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Correction

Table I in the text should read Table II. This, like the entry, NA*, is quite clear from the text; the latter might read 'not achieved'.—*Editor.*

LITHIUM THERAPY IN AGGRESSIVE MENTALLY SUBNORMAL PATIENTS

DEAR SIR,

I read with interest Dr Dales' article (*Journal*, November 1980, **137**, 469–74).

These are a notoriously difficult group of subjects to treat. In the study, one of the patients was withdrawn from lithium because of the onset of tardive dyskinesia. From the discussion it would appear that lithium was involved in the production of this somewhat serious side-effect. This would certainly be a unique finding. I wonder if it is not possible that the patient in question had been receiving neuroleptics prior to entering the lithium treatment (no information is given in the tables with regard to prior medication). If this were indeed the case then this would be an example of a withdrawal dyskinesia which usually takes place some one to three weeks after withdrawal of medication, but in some cases longer.

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DEAR SIR,

The patient in question was receiving chlorpromazine (50–100 mg t.d.s.), orphenadrine (50 mg t.d.s.) and haloperidol (3 mg t.d.s.) at the time of institution of treatment with lithium carbonate (250 mg t.d.s.). Haloperidol was discontinued three weeks later but treatment with chlorpromazine and orphenadrine continued for a further four weeks, by which time the patient's behaviour had so improved that both drugs were stopped. Tardive dyskinesia in the form of tongue movements and sucking was first noticed two months later.

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PERSONALITY CHANGE FOLLOWING ACCIDENTS

DEAR SIR,

Dr Parker's well documented case (*Journal*, November 1980, **137**, 401–409) was of considerable interest; yet the contribution might have been more profitable had the major emphasis been placed on the developmental predisposition rather than on the apparent precipitant. The title might indeed have been "Personality Vulnerability Following Severe Emotional Inhibition in Childhood—The Report of a Double Murder".

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DEAR SIR,

Basil James' criticism of my article (*Journal*, November 1980, **137**, 401–409) expresses a point of view, popular in some circles, which my experience does not support. This has been discussed in detail in a chapter on "Accident Neurosis" (Parker, 1976) and will not be repeated here. In essence, he is saying that "severe emotional inhibition in childhood" is of such overwhelming importance, that any subsequent life events pale into etiological insignificance when explaining disturbed behaviour occurring in a man in his forties.

In my case report the co-twin was subjected to the same disturbing childhood yet did not murder his wife and daughter. One must therefore look for something additional to explain the discordance for homicide, and all professional people involved with the monozygotic twins whom I described were satisfied that two terrifying accidents emerged as the obvious difference in their histories.

The aim of my paper was to highlight the significance of accidents, even of an apparently minor nature, as a precipitating cause in the development of

emotional disorders and one would expect this to be expressed in the title of the article.

Accident neurosis not only affects those people who are highly predisposed to react to stress; well adjusted personalities can become so overwhelmed by the awareness of their vulnerability for the first time, that they may break down following a frightening accident. E. A. Rappaport (1968), a survivor of Nazi persecution, commented that "an insistence of investigators on finding some latent predisposition for personality breakdown betrays their unwillingness to imagine the full impact of the terror". I would recommend this excellent article to Professor James, and others who over-emphasize "severe emotional inhibition in childhood" in the causation of disturbed behaviour in middle age.

NEVILLE PARKER.

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SCHIZOPHRENIA ON PATERNAL AND MATERNAL SIDES

DEAR SIR,

In the December 1980 issue of your *Journal* (**137**, 505–509) Dr Baron using Slater's (1966) computational model of assessing distribution patterns of ancestral secondary cases in first and second degree relatives of 18 schizophrenic probands, reported that the distribution of unilateral to bilateral pairs of affected relatives did not deviate significantly from that expected in polygenic inheritance.

We presented similar findings at the 9th Panhellenic Congress in Neurology and Psychiatry (Athens, December 12–14, 1980)—see Table.

TABLE

Comparison of observed and expected under the polygenic hypothesis pairs

	Observed	Expected
Unilateral pairs	40.0	41.3
Bilateral pairs	22.0	20.7
Total	62.0	62.0

$\chi^2 = 0.06$, 1 df, NS.

These data were obtained from the application of Slater's (1966) computational model in the study of the distribution of the ancestral secondary cases of 26 schizophrenic probands who had two or more first or second degree relatives affected with schizophrenia.

The schizophrenic probands and their affected relatives were diagnosed using Feighner *et al's* (1972) diagnostic criteria for schizophrenia. The probands and all living schizophrenic relatives (34 out of 62) were personally interviewed, while the diagnosis of the affected relatives who were not living at the time of the investigation was based on information gathered from the two closest relatives available for interview.

More details concerning the data of this study could be obtained from the full paper which will shortly appear in the *Proceedings of the 9th Panhellenic Congress in Neurology and Psychiatry*.

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NOMIFENSINE AND DYSKINESIA

DEAR SIR,

A 72-year-old man with a long history of manic depressive disorder was admitted to this hospital in October 1977 with severe retarded depression. He had been receiving Priadel 800 mgm daily for many months to which was added nomifensine (Merital) 25 mgm tds. Within 48 hours he developed choreoathetoid movements of his tongue which disappeared 72 hours after the nomifensine was discontinued.

He was readmitted in April 1980 again depressed and had taken no medication of any sort for 8 months. The doctor who admitted him prescribed nomifensine 25 mgm tds and he not only developed dyskinesia of the tongue, but also choreiform movements of the extremities and shoulders, which all disappeared within 5 days of the nomifensine being discontinued.

It is well known that dopamine agonists can produce dyskinesia, so it is not surprising that nomifensine can have such an effect, though it has not been described previously as far as I know.

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