British Journal of Psychiatry (1985), **147**, 320–327

Correspondence

Vampires: The Nature (and Number) of the Beast

DEAR SIR,

Herschel Prins claims that the vampire myth "was probably given more tangible reality by such physical explanations as Erythropoietic Protoporphyria or its variants" (*Journal*, June 1985, **146**, 666–668).

The signs listed by Prins (red eyes, skin and teeth, anaemia, and severe skin lesions), are actually typical of the rarer, recessively inherited congenital porphyria, not erythropoietic protoporphyria (Kosek, 1982). Of the latter, latent forms may actually be more common than active ones, because of partial penetrance, and patients are not usually anaemic or exhibiting a taste for blood. In any case, the replenishing of the stores of haem by this route would be minimal because of its poor absorption by the gut (Day, 1984).

It may seem churlish to drive a stake through this attractive but unfounded hypothesis, were it not for the harm such macabre tainting of a potentially fatal group of diseases could cause. Known sufferers, their relatives and the general public need reassurance that they are not predisposed to blood-sucking, sadomasochism or murder. The danger is of course that such sensationalism could discourage those with porphyric symptoms or a family history of such symptoms from seeking medical advice.

While on the subject, it would seem timely to remind your readers, particularly those who set the MCQs for the College examination, that the hoary old chestnut that George III suffered from porphyria has been comprehensively refuted (Dean, 1971).

Finally, I cannot help wondering whether the Editor felt obliged to follow a dictum higher than the College motto in his selection of the title-page number:

> "This calls for wisdom: let him who has understanding reckon the number of the beast . . .
> its number is six hundred and sixty-six." (*Revelation*, XIII, 18)

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References

DAY, R. S. (1984) Bloodlust, madness, murder and the press. *New Scientist*, 13 September, 53–54.

DEAN, G. (1971) *The Porphyrias*. London: Pitman.

KOSEK, M. S. (1982) Medical Genetics. In *Current Medical Diagnosis and Treatment* (eds. M. A. Krupp & M. J. Chatton). Los Altos: Lange Medical Publications.

DEAR SIR,

I note with interest that Herschel Prins' article on Vampirism appears on page 666 (the number of "the beast"). Is this more than coincidence?

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Malnutrition and Alzheimer's Dementia

DEAR SIR,

Thygessen *et al* (1970) and Gibberd & Simmonds (1980) have described in former concentration camp prisoners and former Far East prisoners of war a type of dementia with cerebral atrophy which occurred many years after their liberation. They suggested that it was caused by prolonged severe undernutrition which is a cause of cerebral atrophy (Skullerud, 1985) and of intellectual impairment (Leyton, 1946). We have recently seen two patients presenting such a dementia, who fulfilled DSM III criteria for Alzheimer's dementia. These facts suggest a possible identity between these apparently different types of dementia. To our knowledge, existing neuropathological data cannot determine whether they are identical or not. Thus, neuropathological studies should be carried out to this end. If these two types of dementia were identical, this would argue for a nutritional etiology in the Alzheimer variety (Abalan, 1984).

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References

ABALAN, F. (1984) Alzheimer's disease and malnutrition: a new etiological hypothesis. *Medical Hypotheses*, **15**, 385–393.

GIBBERD, F. B. & SIMMONDS, J. P. (1980) Neurological disease in ex-Far East prisoners of war. *The Lancet*, **2**, 135–137.

LEYTON, G. B. (1946) Effects of slow starvation. *The Lancet*, **ii**, 73–79.

SKULLERUD, K. (1985) Variations in the size of the brain, influence of age, sex, body length, body mass index, alcoholism, Alzheimer's changes and cerebral atherosclerosis. *Acta Neurologica Scandinavica*, Supp. 102, **71**, 1–90.

THYGESSEN, P., HERMANN, K. & WILLANGER, R. (1970) Concentration camp survivors in Denmark: Persecution, disease, disability, compensation. A 23-year follow-up. A survey of the long-term effects of severe environmental stress. *Danish Medical Bulletin*, **17**, 65–108.

