presynaptic autoreceptor. This is interesting since this receptor has been implicated as a possible locus for the action of antidepressant treatments (Goodwin *et al*, 1985). In addition, electrophysiological evidence suggests that chronic antidepressants lead to a 'down regulation' of 5-HT_{1A}-mediated inhibition (Blier *et al*, 1988). While this has yet to be confirmed for the action of 5-HT on calcium currents, if it is, then clearly antidepressants have an opposing action to calcium-channel blockers by being able to decrease the ability of 5-HT to reduce calcium influx. Therefore the common pathway for the action of dihydropyridines and monoamines in affective disorders could be at the level of the calcium channel itself.

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References

BLIER, P., DE MONTIGNY, C. & CHAPUT, Y. (1988) Electrophysiological assessment of the effects of antidepressant treatments on the efficacy of 5-HT neurotransmission. *Clinical Neuropharmacology*, **11**(suppl. 2), S1–S10.

GREEN, A. R., DE SOUZA, R. J., DAVIES, E. M., *et al* (1990) The effects of Ca^{2+} antagonists and hydralazine on central 5-hydroxytryptamine biochemistry and function in rats and mice. *British Journal of Pharmacology*, **99**, 41–46.

GOODWIN, G. M., DE SOUZA, R. J. & GREEN, A. R. (1985) Presynaptic serotonin-mediated response in mice attenuated by antidepressant drugs and electroconvulsive shock. *Nature*, **317**, 531–533.

O'SULLIVAN, A. J., CHEEK, T. R., MORETON, B., *et al* (1989) Localization and heterogeneity of agonist-induced changes in cytosolic calcium concentration in single bovine adrenal chromaffin cells from video imaging of fura-2. *The EMBO Journal*, **8**, 401–411.

PENINGTON, N. J. & KELLY, J. S. (1990) Serotonin receptor activation reduces calcium current in an acutely dissociated adult central neuron. *Neuron*, **4**, 751–758.

Investigating psychosurgery

SIR: I was interested to read Al-Sheikhli's letter (*Journal*, June 1990, **156**, 903) and, like him, I wondered why psychosurgery had not been considered in Paykel's review of the relevance of research for the treatment of depression. Professor Paykel helpfully replied (*Journal*, June 1990, **156**, 904) and pointed out that psychosurgery, while having a place with severe resistant depression, needs more research into efficacy and indications.

However, in this letter my main purpose is to question Professor Paykel's statement that "I understand that the Mental Health Act Commission has comprehensive records ...". The Commission have been involved with a total of about 100 patients and the Act requires only opinions in writing from three professionals for each patient. By contrast we have now carried out over 1200 operations since the mid-1960s and we have increasingly detailed information on all these patients over this period of time. Our problem is that money for research is not at all easy to obtain but, given better computer facilities, we have the richest possible information available for investigating psychosurgery.

Professor Paykel suggests that the College could take up the fact that more research into the efficacy and indications of psychosurgery is needed. At the foundation of the College a research committee was set up to investigate the value of psychosurgery. One of the main problems was that there seemed to be no treatments that could be used as a control. A protocol was suggested (Research Committee, 1977) but funding proved impossible.

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Reference

RESEARCH COMMITTEE, THE ROYAL COLLEGE OF PSYCHIATRISTS (1977) Evaluation of the surgical treatment of functional mental illness: proposal for a prospective controlled trial. In *Neurological Treatment in Psychiatry, Pain and Epilepsy* (eds W. H. Sweet, S. Obrador & J. G. Martin-Rodriguez). Baltimore: University Park Press.

WHO consensus statement

SIR: We read with interest the WHO consensus statement on prophylactic use of anticholinergics in patients on long-term neuroleptic treatment (*Journal*, March 1990, **156**, 412). However, like Barnes (*Journal*, March 1990, **156**, 413–414), while we welcomed the statement, we also felt that it was too brief and failed to comment on some benefits of anticholinergic therapy. Such benefits may be of particular relevance in third-world countries where, for example, patients may experience extreme transport difficulties in reaching a hospital should acute neurological side-effects arise. The occurrence of an otherwise preventable acute dystonia may well exacerbate natural fear of 'western' medicine, so adversely affecting future compliance and willingness to return for further psychiatric care. Also, use of those antipsychotics which are less likely to produce side-effects may not be possible in situations where poor availability and relative costs must be taken into account. Other than from Morocco, there were no other African contributions to this WHO statement which may possibly explain why such factors were not considered.

