their living without assistance; such patients would be judged severely disabled on the Glasgow scale.

3. *Severe disability (conscious but dependent)*

Patients in this category are dependent on some other person for some activities during every 24 hours. The worst affected are physically severely disabled, often with spastic paralysis of three or four limbs. Marked dysphasia, which limits communication, is a major handicap in some, and in others dysarthria is a problem. Marked physical deficits of this kind are always associated with markedly restricted mental activity. However, some patients who have little or no persisting neurological disability are so seriously affected mentally that they require permanent supervision by family or in residential care. Their mental problems vary from severe organic dementia to

disinhibited, irresponsible behavior. The least affected patients in the category of severe disability are those who are communicative and sensible (though often with impaired cognitive and memory tests) but who are dependent for certain physical activities on others – perhaps dressing, feeding, or cooking their meals. Because such a person could not be left alone, even for a weekend, he/she is not independent and is classified as severely disabled.

4. *Vegetative state*

The characteristic feature of this condition of non-sentient survival, defined by Jennett and Plum in 1972, is that there is no evidence of psychologically meaningful activity, as judged behaviorally. The criteria for the definition of this state should be strict and patients who obey even simple commands or who utter even occasional words should be assigned to the category of very severe disability. Relatives or inexperienced nurses sometimes interpret reflex grasping, groping or withdrawal as evidence that commands are obeyed or that purposeful movements are carried out. Likewise groans, cries and chewing and pouting are sometimes hopefully interpreted as 'no' or 'Mum' and claimed as evidence of returning speech (see below).

22.2.1 NUMBER OF CATEGORIES, RELIABILITY AND VALIDITY

According to the purpose for which an outcome classification is required, more or fewer categories than those of the original Glasgow Outcome Scale (GOS) may be needed. When analyzing the rate and degree of recovery it can be useful to recognize improvement within one category by subdividing each of the three categories of conscious survivors into a better and a worse grade (Jennett *et al.*, 1981). On the other hand, when one is seeking statistical relationships between early features and outcome the fewer categories the better. For example, dead and vegetative patients may be combined (bad outcomes), or vegetative and severely disabled (dependent), and moderate disability and good recovery (independent).

In the study of 150 Glasgow survivors after severe head injury classified independently by a neurologist and by a neurosurgeon, referred to earlier, there was over 90% agreement on the basis of written reports, both for assessments at 6 months and at 12 months after injury (Jennett *et al.*, 1981). A more detailed analysis of 57 cases confirmed good inter-rater reliability, this time between a psychiatrist and a neurosurgeon (Brooks, Hosie and Bond, 1986). This applied to both the three-point and six-point scales for conscious survivors, most disagreements being between good

recovery and moderate disability. Cognitive tests correlated much more closely with the three-point scale in this study at 3 months than later after injury, but did so at both 3 and 6 months in a later study (Clifton *et al.*, 1993).

22.2.2 ALTERNATIVES TO THE GLASGOW OUTCOME SCALE

The main value of the GOS is in comparing the outcome of groups of head-injured patients who have been treated by different therapeutic regimes or in different places. An obvious criticism is that its simplicity makes it relatively insensitive to improvements occurring late in recovery that are not large enough to make a change in category and yet are significant. It has been claimed that the Disability Rating Scale (Table 22.1) is more useful for this purpose (Rappaport *et al.*, 1982; Hall, Cope and Rappaport, 1985).

A consensus conference on outcome measures for clinical trials in head injury, held in Houston in 1992, concluded that for severe injuries either the Glasgow Outcome Scale or the Disability Rating Scale at 6 months was appropriate, but that for moderate injuries (Glasgow Coma Score 9–12) the Disability Rating Scale at 3 months was better (Clifton et al., 1992).

Outcomes research in medicine in general is making increasing use of state of health measures which indicate the patient's own perception of himself.

Table 22.1 Disability Rating Scale (Rappaport *et al.*, 1982)

| | |
|--|-----------|
| Scoring in eight categories with high indicating maximum disability. | |
| Eye opening | 0–3 |
| Verbal | 0–4 |
| Motor response | 0–4 |
| Toileting | 0–3 |
| Feeding | 0–3 |
| Grooming | 0–3 |
| Dependence | 0–5 |
| Employability | 0–3 |
| Maximum disability | 30 |

} Cognitive ability for

This gives ten degrees of disability.

| | |
|-------|-----------------------------|
| 0 | Nil |
| 1 | Mild disability |
| 2–3 | Partial disability |
| 4–6 | Moderate disability |
| 7–11 | Moderate/severe disability |
| 12–16 | Severe disability |
| 17–21 | Extremely severe disability |
| 22–24 | Vegetative state |
| 25–29 | Extreme vegetative state |
| 30 | Death |

Table 22.2 Nottingham Health Profile (Hunt, McKenna and McEwan, 1981)

This consists of a questionnaire for patient completion, consisting of a series of questions requiring yes/no answers.

Part I has questions about six problems with health – physical mobility, pain, sleep, energy, social isolation and emotional reactions.

Part II deals with seven areas of daily life affected by health – paid employment, looking after the house, social life, love life, sex life, hobbies and holidays.

There are various weights for the 'yes' answers to different questions and these sum to 100 for each category of disability – for comparison with figures for abnormal population or with the patient at some previous stage of their recovery.

Widely used measures are the Nottingham Health Profile (Table 22.2) in the UK (McEwan, 1983) and the Sickness Impact Profile in the USA (Bergner *et al.*, 1976), but both countries are now recommending the Short Form 36. This scores physical and social functioning, physical and emotional limitations, mental health, energy/vitality, pain and general health perceptions (Ware, 1993; Jenkinson, Coulter and Wright, 1993).

22.2.3 COMPONENT DISABILITIES IN DIFFERENT OUTCOME CATEGORIES

The mental component of the disability was judged to be more important in more than half the survivors of severe head injury in Glasgow; in only a quarter was the physical disability more prominent than the mental. This predominance of the mental disability was found in each of the three categories of conscious survivors. In a more detailed study of a subset of these patients Bond (1976) found that mental disability correlated more significantly than did physical deficits with social handicap. Almost half the patients who had made a good recovery had mild changes in personality. In those with moderate disability, personality change and physical disability were recorded in about equal proportions. Common neurophysical sequelae in good recoveries were mild hemiparesis, cranial nerve palsies or infrequent epilepsy. In the moderately disabled, hemiparesis was again prominent and was sometimes severe, while dysphasia was much more frequent than in the good recoveries. Cranial nerve palsies were common in the moderately disabled and ataxia was not uncommon. In both these upper grades of recovery personality changes were common; as expected these were more frequent and more severe in the moderately disabled than in the good recoveries.

All the severely disabled patients had personality changes. Because of the small number of severely disabled patients in whom formal cognitive testing was feasible no marked difference could be shown between measurable aspects of cognitive deficit in the moderate and the severely disabled. This emphasizes the value of making an overall clinical judgment rather than basing a calculation of overall disability on an aggregation of individual disabilities. *Moreover this can be reliably done at a brief outpatient interview by medical, nursing or remedial staff from various disciplines once they have been adequately instructed in the use of a structured questionnaire.* However, according to one study, the GOS underestimates the continuing limitation of psychosocial reintegration of less severely injured patients. Although 52% of 87 patients made a good recovery, only 24% made a good level of reintegration. There was a substantial limitation of reintegration in more than half of the good recoveries (Tate *et al.*, 1989).

22.3 When to assess outcome

Reports on outcome sometimes fail to specify how long after injury the assessments were made, or give only the minimum or mean periods of follow-up. The process of recovery in survivors can continue for months, and claims are often made that substantial recovery has occurred over a period of years. What interval is chosen for assessment depends to some extent on how detailed a categorization is used. Deaths and survivors can reasonably be identified within a few weeks, apart from the few late deaths – most of which are in vegetative or severely disabled survivors. Attempts to record the ultimate degree of disability at this early stage usually prove to be predictions rather than assessments. Prognosis becomes more confident as time passes but it should always be made clear whether the expected or the actual state is what is being described, and to what interval after injury each applies.

Anecdotes about unexpected late improvement many months or years after severe head injury lead some clinicians to encourage patients, relatives and therapists to look forward to continued recovery over a long period of time. This is often unrealistic, and what is required for practical purposes is to determine an interval after injury by which most patients will have achieved most of their recovery.

When more than 500 survivors in the Glasgow-based three countries study were assessed at 3, 6 and 12 months after injury (Jennett and Teasdale, 1981) there were more good recoveries and fewer severely disabled patients by the end of the year. Not only had some improved but others with severe disability had died during the period of follow-up. *Of those who by*

12 months had made a good or moderate recovery, two-thirds had already reached this level of recovery on the scale within 3 months of injury, and 90% had done so by 6 months. Only 5% of 82 patients followed for more than 18 months showed sufficient improvement after 12 months to change their outcome category. In the 150 Glasgow patients it was confirmed that 10% of patients who were severe or moderate at 6 months had improved to become moderate or good respectively by 1 year.

This is not to deny that some degree of recovery may continue after 6 months in many patients; however, the degree of improvement seldom justifies a change of category on the three-point Glasgow scale for conscious survivors. With the six-point outcome scale 20% of the 150 Glasgow survivors changed by one category between 6 and 12 months; half of these moved into the next higher category on the three-point scale. *That most recovery occurs in most patients within the first 6 months is supported by other studies, for example the 20-year follow-up by Roberts (1979). Six months after injury is a useful time to assess outcome, because it is practical to maintain contact with the majority of patients for this long. Six-month outcome was recently recommended as the appropriate prime outcome measure for clinical trials of therapies for the acute phase of injury (Clifton *et al.*, 1992).*

Long-term follow-up reports of severe injuries are now available from several centers. For patients aged 15–64 years with at least 2 days PTA the Glasgow Group made assessments at 3, 6 and 12 months and at 2, 5 and 7 years; the psychosocial and neurophysical status of patients were assessed as well as the psychiatric and social consequences for the main caring relatives (Livingston, Brooks and Bond, 1985a,b; Brooks, Campsie and Symington, *et al.*, 1986, 1987). *Although neurophysical deficits (including dysphasia) tended to improve over the first few years no consistent reduction in the symptom level was found at 7 years compared with 2 years. Relatives were more distressed at a year than at 3 months, and they commonly complained of increasing burden over subsequent years.* More psychiatric symptoms and negative social consequences became manifest as time passed with the patient improving so little.

