

citations were identified. By expanding the search with one thesaurus term so it read: ((MRI or explode 'MAGNETIC-RESONANCE-IMAGING/all subheadings') and schizophrenia) 233 additional records were identified. Many of these would have been of considerable interest to the reviewers. We did not test the added value of other thesaurus terms such as 'NUCLEAR-MAGNETIC-RESONANCE/all subheadings'. By making the schizophrenia part of the search more sophisticated, using a published phrase for identifying Medline schizophrenia studies (Adams *et al*, 1998), 182 more records were identified. This subset has a high false positive rate but there are, quite clearly, citations of direct interest for a comprehensive MRI meta-analysis. When a similar exercise was undertaken on EMBASE an additional 1716 unique records were identified. Again, the false positive rate was high but there were studies of relevance to Lawrie & Abukmeil's review. We did not investigate other rich sources of data such as PsychLit and Biological Abstracts. It is unlikely that the hand-searching of journals and references would have picked up most of the studies.

In such reviews being comprehensive is desirable. Studies that are readily accessible by simple searches on Medline may well have systematically different results to those that are more difficult to find (Egger *et al*, 1997).

**Adams, C. E., Duggan, L., Wahlbeck, K., et al (eds) (1998)** *Schizophrenia Module of the Cochrane Database of Systematic Reviews* (updated 4 December 1997). Available in The Cochrane Library (CD-ROM), Issue 1. Oxford: Update Software.

**Egger, M., Zellweger-Zahner, T., Schneider, M., et al (1997)** Language bias in randomised controlled trials published in English and German. *Lancet*, **350**, 326–329.

**Lawrie, S. M. & Abukmeil, S. S. (1998)** Brain abnormality in schizophrenia. A systematic and quantitative review of volumetric magnetic resonance imaging studies. *British Journal of Psychiatry*, **172**, 110–120.

**C. E. Adams, B. Thornley, C. Joy** Cochrane Schizophrenia Group, Middle Way, Summertown, Oxford OX2 7LG

### What counts as clinical research?

**Sir:** Morlino *et al* (1997) report findings on the extent of basic research papers published that they regard as “at variance with the conclusions of Pincus *et al* (1993)”. The difference might be related to how ‘basic research’ is defined. In our study, ‘basic

biological research’ included animal studies and other research reports not involving clinical populations. Many of the papers Morlino *et al* included as “basic research topics such as neurochemistry, neuroanatomy and brain imaging” are likely to have involved clinical populations and probably would have been included in our category of ‘clinical psychobiology’.

It is important to be clear in the use of such terms as ‘clinical’ and ‘basic’ research. The National Institute of Health’s high-level committee to review issues and problems in clinical research spent a great deal of time and effort to develop a standard definition of clinical research (National Institutes of Health Director’s Panel on Clinical Research, 1997). Their definition (which engendered some controversy) includes, in addition to epidemiological, behavioural and health services research studies, “patient oriented research” that is not only conducted with human subjects but also with “material of human origin . . . in which it is necessary to know the identity of the patients”. Thus, ‘basic’ research would be limited to animal research and *in vitro* studies utilising human tissues that do not require dealing directly with patients. With these fairly broad criteria they found, overall, that 27% of National Institute of Health grants met the clinical research definition.

**Morlino, M., Lisanti, F., Gogliettino, A., et al (1997)** Publication trends of papers on schizophrenia. A 15-year analysis of three general psychiatric journals. *British Journal of Psychiatry*, **171**, 452–456.

**National Institutes of Health Director’s Panel on Clinical Research (1997)** *Report to the Advisory Committee to the National Institutes of Health Director*. Bethesda, MD: NIH.

**Pincus, H. A., Henderson, B., Blackwood, D., et al (1993)** Trends in research in two general psychiatric journals in 1969–1990: research on research. *American Journal of Psychiatry*, **150**, 135–142.

**H. A. Pincus** American Psychiatric Association, 1400 K Street, N.W., Washington, DC 20005, USA

### Rapid intravenous detoxification in heroin addiction

**Sir:** Despite its short follow-up period, the study by Seoane *et al* (1997) gives us two crucial pieces of information on the vexed question of accelerated detoxification from opioids. First, the degree of sedation in randomised groups yielded no difference in abstinence rates a month after the proce-

sure. Second, the occurrence of serious side-effects was just under 5% in both the lightly and heavily sedated groups. These reactions included respiratory depression, bradycardia, pneumonia and fever of unknown origin.

