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Correspondence

COMPUTED TOMOGRAPHY IN TARDIVE DYSKINESIA

DEAR SIR,

We read with interest the article 'Tardive Dyskinesia and Dementia' by Famuyiwa *et al* (*Journal*, December 1979, **135**, 500–504). The authors concluded from their well-conducted study that the tardive dyskinesia group had significantly greater proportion of abnormalities in the computed tomography (CT) scans (and on certain neuropsychological tests), and that these may be related to chronic neuroleptic toxicity. We are writing this letter since our results with CT scans have been at variance with these conclusions.

We (Jeste *et al*, 1980) reported earlier an absence of significant differences between the CT scans of elderly hospitalized women with moderate to severe tardive dyskinesia, and those of a matched control group. In view of the possibility that the pathogenesis of tardive dyskinesia may differ between the two sexes and in different age groups, we recently extended our study to men under 50. Two psychiatrists screened 28 hospitalized male schizophrenic patients below the age of 50. Using the same diagnostic criteria as in the earlier study, six patients were found to have moderate to severe tardive dyskinesia. Next, we formed a control group of six patients matched with the dyskinesia group for age, sex, race, primary psychiatric diagnosis, hospitalization, and length of neuroleptic treatment. As in the previous study, the CT scans of the two groups were read 'blind' on the following measures: lateral ventricle/brain ratio (planimetric assessment), bifrontal/bicaudate ratio for caudate atrophy, Huckman's criteria for cortical atrophy, Hahn's cerebroventricular indices, Allen's criteria for cerebellar atrophy, and focal abnormalities. There were no significant differences between the CT scans of the dyskinesia and nondyskinesia groups on any of these variables.

Gelenberg (1976) also found a lack of specific abnormalities in the CT scans of tardive dyskinesia patients. Indeed, the CT scan data of Famuyiwa *et al* (1979) showed that the dyskinesia and control groups did not differ from each other in the mean CT scan scores on radiological grading, Huckman's number, ventricular index and cella media index. Both the groups were within normal limits for the Huckman's number, while their values on the cella media index were almost identical. The only significant difference between the two groups was in the number of patients whose ventricular index was below 1.6. This cut-off point of 1.6 was based on a study by Meese *et al* (1976). The validity of an arbitrary cut-off point in different types of patient populations may be doubtful, especially when the differences between experimental and control groups are small.

Regarding the possibility that chronic neuroleptic treatment produces structural brain damage, there is substantial evidence to suggest that the structural abnormalities described by Famuyiwa *et al* occur independent of neuroleptic drug treatment. Crow and Johnstone (1977) reported that schizophrenic patients who had not received neuroleptics and other physical treatments had larger ventricles than did the patients who had received these treatments. Weinberger *et al* (1979) found that the lateral ventricular enlargement seen in a subgroup of chronic schizophrenic patients could not be correlated with past neuroleptic treatment. Furthermore, several pneumoencephalographic studies which also found ventricular enlargement in chronic schizophrenic patients, were done prior to the advent of neuroleptic drugs. It is, of course, possible that persistent tardive dyskinesia is associated with structural brain abnormalities which may be either too small or so localized (e.g. in substantia nigra) that the available techniques of 'reading' CT scans are not sensitive enough to detect them. We agree with Famuyiwa *et al* (1979) about the likelihood that individual variation in metabolism of neuroleptics, and other biochemical characteristics may contribute to the pathogenesis of at least some types of tardive dyskinesia. Our elderly patients with tardive dyskinesia had significantly higher serum-neuroleptic levels, lower platelet monoamine oxidase activity and higher plasma dopamine-beta-hydroxylase activity as compared to matched controls (Jeste *et al*, 1979a and b). The relevance of these findings to the pathophysiology of tardive dyskinesia is as yet uncertain. We are presently studying these biochemical parameters in younger patients with dyskinesia.

With further refinements in neuropathological and neurochemical techniques in the near future, the

mechanisms underlying tardive dyskinesia(s) will, we hope, be better understood.

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COMPULSORY ADMISSION

DEAR SIR,

I certainly agree with Dr Srinivasan and his colleagues (*Journal*, February 1980, **136**, 200–201) that factual evidence about the multiple effects of compulsory hospital admission is badly needed. However, I doubt that this will be provided by the study they outline in their letter.

I am concerned that the views of 20 out of a sample of 50 patients are brushed aside with the judgement that *some* of them were unable to show enough 'critical judgement and insight'—what exactly was required?

Then there is the host of questions raised by patients 'admitting they were ill' (was this sufficient to justify compulsory admission?) or feeling that the hospital stay was 'helpful'. There is a marked tendency on the part of hospital staff to inculcate both these platitudes upon patients and relatives during the traumatic period surrounding such an admission. It is, indeed, an independently-minded patient who does not answer in appropriate fashion similar questions asked by any hospital authority. The five patients who did answer negatively, not surprisingly, blamed circumstantial features such as ward conditions.

The next-of-kin is the person required to sign the section form: simple theories of cognitive dissonance predict they will agree that the patient was 'ill' and that the hospital stay was 'helpful'.

Leaving aside, then, the strange calculation of percentages, this study needs much improvement before it is extended to other hospitals. Patients still resident in the hospital are likely to include representatives of the least conforming subsample, and it should be possible to obtain the views of some of this group. What is meant by a 'helpful' admission: perhaps the same help could be given in another way? The views of other relatives, social workers and persons concerned around the time of admission, are important.

Without such modifications, publication of the study may encourage unjustified complacency. Let us not increase government inertia on mental health matters!

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EMERGENCY MANAGEMENT OF THE STARVING PSYCHIATRIC PATIENT

DEAR SIR,

We wish to report how the use of a naso-gastric tube relatively easily saved a depressive patient who was determinedly starving herself to death. She was aged 55 with a particularly intractable depressive illness, severely agitated and deluded that she 'had no stomach and therefore could not eat'. Her brother had died of starvation in this institution two years before. When our patient had drunk only two cups of tea in 24 hours we feared for her life and decided to use a naso-gastric tube.

The technique was as follows: A Ch. 8 gauge naso-enteral tube was employed. For convenience and for the comfort of the patient, the tube was inserted while the patient was still unconscious following ECT. It was found that the supplied introducer was not needed, and the narrow-bore tube was easily guided into the oesophagus with a pair of Magill endotracheal forceps, under direct vision with a laryngoscope. The position of the distal end of the tube was checked, firstly by blowing air down it and auscultating over