unwillingness on the part of the psychiatrist [*sic*] to reach this diagnosis." Slater continued, "If the diagnosis of schizophrenia is correct, then there would seem to have been serious deficiencies in the clinical judgment or the open-mindedness of a number of distinguished world psychiatrists ...The diagnostic blindness, the failure to reach an obvious conclusion becomes a problem in its own right, demanding explanation."

Rees, according to Slater, was the key to the error. Rees's understanding of "psychopathological phenomena on exclusively psychodynamic lines" and his contention "that diagnosis is a bagatelle" led to "the results of grave misconceptions of the task of the psychiatrists of what constitutes psychiatric knowledge, and of the function of psychiatry as a medical discipline *vis-à-vis* the individual patient". Rees, as the only psychiatrist on the British examining team and as the only person present with extensive involvement with the case, was in a position to influence the others. This, in Slater's opinion, "led to a trial which seems now to have been a miscarriage of justice".

In his 1972 article Slater called for the release of the subsequent psychiatric records on Hess when he died, to settle the diagnostic issues. From 1946 until his recent death, over 41 years of such records have accumulated. What effect, for example, did Hess's psychiatric status have on the decision not to release him from Spandau? If Hess was psychotic, was he treated during those 41 years? What effect did press reports that Hess might be malingering have on the decision to try him at Nuremberg? Since Hess was a Deputy Führer whose flight to the West was deeply embarrassing to Hitler, did political considerations enter into the diagnostic decision to not label him as insane? If Hess was psychotic, what was his mental status as Deputy Führer when he and Hitler were formulating the policies which led to the war? It is also interesting to contrast the case of Hess, apparently psychotic but found fit to stand trial, with the simultaneous case in the United States of Ezra Pound, who was not psychotic but claimed to be unfit to stand trial on 19 counts of treason (Torrey, 1983).

In the interest of history, the psychiatric records of Rudolph Hess should be made available for professional examination.

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Expressed Emotion and First Episodes of Schizophrenia

SIR: The debate about the validity of the Northwick Park Study in the papers by Mintz *et al* and Macmillan *et al* (*Journal*, September 1987, **151**, 314–323) highlights a number of important issues about the EE concept. The level of discrimination between high and low EE in predicting relapse among first-episode schizophrenics, whether significant or not, is certainly lower than in previous studies of EE in undifferentiated samples. In fact, Leff & Brown (*Journal*, April 1977, **130**, 417) have previously revealed that the nine-month relapse rates for high EE first admissions is 38% (compared with 13% for low EE) and 69% (compared with 17%) for readmissions. If high/low EE are stable characteristics, why should such temporal changes in predictive power be observed? The possibility that EE may in fact be an unstable characteristic is revealed in the recent large-scale study of Hogarty *et al* (1986): 25% of the high EE sample not exposed to family treatment 'spontaneously' reduced to low EE, compared with 39% who reduced under family treatment. In both groups no relapse was observed, suggesting perhaps that the spontaneous reduction in high EE was genuine and not an artefact of measurement. It seems that there is an important developmental dimension in the genesis of stable EE which is concerned with the cumulative interaction of inherent schizophrenic vulnerabilities and the family system (Birchwood & Smith, 1987).

Macmillan *et al* discuss the applicability of EE: only one-third of their sample come from a family home, and of these only a minority were high EE. Our experience is roughly similar, although we have observed that many first admissions return to the family home subsequently; McCreadie & Robinson (*Journal*, May 1987, **150**, 640–644) found that 50% of their undifferentiated sample lived with a relative. It is not at all clear to us that low EE families cope in a 'model' way; in some cases we have observed that families manage the burden of living with schizophrenia in such a way as to exacerbate other clinical or social features, such as social withdrawal (Birchwood & Smith, 1987). The prelevance of high EE does not therefore adequately reflect the therapeutic or service implications of family interventions.

Finally, Macmillan *et al* draw attention to the comments of Hogarty *et al* (1986) that family interventions delay rather than prevent relapse and that

the success of these interventions might be inflated by drug defaulters among the controls. It is important to recall that many have argued that neuroleptics themselves serve to delay relapse (Englehart *et al*, 1967) and that, to quote Macmillan *et al* (*Journal*, February 1986, **148**, 128–133), "relapse rates were lower on... medication than placebo, *but however assessed, outcome at this early stage was poor for many patients*" (our italics).

Clearly, we have no cause for complacency, and both environmental and biological research needs to be actively encouraged and pursued.

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A Comparative Trial of Amitriptyline and Fluoxetine

SIR: The comparative trial of amitriptyline and fluoxetine in depressed out-patients by Young *et al* (*Journal*, September 1987, **151**, 337–340) found no significant difference in outcome except for a slight difference, discounted by the authors, on the Beck Scale for Depression (BSD). Improvement in BSD scores commenced in the first week, and six-week improvement is described as moderate. I may not be alone in being baffled by the conclusions drawn from these results: "Efficacy was similar in the two groups"; "Overall both drugs proved to be effective antidepressants over the six-week trial..."; "The apparent efficacy of fluoxetine..."; "... both drugs proved equally efficacious"; "... the absolute usefulness of both drugs can only be assumed".

With the possible exception of the last of these statements, none of them (*pace* the pharmaceutical industry) is warranted by the evidence presented. Perhaps the most convincing result was that amitriptyline gave patients dry mouths and made them fat; fluoxetine made them sick. This seems a good example of the "me-too" variety of drug trial. A placebo group was omitted on ethical grounds. Similar grounds might have been invoked for the omission of the conclusions.

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Tuberous Sclerosis and Psychosis

SIR: Dennis & Hunt (*Journal*, September 1987, **150**, 413–414) have recently drawn attention to two case reports in which tuberous sclerosis in associated with psychotic symptomatology (Lawlor & Maurer, *Journal*, March 1987, **150**, 396–397; Clarke *et al*, *Journal*, May 1987, **150**, 702–703). In addition, they refer to their own study of ninety children with tuberous sclerosis, over half of whom demonstrated psychotic behaviours (predominantly autistic or hyperkinetic) (Hunt & Dennis, 1987). They propose that profound language delay, severe impairment of social interactions, hyperkinesis and sleep disturbance constitute a behavioural phenotype. They believe that this occurs so frequently as to be included within the diagnostic criteria for tuberous sclerosis.

Within the field of mental handicap, there are few specific organic disorders which are associated with specific psychiatric disorders. Exceptions might include organic mental states in Down's syndrome, self-mutilation in Lesch-Nyhan and Cornelia de Lange syndromes, autistic-like symptomatology in Rett's syndrome, and perhaps a few others.

The problem of associating tuberous sclerosis with autistic symptoms is of interest, but difficult, as it raises our uncertainty over the aetiology of autism itself, although this is presumably the result of some organic lesion. The association really resolves itself around two questions: is tuberous sclerosis *directly* associated with autistic symptoms? Or does it frequently give rise to degrees of mental handicap, which are themselves frequently associated with autistic symptoms?

The authors need to show that, having controlled for degrees of mental handicap, patients with tuberous sclerosis are more likely to develop autistic symptoms than other groups of brain-damaged individuals. Unless this is done, the suggestion that autistic symptoms are specifically applicable to individuals with tuberous sclerosis, any more than to patients with, say, hydrocephalus, may be entirely spurious.

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