Correspondence

EDITED BY LOUISE HOWARD

Co-occurrence of polydactyly and psychosis

Sir: We wish to comment on the preliminary report listing five cases of co-occurrence of polydactyly and psychosis (Cardno *et al*, 1998), which concluded that there was preliminary evidence that polydactyly was over-represented in individuals with familial schizophrenia and related psychotic illnesses.

We were interested in the report as we had a 36-year-old Asian male who suffered from chronic relapsing schizophrenia and also had pre-axial polydactyly with an extra thumb on the left hand. There was no family history of polydactyly but his eldest brother suffered from schizophrenia. Our case was similar to the fifth case described by the authors (a Caucasian young male, 41 years old) except the ethnicity.

O'Callaghan *et al* (1991) concluded that minor physical abnormalities indicated early dysmorphogenesis in schizophrenia, particularly in males (all the cases described by the authors were male), which appeared to be associated more reliably with genetic than obstetric factors and with cognitive impairment. They also found that a family history of schizophrenia was particularly associated with abnormalities of the mouth.

Post-axial polydactyly (little finger side) occurs as an isolated lesion in Black people, inherited as an autosomal dominant trait, but in White people may be associated with other anomalies and syndromes. We note that three out of the four cases of post-axial polydactyly described by the authors also had a family history of polydactyly. Pre-axial polydactyly with extra thumbs is common in White people; it is usually sporadic and unilateral (Nelson *et al*, 1992). (Unfortunately this vital information has been omitted from the recent edition of the same text, which Cardno *et al* cited (Nelson *et al*, 1996).) In our opinion, as isolated pre-axial polydactyly of digits is likely to be sporadic, such cases should not be included in the same group as familial post-axial polydactyly in future research.

We also note that the first case described by Cardno *et al* (an Indian male, 64 years old) did not have any family history of schizophrenia or related psychotic illnesses. We feel that further research should recognise these differences and focus on patients with familial polydactyly and familial schizophrenia.

Cardno, A. G., Murphy, K. C., Jones, L. A., *et al* (1998) Polydactyly and psychosis. Five cases of co-occurrence. *British Journal of Psychiatry*, **172**, 184–185.

Nelson, W. E., Behrman, R. E., Kliegman, R. M., *et al* (1992) *Nelson Textbook of Paediatrics* (14th edn), p. 1720. Philadelphia, PA: Saunders.

—, —, —, *et al* (1996) *Nelson Textbook of Paediatrics* (15th edn), p. 1923. Philadelphia, PA: Saunders.

O'Callaghan, E., Larkin, C., Kinsella, A., *et al* (1991) Familial, obstetric and other clinical correlates of minor physical anomalies in schizophrenia. *American Journal of Psychiatry*, **148**, 479–483.

N. Purandare, S. Plunkett Department of Psychiatry, Withington Hospital, Nell Lane, Manchester M20 8LR

Olanzapine in the treatment of psychotic depression

Sir: In connection with the review by Tollefson & Kuntz (1999), examining the clinical studies involving olanzapine, we would like to report its effective use in the treatment of psychotic depression.

Two patients with severe psychotic depression, responsive solely to electroconvulsive therapy (ECT), refused further treatment necessitating pharmacotherapeutic change. Both patients were receiving maximal doses of venlafaxine (375 mg daily), with lithium augmentation, in combination with an antipsychotic (thioridazine or sulpiride). Severity of illness precluded the use of placebo and so, over a period of three months, we prospectively conducted an open study, involving antipsychotic substitution with olanzapine (10 mg). This was the only change implemented. Objective and subjective mood and psychotic symptoms were rated weekly, blind to treatment status (Hamilton, 1960; Beck *et al*, 1961; Cliffe *et al*, 1995). Prior to olanzapine substitution, ECT had not been administered for at least two months and the maximum tolerable doses of traditional antipsychotics had been prescribed without success.

In both subjects it was found that within three weeks of starting olanzapine the psychotic symptoms diminished and mood began to improve. With ongoing treatment clinical improvement continued and both patients eventually recovered sufficiently to enable discharge from hospital.

