Correspondence

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Transcranial magnetic stimulation

Sir: The timely article by Reid *et al* (1998) is both informative and thought-provoking; the authors raise a number of issues which may have far-reaching implications beyond the treatment of depression. As Reid *et al* are quick to point out, transcranial magnetic stimulation (TMS) has already established its usefulness in a number of clinical and research areas, including brain-mapping research and pre-operative neurological assessment.

In addition to its exciting research potential and existing clinical uses, TMS may also find a role in the treatment of a variety of neurological and neuropsychiatric conditions, for example Parkinson's disease (particularly where there is concurrent speech deficit, as occurs in more than half of these patients). Sandyk (1997) describes the case of a 52-year-old patient with a four-year history of Parkinson's disease complicated by speech impairment (mainly severe stuttering predominantly during 'on–off' periods); a "dramatic and consistent improvement in speech" occurred following regular TMS treatment. Another area of investigation is the efficacy of TMS in combination with serotonergic agents, which may have a synergistic effect (Belmaker *et al*, 1997) with implications again for movement disorders (notwithstanding the motor component of psychoses).

Another issue raised by the growing use of TMS is its safety. Complications of its clinical use are considered in detail elsewhere (Shajahan & Ebmeier, 1988) and Reid *et al* emphasise the potential of TMS for inducing seizures. However, what is not discussed is the possibility that TMS-provoked seizures may have a therapeutic benefit, as they apparently do in conventional electroconvulsive therapy (ECT). Of course, this would have implications for anaesthesia and muscle relaxants, again affecting patient acceptability. Presumably the primary advantage of TMS over ECT lies less in its efficacy and more in its tolerability. If this is the case, the greater acceptability of TMS will doubtless have an effect in the arena of public opinion. The popular notion of unmodified 'fitting' in the 1950s made ECT a public pariah for decades, culminating in a public demonstration against ECT outside the Royal College of Psychiatrists in August 1998. In contrast, the apparent sophistication of TMS in conscious and cooperating patients may prove advantageous to the public perception of psychiatry.

Belmaker, R. H., Grisaru, N., Ben-shahar, D., et al (1997) The effects of TMS on animal models of depression, beta-adrenergic receptors, and brain monoamines. *CNS Spectrums*, **2**, 26–30.

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Definitions of schizophrenia

Sir: While we applaud the design and presentation of the results of the study by Mason *et al* (1997) comparing the predictive validity of various definitions of schizophrenia, we take exception to the conclusion drawn by the authors that the ICD–10 (World Health Organization, 1992) definition of schizophrenia "probably represents the most clinically useful definition for first-episode studies" because it combines both high sensitivity (92.73%) and high specificity (88.64%). This might be true when the ICD–10 definition is compared with the DSM–III–R (American Psychiatric Association, 1994) definition in isolation, but it ignores the clinical reality of the situation. The DSM classification of schizophrenia recognises that the relative importance of sensitivity and specificity depends on the clinical context. Because of the dire consequence of being given a misdiagnosis of schizophrenia, the DSM definition of schizophrenia has been devised with a very low tolerance for false positives, which is borne out by the study's finding of no false positives in the sample (using 13-year diagnostic stability as the gold standard). However, for the purposes of case-finding, first-episode studies of schizophrenia invariably use a combined definition; that is, a case would be included for study if the criteria are met for either schizophreniform disorder or schizophrenia, which in this study would result in a combined sensitivity of 94.12% (superior to the ICD–10 definition). This result, coupled with the superior predictive validity of the DSM–III–R, would seem to argue for a different conclusion – that the DSM–III–R definition performs the best. Given the inclusion of negative symptoms into the DSM–IV definition of schizophrenia, one would expect the sensitivity of the DSM–IV definition to be even better.

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Satisfied with dissatisfaction?

Sir: Leese *et al* (1998) report similar user satisfaction, as indicated by the Verona Service Satisfaction Scale (VSSS; Ruggeri & Dall'Agnola), between an intensive and a standard community mental health service. They conclude that both services were reasonably successful with fairly high levels of satisfaction. We would question such an optimistic interpretation.

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The 54% of those selected for interview who did not participate may have had very different experiences from those who chose to comply, therefore introducing important responder bias. Using non-independent researchers is likely to bias responses.

