AUTHORS' REPLY: Takei and Murray's calculations (as I see it) do not address the issue, which is simple.

O'Callaghan *et al* (1991) concluded that an 88% increase in the births of patients with schizophrenia in March 1958 was due to the fact that their mothers, while in the second trimester of pregnancy, had been infected with the influenza virus. Thirty-four children who later developed schizophrenia (by narrow criteria) were born to the 16 268 mothers in the NCDS who gave birth in the week 3–9 March 1958. If O'Callaghan *et al* were right, 88/188 of these 34 children (=15.9 children) developed schizophrenia because their mothers suffered from influenza in the second trimester. However, influenza infection during pregnancy was recorded in this survey, and 945 mothers are noted as affected in the second trimester. Therefore (according to O'Callaghan *et al*) it is these 945 mothers who must have given birth to 15.9 children with schizophrenia. In reality, only one case of schizophrenia was recorded as being born to these mothers, where (according to the hypothesis) there should have been 15.9.

Therefore (I concluded) O'Callaghan *et al*'s conjecture must be mistaken. For the reasons I gave in my editorial, the other evidence that is put forward in support of the view that prenatal exposure to influenza causes schizophrenia does not seem cogent. For example, O'Callaghan *et al* (1991) stated in their summary that "five months after the peak infection prevalence (following the 1957 epidemic), the number of births of individuals who later developed schizophrenia was 88% higher than the average number of such births in the corresponding periods in the two previous and the next two years" and concluded that the relationship was causal. Sham *et al* (1992) wrote that "our results indicated that 1–2% of all schizophrenic births can be explained by the number of influenza deaths in the preceding months". If these two sets of analyses are both to be interpreted in favour of a relationship, they suggest effects that differ by two orders of magnitude – but Takei and Murray are co-authors of both papers. In the face of such unreconciled inconsistencies I do not see that the theory that prenatal exposure to influenza causes schizophrenia can be taken seriously.

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Night terrors

SIR: There is apparently some diagnostic confusion regarding the classification of the episodic phenomena presented in the case report by Lillywhite *et al* (*BJP*, April 1994, **164**, 551–554). The International Classification of Sleep Disorders (ICSD, 1990) differentiates between several paroxysmal nocturnal anxiety states. A nightmare (307.47–0) is a REM-parasomnia, awakening typically occurring out of REM-sleep. A sleep terror (307.46–1) is a disorder characterised by sudden arousal from slow wave sleep (SWS). It is an unusual NREM parasomnia in adults (<1%). Nocturnal panic episodes (300) most often occur in light, stage 2 NREM sleep.

The patient's sleep recording reported by Lillywhite *et al* shows a superficial, fragmented sleep pattern, as often recorded in patients using alcohol and benzodiazepines. In our opinion, sleep preceding these events seems to deepen, and in fact shows hypersynchronous delta bursts, an EEG pattern reported in adult somnambulism often preceding arousal (Blatt *et al*, 1991). Somnambulism is a sleep NREM stage-dependent parasomnia. The neurophysiological characteristics of this first night recording thus favour the diagnosis of nocturnal panic attacks rather than sleep terrors.

A further argument favouring this interpretation is the observation that the SWS increase reported in the second sleep recording does not deteriorate the clinical picture, as would have been expected for sleep terrors. SWS increase following serotonin agonists is not surprising, as serotonin is a neuromodulator involved in NREM sleep rebound (Sallanon *et al*, 1983). Increased serotonergic drive

https://doi.org/10.1017/S0007125000073827 Published online by Cambridge University Press