money transferred from the hospital account to a nearby bank. He was considered to show a good grasp of financial matters and was given the go-ahead to do so. He continued to play chess and snooker with the staff. His chest condition gradually worsened and he died of bronchopneumonia some sixteen months after admission.

Neuropathological examination of the brain indicated an absence of observable atrophy of either cortex or the basal ganglia. The basal arteries were virtually free of atheroma, and cortical sections failed to reveal any significant abnormalities. Noticeable neuronal depletion and astrocytic gliosis were, however, recorded in the putamen and caudate nucleus leading the neuropathologist to comment: "The histological changes are so characteristic that the diagnosis must be accepted . . ."

Following post mortem confirmation of the diagnosis one of the patient's sons was referred for psychiatric consultation, presenting with depression and observably abnormal involuntary movements. It seems probable that this man has inherited the disease. Even more recently the daughter-in-law has reported hearing that the patient's surviving brother in Germany has developed tremors and jerky movements, though it has not proved possible to confirm this.

Apart from the absence of either clinical, behavioural or psychometric evidence of dementia, and the late age of presentation, other clinical manifestations were quite typical—the personality abnormalities, the emotional impulsivity, progressive self neglect and choreiform movements. These findings raise several interesting and important questions, namely the extent to which any 'sub-cortical dementia' can exist independently of cortical involvement (Albert *et al*, 1974), and the limitations in identifying 'early' psychometric patterns of the disease (Josiassen *et al*, 1982). As Dr. Turner also points out, it raises the interesting possibility of separting the movement disorder from the cognitive impairment in Huntington's disease. Certainly we feel that our case adds substantially to the literature in documenting the possibility of a diagnosis of Huntington's chorea being made in the absence of dementia and indicates caution against viewing dementia as a "constant" feature of the disease (Cummings & Benson, p. 74). Whether or not Huntington's chorea patients without dementia are indeed rare is not clear. For example the last patient dying in this hospital with a positive history for the disease evinced few signs of intellectual deterioration throughout the seven years of his stay.

J. L. WADDELL

Herdmanflat Hospital, Haddington, East Lothian

C. J. GILLARD

University of Edinburgh

References

ALBERT, M. L., FELDMAN, R. G. & WILLIS, A. L. (1974) The sub-cortical dementia of progressive supranuclear palsy. *Journal of Neurology, Neurosurgery and Psychiatry*, **37**, 123–130.

CUMMINGS, J. L. & BENSON, D. F. (1983) *Dementia; A Clinical Approach*. Boston: Butterworths.

JOSIASSEN, R. C., CURRY, L., ROEMER, R. A. & DEBEASE, C. (1982) Patterns of intellectual deficit in Huntington's disease. *Journal of Clinical Neuropsychology*, **4**, 173–183.

Tourette Syndrome and Drug Addiction

DEAR SIR,

I read with interest Bleich *et al's* account of coexisting Tourette syndrome (TS) and mania (*Journal*, June 1985, **146**, 664–665). Although this syndrome is often thought to involve mainly motor and vocal manifestations, Bliss *et al* (1980) described some of the sensory experiences of patients with TS. In some patients the tic manifestation is characteristically preceded by a brief irritative sensation which seems to act as a stimulus for relief by a tic-like motor activity. The motor manifestation can often be voluntarily inhibited for a time, but at the cost of a subjectively-perceived progressive build-up of tension, leading ultimately to some form of motor expression. "What is basic to the TS overt actions... is the (almost) intolerable need to produce a sharp punctuation that will at one and the same time gratify and terminate an almost intolerable urge" (Bliss *et al*, 1980). It appears, then, as if the TS sufferer must endure a continual succession of sensory-motor craving-and-relief cycles.

Another type of patient we see whose life is dominated by cycles of craving and relief is the opiate addict. Research has implicated the locus coeruleus (LC), a noradrenergic brainstem region, as being a prime site of exogenous opiate-endorphin-catecholamine interaction. It has been hypothesised that an effect of chronic opiate administration may be to cause a decrease in endogenous opiate release and synthesis, or otherwise derange the endorphin system's functional activity. Since one of the activities of the endorphin system is to regulate norepinephrine production, a consequence of this derangement is a noradrenergic rebound when the exogenous opiate drug is no longer forthcoming, producing the unpleasant symptoms of withdrawal (Gold & Dackis, 1984). In fact, the LC may be involved in other, naturally occurring physical and psycho-

logical overarousal reactions, such as cardiovascular irregularities, panic and anxiety disorders (Gold & Pottash, 1981).