The VSSS asks patients to indicate their satisfaction on a five-point Likert scale. The choices are: 1=terrible, 2=mostly unsatisfactory, 3=mixed, 4=mostly satisfactory and 5=excellent. The mean satisfaction scores reported by Leese *et al* are less than four (mostly satisfactory) in all but one domain in both services at both time points. Scores which fall short of mostly satisfactory must indicate that users' experiences of services could have been better. Leese *et al*'s interpretation – that this indicates successful services delivering fairly high levels of satisfaction – ignores the discontent that respondents have expressed.

This potential misreading of patients' experiences is compounded by the use of summary scores only. In our survey of psychiatric in-patients (Greenwood *et al* 1999) we report 73% of patients as very or fairly satisfied. Even in this group 60.4% reported significant levels of adverse experiences with the service. The strength of the VSSS is that it questions the respondents in some detail within each domain. Averaged satisfaction scores may well obscure real dissatisfaction including perhaps a number of unpleasant experiences.

If services are to be evaluated with a view to improving them, then the details of dissatisfaction that users voice may be the most valuable and deserve closest attention.

Greenwood, N., Key, A., Burns, T., *et al* (1999) Satisfaction with in-patient psychiatric services. Relationship to patient and treatment factors. *British Journal of Psychiatry*, **174**, 159–163.

Leese, M., Johnson, S., Slade, M., *et al* (1998) User perspective on needs and satisfaction with mental health services. PRiSM Psychosis Study 8. *British Journal of Psychiatry*, **173**, 409–415.

Ruggeri, M. & Dall'Agnola, R. (1993) The development and use of the Verona Expectations for Care Scale (VECS) and the Verona Service Satisfaction Scale (VSSS) for measuring expectations and satisfaction with community-based psychiatric services in patients, relatives and professionals. *Psychological Medicine*, **23**, 511–523.

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Traumatic brain injury and post-traumatic stress disorder

Sir: O'Brien & Nutt (1998) develop the proposition that traumatic brain injury may protect against the development of emotional consequences arising from a traumatic experience (Adler, 1945). Although it may be compelling to think that nightmares or horrific memories associated with such events cannot occur if there has been loss of consciousness, the literature does not support this contention (see review, McMillan, 1997). One study reports on 10 cases who had traumatic brain injury ranging from mild to very severe and had post-traumatic stress disorder (PTSD) (McMillan, 1996). Several ways in which PTSD can develop, despite loss of consciousness and post-traumatic amnesia, have been reported. These include distressing 'windows' in memory, which for minor head injuries includes isolated memories soon before (e.g. of a lorry about to make impact) and after (e.g. being in a car, trapped and smelling petrol) the accident, and for more severe head injuries isolated memories during post-traumatic amnesia (McMillan, 1996). Some suggest that implicit learning which occurs during post-traumatic amnesia is a vehicle (Layton & Wardi-Zonna, 1995). These studies indicate that loss of consciousness and post-traumatic amnesia may not protect an individual from traumatic emotional experiences, but not that this never occurs.

O'Brien & Nutt suggest that by mimicking neurotransmitter changes caused by traumatic brain injury by pharmacological intervention, the development of PTSD might be arrested in people who have sustained no head injury. Given that PTSD occurs even in people who have sustained a severe head injury, and that other emotional consequences such as travel anxiety/phobias are not uncommon, some doubt must be placed upon their premise. Furthermore, traumatic brain injury triggers a cascade of biochemical events resulting in oedema, necrosis, haemorrhage and functional impairment. The complexity of secondary injury processes makes it difficult to elucidate the roles of specific injury mechanisms, including those underlying loss of consciousness and post-traumatic amnesia. Glutamatergic (Faden, 1996; Koura *et al*, 1998) and cholinergic mechanisms (Murdoch *et al*, 1998), each implicated in memory dysfunction, are also postulated to underly coma in man. After traumatic brain injury these and caspase-3-like proteases (Yakovlev *et al*, 1997), endogenous opioids acting on kappa-2 receptors (Faden, 1996) and other acute metabolic responses contribute to coma in experimental animals. It may be premature to pin the pathophysiology of coma or post-traumatic amnesia primarily on glutamatergic mechanisms which are responsible for only a proportion of these post-traumatic sequelae (Myseros & Bullock, 1995). Any pharmacological hypothesis needs to account both for 'windows' of awareness during loss of consciousness and post-trumatic amnesia despite 'excitotoxic surge', and for the registration and consolidation of memories therefrom. As O'Brien & Nutt acknowledge, the pharmacological bases for retrograde and anterograde amnesia are likely to differ, each time frame a potential source of traumatic memories.

Given the present state of knowledge about PTSD after traumatic brain injury, it is premature to recommend the pharmacological intervention of the kind that they suggest for the reasons that they give.

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