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SIR: Crow (*Journal*, June 1990, **156**, 788–797) discusses the hypothesis that schizophrenia results from a disorder in the gene which determines cerebral dominance. In conceptual form the theory is supported by analysis of the unifying feature of the first-rank symptoms. These refer to the reception of vocal input ascribed to an external source, passivity phenomena in which a controlling external agency is implicated in thought insertion or withdrawal, or the imposition of motor or sensory data. In thought broadcast, thoughts are felt to be available to another externally. The subjective experience of the non-dominant hemisphere in its interaction with a dominant vocal hemisphere explains the above if the unifying feature is the awareness of the involvement of an external agency. These symptoms are understandable when considered in systems control theory as the experience of a processing unit which achieves a relative independence and seeks to understand its controller (e.g. reception of information by the right hemisphere from the left would be seen as thought insertion and vice versa as thought withdrawal). The above system would conform to the requirements for a neuropsychology of schizophrenia described by Frith & Done (1988) if the monitor and plan-evolving centre with willed intention represent dominant hemispherical functioning, while synthesis of perception, stimulus intention and resultant action represent non-dominant functioning. Support for the above possibility is mentioned in Birchwood *et al* (1988), where an account is given of the absence of first-rank symptoms in split-brain patients, post-hemispherectomy or in those with agenesis of the corpus callosum.

While there is conceptual support for Dr Crow's hypothesis, in consideration of the phenomenology of schizophrenia, the ascription of the abnormality of cerebral dominance to a single genetic abnormality is questionable. The single strongest point of opposition is Badian's (1983) paper (quoted by Crow, 1986) concerning the seasonal incidence of left-handedness. The results showed an excess of left-handedness in males born between September and February, with a high statistical significance. This pattern of increased left-handedness correlates well with the observed excess of winter births in schizophrenia. However, for cerebral dominance to interact so closely with an environmental variable any genetic theory of dominance must invoke an intermediate stage in development which is subject to the effects of an environmental variable. I am presently researching the possibility that the ratio of light:dark in the first few months following birth affects the left hemisphere to a greater extent than the right, and that genetic sensitivity to its effect would explain the majority of the above findings. In the first few months following birth, with the four-fold increase in brain mass, there would be a particular neurodevelopmental susceptibility to the action of an environmental variable.

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Post-traumatic stress disorder

SIR: Kennedy (*Journal*, January 1990, **156**, 129) wrote questioning whether the data in my recent studies about Australian volunteer fire fighters is capable of generalisation to post-traumatic stress disorder (PTSD) as a whole. He suggests that this may not be the case because this group was trained, well motivated and had previous experience. He goes on to state that the event lacked the element of surprise and unfamiliarity experienced by other PTSD groups examined.

These are important issues to consider. However, the experience of these fire fighters in the Ash Wednesday disaster was far beyond their most extreme expectations. The intensity of the heat in these fires was very great, with five metres of fire-front generating as much energy as a large power station (Webster, 1986). Most of these volunteers were also members of the local community and many had their homes damaged or destroyed. As well, while they were fighting the blaze, most knew that their families were also facing considerable risk. This was particularly difficult for them because they were not in a position to protect their families. Furthermore, often the radio communications used by the fire networks were completely ineffective because the intensity of the heat ionised the atmosphere. This created an atmosphere of isolation and lack of direction. Moreover, much of the fire-fighting equipment

also failed during the fires. Many of the vehicles used were powered by petrol motors, or had petrol pumps working the fire fighting equipment. The intensity of the heat was sufficient to vaporise the fuel in the lines. As a result they were often trapped in extraordinarily dangerous circumstances without equipment to protect themselves.

Having spoken to many of the fire fighters, this experience was both way beyond their wildest expectations of a major fire and had all the elements of extreme surprise and threat. Having interviewed many victims of the disaster (McFarlane, 1986) as well as studied the families of those victims (McFarlane *et al*, 1987), it appears that the experience of these fire fighters was often worse than that of the victims themselves because they were repeatedly exposed to extreme danger as well as having at times to remain on duty for periods of up to three days.

The experience of these fire fighters was thus one similar to all victims of extreme threat and danger. While they had some training, in the circumstances experienced, this offered them little or no protection. Therefore these data, I believe, are generally applicable to the aetiology and phenomonology of PTSD.

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Malaria presenting as atypical depression

Sir: Arun Prakash & Stein (*Journal*, April 1990, **156**, 594–595) mention hysterical stupor and atypical depression as sequelae to cerebral malaria. However, this seems disputable.

The World Health Organization (1986) specifically recommends to restrict diagnosis of cerebral malaria to patients with unarousable coma (showing non-localising or absent motor responses to noxious stimuli), in whom other causes of encephalopathy have been excluded. So, mild or transient cerebral dysfunction in a patient with malaria should not automatically be diagnosed as evidence of sequestration of parasitised erythrocytes in the cerebral vascular bed, which is the underlying pathophysiology of cerebral malaria (Osuntokun, 1985).

In addition, it seems improbable that at the time of admission the patient was actually suffering from malaria. In a non-immune individual not using antimalarial prophylactic medication, normal clinical as well as laboratory tests virtually rule out the diagnosis. Even in low-grade infections, splenomegaly, slight anemia and a raised erythrocyte sedimentation rate are obligatory (Manson-Bahr & Apted, 1982).

Plasmodium vivax malaria, which occurs in Thailand, may remain dormant for a long time, especially after insufficient chemoprophylaxis (Manson-Bahr & Apted, 1982). It can be reactivated among others by immunosuppression. A major depressive episode (Denman, 1986), malnutrition (Chandra, 1983) and, possibly, treatment with a tricyclic antidepressant (Denman, 1986) have been reported to impair immunocompetence. Therefore, it appears more likely that the malarial attack in this patient occurred in the course of and not before her depressive episode. Of course, this does not make treatment any less imperative.

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Anorexia nervosa in people of Asian extraction

Sir: Bhadrinath (*Journal*, April 1990, **156**, 565–568) presented three case-reports of anorexia nervosa in adolescents of Asian extraction which were very interesting. We are led to believe that the condition in non-white populations in the UK is very rare. Despite the small numbers of people from ethnic minority groups in the area of East Suffolk we have seen the condition in a teenage girl from a