10 weeks after delivery, at Magomeni Health Centre in Dar Es Salaam. Mothers who saw themselves as having major socio-economic problems were excluded from the study, as also were unmarried mothers, those who said they were unhappily married, and those who had major ill-health, previous abnormal pregnancies and abnormal deliveries. In addition, the recent pregnancies and deliveries had to have been normal, and mothers had to be breast feeding mothers.

At interview, data were obtained concerning the mother working through the Middlesex Hospital questionnaire (because of cultural differences, some questions were considered inappropriate). 'Blues' were scored using a check list outlined by Brice Pitt, with a score of 0–4 for each symptom.

A total of 38 out of 50 mothers showed some evidence of 'Maternity Blues' with a maximum incidence on the seventh day after delivery, and the remaining 12 showed no evidence of 'Maternity Blues'. When these figures were further broken down in terms of all the mothers, the following findings emerged.

Twenty-one mothers experienced weeping or a feeling of tearfulness, lasting from half an hour to a day at some point in the first two weeks after delivery, eight showed somatic features alone (without tearfulness) such as palpitations, air hunger, chest pains and sleeplessness, and nine experienced relatively minor symptoms such as a feeling of confusion, and irritability.

Preliminary analysis of results indicates a highly significant positive correlation of 'Maternity Blues' with the items of 'anxiety' and 'somatic symptoms' ($P < .001$), as measured on the Middlesex Hospital questionnaire.

These results show that 'Maternity Blues' is a cross cultural phenomenon and occurs in women from a culture which is quite different from that of Western countries. Preliminary comparison with a similar group from Cardiff showed a much stronger positive correlation of somatic symptoms (as measured on the MHQ) with 'blues' in the African group, so these results also illustrate the high degree of somatization of stress which characterizes East Africans.

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PLASMA AND RED CELL LITHIUM

DEAR SIR,

During the last few years, researchers on treatment with lithium for manic-depressive psychosis have been interested in biochemical parameters for an objective classification of the patients.

We have compared a group of 13 primary bipolar outpatients with 12 primary unipolar depressive outpatients, all being in remission for at least one year and taking therapeutic doses of lithium salts of 800–1,600 mg/day. The plasma lithium concentration and red blood cell lithium concentration were determined 12 hours after the last dose was taken, following the method of T. B. Cooper *et al* (1974). When the Spearman's rank correlation coefficient was applied to correlate the plasma lithium and red blood cell lithium values in the two groups of patients, we found that the correlation coefficient was statistically significant ($P < 0.05$) in the bipolar depressive group, and nonsignificant in the group of unipolar patients (Table I). The same results were found when the determinations were made four hours after the last dose was taken. Lithium ratio (LR) data which are normally studied in most work in this field, do not show significant differences between the two groups of patients, so we are not able to establish any relation between this ratio and the clinical unipolar/bipolar diagnosis of the patients.

TABLE I

Correlation between plasma and red blood cell lithium concentration (Spearman's rank correlation coefficient)

	Four hours after last intake	Twelve hours after last intake
Total	0.43*	0.66*
Bipolar patients	0.76*	0.73*
Unipolar patients	0.05	0.37

* $P < 0.05$

It is not possible at the present time to draw conclusions from these results, but it is interesting to note that all the patients have homogeneous characteristics and have been in remission of their illnesses for a long time. Other parameters apart from lithium ratio could exist which would differen-

tiate unipolar from bipolar patients, so that research with improved sampling and methods must continue.

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TARDIVE DYSKINESIA AND DEMENTIA

DEAR SIR,

Famuyiwa *et al* (*Journal*, December 1979, **135**, 500–4) discuss two possible explanations of their findings, both of which invoke neuroleptic drugs in the etiology of tardive dyskinesia. Application of Occam's razor suggests a third possibility, namely, that tardive dyskinesia is simply one manifestation of the cerebral degeneration caused by the schizophrenic disease process in these patients.

Although tardive dyskinesia can be suppressed by neuroleptics, until a survey is carried out of its incidence among chronic schizophrenics never exposed to neuroleptics, the etiology of tardive dyskinesia remains uncertain. The finding that the mean dose of fluphenazine decanoate among the tardive dyskinesia patients was higher than among the non-tardive dyskinesia patients could indicate that the former were more brain damaged by their illness and required higher doses of drug to control their symptoms. Had there been more than three patients on oral drugs in the tardive dyskinesia group, a similar difference might have been found between oral dosage means.

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OXYPERTINE FOR TARDIVE DYSKINESIA

DEAR SIR,

We have read with great interest the paper by Drs Mackay and Sheppard on pharmacotherapeutic trials in tardive dyskinesia (*Journal*, December 1979, **135**, 489–499). Brief reference is made there to oxypertine as a possible therapeutic agent and this substance is at present being investigated clinically by us.

Oxypertine is thought to exert its therapeutic effects by depleting pre-synaptic neuronal stores of neurotransmitters. Compared with reserpine and tetrabenazine, it is a more potentent depleter of brain noradrenaline, but has less dopamine depleting activity. It has been shown to be effective as a neuroleptic in controlling the symptoms of schizophrenia, has not been recorded as causing depression, and is thought to be of value in activating patients with marked negative symptoms. It does not show acetylcholine-like properties, nor does it stimulate GABA-systems, both of which may be concerned in the production of TD.

Chien, Jung and Ross-Townsend (1978) carried out a double-blind study to compare the efficacy of oxypertine, sodium valproate and dimethylaminoethanol in the control of TD, using a group of 17 patients. Only oxypertine was found to be significantly superior to placebo, though the number of patients was too small to allow any final conclusion other than that oxypertine shows promise as a therapeutic agent in TD.

In a so far unpublished paper, Kazamatsuri reports an open study of oxypertine in ten chronic mental hospital patients, all showing clear evidence of TD; out of these, seven experienced complete disappearance of their involuntary movements whilst receiving oxypertine. Neuroleptic drugs that were being administered before the trial were continued and no worsening of psychopathology was observed during the trial, nor did new side-effects emerge. Out of a total of 40 patients in four other uncontrolled studies, 22 are said to have shown either disappearance of TD or a marked improvement.

Our study has consisted of a double-blind comparison of oxypertine versus placebo in in-patients, aged between 18 and 70, using the AIMS score. Patients selected for the trial had a drug-free period of two weeks, followed by drug or placebo for a month, another wash-out period and then a month on the other medication. The requirements listed by Mackay and Sheppard for a therapeutic trial in TD were very largely fulfilled.

Only preliminary results are available so far; however, in the first half of the trial, patients receiving oxypertine (N = 10) showed a mean value of 50 per cent improvement, whereas those on placebo (N = 9) showed a mean improvement of 30 per cent. There is thus a significant trend in favour of oxypertine, but certain individual patients showed a dramatic response to the drug. Available information thus suggests that the addition of oxypertine may allow dosages of other neuroleptics to be reduced, thereby diminishing extrapyramidal reactions, treating or preventing TD and possibly improving the treatment