We have since drawn up our own guidelines for the use of anticholinergics in patients on neuroleptics. These may be of interest to those working in similar settings and are as follows.

(a) Routine use is not recommended.

(b) At the *onset* of neuroleptic treatment where prescribing for *in-patients*, anticholinergics should only be used in the presence of acute dystonia and should be gradually withdrawn after one week.

(c) At the *onset* of neuroleptic treatment where prescribing for *out-patients*, anticholinergics should be given for two weeks where: (i) the patient is a younger male or a child; (ii) there is a past history of drug-induced dystonia; or (iii) the patient lives a considerable distance from psychiatric care.

(d) During longer-term neuroleptic treatment, anticholinergics should be prescribed only where Parkinsonism has developed and where reduction of neuroleptic dose has been ineffective. In these cases anticholinergics should be given gradually but withdrawn after two months. If symptoms persist it may be sufficient to prescribe anticholinergics strictly for one week after each depot injection. In a small number of patients with disabling bradykinesia, long-term anticholinergics will be needed in combination with the lowest possible dose of neuroleptic.

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Benefits of routine laboratory investigations

SIR: White & Barraclough (*Journal*, July 1989, **155**, 65–72) concluded that only very limited routine laboratory testing was justified for adult psychiatric admissions. We examined the results of laboratory screening tests, ordered on admissions to our in-patient inner-city general psychiatry unit, to determine the frequency of abnormal results and their clinical significance, and compared our results with those of Drs White & Barraclough. The patient population was primarily male veterans with substance use, psychotic and major mood disorders. A total of 2308 tests on 52 admissions of 50 patients were examined. Each admission had an average of 44.4 tests and 5.5 abnormalities; each patient had at least one abnormal result. A total of 288 (12.5%) of the tests ordered were abnormal (a figure not unlike the 10.2% reported by White & Barraclough). No unexpected physical condition responsible for psychiatric symptoms was detected by the tests.

Of the abnormal results, 26 were positive toxicology screens in 20 patients with an already known substance use diagnosis (and were hence not unexpected results), and four were from a delirious patient with known chronic renal failure. A substance use diagnosis (including alcohol use disorders) accounted for 80 of the abnormal tests, including not unexpected elevations in liver enzymes, macrocytosis, elevated mean corpuscular haemoglobin concentration, bilirubin and low albumin, platelets or folate. Eleven abnormalities seemed to be related to poor nutrition (e.g. ketonuria on urinalysis, decreased blood urea nitrogen and creatinine), and 30 seemed to be associated with dehydration. Eighteen abnormalities were in AIDS patients and were important in monitoring their azidothymidine treatment. One psychotic patient was slightly hyponatraemic, probably secondary to psychogenic polydipsia. The remainder of the abnormalities were classified as without clear significance or diagnostic relevance.

We agree with Drs White & Barraclough that most of the abnormalities were predictable and do not appear helpful in establishing previously unsuspected organic causes for psychiatric symptoms. However, 170 (60%) of them were felt to be clinically significant (i.e., useful in treatment planning), as they: (a) had utility as a gauge of the severity of the patient's psychiatric condition (e.g. dehydration in a psychotic or depressed patient or liver damage in an alcohol abuser); (b) were useful in confronting the denial commonly seen in alcohol and substance abusers (e.g. using elevated liver enzymes in an alcoholic, positive toxicology screen results in a known drug-abuser denying recent use); (c) were useful in the detection of use of drugs other than those acknowledged by the patient; or (d) were important in managing various organic medical and psychiatric therapies. We would add these to the list of laboratory test benefits outlined by Drs White & Barroclough. On the basis of our study, we agree with Drs White & Barraclough's suggestion for only a selective laboratory screen on admission. The only aspect of laboratory screening on admission that should be 'routine'