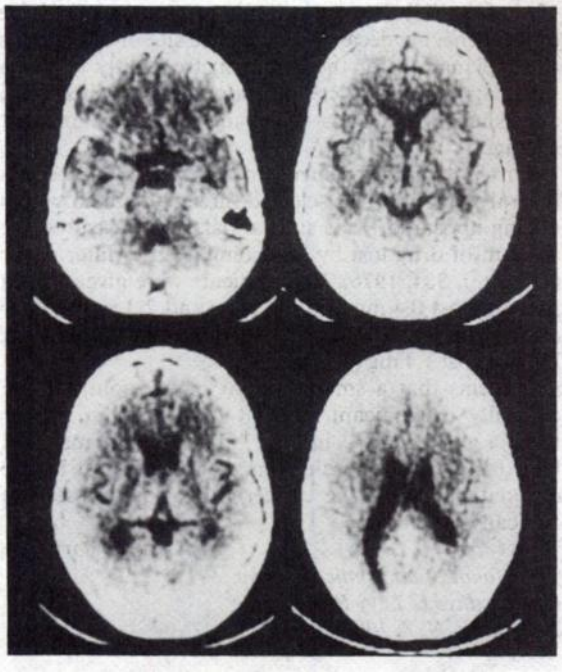

FIG 1.—CT scan showing generalized atrophy.

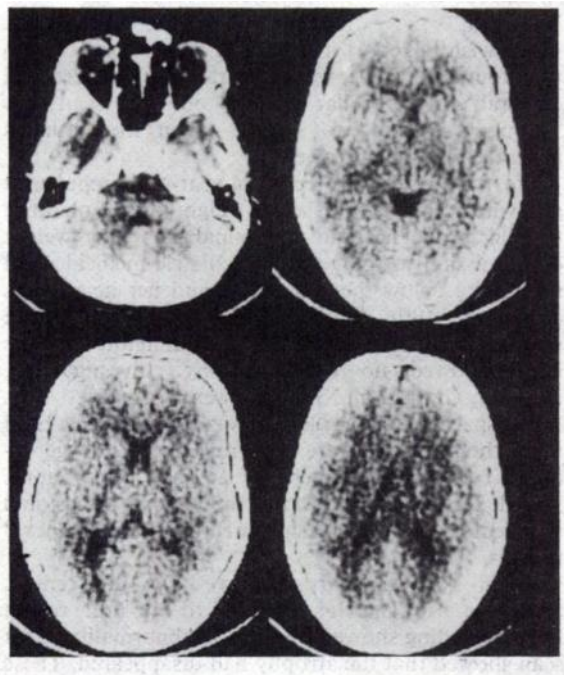

FIG 2.—Repeat CT scan, three months later, showing a normal brain.

EMI scan has become a valuable investigation for excluding space occupying lesions which may masquerade as anorexia nervosa. It is important therefore to be aware of these apparently reversible changes. What causes them and whether they have any bearing on the pathogenesis of anorexia nervosa merit further study.

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THE TWO MANIAS

DEAR SIR,

I am afraid that Dr Hare, in writing his article (*Journal*, February 1981, **138**, 89–99) has fallen into error on a number of matters which I feel I must point out.

(1) 'Partial versus total' insanity has been an issue since before Locke (Causabon, 1655) relating to either the intensity or extension of symptoms, and cannot be said to be conceptually dependent upon the 19th century debate between Associationism and Faculty Psychology. Because of this it is equally wrong to postulate any continuity between this and the concept of 'Einheitspsychose'. The 'unitary psychosis' view developed as a solution to aetiological and taxonomic difficulties that could only have originated during the 19th century as a result of fundamental changes in the concept of illness (Llopis, 1954; Valenciano, 1970; Janzarik, 1969; Vliegen, 1973).

(2) The particular strand of Faculty Psychology that influenced French views on insanity is unlikely to have been Kantian in origin. Instead there is growing agreement on the relevance to this of the Scottish philosophy of common sense (Albrecht, 1970; Klein, 1970; Klemm, 1911; Brooks, 1976) and of the bridging role of Pierre Royer-Collard. At any rate there already

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existed an old Faculty Psychology tradition in European philosophy (Blakey, 1850). For example Pinel's classification is based on Baconian principles which he received via Diderot and d'Alembert (Riese, 1969; Klemm, 1911). Phrenology, yet another of its manifestations during the early 19th century (Young, 1970), also influenced taxonomic and explanatory views on insanity.

(3) It is not necessarily the case that there were 'vagaries' associated with the word 'delirium' and that these caused 'misunderstandings' in psychiatry. The process whereby 'delirium' became 'délire' in French psychiatry is certainly a long one (Ey, 1955), but it can be understood against the background of changing psychological theories and observational bias (Berrois, 1981). The rendition of 'délire' as 'delirium' in the translation of Pinel's book reflects more Davis' ignorance (he was an obstetrician) than the semantics of the term at the beginning of the 19th century. Textual analysis shows that Pinel meant 'idée délirante' or 'delusion' and not delirium (in the sense of a symptomatic insanity). There is, however, good evidence for this latter usage at the time (Middleton *et al*, c. 1780; Sutton, 1813; Georget, 1820).