The LC alpha adrenergic-blocking properties of clonidine are thought to account for this drug's usefulness in treating such disorders as hypertension, panic disorder and drug withdrawal. It is therefore intriguing that, as Bleich *et al* point out, and as has been reported by others (e.g. Borison *et al*, 1979; Cohen *et al*, 1980), clonidine is effective in the treatment of some TS patients. If, as Bleich *et al* suggest, disorders like TS, which involve compulsive thought and action, are due to abnormalities in brainstem catecholamine systems (which, in part, renders them sensitive to clonidine), then the recurring sensory tension-motoric release experiences reported in some TS patients could be seen as actual brief withdrawal episodes in a rapidly-cycling auto-addiction syndrome. For those who study and treat both drug addition and compulsive neurobehavioural disorders, this possibility deserves further examination.

LAURENCE MILLER

Fair Oaks Hospital,
19 Prospect Street,
Summit, New Jersey 07901

References

BLISS, J., COHEN, D. J. & FREEDMAN, D. X. (1980) Sensory experiences of Gilles de la Tourette syndrome. *Archives of General Psychiatry*, **37**, 1343–1347.

BORISON, R. L., ANG, L. & CHANG, J. G. (1979) Clonidine in Tourette syndrome. *Lancet*, **2**, 551–552.

COHEN, D. J., SHAYWITZ, B. A. & YOUNG, J. G. (1980) Clonidine ameliorates Gilles de la Tourette syndrome. *Archives of General Psychiatry*, **37**, 1350–1357.

GOLD, M. S. & DACKIS, C. A. (1984) New insights and treatments: Opiate withdrawal and cocaine addiction. *Clinical Therapeutics*, **7**, 6–21.

—— & POTTASH, A. L. C. (1981) Endorphins, locus coeruleus, clonidine and lofexidine: A mechanism for opiate withdrawal and new nonopiate treatments. *Advances in Alcohol and Substance Abuse*, **1**, 33–52.

What Price Psychotherapy?

DEAR SIR,

An exchange of views between Professor Shepherd and ourselves in the *Journal* columns may be useful in bringing the debate about the effectiveness of psychotherapy to the fore but is unlikely to facilitate progress in determining how the issue should best be tackled in terms of evaluating existing knowledge and conducting research in the future. Lambert and I (*Journal*, January 1985, **146**, 96–98) remain persuaded that the views we expressed in our rejoinder to Professor Shepherd (*British Medical Journal*, 1984, **288**, 809–810) are valid. Professor Shepherd no doubt will stick to his guns.

Perhaps the most constructive next step to take is one we seem to agree about: Shepherd refers to the need for a "vigorous initiative on the part of the College" to examine the nature and role of psychotherapy (*Journal*, May 1985, **146**, 555–556); Lambert and I suggest that "psychotherapy requires an unswerving commitment to its intelligent and rigorous study as well as the exploration of new paradigms for research". Shepherd feels that the appraisal should not be left to the psychotherapist alone; we indicate that psychiatrists *generally* can "play a crucial, constructive role".

Would it therefore not be reasonable and timely for the College to launch an objective, scientific study of the current status of psychotherapy and of future research options? The Australian and New Zealand College of Psychiatrists has demonstrated through its Quality Assurance Project (see for example the *Australian and New Zealand Journal of Psychiatry*, 1983, **17**, 129–146) that such a project is both feasible and worthwhile.

SIDNEY BLOCH

Warneford Hospital,
Oxford OX3 7JX

Transcultural Psychiatry

DEAR SIR,

Apropos of Roland Littlewood's passionate plea (*Journal*, July 1985, **147**, 93) for closer examination of the exotic phenomena beloved by transcultural psychiatrists, I was amused by the following entries on the subject of *whitigo (windigo)* in the latest edition of *Comprehensive (sic) Textbook of Psychiatry IV* (Kaplan & Sadock, 1985):

Having treated "a sizeable number of Cree Indians" without ever encountering a patient presenting with *whitigo*, H. E. Lehmann (Vol. II, 1235) considers the disorder to have become "extremely rare... if it exists at all. In the most comprehensive review (not cited) of this condition, one author in 1961 collected some 70 cases from Canada." A. R. Favazza (Vol. I, 258), however, concludes from "a thorough review" (again not cited) that since "no case of *windigo* psychosis has ever been observed by an anthropologist or psychiatrist... it is extremely unlikely that (any) has ever existed"!

Davis (1983), on the other hand, has gathered fascinating laboratory evidence that, together with fieldwork and existing medical literature, argues strongly for an "ethnopharmacological basis" to the zombi phenomenon. Analysis of the potions