drug and alcohol misuse and other predictors of negative outcome (Vestergaard *et al*, 1998) select themselves to the non-compliant patient group. Therefore, a finding that non-compliant patients fare worse than compliant patients may testify only to the existence of negative predictor variables among patients who were non-compliant, instead of supporting the efficacy of lithium treatment. Neither our study nor Kallner *et al*'s allow conclusions as to whether or not lithium has specific antisuicidal effects exceeding what can be inferred from its ability to prevent recurrent illness episodes in affective disorder patients.

The efficacy of long-term prophylactic treatment with lithium has been questioned frequently (Moncrieff, 1995). We believe, as apparently do Gracious & Falodun, that despite its shortcomings lithium is a very helpful tool in the psychiatric armamentarium. Arguments that support the efficacy (or inefficacy) of long-term lithium treatment should, however, rest on sound scientific evidence.

Brodersen, A., Licht, R. W., Vestergaard, P., *et al* (2000) Sixteen-year mortality in patients with affective disorder commenced on lithium. *British Journal of Psychiatry*, **176**, 429–433.

Kallner, G., Lindellius, R., Petterson, U., *et al* (2000) Mortality in 497 patients with affective disorders attending a lithium clinic or after having left it. *Pharmacopsychiatry*, **33**, 8–13.

Moncrieff, J. (1995) Lithium revisited. A re-examination of the placebo-controlled trials of lithium prophylaxis in manic–depressive disorder. *British Journal of Psychiatry*, **167**, 569–574.

Vestergaard, P., Licht, R. W., Brodersen, A., *et al* (1998) Outcome of lithium prophylaxis: a prospective follow-up of affective disorder patients assigned to high and low serum lithium levels. *Acta Psychiatrica Scandinavica*, **98**, 310–315.

P. Vestergaard, R. W. Licht Mood Disorders Research Unit, Aarhus University Hospital, Skovagervej 2, DK-8240 Risskov, Denmark

Finding the evidence in forensic rehabilitation

Cure & Adams (2000) suggest that we managed to overlook 22 000 potential references including 2000 which apparently contain data relevant to our inquiries. Contrary to our belief, they also claim that the randomised trial is the preferred research methodology in forensic psychiatric rehabilitation.

These criticisms are, in our view, based on a poor understanding of the process of rehabilitating mentally disordered offenders, and reveal a blinkered approach to novel research strategies which may be of value in such atypical settings.

Cure & Adams cited three examples of the many quality studies they allege we overlooked in our review. All were published after the final submission of our paper, but are presumably presented as examples of the treatment and rehabilitation of mentally disordered offenders. Two of the cited reviews examine antipsychotic treatment (in people with learning disabilities and with acute schizophrenia) and the other is a review of sex offender treatment. These studies are, without doubt, most relevant to clinical forensic psychiatric practice. They do not, however, target the process of rehabilitation in a more general sense, as outlined in our paper. There is more to forensic work than drugs and specific programmes for certain offender groups.

Apparently, Cure & Adams fail to appreciate the difference between psychiatric work among forensic and non-forensic populations. That difference is the rationale for our remark that a randomised trial is not the method of choice in evaluating the outcome of forensic psychiatric rehabilitation. The crucial point is that allocation to forensic psychiatric treatment is not controlled by medical professionals but by legal authorities, refractory to the systematic and premeditated manipulation that some research requires. Although mentally disordered offenders, delivered by the courts to the hospitals, can be diverted into different treatment schemes, it is not feasible to maintain a predetermined course of rehabilitation. Important factors such as the length of incarceration, number and duration of leaves as well as external support by non-forensic caregivers, are not possible to randomise and control.

