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Correspondence

Correspondents should note that space is limited and shorter letters have a greater chance of publication. The Editors reserve the right to cut letters and also to eliminate multitudinous references. Please try to be concise; strictly relevant and interesting to the reader, and check the accuracy of all **references in Journal style.**

PARENTAL AGE IN SCHIZOPHRENIA

DEAR SIR,

There are few areas of schizophrenia research where one finds such an impressive concordance of results from different studies as that of parental age. Both maternal (Goodman, 1957; Johanson, 1958; Gregory, 1959; Bojanovsky and Gerylovova, 1967; Hare and Moran, 1979) and paternal (Johanson, 1958; Gregory, 1959; Bojanovsky and Gerylovova, 1967; Hare and Moran, 1979) age have been consistently found to be raised. I would like to report the results of a multi-hospital study from the Newcastle region which confirm previous evidence. These are set out in the following table.

TABLE
Table showing parental age at birth patients v controls

Mean maternal age		Mean paternal age	
Patients (n = 342)	Controls (n = 1817)	Patients (n = 320)	Controls (n = 1788)
30.073	29.06	33.803	32.01
SE of Means		SE of Means	
0.349	0.15	0.412	0.18
t (2.667) significant at .01 level		t (3.988) significant at .001 level	

The control ages were derived as described by Hare and Moran (1979, 1978). The latter authors have fully discussed the implications of such findings, and they conclude that the facts best fit the hypothesis of a biological parental trait leading to delayed marriage, the raised maternal age being secondary to the advanced paternal age.

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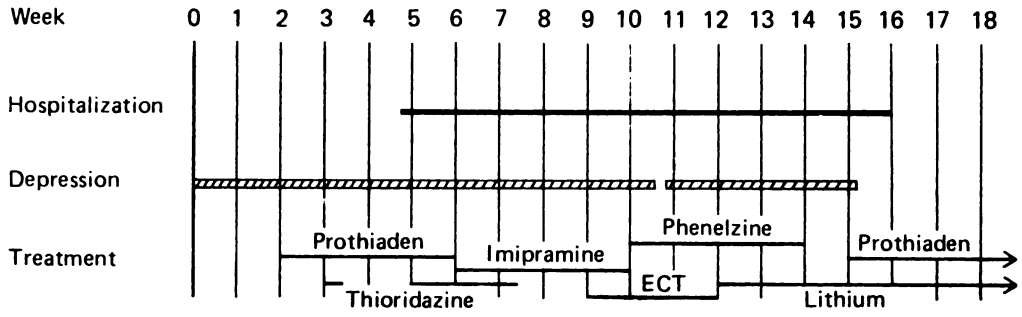
RAPID RESPONSE TO LITHIUM IN TREATMENT-RESISTANT DEPRESSION

DEAR SIR,

The recent reports by De Montigny *et al* (1981) and Nelson and Byck (1982) who describe a rapid relief of depression with lithium after no response to tricyclic antidepressants and phenelzine respectively, lead us to report on two patients in whom a lithium-antidepressant combination was effective after no response to one or more antidepressants and ECT.

Mr A was a 66-year-old happily married man who presented with a one month history of a severe endogenous depression (DSM-III diagnosis: recurrent major depressive disorder with melancholia) which had not responded to prothiaden 100 mg daily prescribed by his general practitioner. In the past he had suffered from three prolonged depressive episodes requiring hospitalization, the first when he was 45 years old. He had no other past psychiatric history and no family history of psychiatric disorder. After not responding to prothiaden (225 mg), imipramine (175 mg), ECT × 10 (5 unilateral and 5 bilateral), phenelzine (75 mg), phenelzine and lithium carbonate (500 mg), he responded rapidly and dramatically when prothiaden (100 mg) was added to his lithium. Except for one day after his sixth ECT his mood remained one of severe depression until the third day after prothiaden (100 mg) was added to the lithium carbonate. Over a twelve month follow up, attempts to stop either the lithium carbonate (blood level approximately 0.5 mmol/L) or the prothiaden have led to a relapse of his

Fig. Sequence of treatment for Patient A



FIG—Sequence of treatment for Patient A.

depression. The figure details the sequence of his illness and treatment.

Mr B was a 64-year-old socially isolated single man who presented with a six week history of endogenous depression (DSM-III diagnosis: recurrent major depressive disorder with melancholia and mood-congruent psychotic features). In the past he had abused alcohol, but this had not been a problem over the past twelve years. Over the past eleven years he had had four hospital admissions for severe depression. The last two admissions in the previous two years had each been of four to five months duration, had been especially difficult to treat and had not responded to various tricyclic antidepressants. On the last admission he eventually responded to mianserin 120 mg daily, chlorpromazine up to 400 mg daily and ECT. During the present admission he refused to eat or drink at times because of his delusional belief that he had no stomach, and at times it was necessary to maintain hydration via a nasogastric tube or intravenously. He failed to respond to mianserin up to 150 mg daily, chlorpromazine 400 mg daily and ECT × 12 (5 unilateral and 7 bilateral) over one month in hospital. Lithium carbonate 1000 mg daily was added to his mianserin and chlorpromazine. Within 48 hours he was readily eating and drinking. He continued to steadily improve and was discharged two weeks later.

These two patients provide further evidence for the value of lithium in antidepressant non-responders. They also differ from the previously described patients in a number of ways. Firstly, both had not responded to ECT as well as to various antidepressants. In patient B the antidepressant was mianserin, a tetracyclic, and not a tricyclic or monoamine oxidase inhibitor. In patient A the rapid relief from depression occurred when prothiaden was added to lithium, rather than vice versa, yet previously neither prothiaden alone, nor phenelzine and lithium had been effective.

We have also seen this rapid relief from depression after the addition of lithium in four other patients. Three of these patients had a bipolar affective illness and the lithium was added to amitriptyline, mianserin and tranylcypromine. The fourth patient had unipolar affective illness and after not responding to amitriptyline 150 mg and tranylcypromine 30 mg daily over three weeks, started to improve within 48 hours of adding lithium (750 mg daily) to this antidepressant combination.

Whether the hypothesis of De Montigny *et al* (1981) of postsynaptic serotonin supersensitivity following tricyclic antidepressant treatment, with presynaptic facilitation of serotonin neurons by lithium, is correct, remains to be determined. It appears however that this observed rapid relief from depression with lithium is a clinical phenomenon which deserves further attention, and it would be interesting to observe the changes in biological markers (e.g. REM latency, dexamethasone suppression) during such sequential treatment strategies, especially as the dexamethasone suppression test has been reported to normalize before clinical improvement (Holsboer *et al* 1982; Greden *et al* 1980).

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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 χ^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.

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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