Correspondence

EDITED BY GREG WILKINSON

Premorbid adjustment and schizophrenia

Sir: Although there had been previous evidence for premorbid social adjustment and personality being predictive for schizophrenia, Malberg *et al* (1998) are the first to show their immense impact in a cohort study. But how can premorbid functioning be measured? The Premorbid Adjustment Scale (PAS) by Cannon-Spoor *et al* (1982) covers social accessibility – isolation, peer relationships, functioning outside the nuclear family, and capacity to form intimate socio-sexual ties. Additionally, the highest level of functioning achieved before becoming ill can be estimated, as well as the time span and characteristics of the onset of illness, and general information such as education. The PAS gives a total score from 0 representing good, to 1 representing bad premorbid adjustment.

Using the PAS, we investigated a German sample of 86 unrelated patients (average age 39 years; s.d.=11.2) with a schizoaffective or schizophrenic disorder. Additionally, 38 healthy parents (average age 64 years; s.d.=12.3) were examined. DSM–IV diagnoses (American Psychiatric Association, 1994) were based on a standardised interview.

Here, we report that patients and controls differed significantly in every item of the PAS. Using a threshold between affected and healthy subjects (PAS score 0.23) an odds ratio of 27.9 (95% CI 9.39–82.89) appeared in our sample. This observation is in accordance with the findings of Malberg *et al* (1998) and supports the importance of premorbid functioning.

However, the PAS does not cover items which Malberg *et al* (1998) found to be associated with schizophrenia, such as delinquency, breach of regulations, and substance misuse. Nevertheless, the PAS can be recommended for the measurement of premorbid social adjustment. Furthermore, measuring premorbid functioning seems to be helpful in predicting the risk of developing schizophrenia. Later, it may help to estimate the course of the disorder, since high PAS values were related to an unfavourable course.

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Pindolol augmentation of antidepressant therapy

Sir: We read with interest the timely review of the augmentation of antidepressants with pindolol by McAskill *et al* (1998). They detail open studies supporting a role for pindolol in accelerating and augmenting the action of antidepressants, an effect thought to be mediated by the blockade of somatodendritic (presynaptic) 5-HT_{1A} autoreceptors. However, the controlled studies completed to date are somewhat contradictory. McAskill *et al* (1998) argue for larger-scale trials using higher doses of pindolol than those previously employed. We would suggest that such strategies require caution for interpretable results to be obtained.

McAskill *et al* (1998) imply that pindolol selectively blocks 5-HT_{1A} receptors at the somatodendritic location but not at the post-synaptic site. However, positron emission tomography (PET) imaging in man of 5-HT_{1A} binding shows that pindolol binds to both somatodendritic and post-synaptic receptors (Rabiner *et al*, 1998). Depression is thought to be associated with an impairment of post-synaptic 5-HT_{1A} function (Power & Cowen, 1992) and an increase in transmission through this receptor is hypothesised to underlie the mechanism of action of antidepressants (Blier & de Montigny, 1994). Therefore, pindolol may augment antidepressants by blocking somatodendritic 5-HT_{1A} receptors but this may be counterbalanced by a deleterious action of post-synaptic 5-HT_{1A} receptor blockade. McAskill *et al*'s (1998) suggestion of using higher doses of pindolol may compound this problem since PET studies suggest that the ratio of somatodendritic to post-synaptic blockade decreases with increasing doses (Rabiner *et al*, 1998).

The use of pindolol as a somatodendritic 5-HT_{1A} antagonist is complicated further by the possibility that pindolol may be a partial agonist at this site (Clifford *et al*, 1998). This may, together with the mixed somatodendritic and post-synaptic binding, explain the lack of a consensus in the controlled trials of pindolol augmentation of antidepressants. Rather than rushing into larger studies with higher doses of pindolol it is perhaps more sensible to first investigate whether a dose of pindolol can be found that leads to a high ratio of somatodendritic to post-synaptic binding. This may indeed be lower than the doses currently employed. Ultimately, it will only become clear if blockade of somatodendritic 5-HT_{1A} receptors is an effective means of accelerating and augmenting antidepressant actions when a pure antagonist, rather than a partial agonist like pindolol, becomes clinically available.

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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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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