

in man using PET. *International Journal of Neuropsychopharmacology*, **1** (suppl. 1), S65.

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### Neurosurgery for obsessive-compulsive disorder

**Sir:** In a valuable review Jenike (1998) details the psychosurgical procedures currently available worldwide for the treatment of obsessive-compulsive disorder. In the description of the technical procedure of stereotactic subcaudate tractotomy (SST) it is stated that the brain lesion is created by means of radioactive yttrium ( $Y^{90}$ ). However, we feel it is important to mention that the operation was modified in 1995 and a new procedure using the Leksell instrument and frame to create thermo-controlled high-frequency electrocoagulation has been in place since 1996 (Malhi & Bartlett, 1998). The new procedure successfully replicates the original method and has enabled the operation of SST to continue without any fundamental change in the characteristics of the lesions. The operation is not longer dependent on the availability or optimum activity of  $Y^{90}$ , and this affords greater flexibility in terms of scheduling surgery. Of particular importance is that the new procedure incurs less expense and may produce clinical response sooner. The indications for SST have not changed and it is still made available to those with treatment-resistant depression, intractable anxiety disorders and obsessive-compulsive disorder.

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### Loss of consciousness and post-traumatic stress disorder

**Sir:** In their editorial on post-traumatic stress disorder (PTSD) and loss of consciousness, O'Brien & Nutt (1998) highlight the

lack of information and research into the prevention and treatment of PTSD, despite its increased prevalence in the literature and its medico-legal implications. They note the importance of coma as a protective element. We would like to report our findings in support of this and highlight some of the conflicting reports from the literature. Although there have been few studies, when rates of PTSD were looked at in head-injured patients they were found to be quite low, which led to the theory that loss of consciousness and post-traumatic amnesia may be protective. Mayou *et al* (1993) found that among 188 victims of road traffic accidents, 19 met PTSD criteria but among 51 traffic accident patients who had sustained loss of consciousness for more than five minutes, none developed PTSD. The recurrence of memories of the injury/event was predictive of PTSD. Creamer *et al* (1992) suggest that adjustment in PTSD involves cognitive processing of threat-related information in a way that permits resolution of anxiety. The cognitive impairment associated with head injury may impede the individual's ability to process information in a manner that permits resolution. It also appears that a proportion of head-injured patients experience intrusions about events for which they are amnesic (Bryant & Harvey, 1995). Bryant & Harvey also found rates of PTSD of 42% in non-head-injured *v.* 26% of head-injured motor vehicle accident victims.

In our population-based study of head-injured patients, we looked at 196 adults attending the emergency department in South Glamorgan (catchment population 400 000) over a one-year period (1994–1995) who required in-patient admission with traumatic brain injury (defined by loss of consciousness and/or Glasgow coma scale 14 or less and/or post-traumatic amnesia and/or radiological evidence of skull fracture and/or localising neurological signs). As well as psychiatric screening questionnaires, all patients were administered a questionnaire specifically designed to identify the symptoms of PTSD. Of the entire cohort only five patients had experienced PTSD, and of these one had recovered at time of interview (one year after the head injury), as she had been treated by a psychiatrist specialising in the illness. All five had no or minimal loss of consciousness and had recollection of the traumatic event. These rates are far lower than would be expected when rates of

PTSD are looked at in other physical injuries such as burns (Perry *et al*, 1992). PTSD is now recognised to develop in the context of bodily injuries as well as emotional trauma, and physical injury may be a risk factor. However, head injury can be considered to differ from other injuries because the injury itself may interfere with recollection or memories of the accident. As O'Brien & Nutt (1998) point out, brain injury and its resultant loss of consciousness may have a paradoxical beneficial effect on the psychological recovery from trauma. We feel our findings support this theory and hope that further research can lead to the development of therapeutic approaches to prevent PTSD.

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**Creamer, M., Burgess, P. & Pattison, P. (1992)** Reaction to trauma: a cognitive processing model. *Journal of Abnormal Psychology*, **101**, 452–459.

**Mayou, R., Bryant, B. & Duthie, R. (1993)** Psychiatric consequences of road traffic accidents. *British Medical Journal*, **307**, 647–651.

**O'Brien, M. & Nutt, D. (1998)** Loss of consciousness and post-traumatic stress disorder. A clue to aetiology and treatment. *British Journal of Psychiatry*, **173**, 102–104.

**Perry, S., Difede, J., Musngi, G., et al (1992)** Predictors of posttraumatic stress disorders after burn injury. *American Journal of Psychiatry*, **149**, 931–935.

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### Recovered memories of abuse and dissociative identity disorder

**Sir:** Brandon *et al* (1998) and Pope *et al* (1998) have claimed that there is no evidence for delayed recall of authentic childhood trauma, implying that this recall involves pseudo-memories.

Although no relevant retrospective, prospective or case study is without its methodological limitations, all such studies have found evidence consistent with the hypothesis that a proportion of cases retrieve delayed memories of trauma (Brown *et al*, 1998). This convergent evidence is strengthened by recent data from studies which circumvent such limitations (e.g. Duggal & Sroufe, 1998). The question is not whether trauma can be partially or completely forgotten and recalled after a substantial delay, but what