Seoane *et al*'s assertion that the incidence of side-effects is lower than with ‘conventional’ detoxification is only supported by reference to another ‘rapid’ detoxification series with a complication rate of 5.8%. Traditional detoxification is believed to have a complication rate close to zero.

The prior use of methadone was not revealed. Despite being based on work done prior to 1994, there is no follow-up of data beyond four weeks. This is surprising considering the novelty and controversial nature of the treatment. A study of accelerated opioid detoxification under anaesthetic showed that 43% of patients who could be contacted had ceased their prescribed naltrexone and returned to daily heroin use at 18 months’ follow-up (Rabinowitz *et al*, 1997).

**Seoane, A., Carrasco, G., Cabré, L., et al (1997)** Efficacy and safety of two new methods of rapid intravenous detoxification in heroin addicts previously treated without success. *British Journal of Psychiatry*, **171**, 340–345.

**Rabinowitz, J., Cohen, H., Tarrasch, R., et al (1997)** Compliance to naltrexone treatment after ultra-rapid opiate detoxification: an open label naturalistic study. *Drug and Alcohol Dependence*, **47**, 77–86.

**A. Byrne** 75 Redfern Street, Redfern, New South Wales 2016, Australia

### Moclobemide in social phobia

**Sir:** The claim made by Schneier *et al* (1998) that moclobemide is not indicated as a first-line therapy in social phobia should be challenged. Social phobia is a relatively common anxiety disorder, which rarely presents to psychiatrists even when there is marked impairment in occupational and social functioning (Weiller *et al*, 1996). Thus, a first-line therapy for social phobia should be effective, well tolerated and suitable for prescription within primary care.

Addressing the latter two issues, moclobemide has a simple dosing regime and is well tolerated; Schneier *et al* found eight-week drop-out rates were 24% on moclobemide *v.* 25% on placebo. Their most serious objection to the use of moclobemide

as a first-line treatment is one of efficacy. They found 23% of patients with severe or very severe social phobia treated with moclobemide for eight weeks were rated as much or very much improved (*v.* 0% in the placebo group), although numbers were too small to reach statistical significance. This finding of greater efficacy in more severe social phobia is also supported by the International Multicenter Clinical Trial Group on Moclobemide in Social Phobia (1997) who found patients with severe social phobia treated with 600 mg moclobemide had a 52% response rate (*v.* 32% on placebo).

**International Multicenter Clinical Trial Group on Moclobemide in Social Phobia (1997)** Moclobemide in social phobia. A double-blind, placebo-controlled clinical study. *European Archives of Psychiatry and Clinical Neuroscience*, **247**, 71–80.

**Schneier, F. R., Goetz, D., Campeas, R., et al (1998)** Placebo-controlled trial of moclobemide in social phobia. *British Journal of Psychiatry*, **172**, 70–77.

**Weiller, E., Bisserbe, J. C., Boyer, P., et al (1996)** Social phobia in general health care. An unrecognised under-treated disabling disorder. *British Journal of Psychiatry*, **168**, 169–174.

**R. Duffett** The Royal London Hospital (St Clement's) 2A Bow Road, London E3 4LL

### Liaison between adolescent and adult services in early-onset schizophrenia

**Sir:** Pelkonen *et al*'s (1998) follow-up study of occupational functioning of adolescent in-patients emphasises the importance of active intervention in early adulthood in those with psychotic disorders. However, they do not highlight the importance of close liaison between adolescent and adult services in order to achieve this. In clinical practice there is lack of clarity about which service should serve those aged 16–18 years. Traditionally, this had depended on the young person's educational status at the time of presentation. However, some 16 to 18-year-olds may have left school as a result of developing psychosis. Their needs in terms of re-integration into educational services and addressing family issues (such as expressed emotion interventions) may be better met by the resources of adolescent services.

