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M. Willis Specialist Registrar, Great Ormond Street/Royal London Rotation, Simmons House Adolescent Unit, St Luke's-Woodside Hospital, Woodside Avenue, London N10 3HU, UK.
Email: willismarcia@yahoo.com
doi: 10.1192/bjp.189.5.467a

Acute and transient psychotic disorders and puerperal psychosis

Marneros (2006) addresses an important issue in his editorial on the concept of acute and transient psychosis, which is a challenge to the Kraepelinian dichotomy. He argues that acute and transient psychoses are separate from schizophrenia, schizoaffective disorder or affective disorder, based on the clinical manifestations, but he did not mention puerperal or post-partum psychosis, which also lacks a consensus of definition (Kohl, 2004). Post-partum psychosis has been described as functional psychosis with good prognosis and clinical presentation similar to acute and transient psychosis (Kendell *et al*, 1987). Despite a varying symptomatology, women with schizophrenia rarely experience arousal of their symptoms after childbirth (Meltzer & Kumar, 1985). Puerperal psychosis appears to occupy a clinical position which is different from schizophrenia and affective disorder.

It is of interest that acute and transient psychosis mainly affects females (Marneros, 2006), and suggests a link between puerperal psychosis and acute and transient psychosis. I therefore suggest that the concept of puerperal psychosis should be included in discussions of the concept of acute and transient psychosis.

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Kohl, C. (2004) Postpartum psychosis: closer to schizophrenia or the affective spectrum? *Current Opinion in Psychiatry*, **17**, 87–90.

Marneros, A. (2006) Beyond the Kraepelinian dichotomy: acute and transient psychotic disorders and the necessity for clinical differentiation. *British Journal of Psychiatry*, **189**, 1–2.

Meltzer, E. S. & Kumar, P. (1985) Puerperal mental illness, clinical features and classification: a study of 142

mother-and-baby admissions. *British Journal of Psychiatry*, **147**, 647–654.

J. Lewin Coombe Wood Perinatal Service, Park Royal Centre for Mental Health, Central Way, London NW10 7NS, UK. Email: jona.lewin@nhs.net
doi: 10.1192/bjp.189.5.468

Author's reply: Dr Lewin is right that puerperal psychoses are of special interest in the context of acute and transient psychoses. To our knowledge there is consensus that post-partum disorders are not distinct nosological entities (Brockington, 2004; Riecher-Rössler & Rohde, 2005) with neither 'post-partum depression' nor 'post-partum psychosis' having specific aetiology. 'Giving birth to a child with all its biological and psychosocial consequences seems to act as a major stressor, which – within a general vulnerability–stress–model – can trigger the outbreak of all classical disorders in predisposed women' (Riecher-Rössler & Rohde, 2005). Hence it is evident that the situation after delivery can be typical for triggering acute and transient psychosis.

Re-evaluation of our own sample of 61 women (Rohde & Marneros, 1993) with first onset of psychosis after delivery showed that according to ICD–10 criteria 18 (29.5%) should be classified as having acute and transient psychosis (Rohde & Marneros, 2000); all other diagnostic categories were also present (schizoaffective and affective disorders, schizophrenic and organic psychoses). In our sample the frequency of acute and transient psychoses was much higher than expected from the general prevalence. This might be a reason for the frequent observation that puerperal psychoses are mainly very acute, short episodes with a 'colourful' psychopathology and good prognosis.

Considering the few available studies we conclude that in the post-partum period acute and transient psychoses represent a disorder that is different from other psychiatric disorders but is part of a psychotic continuum.

Brockington, I. (2004) Postpartum psychiatric disorders. *Lancet*, **363**, 303–310.

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Rohde, A. & Marneros, A. (1993) Postpartum psychoses: onset and long-term course. *Psychopathology*, **26**, 203–209.

Rohde, A. & Marneros, A. (2000) Bipolar disorders during pregnancy. In *Bipolar Disorders 100 Years After Manic Depressive Insanity* (eds A. Marneros & J. Angst), pp. 127–137. Dordrecht: Kluwer Academic Publishers.