Brooks *et al.* (1987) emphasized that their Glasgow patients did not have access to a well-structured scheme of rehabilitation, coming as they did from a wide area; they postulated that they might therefore have fared less well than patients reported from elsewhere.

However, a 7-year follow up of a series culled from one of the few dedicated neurological rehabilitation centers in Britain (Oddy *et al.*, 1985) showed similar results to those of Brooks. These patients were all

under 40 years of age and had a PTA of 7 days or more. They were assessed 2 years after injury and again at 7 years, when no significant change was found in neurophysical status or in performance of cognitive tests. Personality changes had persisted and some patients had developed major psychiatric problems. There had been no major changes in employment status; no patient without a job at 2 years having subsequently obtained work. Limited improvements in social adjustments had occurred in a few, but these were patients who were already well recovered by 2 years – most of them from the minority who had by then already returned to some kind of work. Reporting some late improvement in their somewhat less severely injured series Brooks *et al.* (1987) emphasized that they could find no reliable predictors of who might have delayed improvement. Similar results have been reported from Denmark, where Thomsen (1984) studied 40 patients under 45 years of age with PTA of a month or more. *Although there is a small added risk of accidental death, sometimes related to physical disability, to epilepsy or to suicide in disabled conscious survivors of severe head injury, this makes for very little reduction in the normal expectation of life (Roberts, 1979). Therefore most patients left with severe disability will have to endure this for 30–40 years: the average age of such patients at injury is 27 years.*

The conclusion must be that it is unrealistic to expect significant improvement in physical deficits, cognitive functions, behavior or personality problems after 2 years. The improvements in social functioning after that time (and indeed most such improvements after the first year) probably reflect gradual acceptance of, and adaptation to, a relatively fixed disability, on the part of both the patient and the family. These studies have all emphasized that the patients' complaints are fewer and less marked than the deficits perceived by their carers, who are more aware of the relatively static disability and of the contrast with the patient's

previous state or their expectations for this future. This may account for carers reporting increasing burden as the years pass, and for their adverse reactions to the situation.

The prognosis for patients in a vegetative state is dealt with later.

(a) Distribution of outcomes

There have been numerous reports of outcome after severe head injuries in the last decade or so, with varying definitions of initial severity and of the time when outcome was assessed. Three large series indicate a similar distribution of recovered patients among categories (Table 22.3). The lower mortality of the two more recent series is partly explained by the exclusion of patients who deteriorated because of intracranial hematomas without having been in persisting coma initially.

22.4 Ethical issues

The main ethical issue arises when active treatment seems so unlikely to benefit the patient that a decision to limit treatment has to be considered (Jennett, 1992a). This is a response to the four principles of medical ethics – to maximize benefit but to minimize harm for the patient, to respect his/her autonomy and to have regard to justice in the use and distribution of health care resources. *Two circumstances are now widely agreed to justify the withdrawal of treatment, namely when a patient is brain-dead and when he/she has been vegetative for so long that no prospect of recovery remains.* These two situations are dealt with in detail below. However, there are two less clear-cut situations when a decision to limit treatment may be considered. One is when, soon after admission, it is judged that the patient has sustained an irrecoverable injury – whether only to the head or from multiple injuries. This decision will usually be reached only

Table 22.3 Outcome after severe head injury (TCDB = Trauma Coma Data Bank)

| Study | Ref | Years | n | Interval* | Severe | | | | | Moderate/ |
|----------------------------|-------------------------------|---------|---------|--------------|----------|----------------|----------------|--------------|----------|-----------|
| | | | | | Dead (%) | Vegetative (%) | disability (%) | Moderate (%) | Good (%) | Good (%) |
| Three countries | Jennett <i>et al.</i> , 1979 | 1970–77 | 1000 | 6 months | 49 | 2 | 10 | 17 | 22 | 39 |
| Trauma Coma Data Bank, USA | Marshall <i>et al.</i> , 1991 | 1984–87 | 746 | Discharge | 33 | 14 | 28 | 19 | 7 | 26 |
| | | | | Last contact | 36 | 5 | 16 | 16 | 27 | 43 |
| Four UK centers | Murray <i>et al.</i> , 1993 | 1986–88 | A: 1067 | 6 months | 45 | 1.6 | 20 | 18 | 15 | 36 |
| | | | B: 1353 | 6 months | 37 | 1.3 | 18 | 18 | 23 | 45 |

Entry criteria: Three countries – coma for ≥ 6 h; Trauma Coma Data Bank – GCS ≤ 8 on admission or deteriorated to this in ≤ 48 h; UK centers – A, coma ≥ 6 h; B, including coma ≤ 6 h

***Time of outcome in TCDB:** at discharge from Neurosurgery – 46% in 30 days (mostly deaths), 79% in 60 days; last contact – two-thirds > 1 year, one-third > 2 years

after attempts at resuscitation, sometimes when a CT scan of the head has also been done. Even if the latter shows a large intracranial hematoma it may be judged irrecoverable as the patient has been deeply comatose with fixed pupils for some time, particularly if he/she is elderly. Confidential inquiry of perioperative deaths in the UK has identified a number of patients whom it was considered should not have been subjected to surgery because they were moribund, and this included some with severe head injuries. In an audit of deaths in a neurosurgical unit we found that decisions to limit treatment were quite frequently made, including a number where surgery was withheld after initial assessment (Barlow and Jennett, 1991). *More often a decision to limit treatment is made when the patient shows no sign of recovery after a trial of treatment – for example ventilation and perhaps evacuation of an intracranial hematoma.* There are now formal computer programs for estimating the probability of survival and recovery, given a limited number of variables that include the depth and duration of coma, pupil reactions and the patient's age. We found that providing such data to neurosurgeons led to less use of certain active treatments for patients with poor prognosis and greater use in those with a better outlook (Murray *et al.*, 1993). There was no change in the proportion of patients for whom a written decision to limit treatment was made when predictions were available. The commonest treatments to be withheld are cardiopulmonary resuscitation and antibiotics for new infections. Surgery may be withheld either initially, as described above, or when a secondary complication develops. Sometimes a patient on a ventilator who is not brain-dead remains deeply comatose with no sign of recovery. It may then be decided to withdraw the ventilation, accepting that spontaneous ventilation will probably be inadequate. When the time comes for decisions such as these it may be wise to consider discharge from the intensive care unit (or the neurosurgical unit) to a less intensive and less expensive setting.

In head-injured patients there is no prospect of respecting the patient's autonomy because he/she cannot express his/her preferences. The family (or friends) then become important and they should be kept fully informed of the expectations of the doctor regarding survival or useful recovery. They will then often volunteer that they believe that this patient would not want his/her life prolonged in such circumstances. Doctors should make every effort to be sure that the relatives are genuinely attempting to put forward the patient's viewpoint rather than their own. Only occasionally, but hopefully more often in the future, patients may have an advance directive, which can make such decision-making easier. What is clear is that, if the patient has made such a declaration,

the doctor should respect it and act accordingly. So should he/she act in accordance with the family wishes if he/she is satisfied that these are in the patient's best interests. Under such circumstances there should be no risk of civil or criminal liability for the subsequent death of the patient.

There is increasing evidence that people do not want life at any cost, and in particular are anxious to avoid the risk of survival with severe brain damage. In a survey of 500 Americans about their wish to refuse life-saving or life-sustaining treatments given four scenarios, more than 80% would refuse such treatments if vegetative while more than half would refuse them if in coma with a small chance of complete recovery (Emanuel *et al.*, 1991). *When a group of 59 neurosurgeons from several countries were asked at what level of probability of a poor outcome after severe head injury (dead, vegetative or severely disabled) they would withhold ventilation or surgery, most wanted more than 95% certainty (Barlow and Teasdale, 1986). However, when then asked to imagine that they themselves were injured, many wanted treatment to be limited at a much lower probability of a poor outcome. They were not prepared to accept the risk of survival with severe brain damage that they would recommend for their patients.* Whatever the generality of views on such matters, however, what matters is to try to determine what a particular patient's previous attitudes were and thus to make a decision that is both in his/her best interest and likely to reflect what he/she would have wanted.

To assist in making such decisions it is helpful to have guidelines, agreed by medical and nursing staff, about the kinds of situation that would lead to a treatment-limiting decision. These can be useful in signaling that such decisions are part of good practice, as well as reminding decision-makers of the factors to consider when faced with such a situation. They are therefore supportive of the doctor who makes such a decision by indicating that this is in accordance with agreed practice.

22.5 Brain death

Essential to the concept of brain death is the recognition that death is a process rather than an event. Organs and tissues cease to function and later necrose at different stages in the process of death, and when death is declared is to some extent arbitrary. The World Medical Association declaration of 1968 in Sydney proposed that death is when the body as an integrated whole has irreversibly ceased to function, rather than when all organs and tissues are dead.

