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

satisfy the criteria for a diagnosis of PTSD. Some survivors, however, may have had a fluctuating state of consciousness and therefore may have had some experience of the event.

Some symptoms of a concussive head injury and of PTSD overlap, and where there is amnesia for the event associated with a head injury, it is important to determine, from ambulance and casualty records, the level of consciousness and whether or not this fluctuated. In the ambulance officer's and casualty reports of two such cases I have recently seen (one eighteen months and one three-and-a-half years following very serious road traffic accidents) there was evidence of patchy awareness of their circumstances, both at the scene of the accident and in the casualty department. In both cases there was, initially, total amnesia for the accident and a significant period of amnesia in relation to their subsequent admission to hospital. Both had shown post-concussional symptoms for some months and both were referred for psychiatric assessment in relation to an exacerbation of their physical symptoms. Since there was total amnesia for the accident, there was no 're-experiencing of the event' but otherwise both had symptoms satisfying the other criteria of a PTSD. It appeared that evidence of re-experiencing the event was limited to re-experiencing specific pain and other physical symptoms. This would have come as no surprise to Pierre Janet 100 years ago (van der Kolk *et al*, 1989) who argued that when such traumatic experience is not properly digested it can return as "fragmentary reliving of the trauma, as emotional conditions, somatic states, visual images or behavioural re-enactments" (p. 366).

Clearly such patients need a very careful evaluation. Interviewed under intravenous sodium amytal, both the patients referred to above regained some patchy memories of the experience and some of the affect experienced at the time and during their admission to hospital. In both cases there remained, on neuropsychological assessment, evidence of significant but not incapacitating cognitive deficits.

Dr Medley *et al's* current investigation of the survivors of the airliner crash may indeed help to clarify some of the many interesting issues raised by the diagnosis of PTSD.

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SIR: Medley *et al* (*Journal*, January 1990, **156**, 134) raise the question of whether survivors who have amnesia for a traumatic event can develop PTSD. Our experience is that such survivors indeed can develop PTSD, although the presentation may be slightly altered.

Case report: A 22-year-old male soldier was travelling in a coach which was blown up. Eight other passengers were killed. He suffered a fractured mandible and a significant closed head-injury. He was unconscious for 36 hours and subsequently had a post-traumatic amnesia of 72 hours and a retrograde amnesia of eight hours. He made a full recovery physically from his injuries, but developed PTSD as defined by DSM–III–R within three weeks of the incident. In particular he experienced intense psychological distress and physiological reactivity on seeing a coach, with marked phobic avoidance. However, intrusive recollections did not occur and although he awoke with bad dreams he was unable to remember the content. He was treated using a cognitive-behavioural approach including *in-vivo* exposure to coaches. His symptoms improved considerably over a six month period and at the end of treatment he was able to travel by coach with only moderate anxiety.

Despite a total amnesia for the traumatic event this patient developed a significant PTSD. The lack of intrusive recollections, flashbacks or dreams is presumably related to the absence of any accessible memory. Psychogenic amnesia is a recognised feature of PTSD which could be a source of confusion, although the consistency and pattern of amnesia in this case is highly suggestive of an organic cause. It is uncertain whether the symptoms relating to coaches were as a result of being told afterwards of the event or because of a memory of it which he was not able to retrieve consciously. This man knew what had happened to him because there was much media attention and other survivors to talk to. It would be interesting to speculate how the presentation might differ in cases where such information were not available to survivors with amnesia.

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