stands out as the sole correlate of schizophrenia. Yet Table 2 of Thomas *et al* indicates that abnormalities of foetal heart rate (<100 or >160 b.p.m.), a quantifiable component of the Apgar score, did not distinguish cases from controls in any of the subgroups examined. In other words, the heart rate was unchanged in the cases the authors judged to be asphyxiated at birth.

Two recent case–control studies cast doubt on the pathogenic influence of asphyxia that Dalman *et al* claim to have detected. Kendell *et al* (2000) found no evidence that an Apgar score of <7 distinguished the birth records of 156 cases of schizophrenia from 156 matched controls (nor did the Apgar score distinguish cases from controls in their study of 217 probands with affective psychoses; Bain *et al*, 2000). Byrne *et al* (2000) found that the assessments of 'incubator/resuscitation/blue' and 'asphyxiation', both of which would appear to have qualified a case for the category of 'asphyxia' as defined by Dalman *et al*, did not distinguish 431 individuals with schizophrenia from 431 gender-matched controls. Both the studies of Kendell *et al* and of Byrne *et al* drew negative conclusions regarding the role of a range of labour and delivery complications in the aetiology of schizophrenia. The negative findings of Thomas *et al* add to this consensus, while the positive claims regarding asphyxia of Dalman *et al* stand in contrast to other recent studies and may be attributable to observer bias.

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MORE LARGE STUDIES NEEDED

We need more large studies and cumulative meta-analyses of individual obstetric complications and their effects on the neonate

Obstetric complications are 'one' of the few putative causes of schizophrenia for which there is relatively good evidence (Geddes & Lawrie, 1995), but only a few particular obstetric complications are likely to be important (Geddes *et al*, 1999) and they may have at most non-specific effects, for example in bringing forward the age at onset (Verdoux *et al*, 1997). The importance of the papers by Dalman *et al* (2001, this issue) and Thomas *et al* (2001, this issue) is that they attempt to relate obstetric complications to their effects on the neonate, and to the subsequent development of schizophrenia. They are a valuable contribution to the ongoing debate about the role of obstetric complications in schizophrenia, despite some inevitable methodological limitations.

The investigators identified the obstetric records of 524 cases and 1043 controls ascertained from the Stockholm County In-Patient Register, thus avoiding the potential pitfalls of maternal recall bias. Apgar scores were recorded at the time of delivery in only 20.5% of the sample and the majority of the scores were therefore calculated retrospectively, albeit blind to case/control status. An Apgar score of 6 or less at 1, 5 or 10 minutes was taken as evidence of asphyxia and found in 44 obstetric records. These 'positive' records were then scrutinised by experienced paediatricians, although negative records were not subject to the same scrutiny. Interrater reliability was high. The methodological limitations may have resulted in some bias but are unlikely to have lead to a false positive result.

Sample characteristics for cases and controls differed in a few important respects. A higher proportion of cases were unmarried or divorced, many received inadequate antenatal care and cases were more likely to have a history of maternal psychotic illness. The risk each complication contributed to the development of schizophrenia was calculated using the odds ratio (OR) by conditional logistic regression.

Most obstetric complications were not found to contribute any additional risk, with the exception of signs of asphyxia which were found significantly to increase the odds of the subsequent development of schizophrenia (OR 2.7, 95% CI 1.5–4.8). This result remained significant and was in fact strengthened once potential confounders (maternal history of psychotic illness, maternal age, socio-economic class, marital status, attendance at antenatal care) were taken into account (OR 4.4, 95% CI 1.9–10.3). Notably, however, no dose–response relationship was found between the severity of asphyxia and the risk of schizophrenia. This does not support an aetiological relationship, but one could argue that collapsing Apgar scores of less than seven over three time points (presumably to increase statistical power) added 'noise'. A large or consistent effect of gender, age at diagnosis or maternal history of psychosis was not found.

These results are in keeping with the results of meta-analyses suggesting that obstetric complications are not simply a manifestation of genetic risk and may be pathogenic via a potential final common pathway of hypoxic brain damage (Verdoux *et al*, 1997). Although the age at onset effect was not significant, the results are in the expected direction. Single studies are often underpowered, frequently fail to find significant differences between cases and controls and tend to rely on summary scales of mainly maternal complications. The current studies avoid the problems of summary scales and their uncertain interpretation. It is, however, sobering to realise that despite a total sample of over 1500 they may have been too small to detect some important effects. More large studies of specific complications, and cumulative meta-analyses of them, are required before the case for or against the potential role of obstetric complications in schizophrenia is conclusive.

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