The atypical antipsychotic olanzapine may, therefore, have a place in the management of treatment-resistant psychotic depression. This would be in keeping with its effects on comorbid mood symptoms in schizophrenia (Tollefson & Kuntz, 1999) and its suggested adjunctive role in the treatment of bipolar disorder (Carlos *et al*, 1998).

Beck, A. T., Ward, C. H. & Mendelson, M. (1961) An inventory for measuring depression. *Archives of General Psychiatry*, **4**, 561–571.

Carlos, A. Z. Jr, Narendran, R., Tohen, M., *et al* (1998) Clinical predictors of acute response with olanzapine in psychotic mood disorders. *Journal of Clinical Psychiatry*, **59**, 24–28.

Cliffe, M., Possami, A. & Mulhall, D. J. (1995) Modified personal questionnaire rapid scaling technique for measuring delusional beliefs. *British Journal of Clinical Psychology*, **34**, 251–253.

Hamilton, M. (1960) A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, **23**, 56–62.

Tollefson, G. D. & Kuntz, A. J. (1999) Review of recent clinical studies with olanzapine. *British Journal of Psychiatry*, **174** (suppl. 37), 30–35.

G. S. Malhi, S. A. Checkley National Unit for Affective Disorders, Maudsley Hospital, London SE5 8AZ

Use of long-acting benzodiazepines in older people

Sir: Taylor *et al* (1998) produced data from Liverpool showing no reduction in overall benzodiazepine use among the elderly during a 10-year period, and that there was a high rate of inappropriate use of the drugs.

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There is another aspect of this issue that is worthy of particular emphasis – the use of long-acting benzodiazepines in older people, as opposed to short-acting agents. There is evidence that elderly people who are taking benzodiazepines with a long duration of action are particularly prone to suffer falls, and Hemmelgarn *et al*, (1997) have shown that there is a significantly increased risk of being involved in a motor vehicle crash for an older person on a long-acting, but not a short-acting, benzodiazepine. While Taylor *et al* (1998) refer to advice that diazepam and chlordiazepoxide may be inappropriate for use in older people, they do not specifically address this aspect of benzodiazepine use. On the basis of the information given with regard to benzodiazepine type, 274 (44%) of 621 benzodiazepine users in the Liverpool sample were taking a long-acting agent (elimination half-life $\geq$24 hours). Comparative data for 1985 in North America (Ray *et al*, 1989) showed that one-third of older benzodiazepine users were taking long-acting drugs and, therefore, the even higher use in the 1990s is of concern, given the increasing availability of short-acting alternatives. The Liverpool data would also appear to suggest that the use of long-acting benzodiazepines is more common in the anxiolytic class (88/135, 65%) than in the hypnotic class (186/486, 38%) of benzodiazepines. This may be explained by the frequent attention placed on the hangover effect of long-acting hypnotics, with the adverse effects of long-acting daytime anxiolytics receiving less attention.

It is worth noting that these findings refer to the community-dwelling elderly and that many such older people remain independent and continue to drive and, therefore, may be vulnerable to the adverse effects of long-acting benzodiazepines in the course of their normal daily activities.

Hemmelgarn, B., Suissa, S. & Huang, A. (1997) Benzodiazepine use and the risk of motor vehicle crash in the elderly. *Journal of the American Medical Association*, **278**, 27–31.

Ray, W. A., Griffin, M. R. & Downey, W. (1989) Benzodiazepines of long and short elimination half-life and the risk of hip fracture. *Journal of the American Medical Association*, **262**, 3303–3307.

Taylor, S., McCracken, C. F. M., Wilson, K. C. M., *et al* (1998) Extent and appropriateness of benzodiazepine use. Results from an elderly urban community. *British Journal of Psychiatry*, **173**, 433–438.

