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Correspondence

Contents: Steroid-induced prepartum psychosis/Schizophrenia in twins/Screening for pathological gambling among substance misusers/Attention deficit disorder/In-patient psychotherapy/Antipsychotic drug-induced dysphoria/Inappropriate secretion of antidiuretic hormone and SSRIs/Seizure length during ECT/Suicide prevention versus alcohol promotion/Quaker belief and child psychiatry.

Steroid-induced prepartum psychosis

SIR: The possibility of puerperal psychosis having an onset before delivery has been suggested by Brockington *et al* (1990), but only a small number of cases have been reported in the literature. Glaze *et al* (1991) discussed the nature of the association between prepartum and postpartum psychoses and hypothesised that prepartum psychosis could occur as a result of sensitisation by earlier postpartum illness.

Case report

A 31-year-old woman presented at 36 weeks gestation with symptoms suggestive of hypomania. Two weeks previously she had been treated with prednisolone (20 mg/day), for an infective exacerbation of asthma. The total dose prescribed was 60 mg. There was no relevant past psychiatric or family history.

The patient responded to haloperidol (dose range: 5–15 mg per day). Her symptoms gradually settled over the next month before being discharged home on no neuroleptic medication.

The patient remained stable until her subsequent (fourth) pregnancy. One week after delivery, she was admitted with a relapse of psychotic illness. During this admission the patient was treated with electroconvulsive therapy for escalating disturbance. She was discharged on lithium and haloperidol.

In this case the illness may have been precipitated by the use of steroids, predisposing the patient to later episodes of post-natal illness. Steroids are frequently prescribed in pregnancy – most commonly dexamethasone sulphate for premature rupture of membranes. If indeed steroid therapy has the potential to precipitate severe and recurring puerperal illness then prospective studies of pregnant women treated with dexamethasone would appear to be an appropriate focus of future research.

BROCKINGTON, I. F., OATES, M. & ROSE, G. (1990) Prepartum psychosis. *Journal of Affective Disorders*, **19**, 31–35.

GLAZE, R., CHAPMAN, G. & MURRAY, D. (1991) Recurrence of puerperal psychosis during late pregnancy. *British Journal of Psychiatry*, **159**, 567–569.

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Schizophrenia in twins

SIR: Congratulations go to Kläning *et al* (1996) for their epidemiological demonstration that twins are more prone to schizophrenia than singletons. This is an important finding. It was one which we (Murray *et al*, 1985) predicted in an early formulation of the neurodevelopmental hypothesis of schizophrenia. If obstetric complications do constitute a risk factor for schizophrenia, and twins are known to be more prone to obstetric complications, then it had to follow that twins were at higher risk of schizophrenia. This is certainly the case with other neurodevelopmental disorders such as cerebral palsy and epilepsy.

An important implication which the authors do not discuss is that their finding effectively invalidates the classical twin method as a basis for estimating heritability in schizophrenia. A central assumption of the twin method is that the disorder under examination is no more common in twins than in singletons. That this might be the case was always hinted at by the apparently anomalous finding that concordance rates for schizophrenia between dizygotic (non identical) twins were consistently higher than those recorded amongst ordinary siblings despite the genetic relationship being the same in both cases. I hope that Kläning and colleagues can use their dataset to test further explanatory hypotheses about their findings.

KLÄNING, U., MORTENSEN, P. B. & KYVIK, K. O. (1996) Increased occurrence of schizophrenia and other psychiatric illnesses among twins. *British Journal of Psychiatry*, **168**, 688–692.

MURRAY, R. M. LEWIS, S. W. & REVELEY, A. M. (1985) Towards an aetiological classification of schizophrenia. *Lancet*, **i**, 1023–1026.

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Screening for pathological gambling among substance misusers

SIR: Pathological gambling is a recognised psychiatric disorder with an estimated prevalence of 1 to 2% of the population (Roy *et al*, 1988). To aid early identification Lesieur & Blume (1987) developed the South Oaks Gambling Screen (SOGS). This is a 20-item self-report questionnaire based on DSM–III criteria for pathological gambling. It has been shown to have internal consistency and high test–retest reliability and to correlate well with the criteria set out in DSM–III–R for pathological gambling.

We administered the SOGS to a consecutive series of 376 substance misusers admitted as inpatients to the Substance Abuse Rehabilitation Unit (ARU) at the Veterans Administration Medical Center, East Orange, New Jersey. The SOGS was administered by the admitting nurse as part of the admission process to the ARU on the first day of admission.

Forty-nine of the 376 patients (13%) scored 5 or greater on the SOGS, placing them in the pathological gambling category of the SOGS. Only three patients refused to complete the SOGS.

The possible clinical implication of this result is that substance misuse programmes need to be aware that a meaningful percentage of their patients may also have problems with gambling. Identification of such patients would allow for treatment plans to incorporate interventions aimed specifically at the problem gambling.

LESIEUR, H. & BLUME, S. (1987) The South Oaks Gambling Screen (SOGS): a new instrument for the identification of pathological gamblers. *American Journal of Psychiatry*, **144**, 1184–1188.

ROY, A., ADINOFF, B., ROEHRICH, L., *et al* (1988) Pathological gambling: a psychobiological study. *Archives of General Psychiatry*, **45**, 369–373.

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Attention deficit disorder

SIR: Sandberg (1996) provides an interesting review on the recent thinking about hyperkinetic syndrome and attention deficit disorder. The breadth of the topic does make a comprehensive review difficult; however I think that there were several important aspects to these disorders which were not referred to.

The review does not mention the association between deficits in attention and tic disorders such as Gilles de la Tourette syndrome. This is an important area since studies show that between 21 and 90% of children with Tourette's are restless and overactive (Robertson, 1994). In addition treatment of hyperactivity with stimulants such as methylphenidate can exacerbate pre-existing tics or possibly even precipitate tics in children predisposed to developing them.

Sandberg, while mentioning the association with reading disorder, did not refer to reported links between attention deficit disorder and other developmental difficulties such as dyspraxia. Gillberg *et al* (1989) have described children with deficits in attention, motor functioning and perception (DAMP) as a syndrome overlapping with other forms of overactivity.

The section on medication refers to the use of stimulants but fails to mention alternative drug treatments such as tricyclic antidepressants (mainly imipramine) or clonidine. Admittedly these are not first-line treatments and the use of tricyclics in children is not without risks but clonidine is proving to be a useful alternative. There are some reports suggesting that clonidine may be particularly recommended in cases where overactivity and tics co-exist (Hunt *et al*, 1985).

GILLBERG, I. C., GILLBERG, C. & GROTH, J. (1989) Children with preschool minor neurodevelopmental disorders V: neurodevelopmental profiles at age 13. *Developmental Medicine and Child Neurology*, **31**, 14–24.

HUNT, R. D., MINDERAA, R. B. & COHEN, D. J. (1985) Clonidine benefits children with attention deficit disorder and hyperactivity: report of a double-blind placebo-crossover therapeutic trial. *Journal of American Academy of Child Psychiatry*, **24**, 617–629.

ROBERTSON, M. M. (1994) Gilles de la Tourette syndrome – an update. *Journal of Child Psychology and Psychiatry*, **35.4**, 597–612.

SANDBERG, S. (1996) Hyperkinetic syndrome or attention deficit disorder. *British Journal of Psychiatry*, **169**, 10–17.

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