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

ENRIQUE ALVAREZ
M. CASAS-BRUGUE
C. UDINA ABELLO

Department of Psychiatry,
Hospital de la Sta. Cruz y San Pablo, Medical School,
Barcelona, Spain

J. M. QUERALTO-COMPANO
Department of Biochemistry

Reference

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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.

A. A. SCHIFF

E. R. Squibb & Sons Ltd,
Regal House,
Twickenham TW1 3QT

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

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of the anergic schizophrenic. Further subjects are at present being added to this trial.

HUGH FREEMAN
SOM D. SONI

Hope Hospital,
Eccles Old Road,
Salford M6 8HO

Reference

CHIEN, C. P., JUNG, K. & ROSS-TOWNSEND, A. (1978) Efficacies of agents related to GABA, dopamine and acetylcholine in the treatment of tardive dyskinesia. *Psychopharmacology Bulletin, 14*, 20.

UP TO DATE RECORDS OF LONG-STAY PATIENTS

DEAR SIR,

Dr Henryk-Gutt (*Journal*, February 1980, **136**, 203–4) describes the difficulty of quarrying the gems of information that are contained in the petrified pages of a thick file of old case notes. The problem concerns every patient under long-term care and not only those who are long-stay in-patients. A remedy similar to the one she suggests has been in use in this hospital since 1961 and it works quite well. Dealing as we do entirely with chronically ill patients who have on average upwards of ten years illness (and notes) behind them and who stay here on average for some 3–4 years, our need to make and keep key information accessible is important to us.

There are two parts to the method we use. Part one is a summary of the patient's history up to the time when he comes under care here. His old notes come with him when he is transferred from his previous hospital. They are then sent to the Night Superintendent. He and one particular Night Charge Nurse have shared the task of summarizing the notes of about 1,500 patients to date. Delivery of the typed summary is within three or four days and the service provided is well done and extremely valuable. A sample is appended which describes a real case disguised by a fictitious name. The headings have suited our needs. If the space allocated to any one of them is too small, the narrative continues on the back of the page.

Part two is the handwritten record of the patient's Case Conferences, compiled by the chairman of the Review team. Another sample is appended. The rating scores, abbreviations etc. are understood by the people using them so there is no point in explaining them here. The problem is to reach a compromise between being too brief and too wordy which must depend on individual taste. This sheet contains as you see a mixture of relatively permanent items (e.g. IQ, bereavements) together with very potted progress notes. Again the narrative can continue on the back of the page. It can readily be found at a set place in the file, separate from the routine continuation notes and other records which are kept and filed in the conventional way.

It feels logical in practice to keep the up-dated record in these two parts. The division between the two is the point in time when some new initiative in the management of patient care begins. Whether this involves (as in our case) an inter-hospital transfer or as is more usual a move to the hospital's own rehabilitation unit is as you might say neither here nor there. There may well be a need for a further summary say ten years later when a further stack of notes has accumulated, but I will leave it to somebody else to write to you about that in 1990.

ROGER MORGAN

St Wulstan's Hospital,
Malvern, Worcs

Summary of Previous Casepapers

Name: A. Patient. *Ref. No.:* 1257. *Date:* October 1978.

Previous admissions	*Hospital*	*Admitted*	*Discharged*
1.	St Elsewhere's	10. 5.54	2.10.54
2.	,,	8. 1.55	15. 6.55
3.	,,	30. 6.77	2.10.78
4.	St Wulstan's	2.10.78	
5.			

History before first admission

Born 18.1.23 in Birmingham. C of E. Single Storekeeper.

Only child. Normal childhood, school etc. Machine hand, National Service in Far East, then returned to engineering. Quiet, no friends, no hobbies. Liked football, cricket, cinema. No interest in opposite sex. Heavy smoker, seldom drank.

Onset 18 months before first admission. Took time off work and given the sack. Thought people were after him. Stayed at home wandering about at night and sitting about all day silently staring into the fire. (Father's maternal uncle was at one time inpatient in mental hospital).

History since first admission

(In 1954 aged 31). Quiet detached apathetic strange. Believed some influence had prevented him getting jobs. Believed he had VD following a rash on his thighs. ECT × 6, brighter but solitary manneristic and fatuous. Given Deep Insulin (32 comas) with no change. Gave notice and left. Second admission, dull childish mute no acute symptoms. ECT × 17, no improvement. Had weekend leave and from one of these refused to return. Discharged not improved. Then 22 years at home alone with father (mother was dead). At first helped but later expected everything done for him. No treatment, hearing voices, sometimes aggressive, never violent. Father finally

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