Randomised trials do not provide the only source of data on treatment efficacy, although where these trials are possible, valid and important data may be presented. Our paper did not pretend to review all articles related to the field of forensic psychiatric practice. Such magnificent and ambitious endeavours can only be embarked upon by the privileged few who are provided with considerable support from national funding institutions. Their reports may prove invaluable in guiding clinicians, assuming that the issues are correctly presented – a considerable responsibility. One obvious risk of the rapid growth of evidence-based medicine is its inhibiting effect on the advancement of the theory of clinical practice and its potentially discouraging effects on active contributors and reviewers of articles to medical journals.

Cure, S. J. & Adams, C. E. (2000) Forensic trials inform the present and future (letter). *British Journal of Psychiatry*, **177**, 182.

Lindqvist, P. & Skipworth, J. (2000) Evidence-based rehabilitation in forensic psychiatry. *British Journal of Psychiatry*, **176**, 320–323.

P. Lindqvist Örebo Forensic Psychiatry Service, Eken, S-70185 Örebo, Sweden

J. Skipworth Regional Forensic Psychiatry Service, Aukland, New Zealand

Arachnophobia: a practical management device

While not wishing to endorse a particular product or brand, I would like to report the effectiveness of a cheap and readily available device in the management of insect and spider phobia (the 'Bug Katcha', from Betterware). The device consists of a clear Perspex box with a sliding door mounted on a long handle, allowing the offending insect to be entrapped from a distance and released without manual contact.

Having in jest presented a severely spider-phobic psychiatrist friend with such an item, I was pleased to hear that its use had provided effective exposure *in vivo* and led to a marked reduction in symptoms of anxiety. She became able to talk about and to be in a room with spiders without displaying visible signs of arousal. As many non-arachnophobes prefer not to handle spiders directly, her functioning seems to have been restored to an acceptable level.

This device may provide a practical and cost-effective way to reduce the manifestations of simple insect and spider phobias.

S. Smith Department of Psychiatry, Kidderminster General Hospital, Bewdley Road, Kidderminster DY11 6RJ

Thrombocytosis due to clozapine treatment: working towards an early marker for clozapine-induced agranulocytosis

Recently, Hampson (2000) reported thrombocytosis with clozapine, and serious consideration must be given to reports that

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identify potential markers for the development of agranulocytosis. Haematological side-effects include leucopenia, neutropenia and thrombocytopenia (1–3% of patients); and anaemia, leucocytosis and thrombocytosis (<1% of patients) (American Hospital Formulary Service, 1997). Thrombocytosis reported with clozapine treatment may give evidence of the mechanism of agranulocytosis in some patients.

In some cases clozapine is discontinued if the differential white blood cell count shows an initial drop with starting clozapine treatment. If a re-challenge on clozapine results in either thrombocytosis or thrombocytopenia, this may be a result of an immune reaction, as both these platelet abnormalities are recognised features of such a reaction. (Note that it is now recommended that permanent withdrawal of clozapine should occur for leucopenia below 3×10^9/l or neutrophil count below 1.5×10^9/l (British Medical Association & Royal Pharmaceutical Society of Great Britain, 2000)).

Clozapine has a direct action on the haematopoietic stem cells of the bone marrow and can therefore trigger a reaction similar to an acute myeloid leukaemia or myeloproliferative disorder. It is hypothesised that an abnormal haematocrit and platelet abnormality could be seen if clozapine caused these side-effects via an immune reaction on the haemopoietic tissue. Karyotype analysis provides useful prognostic information in myelodysplastic syndrome (Provan, 1997), and is associated with clozapine response (Arranz *et al*, 1995). A high index of suspicion when reviewing the full blood count or karyotype analysis could lead to a marker before fatal agranulocytosis occurs as a result of clozapine.

American Hospital Formulary Service (1997) *Drugs Information* (ed. G. K. McEvoy), pp. 1743–1744. Bethesda, MD: American Society of Health-System Pharmacists.

Arranz, M., Collier, D., Sodhi, M., *et al* (1995) Association between clozapine response and allelic variation in 5-HT2A receptor gene. *Lancet*, **346**, 281–282.

