pertained to. There was no indication on the tapes whether the patients were first admissions or readmissions. As reported in our 1976 paper, the interrater reliability was 0·86 (product moment correlation). We hope this finally puts to rest the qualms of Dr Scott's group.

With regard to their second point that their test may measure some of the same factors as ratings of Expressed Emotion, we consider that this is entirely possible but needs to be demonstrated by the simultaneous use of both instruments in the same families. Our Family Interview has gone through a long process of refinement and streamlining. We have recently shown that for ratings of Expressed Emotion the original 4- to 6-hour interview can be legitimately reduced to one hour. We are concerned to continue this process and are currently experimenting with further modifications with a view to producing a practical clinical tool.

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ALCOHOLISM AND PSYCHIATRIC DISORDER: SOME FURTHER DATA

DEAR SIR,

Theories which suggest a possible relationship between psychiatric disorders and alcoholism alternatively hypothesize that alcoholism is (1) a *secondary* symptom which through chronic abuse or attempts at self-medication acts to mask an underlying psychiatric disorder, or (2) a *primary* disorder equivalent in relation to another psychiatric disorder in the sense of being a pleiotropic expression of the same etiological process (1–3).

In a recent study of the effects of the repeal of alcohol regulation laws in Sweden in 1955 on the rates of psychiatric hospital admissions and treatment for alcohol addiction and abuse over several subsequent years, Herner (4) reported data which bear on the validity of these theories. From the Table comprising Appendix I in Herner's publication we subtracted the male admissions for alcoholic disorders from the total number of admissions, and reanalysed the data in terms of the effect of repeal on both alcoholism and other psychiatric disorders separately. In his original evaluation, Herner accounted for the extraneous effects of increased population, the enlargement of treatment facilities and available beds, and the rates of alcohol consumption following repeal, all of which were consistent with natural and expected increases in the total rate of admissions. The data represented admissions to all but a few treatment facilities in Sweden for the period evaluated. We deleted data for rates beyond 1961 due to spurious increases attributable to change in the reporting method and because these data were well removed from the period of interest.

The above theories predict that an increase in admissions for alcoholism will reveal a compensatory reduction in admissions for psychiatric disorder. As shown by the Figure below, repeal more than doubled the proportion of admissions for alcoholism from 1954 baselines, while the rate of admissions for psychiatric disorders remained constant and within expectancy for the period. The respective increases in admissions for alcoholism and psychiatric disorder between 1954 and 1955 were 555 and 325 cases, the 1955–56 increases were 1,234 and 356 cases, and the 1956–57 increases were 156 and 270 cases. Proportionately, admissions for alcoholism which comprised 19 per cent of all admissions in 1954 increased to 32 per cent by 1956. This proportion was thence maintained for the remainder of the assessed period.

At face value, these data tend *not* to confirm general explanations of alcoholism as a phenomenon secondary to an underlying psychiatric disorder or as a primary disorder equivalent. Reservations regarding this conclusion include the possibilities that (1) some cases of alcoholism had been misclassified as psychiatric disorder and thus masked the reduction and (2) that alcohol precipitated admission to hospital of persons predisposed to psychiatric disorder misclassified alcoholic. The applicability of these explanations to the non-observed expectancy, however, appears somewhat remote in the face of the large increase in admissions for alcoholism which resulted. Alcoholism in the study was defined as WHO classifications 307 and 322 (5).

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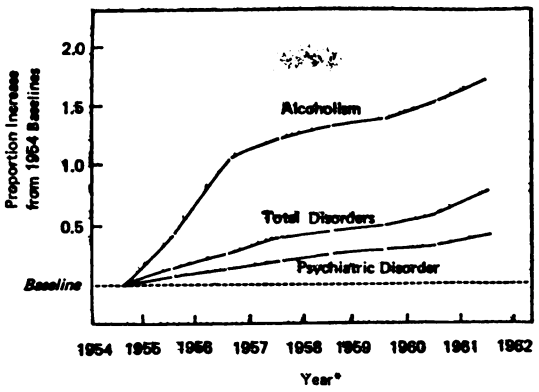

FIG 1.—Proportion increases in male admissions to Sweden Mental Hospitals between 1954 and 1961 calculated from 1954 baselines.

* Data for the year is plotted at the mid-year.

MANAGEMENT OF AFFECTIVE DISORDERS

DEAR SIR,

I found Dr David Shaw's review article on 'The Practical Management of Affective Disorders (*Journal*, May 1977, 130, pp 432–51) stimulating and full of information. However, there are some points with which I would take issue. It is difficult to know how he justifies giving as many as 14 ECTs in a case of depression. I would have thought if the patient did not respond to a maximum of 8 ECTs, the possibility of improving with further ECT is small. Perhaps he has some basis for his figure of 14, but he does not state it. There is also a conspicuous failure to mention the remarkable effect of ECT given in a brisk, brief course in mania. This would seem to be far safer than the heroic doses of haloperidol which Dr Shaw recommends, namely 10–30 mg i.m. initially and further doses 1–1½ hours later repeated till either hypotension ensues or the mania subsides. There is also no mention of the role of barbiturates, which can be most helpful in the severe sleep impairment of agitated depression and mania. Nitrazepam and similar compounds are just not adequate in these instances, and while one would rarely prescribe barbiturates otherwise they are surely indicated here. I would applaud Dr Shaw's emphasis on using adequate doses of tricyclic antidepressants, but he makes no reference to work done on the cardiac effects of different tricyclic antidepressant drugs (see the work of Burrows *et al*, *Brit. J. Psychiat.*, 1976, 129, pp 335–41). This work would suggest that doxepin is safer than the other tricyclics in patients with heart disease. Dr Shaw gives the impression of therapeutic zeal, which is refreshing, but I feel that in cases of so-called resistant depression it is important to emphasize re-examining closely one's diagnosis before embarking on a series of drugs, some of which carry significant problems of toxicity.

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HALOPERIDOL IN NORMALS

DEAR SIR,

Haloperidol is an effective antipsychotic agent which is a relatively specific blocker of dopamine transmission in the brain (Anden *et al*, 1970). As part of the preliminary trials in a study of possible dopaminergic mechanism in affective disorder, the two authors each were given haloperidol 5 mg intravenously in a two-minute push. The effect was marked and very similar in both of us: within ten minutes a marked slowing of thinking and movement developed, along with profound inner restlessness. Neither subject could continue work, and each left work for over 36 hours. Each subject complained of a paralysis of volition, a lack of physical and psychic energy. The subjects felt unable to read, telephone or perform household tasks of their own will, but could perform these tasks if demanded to do so. There was no sleepiness or sedation; on the contrary, both subjects complained of severe anxiety.

The present experience was similar to that previously reported of neuroleptic effects in normal subjects (DiMascio *et al*, 1963; Heninger *et al*, 1965), though previous studies used neuroleptics which block both dopamine and noradrenaline receptors (Anden *et al*, 1970). We used a relatively specific dopamine blocker, haloperidol, and experienced profound cognitive and emotional restriction. Dopamine blocking by neuroleptics may function to restrict cognitive and emotional processes in normals as well as in schizophrenics, and thus it is possible