Epilepsy and Psychosis

DEAR SIR,

In a recent issue of the *Journal* (February 1985, **146**, 155–163) Drs. Perez, Trimble, Marion and Reider make a significant contribution to the longstanding discussions and debate on the relationship between epilepsy and psychosis in general and between temporal lobe epilepsy (TLE) and schizophrenia in particular. The essence of that debate is as follows. Numerous anecdotal reports in the literature document a frequent association between TLE and a variety of generally unpleasant personality traits, aggression and psychosis. Many such studies examine patients with TLE for such traits excluding other epilepsies from purview (Flor-Henry, 1969; Bear & Fedio, 1977; Lindsay *et al*, 1979). Another group of investigations surveys age and otherwise matched patients with both TLE and generalised epilepsy (GE) utilising psychological tests or interviews by blind observers; these studies generally fail to show significant differences in personality or psychopathology between the two groups (Small *et al*, 1966; Mignone *et al*, 1970; Standage & Fenton, 1975; Rodin *et al*, 1976; Hermann *et al*, 1981; Parnas & Korsgaard, 1982).

Ever since the now classic study of Slater, Beard & Glithero (1963) reported that 52 of 69 patients with epilepsy and schizophreniform psychosis had a temporal lobe focus, attention has been drawn to the possible association between these two common disorders. However, as has been pointed out elsewhere, these authors' finding of 70% TLE in a population of adult epileptics with psychosis is close to the anticipated incidence of TLE in adults with epilepsy—variously reported as between 55% and 80% (Stevens, 1966; Marquis-Assis, 1976). It is of interest then that Perez *et al*, in a fresh prospective study of consecutive patients with epilepsy and psychosis have come up with almost exactly the same percentages of TLE as Slater *et al* (71% TLE; 29% GE), a figure falling well within the expected percentage of adults with epilepsy who will have TLE.

However, by delineating certain differences in interictal syndromes for TLE and GE, Perez *et al* have made a distinct contribution. Despite the persistent emphasis on TLE as the culprit in the epilepsy-psychosis constellation, patients with TLE and GE in their series of patients with epilepsy and psychosis are represented in approximately the same percentage as their proportion in the adult epilepsies. Moreover, the interictal psychoses of patients with GE were apparently just as chronic and apparently even more severe than those of patients with TLE. However, important differences emerged between TLE and GE in the Present State Examination (PSE). By using this structured diagnostic interview and the specific criteria of CATEGO to define a group of designated *nuclear schizophrenics* (NS) based on the possession of a requisite constellation of Schneider's first rank symptoms, the authors reported that the NS syndrome was exclusively found in patients with TLE ($n = 11$). In contrast, the remainder of the TLE ($n = 6$) and GE ($n = 7$) patients who failed to meet CATEGO criteria for NS but who were also chronically psychotic had a variety of other psychoses. Although not as well defined by the authors, these psychoses apparently included more non-productive symptoms including cognitive and affective defects, thought incongruity and blocking characteristic of what Kraeplin described as *dementia praecox* and more recently has led Crow (1980) and others to characterise as schizophrenia with "negative symptoms". It also appears that the authors' patients with non-NS and GE score higher on some 12 of the PSE items than non-NS schizophrenic patients with TLE and higher on 18 items than patients with either TLE and NS or NS without epilepsy.

The authors also give new insight to the much debated issue of a critical interval between onset of epilepsy and development of psychosis. Slater *et al* initially reported that a duration of epilepsy around 16 years was a critical factor in the subsequent development of schizophreniform psychosis, although in a subsequent analysis of the same population (Slater & Moran, 1969) they noted a difference from expected age of onset only in the females. Perez *et al* noted that their TLE group with NS has the shortest interval between onset of epilepsy and development of psychosis with a mean of 16 years. In contrast, their non-NS groups with either GE or TLE have a seizure history of 26.7 and 25.1 years, respectively. Inspection of their data indicates that this is at least partly due to the earlier onset of seizures in patients with GE (mean age 6) and of TLE without NS (mean age 9) while patients with the NS syndrome and TLE have a mean age of onset of 11 years.