There are three common sequences that lead to death. Most often, cardiac arrest is the initial event and soon the cerebral cortex ceases to function; later the

brain stem also fails and respiration then stops. Less often, respiratory arrest begins the sequence, leading to anoxic cortical and then brain-stem failure, while cardiac arrest may not occur for 15–30 minutes. Sometimes it is the brain stem that fails first, followed by respiratory arrest with anoxic cardiac arrest occurring later. If artificial ventilation restores oxygenation after the brain stem is dead but before the heart stops, then ultimate cardiac arrest may be delayed for many days. It is, however, a mistake to consider that there are two kinds of death – that evidenced by cardiorespiratory arrest and that by lack of brain-stem function. This is because cardiorespiratory arrest is considered to indicate death only when it has lasted long enough to produce brain-stem death. When patients are successfully resuscitated from cardiac arrest or have been subjected to therapeutically controlled stoppage of the heart during surgery, we do not claim that they have been dead. *It is therefore brain-stem death that is the central feature of all sequences of death, while the state of continued cardiac function after this has occurred is an artifact of nature resulting from technological intervention.*

A distinction is sometimes drawn between brain-stem death and whole brain death. However, the function of the cerebral cortex is dependent on upward impulses from the reticular formation in the brain stem, and therefore when the brain stem is dead the brain as a whole cannot function. This is not to deny that some cells in the cerebral cortex and basal ganglia may not continue to survive for a time, but they are not able to maintain the function of the brain as a whole. The logic of the situation is therefore that if the brain stem is dead the brain is dead. It is also now accepted that if the brain is dead the person is dead. This last concept is explicitly stated in the UK Royal Colleges memorandum of 1979, which states that the time of death is when brain-stem death is confirmed and not some later time when the heart stops (Conference of Medical Royal Colleges, 1979). It is important to explain this to those involved in procedures associated with organ donation, when there may be a delay of several hours before the ventilator is withdrawn and the heart stops. *It is easy to refer carelessly to withdrawing life support or letting the patient die, when in fact ventilation is being stopped in a patient who is already dead.*

22.5.1 THE DIAGNOSIS OF BRAIN DEATH – THE UK CRITERIA (Table 22.4)

These criteria were published by the UK Conference of Medical Royal Colleges in 1976, and a further memorandum in 1979 confirmed these and indicated that death could be declared once the criteria were satisfied. A feature of the diagnostic criteria is the

emphasis on satisfying the preconditions before considering the tests to confirm that the brain stem is dead. There are four preconditions. The patient must be in deep coma, must be apneic (and therefore on a ventilator), must have irrecoverable structural brain damage and reversible causes of brain-stem depression must have been excluded. Common causes of brain damage leading to brain death are severe head injury and spontaneous intracranial hemorrhage, but a few result from brain tumor or intracranial infection. Some cases follow delayed resuscitation after cardiac arrest from various causes, including anoxia and drug overdose. Reversible causes of brain-stem depression include depressant drugs, neuromuscular blocking agents used for intubation during resuscitation or as an adjunct to mechanical ventilation, hypothermia and gross metabolic abnormalities. These various factors may not be the sole cause of brain-stem depression but can aggravate the effect of structural lesions. Screening for drugs will not normally be necessary when there is a clear-cut story of sudden coma from injury or hemorrhage. As for establishing the irrecoverability of the brain damage, enough time should elapse to correct temporary causes of brain-stem depression such as hypotension, hypoxia, raised intracranial pressure and barbiturate therapy. Normally the diagnosis would not be considered in less than 6 hours but when the cause is anoxic damage or when drugs are suspected the diagnosis should be delayed for at least 24 hours.

The tests to confirm that there is no residual brain-stem function are simple to perform and to interpret. There should be no pupillary or corneal reflexes, no movement of the facial muscles to pain or of the throat muscles to movement of the endotracheal tube. The caloric vestibulo-ocular reflex should be absent (no eye movements following irrigation of the external auditory meatus with at least 20 ml of ice cold water on each side). Only when these reflexes are found all to be absent is the final crucial test applied, to confirm apnea. There should be no respiratory movements when disconnection of the ventilator allows the $P_a\text{CO}_2$ to rise. The UK criteria require $P_a\text{CO}_2$ to reach 50 mmHg (6.65 kPa) but American codes recommend 60 mmHg (8.0 kPa). The rate of rise of $P_a\text{CO}_2$ in brain-dead patients can be slow (Benzel *et al.*, 1989), and to attain this level in 10 minutes requires that the $P_a\text{CO}_2$ be greater than 40 mmHg (5.3 kPa) before disconnection. This can be achieved by reducing the tidal volume or by ventilating with 95% oxygen and 5% CO_2 for 5 minutes. To ensure that damaging hypoxia does not occur during disconnection, preoxygenation with 100% oxygen for 10 minutes before disconnection is recommended and the maintenance during disconnection of 6 l/min of oxygen delivered down a catheter in the trachea. Advice from experts is

Table 22.4 UK brain death criteria**Three preconditions**

- Patient on a ventilator
- Coma due to irremedial structural brain damage
- Exclusion of reversible factors
 - depressant or neuromuscular blocking drugs
 - primary hypothermia
 - metabolic or endocrine abnormalities

Five tests

- No pupillary response to light
- No tracheal, gag or cough reflex
- No response to facial and peripheral pain
- No cold caloric responses
- No respiratory effort after achieving a $P_a\text{CO}_2$ of 50 mmHg for 10 min or more

required for patients with pre-existing chronic respiratory insufficiency who normally depend on a hypoxic drive for respiration and may be unresponsive to raised $P_a\text{CO}_2$.

The UK criteria specify that two doctors should be involved in testing, one of them a consultant and the other a senior registrar or consultant, and that the tests be done on two separate occasions. Notice that these criteria require no confirmatory laboratory tests.

Provided the preconditions have been met before the first test the interval between the two assessments need be no more than half an hour. The Ad Hoc Committee of the Harvard Medical School (1968) recommended demonstrating absence of cerebral activity on EEG, but this was declared optional by that institution a year later (Beecher, 1969). This is still frequently used in the US and in other countries, and sometimes in the UK. In practice it is less useful than might be expected, partly because it reflects activity in the cerebral hemispheres rather than the brain stem (and some residual activity may persist after unequivocal brain-stem death), and because securing an isoelectric recording can be technically difficult in the electronically active environment of an intensive care unit. Those who use it sometimes say that they do so to impress the family rather than to make a diagnosis. Another confirmatory test is to demonstrate absence of cerebral circulation over a period of time, either visually by angiography or by showing no entry of radioactive agents injected systemically. Both require technical equipment and expertise and neither is wholly reliable; they are virtually never used in the UK and rarely in the USA.

Definitive guidance on the diagnosis of brain death in children has been given by a US Task Force (Task Force for the Determination of Brain Death in Children, 1987). This counseled that the diagnosis should not be made in the first 7 days of life and the UK Conference of

Medical Royal Colleges (1988) subsequently endorsed this for organ donation in the UK. *From 7 days till 2 months of age the Task Force recommended two isoelectric EEG records 48 hours apart, but for 2–12 months old the interval need only be 24 hours. In children a year or more old the diagnosis by adult criteria with up to 12 hours observation was considered adequate, without EEG confirmation.*

22.5.2 VALIDITY OF THE CRITERIA

These criteria have now been applied to many thousands of patients, many of whom were ventilated until asystole before clinicians gained the confidence to discontinue ventilation once brain death had been diagnosed. Not one case is on record as having recovered after the UK criteria were satisfied, according to Pallis (1990), who listed over 1900 published cases. Nonetheless, sporadic press reports of patients allegedly recovering after supposedly having been brain-dead appeared during the 1970s and these culminated in a challenge on BBC TV in 1980 about whether organ donors were in fact always definitely dead before organs were removed. The critics were mostly from other countries (particularly the US), and seemed mainly concerned that the UK criteria did not require an EEG. In the event the original criteria did not need to be modified (Robson, 1981), while subsequent guidelines in the US stressed that the use of EEG was optional (Medical Consultants to the President's Commission, 1981).

It is, however, wise to be sensitive to misunderstandings that can arise in this sensitive area of medicine. Most center on the issue of organ donation and the suspicion that eagerness to secure organs might tempt doctors to make a premature diagnosis of brain death. An allegation may be made that a patient recovered after 'they nearly removed his kidneys'. A likely explanation is that the family was told soon after an acute episode of brain damage that the outlook was almost hopeless, but that the results of resuscitation were still awaited. When such a prognosis is given families now sometimes immediately themselves raise the possibility of organ donation. The response to this should be to indicate that it is too soon to be sure that there will be no recovery (and certainly too soon to diagnose brain death). However, doctors may be tempted to accept this offer even though noting that diagnostic tests will be needed later. As occasionally such patients do recover or at least survive for a time, it is easy to see how in retrospect it may seem as though organs were nearly taken. The same may happen when a reversible cause of brain-stem depression such as drug overdose is discovered. Clearly these are not examples of recovery after the formal diagnosis of brain death.

Misunderstandings can also arise in the intensive care unit when bystanders observe the activity of spinal reflexes in patients declared brain-dead. In fact these become more active the longer ventilation is continued after brain death, and they may be precipitated by the removal of organs. The best safeguard against such embarrassing allegations is never to consider the diagnosis of brain death until the preconditions have been met, to use widely accepted diagnostic criteria, to have two doctors involved and always to allow enough time to be certain that the situation is not reversible. These conditions have been established in the UK by the Health Department's Code of Practice published in 1979, and revised in 1983 (Health Departments of Great Britain and Northern Ireland, 1983), which reproduces the UK Colleges' criteria and memorandum as well as giving detailed guidelines for the removal of organs for transplantation. This has been widely distributed and the diagnostic criteria have recently been reproduced yet again in the form of a checklist for inclusion in the patient's notes (O'Brien, 1990). In practice there is now little continuing controversy in the UK about brain death, a concept that the public at large and the families actually involved seem able to accept. A recent review of the UK criteria has emphasized certain features, but broadly endorsed the original recommendations (Royal College of Physicians, 1995).

22.5.3 ORGAN DONATION

It is important to emphasize that the diagnosis of brain death and the subsequent withdrawal of ventilation is part of good medical practice and is required regardless of any need for organ donation. Indeed in many instances the patient is medically unsuitable to become an organ donor. Nevertheless, brain-dead patients do present the best source of kidneys for donation (although these can be removed after terminal cardiac arrest), and are the only source of hearts, lungs and livers. It is accepted that it is ethically acceptable to maintain the blood pressure and fluid intake of brain-dead patients in order to optimize the condition of organs to be transplanted. There is, however, some controversy about the elective ventilation of patients in order that they might become organ donors (i.e. patients who would not otherwise be ventilated). Investigations in England (Feest *et al.*, 1990), in Wales (Salih *et al.*, 1991) and in New South Wales (Hibberd, Pearson and McCasker, 1992) suggest that such a practice could lead to considerably more organs being available. Concerns about the lawfulness of interventions that are not in the best interests of the patient who is a potential donor has led to this practice being stopped in the UK, but there is debate as to how this legal problem might be circumvented (Riad *et al.*, 1995).