Poor psychosocial outcome in adulthood in those with adolescent psychotic disorders is a robust finding (Gillberg *et al*, 1993). Although early-onset schizophrenia

is more likely to be associated with a poor prognosis than adult-onset schizophrenia is (Jacobsen & Rapoport, 1998) this could be partly ameliorated by commencing treatment early (Turetz *et al*, 1997) and the potentially greater compliance with atypical antipsychotics. Familiarity with, and experience of, use of atypical antipsychotics by adolescent psychiatrists can be enhanced by liaison with colleagues in adult services.

Close liaison between services at an early stage, therefore, has potential benefits for both services and, particularly, for the young person in terms of addressing all aspects of care and providing continuity of follow-up into early adulthood. The personal and economic implications of years of functional impairment and disability are too great to ignore.

**Gillberg, C., Hellegrén, L. & Gillberg, C. (1993)** Psychotic disorders diagnosed in adolescence: outcome at age 30 years. *Journal of Child Psychology and Psychiatry*, **34**, 1173–1185.

**Jacobsen, L. K. & Rapoport, J. L. (1998)** Research update. Childhood-onset schizophrenia: implications of clinical and neurobiological research. *Journal of Child Psychology and Psychiatry*, **39**, 101–113.

**Pelkonen, M., Marttunen, M., Pulkkinen, E., et al (1998)** Disability pensions in severely disturbed in-patient adolescents. Twenty-year prospective study. *British Journal of Psychiatry*, **172**, 159–163.

**Turetz, M., Mozes, T., Toren, P., et al (1997)** An open trial of clozapine in neuroleptic-resistant childhood-onset schizophrenia. *British Journal of Psychiatry*, **170**, 507–510.

**K. Sayal** Kings College Hospital, Denmark Hill, London SE5 9RS

### Somatoform dissociation is unlikely to be a result of indoctrination by therapists

**Sir:** In a previous letter (Nijenhuis *et al*, 1997) we reported that high scores on instruments measuring dissociation were typical of patients with dissociative disorder and not of those with bipolar disorder. We argued that these data show that dissociative disorders are highly unlikely to be a result of misinterpretation of bipolar disorder. Merskey (1997) commented that our comparison is worthless. Assuming that dissociative disorders results from indoctrination by therapists, he maintained that we have compared un-indoctrinated bipolar patients and indoctrinated 'dissociative' patients.

This assumption is incorrect. For the patients with dissociative disorders we had two groups: one received the Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis *et al*, 1996) prior to, and the other after, the administration of the Structured Clinical Interview for Dissociative Disorders (SCID-D) and diagnosis of 'dissociative identity disorder' or 'dissociative disorder not otherwise specified'. The former group cannot possibly have been indoctrinated. Interestingly, patients from the first group who were unaware of their diagnosis tended to obtain higher SDQ-20 scores than those who were aware of their psychiatric status and who were exposed to therapy (further details available from the author upon request).

The *a priori* assumption that the diagnosis of dissociative disorders must follow from indoctrination seems to be based on prejudice instead of research findings.

**Merskey, H. (1997)** Tests of 'dissociation' and mood disorder (letter). *British Journal of Psychiatry*, **171**, 487.

**Nijenhuis, E. R. S., Spinhoven, P., van Dyck, R., et al (1996)** The development and the psychometric characteristics of the Somatoform Dissociation Questionnaire (SDQ-20). *Journal of Nervous and Mental Disease*, **184**, 688–689.

—, —, —, et al (1997) Dissociative pathology discriminates between bipolar mood disorder and dissociative disorder. *British Journal of Psychiatry*, **170**, 581.

**E. R. S. Nijenhuis, R. van Dyck** Department of Psychiatry, Vrije Universiteit at Amsterdam, Valeriusplein 9, 1075 BG Amsterdam, The Netherlands

**O. van der Hart** Department of Clinical Psychology and Health Psychology, Utrecht University, The Netherlands

**P. Spinhoven** Department of Psychiatry, Leiden University, The Netherlands

### Can transsexualism remit?

**Sir:** The subject of the paper by Marks & Mataix-Cols (1987) is a current patient of ours; Professor Marks was aware of this and discussed this before writing his report, though this is not acknowledged in the paper.

Since the paper contains statements at variance with our understanding of the case, we showed the paper to the patient, who told us of cross-dressing since age 7, self-view as female age 12, and active search for gender reassignment since age