HALSBOER, F., LIEBL, R. & HOFSCHUSTER, E. (1982) Repeated dexamethasone supression test during depressive illness: normalisation of test result compared with clinical improvement. *Journal of Affective Disorders*, **4**, 93–101.

NELSON, J. G. & BYCK, R. (1982). Rapid response to lithium in phenelzine non-responders. *British Journal of Psychiatry*, **141**, 85–6.

## SMOKING PROFILES OF PATIENTS ADMITTED FOR NEUROSIS

DEAR SIR,

I would like to reply to the response of Salmons and Sims (*Journal*, July 1982, **141**, 103) to criticism of their methods and conclusions (*Journal*, January 1982, **140**, 103) reported in the article titled "Smoking Profiles of Patients Admitted for Neurosis" (*Journal*, July 1981, 43–6). Smoking is too important a public health problem for associative or etiological factors to be imputed on scant evidence.

Salmons and Sims appear bent on concluding their hypotheses "upheld", despite the absence of results which would warrant rejection of the null hypotheses (i.e. no significant differences between groups). Inappropriate use and interpretation of statistical tests is not unusual; however, it is not common to find authors willing to present such a large number of non-significant differences in support of their argument.

In acknowledging the direction of the only significant difference between groups reported in their Table III, Salmons and Sims claim this supports their original contention of earlier age onset of smoking by neurotics, as ". . . those who are smokers have started in a much younger age group, leaving only a smaller number to start later". In the younger male age group cited, the (non-significant) difference reported was 49.7 per cent in the general population sample vs. 62.9 per cent in the neurotic patient sample. Salmons and Sims argue that this leaves only 8.1 per cent of the neurotic male sample vs 22.5 per cent of the general population sample to start smoking later. These last figures are interesting, as they indicate the authors have noted that roughly the same proportion of the general population male sample and the neurotic male sample reported having started to smoke by age 30 (i.e. 72 per cent and 71 per cent). However, absence of a significant difference between groups in the younger age group would appear to weaken the argument of Salmons and Sims that neurotics start smoking at an earlier age.

To focus on Table III, describing only contrasts regarding 'Age at Starting to Smoke', may be seen as unfair, as this represented only one component of the authors' search for an association between neuroticism and smoking. Salmons and Sims also reported a higher proportion of smokers in both age and sex groups in the neurotic sample than in the general population sample, that this higher proportion was maintained over all social classes, and that neurotic patients inhaled more deeply. In all, seventeen statistical comparisons were reported in Tables I-IV of Salmons' and Sims' original paper. Of these seventeen contrasts, only four comparisons resulted in $\chi^2$ values which had a probability of occurring by chance .05—findings generally accepted as reason to 'reject the null hypothesis'. Salmons and Sims have apparently chosen to break with this convention. In cases where statistical tests have indicated a significant difference, they view the difference between comparison groups as "especially marked".

If an association is *not* "especially marked", it often does require a relatively large sample size to be demonstrated. Medical researchers are frequently frustrated by this, as samples generally do not come in 'jumbo' sizes in clinical series. Yet, clearly, there is danger in generalizing from a trend in the data obtained from a small and select sample. Salmons and Sims report their findings as contradicting those obtained in earlier research (e.g. Eysenck, 1963; Eastwood and Trevelyan, 1971), and suggest the factor of "neuroticism" must be taken into account in planning community health strategies directed at reducing smoking in the general population. As the earlier studies were conducted on much larger, community samples, perhaps the previous absence of a significant association should be taken more seriously than this more recent interpretation of data from an inpatient sample.

M. R. EASTWOOD

*Clarke Institute of Psychiatry,*
*250 College Street,*
*Toronto M5T 1R8*

## TARDIVE DYSKINESIA IN A PHANTOM LIMB

DEAR SIR,

In their article on the phantom limb phenomenon (*Journal*, 1982, **141**, 54–58), Shukla, Sahu, Tripathi and Gupta discussed the various theories of the phantom limb, including central and psychological mechanisms. We present here a case of tardive dyskinesia in a phantom limb which would support a central mechanism for the phantom limb phenomenon.

A 42-year-old white single male with a DSMIII diagnosis of schizoaffective disorder, antisocial personality, and alcohol abuse, was referred to us for management of persistent dyskinesia of two years duration. The patient had lost his right foot and three toes on his left foot 11 years prior to admission due to

frostbite. The patient had a long history of neuroleptic drug intake and the typical oro-lingual movements, as well as choreiform movements in his fingers and remaining toes of the left foot.