Meanwhile, surveys in intensive care units in England and Wales (Gore, Hindes and Rutherford, 1989; Gore, Taylor and Wallwork, 1991; Gore, Cable and Holland, 1992) and in one neurosurgical unit (Gentleman, Easton and Jennett, 1990) showed that more than a quarter of potentially brain-dead patients are never tested for brain death while 30% of relatives refuse consent for organ donation when testing has confirmed that their relative is brain-dead.

The mechanisms to be followed in arranging organ donation will vary from place to place, but clear guidelines should be available, as in the Code of Practice of the Health Departments of Great Britain and Northern Ireland (1983). *Most will require that the management of the patient up to and including the tests for brain death should be in the hands of the clinicians who are not part of the transplantation team.* While some places require additional tests to confirm brain death when organ donation is under consideration, this seems illogical, in that it suggests that less than reliable criteria are being used to reach a decision to discontinue ventilation when organ donation is not an issue.

22.6 The vegetative state

The management of patients in this state, with no evidence of a functioning cerebral cortex, has given rise to considerable debate in medical, ethical and legal circles in recent years. There is a growing consensus that once there is no prospect of recovery survival in this state is of no benefit to the patient, and that withdrawal of life-sustaining treatment may therefore be morally and legally justified. However, particularly after trauma, the vegetative state may be temporary and before such a decision is made it is essential that both the diagnosis and prognosis be reliable.

The diagnosis remains a clinical one – depending on doctors and nurses being sure that there is no sustained, reproducible purposeful activity or response to external stimuli, and no words uttered or commands obeyed. Reflex responses to light, sound and pain need to be discounted, and spontaneous emotional behavior patterns unrelated to relevant events. No investigations can reliably confirm the diagnosis. CT scanning will show progressive atrophy but similar degrees have been found in demented patients who retain some conscious behavior. However, the absence of atrophy would indicate the possibility of recovery. EEG recordings vary from isoelectric to near normal, but are usually unresponsive to external stimuli. Absence of a cortical response to somatosensory stimuli is common but neither its absence nor its presence confirms or excludes this diagnosis. PET scanning has shown CMRO₂ for glucose in the cortex and basal ganglia to

be less than 50% of normal in vegetative patients, a level associated with deep barbiturate coma.

The commonest pathological finding is severe diffuse axonal injury, but some patients have severe ischemic brain damage in the cerebral cortex and basal ganglia, while others have both types of lesion (McLellan *et al.*, 1986; McLellan, 1989). There is considerable overlap between the findings in vegetative and severely disabled patients. *Vegetative survival is the long-term outcome when the aggregate of damage in the cortex and subcortical structures is such that there is no longer the critical amount of surviving or connected cortex needed for consciousness.*

Estimates of the incidence and prevalence of vegetative survivors varies considerably according to how soon after injury patients are considered to be in a vegetative state, because many of those vegetative for months after insult will die or recover in the course of the next few months (Table 22.5).

*Head injuries accounted for some 40% of patients found to be vegetative 3–6 months after an acute insult in surveys in Japan and the Netherlands. However, an American review of over 700 published series of patients vegetative 1 month after insult revealed 70% due to head injuries in both adults and children (Multi-Society Task Force, 1994). In the US Trauma Coma Data Bank, 14% were vegetative on discharge from the neurosurgical unit but only 5% at last contact many months later (Levin *et al.*, 1991).* In the three-countries data bank 10% were vegetative at 1 month, 2% at 6 months and only 1% at 1 year (Braakman, Jennett and Minderhoud, 1988). A more recent study in four UK units showed only 6% of severe injuries were vegetative 1 month after injury and 2% at 6 months (Murray *et al.*, 1993). The potential for recovery from the vegetative state is greater after traumatic cases than others. However, a distinction should be made between recovery of a limited degree of consciousness and the restoration of useful function. Most patients whose recovery begins many months after being vegetative

either do not speak or are capable of only occasional monosyllabic utterances or of obeying simple commands. Some, however, regain some degree of independence, and a few do become able to function on a day-to-day basis without support – the definition of independence on the Glasgow Outcome Scale (= moderate or good). By this standard 10% of the patients vegetative 1 month after severe injury in the three-countries study were independent by 1 year, but such recoveries were limited to patients under 40 years of age and were twice as frequent in those under 20 years (Braakman, Jennett and Minderhoud, 1988). After 3 and 6 months in a vegetative state 20% and 16% of patients in this series became conscious but none became independent. *In the aggregate of 754 published cases of patients vegetative 1 month after head injury a quarter of both adults and children became independent at 1 year but for those vegetative at 3 and 6 months, only children had a reasonable chance of becoming independent.* It is now widely accepted that, although occasional patients regain some limited consciousness after a year, it is reasonable to consider the vegetative state as permanent after 1 year following treatment.

Declarations to this effect have been made by, *inter alia*, the American Medical Association (1990), the American Neurological Association (1993), the British Medical Association (1993), Medical Council of New Zealand (1993) and the Multi-Society Task Force (1994). Some consider 6 months long enough to wait in adults, particularly in those over 50 years of age.

Whilst almost 50% of patients vegetative a month after head injury are dead by the end of a year, those still alive then may survive for long periods. Several have been recorded for 15–20 years and some even longer. Death is eventually usually due to pulmonary or urinary tract infection.

In view of the potential for recovery in the early months it is important to maintain full supportive care and active efforts at rehabilitation until the condition is considered to be irreversible. It is then usual to

Table 22.5 One-year outcome of patients vegetative 1, 3 and 6 months after head injury (Source: derived from data in Multi-Society Task Force, 1994)

| | Dead (%) | Vegetative (%) | Severely disabled (%) | Independent (%) | Conscious (%) |
|-------------------------------|----------|----------------|-----------------------|-----------------|---------------|
| Vegetative at 1 month | | | | | |
| Adults (<i>n</i> = 434) | 33 | 15 | 28 | 24 | 52 |
| Children (<i>n</i> = 106) | 9 | 29 | 35 | 27 | 62 |
| Vegetative at 3 months | | | | | |
| Adults (<i>n</i> = 218) | 35 | 30 | 19 | 16 | 35 |
| Children (<i>n</i> = 50) | 14 | 30 | 24 | 32 | 56 |
| Vegetative at 6 months | | | | | |
| Adults (<i>n</i> = 123) | 32 | 52 | 12 | 4 | 16 |
| Children (<i>n</i> = 28) | 14 | 54 | 21 | 11 | 32 |

decide not to treat infection with antibiotics and to have a 'do not resuscitate' order. In spite of this, prolonged survival is not unusual and the question of discontinuing tube feeding arises. A number of medical bodies and more widely based ethical committees have declared that this is a morally acceptable course of action, amounting to the withdrawal of treatment that is futile in that it brings no benefit to the patient. Many courts in the US, including the Supreme Court, have sanctioned this, declaring that tube feeding is medical treatment, as have the High Court, Appeal Court and the House of Lords in England (Jennett, 1992b; Dyer, 1993). While the US bench recommends that such decisions now be made by doctors in consultation with families, a recent House of Lords Committee (1994) considers that each such case should come for judicial review and suggests a new form of Court to deal with such cases. It is, however, only a small proportion of families who request such treatment withdrawal even though they accept that there is no prospect of recovery (Tresch *et al.*, 1991).

22.7 Neurophysical sequelae in conscious survivors

The true frequency of various kinds of deficit at various intervals after injuries of differing severity is difficult to determine, because most reports are based on series of patients who have been referred to neurologists, otologists or ophthalmologists because of persisting complaints. Moreover many deficits are temporary and resolve during the first few months after injury.

Persisting neurological deficits in the limbs are common after severe injuries. Three main patterns of neurological disability were found in a series of over 300 patients followed for 20 years after injuries severe enough to have caused unconsciousness or post-traumatic amnesia of a week or more (Roberts, 1979). Hemiparesis was the main disability in 40%, although it was slight in the majority of these. Some 20% had a brain-stem syndrome with asymmetrical cerebellar and pyramidal signs. In 5% the state was described as

athetoid pseudobulbar, a combination of bilateral pyramidal and extrapyramidal signs. About 25% of this series had no neurological deficit; the others had abnormalities outside this classification.

Neurophysical disabilities 6 months after severe head injury in 150 Glasgow survivors who regained consciousness (Jennett *et al.*, 1981) were most often related to dysfunction in the cerebral hemispheres and in the cranial nerves (Table 22.6). The brain-stem and pseudobulbar syndromes described by Roberts (1979) were seldom encountered, but his finding that a quarter of the patients had no neurological abnormalities was confirmed.

22.8 Cerebral hemispheres

Pathological evidence indicates that the brunt of the impact damage from blunt injury falls on the cerebral cortex (contusions) and on the subcortical white matter (shearing lesions). Secondary ischemic damage is common in fatal cases, most often affecting the cortex and the basal ganglia, and this probably also affects some survivors. Many patients who remain disabled have had an intradural hematoma, which caused secondary focal brain damage in one cerebral hemisphere. Of 372 consecutive head injuries of all severities discharged from the Newcastle neurosurgical unit 15% had hemiparesis when they left hospital, which was severe in 4% (Cartlidge and Shaw, 1981). More than half these patients had no deficit 2 years later, and only two of the originally severe deficits were still severe. In a large series of patients with compound depressed fracture in Glasgow focal signs of damage to the cerebral hemisphere were found soon after injury in 20%, but only half of these had residual deficits 6 months after injury. Almost two-thirds of patients recovering after evacuation of an acute intracranial hematoma in Glasgow had hemiparesis soon after operation, but 6 months later only half of them still had a deficit.