M. Kirby Mater Hospital, Department of Psychiatry of Old Age, Dublin 7, Republic of Ireland

Early psychosis

Sir: The June 1998 supplement of the *British Journal of Psychiatry* was devoted to a discussion of early psychosis. Patrick McGorry, the Guest Editor, introduced the subject with an extract from a 1938 article by D. Ewen Cameron. McGorry credited Cameron with originally foreshadowing this form of preventive intervention and he was apparently using Cameron as a source of authority. It is uncertain whether McGorry is aware of Cameron's unsavoury reputation as a CIA-funded unethical experimenter. Cameron attempted to erase his patients' self-identities using electroconvulsive therapy and deep sleep. McGorry's judgement in openly citing Cameron as a source of authority is unsound.

Perhaps McGorry has not read the Discussion which follows Cameron's 1938 article. Harry Stack Sullivan's adverse comments about Cameron's thinking and the general idea of pre-psychotic detection and intervention do not accord with McGorry's beliefs. Sullivan (1938) wrote:

> "I would be very deeply disturbed if, as is implied by the last speaker [Cameron], people who show signs of personality disorders, early mental disorder of an indeterminate kind, were to be rushed through treatment with insulin, metrazol and camphor on the chance that they might otherwise have developed schizophrenia. I privately have a suspicion that might have a distinctly unfavourable effect on the general intelligence level and so on of the community. What does it mean that a person will have schizophrenia which can be detected by the intelligent layman months to years before the schizophrenia appears? In seven and half years of exclusive preoccupation with the schizophrenia problem I was unable to put my finger on anything sufficiently simple and obvious to service this purpose".

Was Sullivan right to nip this scheme in the bud in the late 1930s? Cameron proved to have many very bad ideas yet McGorry draws on Cameron as a source of authority. Should we allow McGorry to persuade us that among all of Cameron's bad ideas, this one is an exception?

McGorry, P. (1998) Preventive strategies in early psychosis: verging on reality. *British Journal of Psychiatry*, **172** (suppl. 33), 1–2.

Sullivan H. S. (1938) 'Discussion' in D. Ewen Cameron. 'Early schizophrenia'. *American Journal of Psychiatry*, **95**, 579.

R. Gosden University of Wollongong, Science and Technology Studies, NSW 2522, Australia

Author's reply: Gosden takes me to task for quoting Ewen Cameron in the *British Journal of Psychiatry* Supplement 33 on early psychosis, because of his subsequently highly problematic career. I was made aware for the first time by Canadian colleagues, after the acceptance of the Supplement for publication, of Cameron's dubious later activities, but originally quoted him on the merits of his landmark paper from the *American Journal of Psychiatry* in 1938. Although he was not the first to emphasise the importance of early intervention in schizophrenia and related psychoses (in fact, Sullivan had done so a decade earlier), his paper highlighted how little was known about the appropriate ways and means of intervening at such an early stage. This challenging yet cautionary note, stressing the level of ignorance which existed and continued for a prolonged period afterwards, is actually the point of the quote I selected. Ironically, it is quite at odds with the interpretation adduced by Gosden, namely a feared headlong rush to apply dubious treatments to arguably healthy individuals. Furthermore, the remainder of the introduction and the Supplement as a whole clearly reflects a belief in the need to collect and weigh the evidence in relation to all aspects of early psychosis.

In the discussion which accompanied the paper, which I had certainly read and from which I have also quoted in scientific forums, Sullivan, while not eschewing his support for a preventive approach, quite appropriately argued against the widespread application of treatments which were unproven and carried the potential for harm. He stressed the need for very accurate prediction of incipient psychosis, if specific treatment were to be considered prior to onset of psychosis. Such caution is appropriate today, particularly in relation to those at increased risk of psychosis, but who have not yet met diagnostic thresholds, and we have been active in research aimed at improving our predictive capacities (Yung & McGorry, 1997). In such potentially prodromal cases, desire for treatment, level of distress and disability, risk of harm and risk of early transition to psychosis are factors influencing the decision to offer treatment when it is sought. It is clearly premature to offer antipsychotic medications in a widespread fashion to such potentially pre-psychotic patients until a series of well-conducted randomised controlled trials from several

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