During the assessment of the patient with the Abnormal Involuntary Movement Scale (AIMS), he spontaneously volunteered that he has had ongoing sensations of wiggling in the nonexistent toes of his amputated foot. He added that he had been aware of these movements daily on his phantom limb for about two years, regardless of whether he was wearing his prosthesis or not. In addition, the patient denied that he had a phantom limb sensation immediately after the amputation, which took place 9 years before the onset of the involuntary movements of tardive dyskinesia in his mouth, fingers, and toes. Thus, his phantom limb sensation appeared to be triggered by the development of tardive dyskinesia.

It is widely accepted that tardive dyskinesia is a state of post-synaptic dopamine receptor supersensitivity in the nigrostriatal tract, secondary to chronic treatment (chemical denervation) with neuroleptic drugs (Gerlach et al, 1974). The implication for the mechanism of phantom limb in our patient is not only that it is of central origin, but that a motor abnormality in the extrapyramidal system may trigger an otherwise "dormant" phantom limb.

MONA McCALLEY-WHITTERS
HENRY A. NASRALLAH

VA Medical Center,
University of Iowa College of Medicine,
Iowa City, Iowa 52240

### Reference

GERLACH, J., REISBY, N. & RANDRUP, A. (1974) Dopaminergic hypersensitivity and cholinergic hypofunction in the pathophysiology of tardive dyskinesia. Psychopharmacologia, 34, 21–35.

## TARDIVE DYSKINESIA LESS SEVERE IN DEPRESSION

DEAR SIR,

Recent studies have suggested a high incidence of tardive dyskinesia among depressed patients (Davis et al, 1976; Rosenbaum et al, 1977). Cutler et al (1981) have reported the occurrence of state-dependent dyskinesias in two cases of manic depressive psychosis, pointing out that the observed exacerbation of tardive dyskinesia at the outset of depression and its alleviation during mania are in the opposite direction of what might be predicted from current neurochemical theories of these disorders.

We have data on a patient in whom the dyskinesias consistently subsided during the depressive periods. He was a 40-year-old man with a two year history of rapid cycling bipolar affective psychosis previously treated with chlorpromazine equivalents in an average daily dose of 300 mg. He developed tardive dyskinesias six months before, and since then had taken only lithium 900 mg daily. He was admitted in a severe depression and his dyskinesias rated using the Simpsons et al scale (1979) twice weekly at 10 am during 7 months stay in the hospital. The mean ratings of the dyskinesias during the three depressive episodes that followed were 55 ± 10.8, significantly lower than during the euthymic periods (92 ± 16.8). The patient was on lithium carbonate 900–1200 mg daily throughout, and had tricyclic antidepressants and ECT in two of the three depressive episodes. (These treatments did not significantly alter the scores between the episodes in which they were employed and the other episode).

There is evidence that tardive dyskinesias result from a functional dopaminergic hypersensitivity and possibly a cholinergic hypofunction (Gerlach et al, 1974). In depression there are reports of decreased dopaminergic activity (Schildkraut, 1975), and cholinergic hyperfunction (Sitaram and Gillin, 1980), so that depression and tardive dyskinesia may involve biochemically opposite mechanisms. Our observation of a reduction in tardive dyskinesia seems to fit in well with this possibility. As the earlier findings were in the opposite direction, we suggest that our finding adds support to the possible biochemical heterogeneity of tardive dyskinesia (Casey and Dunney, 1977).

M. S. KESHAVAN
U. GOSWAMY

National Institute of Mental Health,
and Neurosciences, Bangalore, 560029, India

### References

CASEY, D. E. & DUNNEY, D. (1977) Pharmacological characterization of tardive dyskinesia. Psychopharmacology, 54, 1–3.
CUTLER, N. R., POST, R. M., REY, A. C. & BUNNEY, N. E. (1981) Depression-dependent dyskinesias in two cases of manic-depressive illness. New England Journal of Medicine, 18, 1088–9.
DAVIS, K. L., BERGER, P. A. & HOLLISTER, L. E. (1976) Tardive dyskinesia and depressive illness. Psychopharmacological Communications, 2, 12–30.
GERLACH, J., REISBY, N. & RANDRUP, A. (1974) Dopaminergic hyperactivity and cholinergic hypofunction in the pathophysiology of tardive dyskinesia. Psychopharmacologia (Berl.), 32, 21–35.
ROSENBAUM, A. H., NIVEN, R. G., HANSEN, N. P. & SWANSEN, D. W. (1977) Tardive dyskinesia; relationship with primary affective disorder. Diseases of Nervous System, 38, 423–7.
SCHILDKRAUT, J. J. (1975) Depression and biogenic amines. In American Handbook of Psychiatry, Vol. 6 (eds. D. A. Hamburg and H. K. H. Brodie). New York: Basic Books.