In 935 cases of severe head injury in the International Data Bank study cerebral hemisphere damage was clinically evident during the acute stage in

Table 22.6 Neurophysical sequelae (%) at 6 months after severe injury (coma \geq 6 hours) (Source: from Jennett *et al.*, 1981)

| | All cases (n = 150) | After intracranial hematoma (n = 77) | No intracranial hematoma (n = 73) |
|-------------------------------------|------------------------|---|--------------------------------------|
| Any cerebral hemisphere dysfunction | 65 | 62 | 67 |
| Cranial nerve palsy | | | |
| All cases | 37 | 38 | 36 |
| As only sign | 13 | 10 | 15 |
| Ataxia | 9 | 4 | 14 |

89%; a third of these (28% of all cases) had evidence of bilateral hemisphere damage. In the 150 Glasgow survivors 6 months after severe injury, 49% had hemiparesis and 29% dysphasia, while 21% had both (Jennett *et al.*, 1981). Hemianopia occurred in 5%, usually in association with other signs of hemisphere damage. About half the 150 patients had had an intracranial hematoma removed.

22.9 Cranial nerve deficits

In the 150 severe patients assessed 6 months after severe injury, cranial nerve palsies were found in 32% and in 14% of the series these were the only persisting signs (Jennett et al., 1981).

22.9.1 ANOSMIA

Loss of sense of smell occurs in about 5% of all patients admitted to hospital with a head injury. The incidence is higher (20%) in patients who have been unconscious, but a fifth of one large series of patients with anosmia had never been unconscious (Zusho, 1982). The site of injury was occipital in 30%, facial in 20%, frontal in 19% and temporoparietal in 15%. Less than half the cases have a fracture, but anosmia occurred in about 50% of patients with CSF rhinorrhea from an anterior fossa fracture; after surgical repair 80% were anosmic. Recovery rates of 15–50% are quoted, a variation that may reflect how carefully the deficit was sought soon after injury. Recovery after 3 months is rare, and long-delayed recovery suggests a central lesion.

The significance of anosmia for the patient can easily be underestimated. Anosmia can rightly form a basis for compensation, not only for the loss of many of the pleasures of life but for interference with occupation (e.g. in cooks, food handlers and tasters of wine and tea); also for loss of the ability to detect dangerous smells (e.g. of escaping gases or of burning). The most reliably recognized test odors are coffee, tar, oil of lemon, and almond (benzaldehyde).

22.9.2 VISUAL PATHWAYS

These may be affected anywhere from the retina to the calcarine cortex. Penetrating injuries may affect any part of the system and are the only frequent cause of lesions to the optic radiation. In a prospective survey of 363 patients with midfacial fractures 56 (15%) had temporary or permanent visual loss (Al-Qurainy *et al.*, 1991). In a review of 1800 cases of head injury in hospital optic nerve lesions were detected in 3.6% (Rowbotham, 1964). Blunt injuries affect the intracanalicular part of the optic nerve five to 25 times more often than the chiasm. With optic nerve lesions

in the canal it is often difficult to show a fracture, but there is frequently a nearby orbital or anterior fossa fracture. Autopsy has shown hemorrhagic, ischemic and shearing lesions in the optic nerve (Heinze, 1969). Usually there is complete monocular blindness of immediate onset (with an unreacting pupil). Recovery is rare and the disc usually becomes pale within 3–4 weeks. The mechanism of damage is probably ischemic and no more than 20–40% show any recovery.

Chiasmal lesions cause bitemporal hemianopia, probably owing to ischemia of the vulnerable central part of the chiasm; like nerve lesions these are usually present immediately after injury and neither progress nor improve. Lesions of the calcarine cortex are not uncommon, but temporary hemianopia or cortical blindness may be overlooked. Cortical blindness is often delayed for hours or days after injury and may last for only hours or days. The delay in onset and the temporary nature of the disorder suggests hypoxia or ischemia. Cortical blindness that is marked and persistent is often associated with aphasia and agnosia.

22.9.3 DISORDERS OF THE OCULOMOTOR NERVES AND CONNECTIONS

In the acute stage after injury, temporary abnormalities of eye movements are common. Patients who are in coma for hours or days may have dysconjugate roving or reflex (vestibulo-ocular) eye movements, which return to normal as consciousness is regained, probably reflecting transient dysfunction in the brain stem rather than structural lesions that will lead to sequelae.

Diplopia is common after recovery from the acute stage of head injury. Often, the problem lies in the orbit and need not indicate intracranial damage, nor even involvement of the cranial nerves. Even minimal dislocation of the globe or mechanical restriction of movement can produce ocular imbalance, as a result of orbital fractures, blood, edema or the escape of air or CSF into the orbit, and the ocular muscles or their nerve supply may also be involved. When no definite mechanical or neurological lesion can be found this symptom frequently responds to orthoptic treatment, as it is probably due to breakdown of an existing latent squint.

In a review of 170 ocular nerve palsies due to trauma the sixth nerve was affected in 34%, the third in 30%, the fourth in 15% and more than one in 22% (Rucker, 1966). Third-nerve palsy may be the result of impact injury or (more often) of tentorial herniation. Impact lesions are most often in the superior orbital fissure. Recovery is the rule, but upward movement may remain restricted; aberrant regeneration may result in lid elevation when eye movement is initiated.

Sixth-nerve palsy is usually associated with fracture of the petrous temporal or sphenoid bones, but can occur because of phenytoin intoxication. Recovery is usual. When squint persists from any nerve palsy after 6 months, muscle shortening surgery may improve appearance and it may also restore binocular vision.

22.9.4 SEVENTH AND EIGHT CRANIAL NERVES

These nerves or their end-organs are frequently damaged by petrous fractures (Healy, 1982; Togliola and Katinsky, 1976). Most are longitudinal or horizontal, the fracture running parallel to the long axis of the petrous bone in front of the nerves, but often damaging the middle ear. Transverse fractures occur with more severe injuries, and run at right angles to the petrous axis, often disrupting the bony and membranous labyrinth, the inner ear and the facial nerve.

(a) Facial palsy

With transverse fracture the nerve is disrupted and the paralysis is usually immediate and complete and is often permanent (Potter and Braakman, 1976). With longitudinal fractures paralysis is often delayed by 2–3 days, and is incomplete and temporary, with recovery over a period of 6–8 weeks. Surgical decompression is frequently recommended but most clinicians consider that intervention is seldom justified, since most delayed palsies recover (at least partially) while most immediate lesions remain permanent whatever is done.

(b) Vestibular dysfunction

Transverse fractures that have caused disruption of the labyrinth and utricle usually result in severe vertigo and spontaneous nystagmus for 6–12 weeks, until there is compensation. Testing series of patients months after head injury reveals many with eighth-nerve dysfunction. Some have clinically evident nystagmus, but many more patients have abnormalities when electronystagmography is employed. These data come from series of patients sent to otologists with persisting symptoms long after injury. The severity of injury is seldom stated and it is difficult to assess the frequency of these abnormalities in unselected head injuries of different severities.

(c) Hearing loss

Many patients with severe head injury have some hearing loss, usually sensorineural, and often associated with a transverse fracture; it may be bilateral. Sensorineural impairment also occurs without a fracture, probably due to concussive damage to the organ

of Corti. It resembles damage due to high-intensity noise, affecting the high-frequency range. It may be temporary and missed without early testing. In severely injured patients who cannot cooperate, brainstem evoked response audiometry can be helpful; even if sedation or anesthesia is required to perform this test the response is not affected (Hall, Huang-fu and Gennarelli, 1982).

Conductive hearing loss is much more common because it can occur after less severe injuries, is often temporary and is related to hemotympanum or a lacerated drum. However, the recognition of ossicular chain damage is important because this can often be corrected surgically, if deafness persists.

22.10 Delayed complications

Head injury is so common that it is inevitable that some patients who develop one or other of a wide variety of neurological conditions will be found to have previously sustained an injury. Sometimes, when there is a valid causal relationship (e.g. epilepsy or meningitis), the association with injury may not be recognized by the clinician. The patient may not mention the head injury because the patient sees no connection with his present complaint, or the clinician may dismiss a known head injury as unrelated to the present condition either because it had been relatively mild or was so long ago. *However, epilepsy declares itself more than 4 years after injury in 25% of cases of traumatic epilepsy, while meningitis related to a basal skull fracture can occur 10 years or more after injury.*

22.10.1 POST-TRAUMATIC HYDROCEPHALUS

The availability of CT scanning is making known the frequency and extent of ventricular dilatation after various kinds of head injury and the natural history of this condition (Cardoso and Galbraith, 1985). Three kinds occur; that caused by wasting of the white matter after severe injury (*ex vacuo*); that caused by acute impairment of the circulation of the CSF, probably secondary to bleeding (obstructive); and chronic communicating hydrocephalus (normal pressure). A firm diagnosis of normal pressure hydrocephalus is made only when a patient who has already recovered to a considerable degree from injury develops new symptoms (mental impairment and disorder of gait); diagnosis depends on these characteristic clinical features and on evidence of retarded CSF circulation. *In a large study of hydrocephalus of all kinds after head injury, only a quarter of those diagnosed as having normal-pressure hydrocephalus responded well to shunting (Zandler and Foroglou, 1976).*

22.10.2 LATE TRAUMATIC EPILEPSY

This is by far the most frequent of delayed complications, although it occurred in only about 5% of all patients admitted to hospital in the UK after non-missile head injury (Jennett, 1975). An epilepsy rate of 2.5% was recorded for a large series of patients in Olmstead County, but this included some who did not attend hospital and many who were not admitted (Annegers *et al.*, 1980). After some types of injury the risk is much higher (see below). *Of 150 severe injuries followed for more than a year after injury, 17% had epilepsy (Jennett et al., 1981); this would certainly have been higher had the follow-up been longer. The incidence was twice as great in those who had had a hematoma and in those with severe disability; 20 of 22 severely disabled patients with epilepsy had either an intracranial hematoma or a depressed fracture.*

The significance of epilepsy for the patient depends on whether he/she has other disabling sequelae and on how it impinges on his/her particular life-style. Epilepsy was the only physical disability in almost half the patients in whom it occurred after severe injury. Many are young men on the threshold of their careers whose future options can be appreciably limited by the occurrence of epilepsy, even by the threat that it may develop. Many patients regard the restriction on car driving that epilepsy entails as one of the most disabling aspects of this complication, even for those who are not vocational drivers.

