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Multiple choice questions

1. Depressive disorder following brain injury:
   a rarely responds to antidepressant medication
   b can be easily identified by carers and professionals
   c may be the cause of a global deterioration in someone who was previously improving
   d may be obscured by extrapyramidal problems and aprosodia.

2. Abulia following brain injury:
   a is a form of movement disorder
   b may occur in the context of a depressive disorder
   c is generally improved by major tranquillizers
   d may be improved by dopamine agonists such as bromocriptine.

3. Temper disorders following brain injury:
   a are generally improved by minor tranquillizers
   b are often caused by factors other than malfunctioning brain
   c should in the first instance be treated by major tranquillizers
   d may arise in as a result of psychosis or depression.

4. Hypoglycaemic and hypoxic brain damage:
   a often occur in someone who was depressed at the time of injury
   b generally lead to a uniform pattern of brain damage
   c often result in persistent memory problems
   d can result in bizarre dissociative disorders.

MCQ answers

1	2	3	4
a F	a F	a F	a T
b F	b T	b T	b F
c T	c F	c F	c T
d T	d T	d T	d T

Commentary

Peter Eames

Causes of acquired brain injury

It is helpful to consider two different kinds of acquired brain injury. The more common is stroke, which, like low-velocity penetrating injury, produces severe, often total destruction of a circumscribed area of brain substance. It is remarkable how rarely psychiatric help is sought for those with this kind of injury. The other kind is found in the injuries discussed by Ken Barrett: non-penetrating trauma, subarachnoid haemorrhage, hypoxia, hypoglycaemia and encephalitis. What distinguishes these is the combination of localised and diffuse insults,

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in usually unpredictable permutations of degree and distribution. Because many brain mechanisms are likely to be disturbed, particularly diffuse control mechanisms, the clinical effects are pervasive for the person's experiential, interpersonal and social functioning. Between different categories of this kind of injury, the details of pathogenetic mechanisms do differ, and allow some prediction of the clinical problems to be expected, but it is the shared feature of the combination of localised and diffuse disorders that makes them psychiatrically challenging.

Assessing psychiatric disorders following brain injury

Delirium

This term is virtually never heard in acute hospital settings. It is probably not an appropriate term in the majority of those who are confused after an acute brain injury, because it implies disorders of the sensorium that are relatively rare in this context. The description 'post-traumatic confusion', putting the focus on symptomatic behaviour rather than on a diagnosis, is preferable in most cases. One of the main difficulties confronting psychiatrists in the assessment of patients who have suffered acute brain injury is that their training tends towards finding a 'psychiatric diagnosis', whereas more often than not, a simple behavioural description is more useful and more accurate. The main purpose of diagnosis is to suggest the most appropriate treatment; if diagnosis is pressed into an explicitly psychiatric framework, then psychiatric treatments are likely to be recommended. In the acute stage after traumatic brain injury what really matters for treatment is the patient's behaviour, coupled with a clear recognition of the pharmacological characteristics of the drugs whose use is contemplated. Medical training tends to encourage thinking about drug treatments in terms of therapeutics (in which symptoms and disorders are linked directly with specific drugs) rather than pharmacology (see Eames, 1989). There is then a risk of omitting the step of thinking about all the effects of a drug whenever it is prescribed. This is especially important in the context of acquired brain injury because the recently damaged brain is particularly vulnerable to drugs that block dopaminergic and cholinergic activity. These include many of the drugs of which psychiatrists have most experience and which are most commonly used in the treatment of disturbed behaviour in the context of primary psychiatric disorder.

The drug most commonly prescribed in acute hospital wards for people whose behaviour is disturbed and difficult following brain injury is haloperidol. Not surprisingly this is also the drug most often recommended by junior psychiatrists when a specialist opinion is requested. In the past 10 years, 20 of the referrals to Grafton Manor Brain Injury Rehabilitation Unit because of difficult behaviour had been 'controlled' with substantial doses of haloperidol and other 'typical' neuroleptics. They exhibited severe extrapyramidal motor disorders, most often including frank akathisia, coupled with persistent confusion that resolved briskly once the neuroleptic was stopped (and therefore owed much to adverse anticholinergic effects on the already acetylcholine-depleted brain). The extrapyramidal disorders often took as much as a year or more to resolve, despite dopaminergic treatments. Such iatrogenic disorders can be avoided simply by arranging for practical management of the patient so as to minimise danger to self and others, for example the provision of 'special' nursing by supernumerary nurses with experience of difficult behaviour. Effective though this almost always is, and despite the palpable benefits to both ward staff and other patients' safety, it can be extraordinarily difficult to achieve on acute trauma or surgical wards, because of budgetary constraints. Nevertheless, in many cases the provision of suitable 'specialling' or 'shadowing' staff would be the most appropriate recommendation that could be made by the psychiatrist whose help and advice are sought. When the patient has no immediate needs for physical or communicative rehabilitation, it could be both useful and appropriate to offer to transfer him or her to a safe psychiatric setting, but this seems almost more difficult to achieve, in the current climate, than the provision of extra hands on the general hospital acute ward.

Organic affective disorder

It is likely that the alleged high incidence of affective disorders in some studies results from the use of mood rating scales as the main means of diagnosis. Given the many causes of reactive disgruntlement after brain injury, a high yield is not surprising. There is, however, an important reason for diagnostic caution: all currently available antidepressant drugs are suspected of precipitating epilepsy, at least in the predisposed; post-traumatic epilepsy radically worsens the long-term outcome in terms of achievement and quality of life (Evans, 1989), and even a small risk of exacerbation will mean a substantial number of individuals whose lives are disadvantaged.

Two specific results of brain injury are readily mistaken for affective disorder. The first is so-called pathological laughing and crying (or dysprosopeia; Eames & Papakostopoulos, 1990), in which the

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disorder appears to be of affect, but is in fact of emotional expression. The inner feelings described by the person at the time of their unhappy facial expression do not match their appearance; the most common feeling is simple embarrassment. The distinction is not of much practical importance, because the treatment of choice is a selective serotonin reuptake inhibitor; a positive response is almost always seen within just a few days. The second is the anterior cingulate syndrome seen most typically after anterior cerebral artery occlusion: the individual looks normal in most respects, but emotionally empty and almost immobile; depression of mood is consistently denied.

Organic personality disorder

This is an unfortunate term because, as in general psychiatry, it is too easy to distinguish clearly between disorders of personality and behaviour. Such a distinction encourages more careful analysis of the nature of the person's aberrant behaviours, which provides a better guide to treatment possibilities and prognosis than can the catch-all term. My only disagreement here with Ken Barrett is that the published studies of behavioural rehabilitation (as distinct from a few publications not based on structured study) do not support the view that generalisation to non-residential settings is difficult to achieve. Indeed, the usual outcome is for continuing social improvement during follow-up after discharge (Eames & Wood, 1985; Eames *et al*, 1996; Ashley *et al*, 1997).

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