Inter-relationships among mental and motor symptoms, including gaze and thought disorders, sequentially analysed on a time-base, handedness, gender, lateralised brain functions and detailed gyrus-by-gyrus analysis of the frontal lobe (*Journal*, February 1990, **156**, 216–227) may provide an understanding of how specific patterns of monaminergic overactivity lead to the cognitive disturbances that are clinically significant and a hallmark of schizophrenia (Braff & Geyer, 1990).

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Failure of progesterone treatment in puerperal mania

SIR: Progesterone is widely used in the treatment and prophylaxis of post-natal depression (Dalton, 1985). Sedative effects have been reported when this hormone is given intravenously (Merryman *et al*, 1954). If the onset of psychosis in the early puerperium is related to the precipitous fall in circulating progesterone after childbirth, progesterone therapy might be of benefit in early-onset puerperal mania.

We tried this therapy with three patients, all of whom expressed a preference to try hormonal therapy rather than neuroleptic drugs. The first patient, with a seven-day history of mania starting six days post-partum, reported a subjective calming effect with progesterone (50 mg intramuscular) before and after neuroleptic therapy was commenced. The second patient had suddenly become manic on day four post-partum; administration of progesterone (100 mg) on day six, and repeated after 12 hours, was associated with a surprising return to normality over 24 hours. She had, however, received chlorpromazine (50 mg intramuscular) and haloperidol (40 mg intramuscular) over the 24 hours before being given progesterone. These experiences suggested that intramuscular progesterone was well tolerated and might have an antimanic effect, and encouraged us to use it as the sole therapy in a typical case of puerperal mania.

The third patient had suddenly developed puerperal mania on day seven post-partum and had only been given oral chlorpromazine (100 mg) before progesterone was started. Progesterone (100 mg intramuscular, 12 hourly) was commenced as the sole therapy 12 hours after the onset of the disorder and continued for one week. A random blood level late in this week was 476 nmol/l (about average for levels in late pregnancy). There was *no effect at all* on her mental state with this regime. After switching to standard neuroleptic therapy, a significant improvement occurred within a few days. While we would not wish to discount the possibility of a beneficial effect of progesterone on the basis of one case, we think it important to give publicity to this definite therapeutic failure.

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Post-traumatic stress disorder

SIR: Medley *et al* (*Journal*, January 1990, **156**, 134) raise two important issues in relation to post-traumatic stress disorder (PTSD).

The debate over the relative importance of the trauma itself and pre-existing personality factors and psychological morbidity is likely to be around for some time to come. Of more immediate clinical importance is the diagnosis of PTSD in survivors of catastrophic accidents who have sustained significant head injury. DSM–III and DSM–III–R both indicate that the first criterion for the diagnosis of PTSD is that the individual has 'experienced' an extreme or catastrophically stressful life event. Survivors, such as those who survived the recent crash on the M1, who were immediately rendered unconscious and remained so while they were being rescued, would not have 'experienced' the event. It should not therefore be expected that they would