(4) It is misleading to claim that tendency 'to permanent mental enfeeblement' was a necessary feature of the 19th century view on dementia. In fact there was no such unified view, and it is essential to specify to what period it is referred. For example Georget's 'démence aigüe' (1820), Ball's 'démence vésanique' (1822) or Morel's 'démence précoce' (1960) were not considered as irreversible. The term acquired this extra meaning once its links with neuropathology strengthened (Marie, 1907).

The meaning of 'mania' seems to have changed during the early 19th century (Gauchet and Swain, 1980). It is true that its history constitutes a difficult exercise, but this, I feel, has been tried more successfully in the past (Linas, 1871; Couchard, 1913; Ey, 1954). The fact that the current clinical picture of mania cannot be easily identified in 19th century writings should not be so distressing as to make one resort to a 'mutability of disease' hypothesis. Evidence for this is scanty and certainly Dr Hare's paper provides none. One must not forget that even nowadays the clinical picture of mania is far less discrete than theoretical views might lead one to believe. Genetic studies and psychological assumptions (i.e. mania as a 'primary' affective disorder) may well be intellectually satisfactory but are not very helpful in the actual differential diagnosis of the condition.

Therefore, instead of assuming too quickly a 'mutability' of illness view, would it not be more profitable for the historian of psychiatry to explore how descriptive languages seem to control (in a Whorfian manner) the perception of the opaque biological phenomenon of mental illness? This is no Byzantine exercise, as the elucidation of the ways in which conceptual systems influence psychiatric classifications should contribute to the identification of predictive clinical categories and thence to clinical practice. Wittgenstein's dictum is also valid for historians of psychiatry: "Wovon man nicht sprechen kann, darüber muss man schweigen" (1921).

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DEAR SIR,

Hare's argument (*Journal*, February 1981, **138**, 89–99) that insanity has changed in course and outcome due to changes in 'host immunity' may be correct to a certain extent, but he seems to have overstated the case considerably, a disproportion due to neglect of the frequency of organically caused psychosis. It is surely not controversial to state that certain cerebral affections (such as amphetamine intoxication and brain disease associated with focal epilepsy) may mimic more or less exactly so-called functional psychoses, and I suspect that these covertly organic psychoses were common and accounted for the deteriorations described, the two main causes being infections and toxins.

Infections are liable to change their geography and their virulence, and their effects are also related to the general health of the community and the individual. For example, in Shakespeare's day the fens of East Anglia harboured malaria, which is now a tropical (or travellers') disease; streptococci now rarely cause scarlet fever which was commonplace 50 years ago; the influenza pandemic of 1918 probably flourished on the debilitation of the surviving population and was associated with severe sequelae; encephalitis lethargica, too, is now hardly a common condition.

The same principles of availability and host susceptibility apply to the numerous poisons which man inflicts on himself, whether from contamination (such as lead from water pipes) or from self-medication (with substances such as bromides and opium): pink disease provides a good example of this latter problem. Alcohol was freely available in the 19th century and it is not likely that alcoholism was less common then than now.

The evidence that deteriorating psychoses were associated with brain disease was clearly presented by Kahlbaum (1874): the clinical features described in those with a downhill course included epileptic seizures and hemiplegias, while the post-mortem findings frequently showed macroscopic changes in the brain (although it is difficult to discern the exact nature of the pathological entities present). The other remarkable feature of the autopsy examinations is the high incidence of tuberculous lesions both pulmonary and intestinal. The idea springs to mind that tuberculous meningitis may have been responsible for a proportion of these cases, as it is notoriously difficult to diagnose, has a chronic course with variable outcome, and preferentially affects the base of the brain, i.e. those structures such as the brain-stem, temporal lobes and rhinencephalon implicated in latter-day neuroanatomical theories of psychosis.

The concept of changes in host resistance to the accession and chronicity of noxious influences is very appropriate if earlier psychoses often had an organic cause; if this is not the case it is difficult to understand why schizophrenia diagnosed today runs a more benign course and has a better outcome in developing than in developed countries (World Health Organisation, 1979). The argument that physical health in developing countries is superior is scarcely tenable, and one must invoke either additional factors accounting for course and outcome or posit an awkward extension of 'health' to cover psychological and social factors, with the addendum that these factors are so superior in developing countries as to more than counterbalance the physical debit.

The motivation of Hare's article apparently derives from past discrepancies in the terminology, descriptive psychopathology and prognosis of psychoses. Yet even now there are severe discrepancies on these points to the extent that consecutive papers on ostensibly the same subject adopt incompatible approaches (Ciompi, 1980 and Kety, 1980). The presence of these discrepancies does not necessarily indicate different events or illnesses but rather differing interpretations of comparable observations. Past discrepancies must be susceptible to similar differences, especially given that they were not contemporaneous, that there were fewer influences uniting the European cultural milieu than now, and earlier observers had the benefit neither of standardized assessments of psychological and physical abnormalities nor of powerful statistical procedures for analysing their data.

Finally, may I take issue with the 'rarity' of profound dementia in schizophrenics. Although it is certainly a minority of schizophrenics who develop