(a) Time of onset

Seizures in the first week are recognized as a distinct category (early epilepsy). The proportion of patients regarded as having begun to suffer from traumatic epilepsy within a year of injury depends on whether early fits are counted and on how long the patients are followed. In a study of 481 patients with late epilepsy (Jennett, 1975), 56% had their first late fit in the first year (27% within 3 months of injury). When there had been early epilepsy the late fits more often began in the first year (74%). About a quarter of cases in this series had their first late fit more than 4 years after injury.

(b) Type of fit

About 40% of patients with late epilepsy have at least some fits with focal features. Over 70% of patients have attacks in which they become unconscious. A fifth of patients have seizures with temporal lobe features and when these attacks begin they may not be recognized for some time as being epileptic in origin. Petit mal has not been encountered after injury.

(c) Persistence of fits

It is crucial for the patient's future to know whether or not fits are likely to persist once the first late seizure has occurred. There are several references to post-traumatic epilepsy having died out, but most were of wartime missile injuries; this led to the concept that seizures were a manifestation of a certain stage in the healing process, and therefore temporary. *Remission of epilepsy is a safer term than cessation; 2 years without fits is a reasonable (if arbitrary) definition of remission.* Clinicians frequently recommend discontinuation of anticonvulsant drugs in adults after 2 years without fits. However, even a remission of 2 or more years is frequently followed by reappearance of post-traumatic fits. It has to be accepted that once a patient suffers even one late fit there is a high probability that he/she will continue to have epilepsy, although this may be relatively well controlled by anticonvulsants and there may be remissions.

(d) Prediction of fits

Because this complication occurs relatively seldom and may not develop until years after injury, there is a premium on the ability to predict the likelihood of its occurrence, in order to advise the patient about his future, and to guide lawyers concerned with claiming compensation for injury.

The findings of a study of over 800 patients with traumatic epilepsy following non-missile head injury have now been confirmed by several observers in other countries (Jennett, 1975). *Three factors increase the risk of late epilepsy significantly: an acute intracranial hematoma evacuated within 2 weeks of injury, an early fit (within the first week) and a compound depressed fracture of the vault (Table 22.7).* The risk is greater after surgery for an intradural (45%) than after an extradural hematoma (22%). When a significant intracerebral hematoma is detected by CT scanning but surgical evacuation has not been necessary, the epilepsy rate is only 23%.

After a compound depressed fracture, the risk of late epilepsy varies according to four risk factors: PTA

Table 22.7 Factors increasing the incidence of late epilepsy (Source: from Jennett, 1975)

| | <i>n</i> | % |
|-----------------------|----------|----|
| No hematoma | 27/854 | 3 |
| Hematoma | 45/128 | 35 |
| No early epilepsy | 29/868 | 3 |
| Early epilepsy | 59/238 | 25 |
| No depressed fracture | 27/832 | 3 |
| Depressed fracture | 76/447 | 17 |

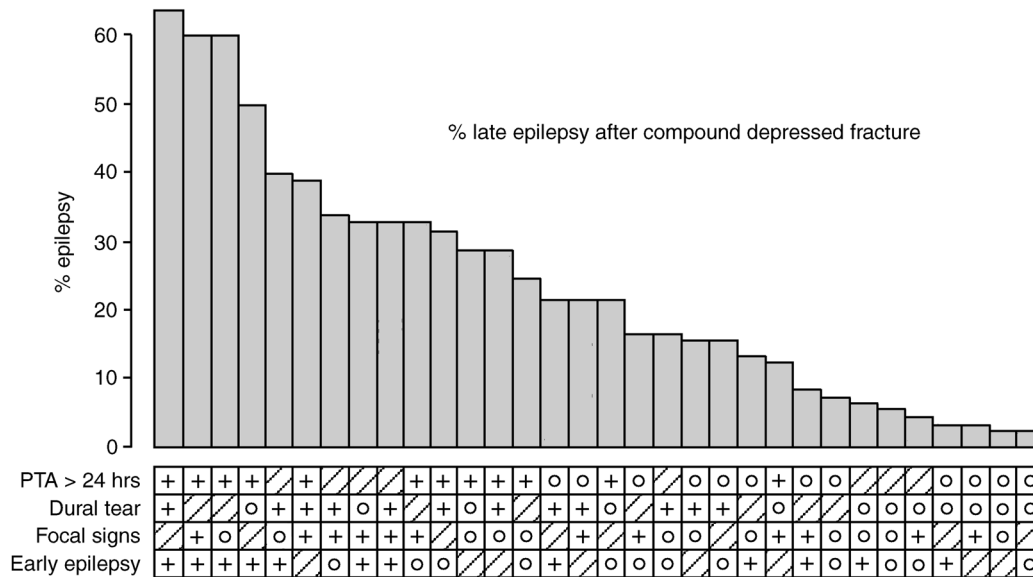


Figure 22.1 Risks of late epilepsy after compound depressed fracture with varying combinations of factors where three are known. (Reproduced with permission from Jennett, *Epilepsy after Non-Missile Head Injury*. Published by Heinemann, 1975.)

exceeding 24 hours, early epilepsy, dural tearing and focal signs. Various combinations of the risk factors enable the clinician to identify patients whose risks of late epilepsy vary between 3% and more than 60% on discharge from hospital after injury (Figure 22.1). High-risk combinations occur relatively seldom and some 40% of patients with depressed fracture can now be reassured that their risk of epilepsy is less than 5%.

In patients with neither a depressed fracture nor an acute intracranial hematoma the risk of epilepsy is low, unless there has been an early fit. This applies whether or not there has been prolonged unconsciousness (PTA > 24 h). The risk of late epilepsy is increased even when early epilepsy was confined to a single fit, and whether the first (or only) early fit was in the first hour after injury or later during the first week. Although

children are somewhat less liable to develop late epilepsy after an early fit, the risk is still significant. The risks of late epilepsy are summarized in Table 22.8, which can be regarded as an algorithm – if no hematoma, was there a depressed fracture; if neither, then was there early epilepsy? The EEG is not helpful in predicting late epilepsy (Terespolsky, 1972).

(e) Prophylactic anticonvulsant therapy

There is increasing doubt about the effectiveness of anticonvulsants in preventing, as distinct from temporarily suppressing, traumatic epilepsy. An attempt to establish therapeutic levels of phenytoin within 24 hours of injury by intravenous and intramuscular injections, followed by oral maintenance checked by

Table 22.8 Summary of risks of late epilepsy

| | | |
|---|---------------------------------|------------------|
| Acute intracranial hematoma | Intradural | Operated 45% |
| | | Not operated 23% |
| | Extradural | Operated 22% |
| Compound depressed fracture | Early epilepsy + PTA > 24 hours | > 50% |
| | Other pairs of risk factors* | 20–40% |
| | Only one risk factor | 5–20% |
| | No risk factors | < 3% |
| Neither hematoma nor depressed fracture | Early epilepsy | 26% |
| | No early epilepsy | < 2% |

*Risk factors: PTA > 24 h, early epilepsy, dural tearing, focal signs

blood levels for 1 year, proved difficult and no convincing benefit could be shown (Young, Rapp and Perrier, 1975). Another trial of phenytoin for post-craniotomy cases, including many with head injury, showed a modest reduction in epilepsy at 1 year. Therapy was then discontinued and by 2 years there was no significant difference between those who had been treated during the first year and those who had not (North *et al.*, 1983). More recent studies on prophylaxis after intracranial surgery for non-traumatic conditions raise further doubts about the efficacy of prophylaxis with phenytoin or carbamazepine, and they reveal a considerable incidence of side effects (Foy, Chadwick and Rajgopalan, 1992). Another controlled trial of phenytoin showed no reduction in post-traumatic seizures (Temkin *et al.*, 1990).

22.11 Mental sequelae

Coma represents the initial disorder of mental functioning after severe head injury, and its depth and duration indicate the severity of diffuse brain damage. Once the patient comes out of coma (opens eyes, speaks or obeys) he remains in a state of disordered consciousness for a much longer period than he was in coma, and is always amnesic for this period (post-traumatic amnesia, PTA). Most patients in coma for 6 hours or more have a PTA of a week or more, half of them of a month or more. *Duration of PTA correlates with ultimate outcome – only patients with a month's PTA remain severely disabled, but a quarter of these patients with a long PTA make a good recovery. However, in patients whose PTA exceeds 3 weeks it is almost always possible to detect impairment of performance on some tests of cognitive function 6 months after injury, and some measurable deficit is often permanent.* Changes in personality are more frequent than altered intellectual function and although they can be equally disabling they are less readily measured. The most frequently encountered mental sequelae are probably related to widespread rather than to focal brain damage. This is consistent with the wide distribution of initial axonal lesions and of secondary hypoxic damage in the brain. If disorders of language and of visuospatial perception are regarded as neurophysical, mental sequelae related to focal brain damage are not common. Some patients, however, do develop features characteristic of frontal lobe damage, while the frequency of memory disorder may be related to the predominance of damage to the temporal lobes. After blunt head injury, however, damage is seldom confined to one lobe, or even to one side of the brain; it is therefore unwise to over-emphasize the localized lesions. What matters is brain damage that persists. Neuropsychological deficits 15–18 months after injury correlate more closely with

late MRI abnormalities than with those seen on MRI or CT soon after injury (Wilson *et al.*, 1988).

Few head-injured patients develop major psychoses but many have reactions such as depression or anxiety as well as behavioral disorders. It seems preferable to use the descriptive term 'mental' to cover these various features and to avoid the terms 'psychological', 'functional' or 'psychiatric', each of which implies a specific (and unverifiable) explanatory interpretation.