British Medical Association & Royal Pharmaceutical Society of Great Britain (2000) *British National Formulary*, no. 39 (March 2000). London: BMJ Books & Pharmaceutical Press.

Hampson, M. E. (2000) Clozapine-induced thrombocytosis (letter). *British Journal of Psychiatry*, **176**, 400.

Provan, D. (1997) Myelodysplastic syndromes. *Prescribers' Journal*, **37**, 17–23.

K. Marlowe Thames Gateway NHS Trust, Mental Health Directorate, Medway Maritime Hospital, Windmill Road, Gillingham, Kent ME7 5NY

Etomidate-induced convulsion prior to electroconvulsive therapy

Choice of anaesthesia for electroconvulsive therapy (ECT) has become more limited in the past year owing to the non-availability of methohexitone. As a result, there has been more use of etomidate in our hospital as an anaesthetic agent for ECT. We would like to draw your attention to a case of a spontaneous seizure after administration of etomidate.

A young man aged 19 years had been an in-patient for 3 months, following the onset of a schizoaffective disorder. He was being treated with a course of ECT and his medication from the start of this treatment was chlorpromazine 1000 mg/day, procyclidine 15 mg/day and fluoxetine 20 mg/day. There was no relevant past medical history or previous history of seizures. Prior to his tenth treatment of ECT he experienced a spontaneous generalised tonic/clonic seizure while being induced under anaesthetic. He was administered etomidate initially 26 mg, which was increased to a dose of 30 mg as facial twitching was evident. He was then administered suxamethonium 75 mg. However, the twitching continued into a full grand mal seizure lasting about 90 seconds, which was terminated by 10 mg diazepam given intravenously.

Recovery from anaesthesia was otherwise normal and there was no evidence of postictal confusion or other physical sequelae. Etomidate was used as anaesthetic agent for this man's nine other ECT sessions, to a maximum dose of 28 mg with no adverse effect. Improvement in this young man's mood was maintained following this incident and it was decided to discontinue ECT.

Generalised seizures after short-term etomidate infusion have been reported during or after recovery from anaesthetic (Goroszeniuk *et al*, 1986; Krieger & Koemer, 1987; Hansen & Drenck, 1988). However, seizures have not yet been reported during induction of etomidate anaesthesia or while undergoing a course of ECT. There is no definite neuropharmacological explanation for this seizure-like activity of etomidate, which is thought to result from a disinhibition of subcortical activity, rather than a specific epileptogenic effect of the compound (Kugler *et al*, 1977).

Concurrent use of fluoxetine and chlorpromazine may have partly contributed, by lowering the seizure threshold. However, in this case the slightly higher dose of etomidate seems the most likely causative agent, as all other medication had been prescribed at the above doses throughout the course of ECT.

This case emphasises the importance of minimising adverse effects during ECT by using the lowest effective dose of anaesthetic agent and carefully considering concurrent usage of other medication.

Goroszeniuk, T., Albin, M. & Jones, R. M. (1986) Generalized grand mal seizure after recovery from uncomplicated fentanyl–etomidate anesthesia. *Anesthesia and Analgesia*, **65**, 979–981.

Hansen, H. C. & Drenck, N. F. (1988) Generalised seizures after etomidate anaesthesia. *Anaesthesia*, **43**, 805–806.

Krieger, W. & Koemer, M. (1987) Generalized grand mal seizure after recovery from uncomplicated fentanyl–etomidate anesthesia. *Anesthesia and Analgesia*, **66**, 284–285.

Kugler, J., Doenicka, A. & Laub, M. (1977) The EEG after etomidate. *Anesthesia Resuscitation*, **106**, 31–48.

K. Nicoll, J. Callender Grampian Primary Care NHS Trust, Block A, Clerkseat Building, Royal Cornhill Hospital, Cornhill Road, Aberdeen AB25 2ZH

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