A. Marneros Universitätsklinik für Psychiatrie und Psychotherapie der Martin-Luther-Universität Halle-Wittenberg, 06097 Halle/Saale, Germany.
Email: andreas.marneros@medizin.uni-halle.de
doi: 10.1192/bjp.189.5.468a

Cognitive impairment in euthymic patients with bipolar disorder

By prospectively verifying euthymia and controlling for the effect of residual affective symptoms Goswami *et al* (2006) make a significant contribution to the existing evidence on cognitive impairments in euthymic patients with bipolar disorder. However, they did not define euthymia and the diagnosis of DSM–IV bipolar I disorder, verification of euthymia and exclusion of current and past psychiatric illness or substance use disorders in patients and controls were made without structured assessments. Controls were relatives of participating patients. In addition, exclusion criteria make no mention of birth injuries, the handedness of patients and whether patients had received electroconvulsive therapy. All these factors influence results of tests for cognitive function (Ferrier & Thompson, 2002).

As the Schedule for Assessment of Psychiatric Disability assesses marital and occupational functioning, details of the patients' marital or occupational status would have helped to better interpret the data. There is also no mention of the duration of illness (in Table 1, p. 368, duration spent in episodes is erroneously labelled as duration of illness). This variable has implications for the generalisability of findings.

A measure of the reliability and validity of the modified Kolakowska battery is not provided. The use of more systematic and better-validated instruments such as the Cambridge Neurological Inventory (Chen *et al*, 1995) and more than one rater to reduce assessment bias would have allowed better characterisation of neurological soft signs. About 92% of healthy controls in the current study had neurological soft signs. This unusually high prevalence could

reflect the inappropriateness of the control group.

In the Rey Auditory Verbal Learning Test, significantly lower scores on lists A1–A5 were taken to infer a reduction in verbal memory. However, there was no difference between patients and controls for lists A6 and A7. The percentage of words retained between trials A5 and A7 would provide a purer index of retention (Thompson *et al*, 2005) and would help to better interpret the data.

In the future, meta-analyses of existing data and studies involving assessment of cognitive function and neuroimaging in euthymic patients with bipolar disorder should help elucidate a profile of cognitive deficits and their underlying neurobiological bases.

Chen, E. Y., Shapleske, J., Luque, R., et al (1995) The Cambridge Neurological Inventory: a clinical instrument for assessment of soft neurological signs in psychiatric patients. *Psychiatry Research*, **56**, 183–204.

Ferrier, I. N. & Thompson, J. M. (2002) Cognitive impairment in bipolar affective disorder: implications for the bipolar diathesis. *British Journal of Psychiatry*, **180**, 293–295.

Goswami, U., Sharma, A., Khastigir, U., et al (2006) Neuropsychological dysfunction, soft neurological signs and social disability in euthymic patients with bipolar disorder. *British Journal of Psychiatry*, **188**, 366–373.

Thompson, J. M., Gallagher, P., Hughes, J. H., et al (2005) Neurocognitive impairment in euthymic patients with bipolar affective disorder. *British Journal of Psychiatry*, **186**, 32–40.

R. Bharadwaj Department of Psychiatry, Postgraduate Institute of Medical Education and Research, Chandigarh 160012, India.
Email: r.s.bh@yahoo.com
doi: 10.1192/bjp.189.5.468b

Authors' reply Certain aspects of methodology were left out of our paper owing to space constraints. Bharadwaj cites Ferrier & Thompson (2002) when questioning the exclusion criteria used in our study. Both are co-authors of our paper, which is a result of collaborative research between the Department of Psychiatry in New Delhi and Newcastle since 1998. Whenever possible, similar tests and criteria for euthymia have been used in both centres with occasional variations to respect cultural differences. Use of spouses and siblings as members of the control group was acceptable, as it brought together people of broadly similar backgrounds. Although this might have resulted in the inclusion of a limited number of controls who were at risk of developing bipolar disorder, it minimised

differences between people with bipolar disorder and controls without greatly confounding our results.

