

will tend to alleviate the difficulty. On the other hand, one of the scales (the first outcome scale: time in hospital) is a proportion—which saturates at 0 and 1, producing a characteristic S-shaped curve!

In any case, even if one was happy about the interval nature of the scales it is improbable that, *a priori*, the relationship between symptomatology and outcome would be linear. As Guttman puts it: "In the social sciences, at least, linearity should be regarded as a departure from non-linearity and not vice-versa".

In view of these difficulties we would like to suggest that even if the method gave clear indications of a non-linear relationship this should at best be regarded as merely indicative and in no way conclusive.

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INTER-RATER RELIABILITY IN MULTI-CENTRE TRIALS

DEAR SIR,

In interpreting the results of the study by Fisch *et al* (*Journal*, February 1981, 138, 100-9), it should be appreciated that they used a drastically reduced version of the Hamilton Depression Rating Scale (HDRS) of only eight items instead of the original eighteen. Furthermore, the HDRS is an instrument for rating change in depressive illness and is not designed for diagnostic procedures. Fisch *et al* used written descriptions of mythical cases for their physicians to rate, but video-tape interviews with patients may be a more useful procedure for increasing inter-rater reliability. Thus, in studies involving members of our group, Tiplady and Loudon (1980) found that during video-tape sessions lasting one day, there was a significant improvement in inter-rater reliability ($P < 0.01$) from first to last rating.

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OILY INJECTIONS THAT OOZE

DEAR SIR,

Catherine Finlay Kinnes states (*Journal*, February

1981, 138, 178) that she strongly suspects that leakage of fluphenazine decanoate from the site of injection is the explanation for the comment frequently heard in psychiatric clinics: "the injection never touched me", and she recommends "Z" injections to prevent leakage.

This possibility occurred to me and my associates several years ago. We selected three patients in whom oozing appeared to be substantial, and measured the amount of drug lost by absorbing it on to filter paper (*Lancet*, *i*, 364, 1976). Two patients were given 25 mg IM, and lost 0.4 mg (1.6 per cent) and 2.3 mg (9.2 per cent) respectively. The third patient was given 37.5 mg IM and lost 1.4 mg (3.7 per cent).

It seems that a small amount of oil, containing a clinically insignificant amount of drug, can appear to be a great deal. Using the "Z" technique may still be worthwhile in those patients who ooze, but I hope our data alleviate the concern that such losses are clinically important.

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ANOREXIA NERVOSA AND PSEUDO-ATROPHY OF THE BRAIN

DEAR SIR,

We wish to report a case of anorexia nervosa who was found to have the appearance of generalized cerebral and cerebellar atrophy on EMI scan: the 'atrophy' reverting to normal when the patient's general condition improved.

We were asked to see a 14-year-old prepubertal girl, who presented with severe weight loss, intolerance to cold, headaches and abdominal pain. Her weight on admission to the general paediatric medical ward was 10 kg below the 3rd centile and her height was above the 10th centile. Neurological examination revealed no abnormalities and examination of the fundi showed no optic atrophy. Investigations, including CXR, skull XR, again were normal.

No space occupying lesion was found in the EMI scan, though there were moderate degrees of generalized cerebral and cerebellar atrophy (see Figures 1 and 2). EEG done at the time shows occasional transient theta discharges occurring in both temporal regions.

When her weight rose above the 10th centile, 3 months after she was admitted to our unit, repeat EEG recording showed no definite abnormality. EMI scan showed that the atrophy had disappeared. These changes in the EMI scan have been reported by Heinz *et al* (1977) and Enzmann and Lane (1977).

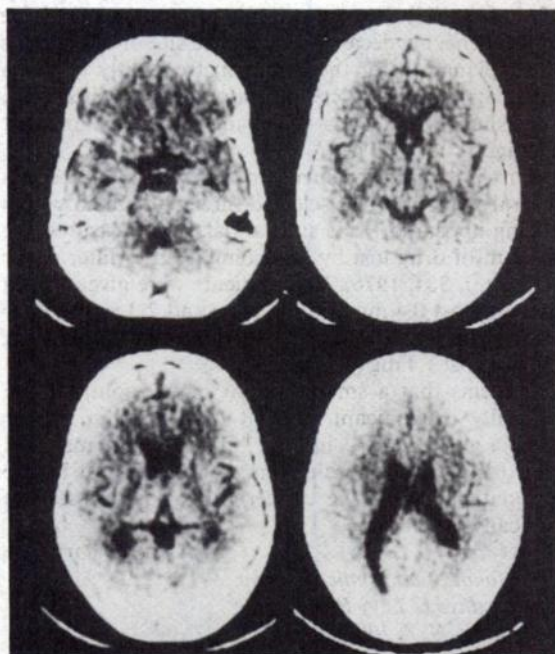


FIG 1.—CT scan showing generalized atrophy.



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