22.12 Deficits of intellectual (cognitive) function

The availability of large numbers of tests of IQ in general, and of subtests for different psychological functions, has led to many reports about the range of abnormalities that can be found after head injury. Many of these tests depend heavily on verbal ability and these put at a disadvantage both patients with lesions in the left hemisphere and those with educational limitations affecting their pretraumatic vocabulary and capacity to manipulate language. Performance on IQ tests tends to reflect what has been accumulated over a lifetime by way of intellectual habits, motivation and cultural expectations. This is the so-called 'crystallized' intellectual ability, as contrasted with the psychophysiological adequacy of the brain to solve new problems at present ('fluid' intelligence). It can, however, be difficult to judge what the functional capacity of an individual's brain was before injury. School performance gives some guide while occupational status provides a crude measure for adults. Vocabulary allows an approximate retrospective assessment because simple verbal tests tend to show little impairment after injury, probably because they test overlearned skills. Non-verbal tests (e.g. part of the Wechsler Adult Intelligence Scale – WAIS – and the Ravens Progressive Matrices) depend on visuospatial ability and on motor performance. But they also test the ability to reason at the time of testing; their value lies in their independence from educational and cultural influences prior to injury. On the other hand, they may be affected by focal brain damage to the non-dominant cerebral hemisphere that has caused perceptual and psychomotor deficits.

The balance between focal and general brain damage differs according to the kind of injury. We owe much of our knowledge about the effects of strictly localized brain damage to the detailed psychological follow-up and testing of military head injuries caused by missile fragments. By contrast, all the evidence from pathological examination of brains that have sustained blunt injury indicates that the damage is usually widespread, although there may be accentuation in certain areas. A study that compared the

psychological test deficits in patients with intracranial tumor, with cerebrovascular accidents and with craniocerebral trauma showed more similarities than differences between these different groups of patients (Reitan, 1973). However, those with head injuries whose brain damage was judged by neurological examination to have been unilateral showed cognitive deficits that were indicative of bilateral pathology more often than did patients in the two other diagnostic groups. Psychological deficits indicative of lesions in the opposite side of the brain from the primary impact were often found in another series of brain-damaged patients who were tested many years after injury (Smith, 1974). *Studies of Vietnam veterans have shown that the more global the cognitive deficit the greater the importance of the volume of tissue loss, whereas lesion location is significant for focal deficits (Grafman et al., 1986).*

Verbal abilities are not only less severely affected in the early stages than are performance IQ tests but they also recover more rapidly; verbal scores have usually largely recovered (if they are going to) within 3–6 months, whereas performance IQ may go on improving for a year or more. Performance tests are more severely impaired, probably because they depend on a wider range of cerebral activities and on the integration of these. They also reflect other aspects of higher mental function, such as motivation and attention, speed of performance and perseverance as well as the ability to organize complex tasks over a period of time. Most complaints by patients and their relatives are in the areas of fluid intellectual function and in memory. Routine IQ testing often fails to demonstrate abnormalities in patients who are clearly not performing normally at home, because the tests commonly used were not designed to discover these kinds of alteration in mental activity. Patients may also perform better during the brief period and relatively structured situation of psychological testing than they do in real life; this applies particularly to patients with frontal damage.

Recently psychologists have begun to focus on various general aspects of mental activity and to devise appropriate means of testing these (Brooks, 1984; Levin, Benton and Grossman, 1982). Tests of attention and vigilance include reaction time to visual and auditory stimuli, and recognizing and checking off repeated letters or words in lists. These may be applied for varying periods (to show fatigue effects) and with the addition of various degrees of distraction. There is evidence that the ability to screen out irrelevant information in order to focus on the task at hand may be one of the mental skills that patients with diffuse brain damage lose. Other tests that require the integration of many different aspects of brain function and may therefore be sensitive indicators of widespread brain

damage are the recognition of faces, the completion of half-finished pictures and the recognition of anomalies in sketches of various life situations. These tests all depend heavily on the integrity of the non-dominant parietal lobe and may be impaired by local damage in that location.

The ability to learn new tasks is another aspect of brain activity that it is appropriate to measure. This may provide a better indication of the state of the brain than the capacity to reproduce previously overlearned material, or to carry out simple tasks, or to solve problems one at a time in a test situation. Indeed, *many patients can continue to undertake activities that were previously routine for them, including their work, but are unable to tackle new tasks or to learn new skills. There is a similarity between the effects of head injury and of the normal aging process; it is characteristic of the elderly that they can perform well in a routine and familiar environment, but react badly to new situations.* If recovery in the brain is partly a learning process it might be expected that the ability to learn after a head injury would correlate with the capacity to recover function as a whole. Learning depends to some extent on memory but this is a function so specifically affected after head injury, sometimes out of proportion to other cognitive defects, that it is considered separately later.

This raises the difficult question of the interdependence of different mental functions and the extent to which one may be affected independently of others. Because blunt head injury produces widespread damage, dysfunction in the brain as a whole is important, but cognitive tests are mostly concerned with certain focal deficits. Although focal dysfunction rarely occurs without some deterioration of mental function as a whole, one or other of these focal deficits (such as memory) may be predominant. It is, however, important not to conclude mistakenly that a patient has general intellectual deterioration when his difficulties stem largely from a specific defect. Research on focal deficits has largely depended on studies of missile injuries and of patients recovering from ischemic strokes, both of which can produce focal lesions without involvement of the brain as a whole. But left-sided lesions can also lead to perceptual deficits (difficulty in figure ground discrimination) and to deficits in certain motor functions, such as copying complex gestures.

22.12.1 LEFT HEMISPHERE LESIONS

Even when patients with clinically detectable dysphasia are excluded, patients with lesions in the dominant hemisphere tend to have particular difficulty with various cognitive tests (Grafman *et al.*, 1986). In some

patients deficits in verbal skills that were not obvious in ordinary speech become obvious when learning and retention of verbal material are tested.

22.12.2 RIGHT HEMISPHERE LESIONS

Some patients with these lesions have clear topographical disorientation or have difficulty in recognizing faces. These deficits may in turn affect memory, in that initial registration is impaired because of the perceptual difficulty. In others the visuospatial difficulties may be so subtle as to require highly specialized tests to uncover them.

22.12.3 FRONTAL LOBE SYNDROMES

The importance of frontal lobe damage is being increasingly recognized. Although its effect is primarily on behavior or personality there is a secondary influence on cognitive performance in many types of test (Stuss and Benson, 1984). Three main types of behavioral change are seen with some correlation with the location of the damage in the frontal lobe. Lesions in the dorsolateral region affect the ability to plan and to correct errors when undertaking complex tasks (such as are tested by mazes). There is a tendency to tackle problems with a fixed strategy with an inability to innovate or change direction in response to the demands of different tasks or failure to succeed with one. *The disinhibition or defect of social restraint that is often considered a classical sign of frontal lobe damage is associated with lesions in the basal or basomedial region. Medial lesions cause lack of drive and motivation, features that clearly impinge on performance tests and on social life. Bilateral lobe damage produces more marked abnormalities.*

22.12.4 MEMORY DEFICITS

Considering the universality of PTA it is not surprising that some disorder of memorizing is a persistent complaint of many patients and is often of concern also to their families. Such reports should not always be taken at their face value because what is loosely described as a bad memory may prove to refer to more generalized cognitive deficit or even to dysphasia ('forgetting names'). Nonetheless, two-thirds of patients 5 years after severe injury complained of their memory in one study. *The phenomenon of PTA indicates that in recovery from unconsciousness the capacity to lay down on-going memory is usually the last function to return.* This may be because it is one of those processes that depends on the integration of several aspects of brain function; it requires that the mechanics of perception be intact and that attention be adequate, so that images are clearly received. Little is known about what is needed to ensure encoding of the

'memory', its persistence, and its availability for retrieval when required; but all are aspects of information-processing, a function that is consistently slow after head injury (Lezak, 1979).

A distinction should be made between recent and remote memory. It is a familiar feature of the elderly demented patient, who cannot remember from day to day or even hour to hour, that he/she can often vividly recall his/her childhood; the same occurs after head injury. Short- and long-term recent memory should also be distinguished; after head injury it is recall over the long term (over half an hour or more) that is impaired. Even patients with devastating deterioration on this time-scale may retain short-term memory (e.g. repeating digits correctly). But even this short-term memory may break down if too much information is presented and the system is overloaded. These patients are also slow at learning because this partly depends on memory, although after much effort and extra time they may eventually achieve a near-normal proficiency. Learning also depends on motivation, attention, information processing and planning – each of which is often affected after head injury.

Failure of recall may be because the 'memory' was never imprinted, or has decayed, or cannot be retrieved. In patients with prolonged retrograde amnesia, there is usually recovery of much of the memory of events that happened prior to the injury, and this indicates that the problem was a defect of retrieval. By contrast there is usually permanent loss of memory for seconds or a minute or so immediately prior to impact, the trace of those happenings presumably never having been imprinted. This is certainly the case with post-traumatic amnesia, which remains stable and which does not yield to attempts to uncover it by abreaction or drugs; such techniques can sometimes accelerate the return of the more distant events that are part of retrograde amnesia.

The question of how discrete memory loss can be without there being parallel deficits in cognitive function is a matter of dispute among psychologists. Of 87 moderate or severe injuries 25% had defective auditory and pictorial memory despite normal Wechsler verbal and performance scores; the deficit was reminiscent of alcoholic Korsokoff amnesia (Levin *et al.*, 1988). In the context of blunt head injury that causes widespread brain damage, marked memory disorder is usually associated with some impairment on standard IQ tests. Some of these tests are themselves directly affected by memory dysfunction and some include specific memory subtests. Even when these are allowed for, however, there are patients with severe memory loss whose routine psychometric test results are otherwise normal; they usually have discrete lesions that affect the temporal lobes bilaterally. Non-traumatic

examples are encephalitis and temporal lobectomy, while bilateral temporal lobe damage is common in head injury.

Several reports (Lezak, 1979; Brooks, 1989) from series of patients with severe non-missile head injuries reveal a good correlation between the duration of PTA and the degree of persisting memory defect in patients with more than a week's PTA. But neither skull fracture nor focal neurological signs (including dysphasia) are related to memory impairment, and neither is the score on standard WAIS IQ tests. Recovery of memory function (to the level of a stable deficit) occurs relatively rapidly once the patient is out of PTA but there is seldom any significant improvement after 6 months.