For verification of euthymia participants were seen at least twice, separated by a minimum of 1 month, before they were tested. Clinical judgement of euthymia was reinforced by a Hamilton Rating Scale for Depression score <8 and a score <20 on Bech's modification of Beigel's Manic State Rating Scale on both occasions. The stability of mood during the intervening period was assessed clinically on a weekly basis. We were not aware of any Hindi version of the Structured Clinical Interview for DSM–III – patient version. The exclusion of other psychiatric morbidity was based on clinical interviews by two highly experienced psychiatrists, complemented by careful mapping of life charts using the techniques of Post *et al* (1998).

Soft neurological signs were assessed with the widely used modified Kolakowska battery. We are unsure whether the use of other batteries, such as the Cambridge Neurological Inventory, would substantially alter our findings. Involving a second rater would perhaps increase reliability but would extend the assessment time unreasonably.

Not surprisingly, soft signs were found in the control group, but only at about one-quarter of the severity seen in people with bipolar disorder. The maximum score on the modified Kolakowska battery was 45. The maximum score for controls was 9 whereas the mean for patients was 13.9. Control scores mainly comprised minimum scores on a few of the 15 items. In a subsequent article (further details available on request) we report high levels of soft signs in the youngest patients with bipolar disorder. There is no evidence that soft signs progress with age in bipolar disorder, whereas in controls there is significant ($P < 0.01$) progression with age.

List A7 of the Rey Auditory Verbal Learning Test measures retention after 20 min. We have further analysed these data and found no difference between the groups.

We agree that 'duration of illness'; actually describes 'duration of illness episodes'. The actual mean duration of illness was 9.1 years (s.d.=6.0). Data concerning marital status and occupation were collected but were omitted for brevity. We did not wish to control for handedness or birth injuries as potential confounders as we regarded these differences to be part of

the spectrum of people with bipolar disorder. We did not include those who had recently received electroconvulsive therapy (≥ 6 months). Finally, we would agree that there is a need for meta-analyses and have recently published such a study (Robinson *et al*, 2006).

Ferrier, I. N. & Thompson, J. M. (2002) Cognitive impairment in bipolar affective disorder: implications for the bipolar diathesis. *British Journal of Psychiatry*, **180**, 293–295.

Post, R. M., Roy-Byrne, P. P. & Uhde, T. W. (1998) Graphic representation of the life course of illness in patients with affective disorder. *American Journal of Psychiatry*, **145**, 844–848.

Robinson, L. J., Thompson, J. M., Gallagher, P., et al (2006) A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. *Journal of Affective Disorders*, **93**, 105–115.

U. Goswami Department of Psychiatry, West Park Hospital, Darlington, UK

P. B. Moore Department of Psychiatry, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP, UK.
Email: pbrianm@aol.com
doi: 10.1192/bjp.189.5.469

Delay in onset of action of antidepressants

In an important editorial, Mitchell (2006) marshalls evidence to show that we do not have to wait 2 weeks for antidepressants to work.

Why has it been so difficult so far to show that they work in the first few days? In addition to the reasons that Mitchell sets out, I should like to mention a further problem. If you analyse the results on a day-by-day basis, it is hard to obtain sufficient statistical power to distinguish the early response to the drug from the response to the placebo, since you have just the scores for that day.

In 1996 my colleagues and I published evidence that the fall in scores on the Hamilton Rating Scale for Depression followed an exponential decay curve with a correlation coefficient of 0.99 (Priest *et al*, 1996; Livingston & Clark, 1997). This observation corresponds with Mitchell's remarks on the steep fall in scores in the first 2 weeks. A comparison of the slope of the curve for the active drug with the placebo, using all of the data, gives a very sensitive way of testing for efficacy.

By plotting the log of the depression scores against time, a straight line is obtained. Thus the recovery from depression