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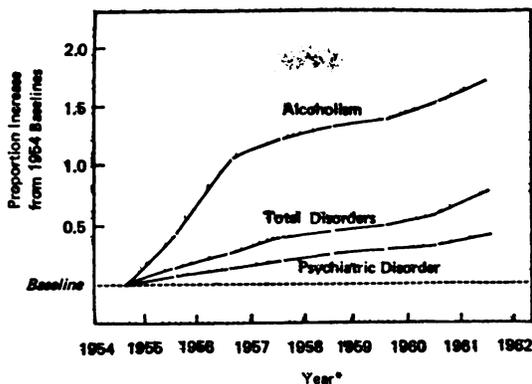


FIG 1.—Proportion increases in male admissions to Sweden Mental Hospitals between 1954 and 1961 calculated from 1954 baselines.

* 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

that it does not specifically antagonize schizophrenic pathology. In the presence of psychotic anxiety or delusions, such cognitive or emotional restriction may be desirable and therapeutic. However, the restrictive effect may be a general one, and is certainly useful in mania as well as in schizophrenia (Shopsin *et al*, 1975). The similarity of the above-described state to that of some cases of agitated depression and post-psychotic depression suggests involvement of dopamine in these affective states (Post *et al*, 1976; Gerner *et al*, 1976), as well as in schizophrenia (Snyder *et al*, 1974).

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LITHIUM AND PSORIASIS

DEAR SIR,

We have recently reported (Skott *et al*, 1977) three patients with bipolar affective disorder who have psoriasis vulgaris and who showed a marked deterioration of their skin disease during lithium treatment. Within the first two months on lithium carbonate or sustained-release lithium sulphate their psoriasis increased heavily and did not respond to conventional means of topical treatment which had earlier been effective. In all cases it was judged inadvisable to

discontinue lithium treatment. Carter (1972) reported exacerbation of psoriasis in three patients on lithium. In his patients lithium treatment was discontinued, which led to prompt improvement of the skin disorder. Voorhees *et al* (1975a) reported three patients, and Bakker and Peppinkhuizen (1975) reported four patients with psoriasis who showed deterioration on lithium.

A possible explanation for the observed effect could be through reduction of cyclic AMP, which is considered to be a stimulus to epidermal proliferation, important in psoriasis. Preliminary data from *in vitro* studies suggest that lithium affects the epidermal cyclic AMP (Voorhees *et al*, 1975b).

Since the completion of our report three similar cases have been referred to us. In one of these cases it was possible to discontinue lithium whereupon the skin disease improved to its previous appearance.

The question whether lithium may actually aggravate psoriasis has practical importance. Further studies are now in progress to confirm this clinical observation.

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