22.12.5 CONCLUSIONS ABOUT COGNITIVE TESTS

There is considerable overlap in the functional effects of separate deficits in cognition, while behavioral changes can also affect performance on tests. Brooks and McKinlay (1983) have suggested a simplification of Prigatono's classification of deficits, as follows:

- learning and memory;
- complex information processing;
- perception and communication.

Although broad correlations can be found between measures of severity of injury and the location of the lesion with the cognitive deficits found, as well as with their rate and degree of recovery, there are wide variations between patients.

In seeking correlations between the site of damage and cognitive deficits new methods of imaging of the brain have been used. The degree of atrophy on CT scan was found to relate well to performance IQ (especially with left frontal atrophy), and to memory quotient (with atrophy in either hemisphere); but atrophy was not related to verbal IQ, representing crystallized intelligence (Cullum and Bigler, 1986). Imaging has its limitations, however, especially if it is assumed that absence of abnormality indicates lack of damage. Studies by MRI in Glasgow showed deep white-matter lesions soon after injury in 30% of a series of patients in whom CT showed such lesions in only 2% (Jenkins *et al.*, 1986). *Neuropsychological tests were frequently abnormal in patients whose lesions had shown only on MRI. But lesions shown 15–18 months after injury correlate more closely with cognitive deficits than do those evident soon after injury (Wilson et al., 1988).* A further caution about assuming focality comes from a study that correlated the frequency of abnormalities on language tests with the apparent laterality of the brain damage (Levin, Grossman and Kelly, 1976). Language deficits were found in only seven of 15 cases judged to have predominantly left hemisphere damage, but in six

of ten whose lesions were considered to be mainly in the non-dominant hemisphere. The greater importance of the volume of brain tissue lost than the location of the lesion after missile injuries has already been discussed.

That the recovery of head-injured patients depends in part on their pretraumatic psychosocial status has been recognized for some time. As a group these patients include a disproportionate number who were already risk-takers, heavy drinkers and unemployed. These factors predict poor social recovery but may also affect performance on cognitive tests. In the Vietnam follow-up study the best predictor of recovery was the preinjury intellectual and educational performance, as indicated by tests carried out at the time of enlistment (Grafman *et al.*, 1986).

The objective of psychometric testing may be summarized as follows. It should allow an accurate assessment of the patient's cognitive behavioral and affective strengths and weaknesses, and the implications for rehabilitation. Moreover, testing regimes should not require prolonged sessions because few severely head-injured patients can complete such tests. Good correlation between cognitive tests and outcome on the Glasgow scale have been shown in two studies (Brooks, Hosie and Bond, 1986; Clifton *et al.*, 1993). *The latter study identified four tests out of 19 that correlated most closely with the Glasgow scale at 3 and 6 months after injury. These were Controlled Oral Word Association, Grooved Pegboard, Trail Making Part B, and Rey-Osterrieth Complex Figure Delayed Recall. Of these Grooved Pegboard accounted for 80% of the variation in the outcome scale.*

22.13 Personality change

This is the most consistent feature of mental change after blunt head injury (Brooks, 1988). In some instances the patient's behavior is clearly abnormal, but in others the change is noticeable only to relatives or close associates; unless they are questioned systematically the doctor may mistakenly believe that the patient has made a complete recovery.

Categorizing a concept such as personality is difficult but it is helpful to consider three aspects of behavior.

Drive is usually reduced and the apathy that results may be described as laziness, or simply slowness. Circumstances may, however, enable a person to carry out his/her work satisfactorily, particularly if this is done in a structured environment. Yet when he/she comes home at night he/she may fail to follow previous leisure pursuits, preferring to dream the evening away in an armchair. In the early stages this lack of drive may be an obstacle to successful rehabilitation, but later it may be dealt with by a near relative acting as a daily goad.

Affect most often changes in the direction of depression, which affects half the patients 2–7 years after injury. Lack of drive and lack of insight may, however, lead patients to passive acceptance of their condition. This may lead them to underestimate their disabilities and to claim that they are better than they really are. More florid aspects of disturbed affect are seen in patients who experience emotional lability. Inexplicable bouts of crying, or less often of laughter, may occur; patients with insight can explain that these represent the outward signs of an emotion that is not mirrored by a corresponding inner feeling. In that event they are more distressing to the onlooker than to the patient. Occasionally a relative will say that a patient is better behaved or easier to live with since suffering a head injury. This will usually be when a previously aggressive individual is now quieter than before.

Social restraint and judgment are qualities that individuals exercise in varying degrees, according to their personality traits and their cultural background. But when a person who is normally well behaved socially, who is sensitive to the needs of others and in control of those inner feelings of dislike and frustration that everyone experiences from time to time, becomes tactless, talkative and hurtful, there is no doubt about the change. Such patients may be no more than a harmless nuisance to those around them, but they may be subject to outbursts of rage that are not only out of character but frightening to the onlooker. It is sometimes questioned whether these might represent episodes of temporal lobe epilepsy, but they can seldom be so explained. More often they result from some trivial frustration that would previously not have led that particular patient to respond in this fashion. The whole picture of lack of social restraint is often referred to as 'childish behavior', reminiscent of a child not yet trained by years of social schooling by parents, relatives, and teachers.

A simple means of scaling personality change used by Brooks (1988) was a five-point scale between two opposing adjectives (Table 22.9). The amount of these

changes were related to the subjective burden recorded by the relatives. More personality change was recorded at 6 and 12 months than at 3 months, perhaps because relatives no longer denied the changes. Preservation of insight into personality and cognitive changes is associated with better prospects for slow improvement over 2–3 years, with possible benefit from behavioral modification techniques.

22.13.1 RELATIONSHIP TO PREVIOUS PERSONALITY

It is useful to obtain an account from relatives about the patient's pretraumatic characteristics soon after injury, when a more unbiased version may be given than when there has been time for reflection about the consequences of injury. Even without the prospect of possible compensation, relatives are apt later to idealize the patient's previous psychosocial status, and this can make it difficult to assess the degree of change. Prior personality can best be assessed using a formal questionnaire; one of the inventories used for self-report may be modified for use by a relative, whose opinion on how the patient would have answered the various introspective questions can then be recorded.

Sometimes the personality change after injury takes the form of exaggeration of that patient's pretraumatic personality traits; or it may be a reversal of them – for example, a person previously quiet, cautious, and kind may become the opposite. It has been suggested that such patients may have been unduly dependent for their previous 'model behavior' on the exercise of marked degree of restraint, probably dependent on the frontal lobes. However, there is no consistent relationship between premorbid personality and the kind of change that follows trauma. Nor is there often a clear relationship between the type of change and the site of brain damage, although patients with frontal damage without prolonged coma sometimes show a degree of change that is more marked than would be expected from the severity of the diffuse damage.

Table 22.9 Assessing personality change by five-point scale between pairs of adjectives (Source: derived from Brooks, 1988)

| | |
|----------------------------------|------------------------------|
| Talkative – Quiet | Down to earth – out of touch |
| Even-temper – quick-temper | Rash – cautious |
| Relies on others – self reliant | Listless – enthusiastic |
| Affectionate – cold | Mature – childish |
| Likes company – dislikes company | Sensitive – insensitive |
| Irritable – easy going | Cruel – kind |
| Unhappy – happy | Generous – mean |
| Excitable – calm | Unreasonable – reasonable |
| Energetic – lifeless | Stable – changeable |

22.13.2 REACTIVE AFFECTIVE (PSYCHIATRIC) SYMPTOMS

To suffer a head injury, even a brief concussion, is a significant experience for anyone. When the incident is mild the patient recovers sufficiently rapidly to remember the scene of the accident, the crowd around him/her, the ambulance, the accident department, and admission to hospital. *By contrast, the more severely injured patient wakes up in hospital, often after several days or sometimes weeks about which his/her mind remains forever blank. The patient finds around him/her relatives who, unbeknown to the patient, have been fearing for his/her life but who are now concerned for his/her sanity.* There may be major physical problems, either related to the brain damage or to associated injuries. But insight into the situation as a whole and its implications for the future seldom develops for some weeks or months. For the moment living day to day is enough. *Only when the patient goes home do he/she and his/her family realize the magnitude of the effects that a severe brain injury has on life as a whole.* At this stage improvement can usually still be recognized on a week-to-week scale but as this process slows down the probability of permanent disability becomes increasingly apparent.

Both the patient and his/her family then enter a new phase of reaction to the situation. Some of the more severely affected patients are so blunted or euphoric that they do not appreciate their plight. But others at this stage become not only aware but also distressed by their condition. They may react to this by frustration and anger, placing blame for their shortcomings either on their relatives or on the doctors and therapists who are trying to help them. Others again become depressed but many deal with the situation by denial of disability, particularly of cognitive and memory deficits that are all too obvious to others. *The relatives may likewise react with frustration, depression or denial, and the psychodynamics of the family can become crucial once the patient returns home (Livingstone, Brooks and Bond, 1985a, b; Brooks, Campsie and Symington, 1986; Brooks et al., 1987).* Prior counseling of families can prepare them for the nature and the time scale of the problems that they may have to face. *It has long been realized that it is the mental disability (cognitive and memory deficits and personality change) that has the most serious consequences for social reintegration.* This is because mental handicaps tend to evoke secondary or reactive psychiatric symptoms both in the patient and in his/her family, and these can aggravate the situation.

The ability to adapt and to cope with new environmental stresses is one of the mental capacities that head injury most consistently impairs. It is never an easy matter to adjust a whole lifestyle to a sudden and

catastrophic change such as that which commonly results from severe head injury. But after head injury this difficulty is compounded by the mental component of this change. It is this that makes parallels with severe physical disability (such as paraplegia) so inappropriate. The youth of the patient is another factor that influences reaction to injury; the average age of survivors after severe head injury is under 30, and many are in their late teens or early 20s. The problems of adolescence or of early married life are then compounded with those of brain damage, which may threaten the completion of education and preparation for a career.

22.14 References

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