dyskinesia in a group of 79 schizophrenic patents studied cross-sectionally. However, we do find that those with dyskinesia have greater cortical atrophy. Indeed the association with cortical atrophy seems due particularly to greater frontal atrophy (assessed separately) in patients over 55 years of age with dyskinesia. While our current study provides a more detailed assessment of CT variables in a larger sample than many previous studies, it also supports a further hypothesis in relation to the development of dyskinesia.

We would suggest that the integrity of fronto-striatal pathways may be relevant to whether or not dyskinesia is evident in patients. It is well recognised that dyskinesia becomes more prevalent with age, and the development of increasing frontal atrophy with age, from the sixth decade onwards, may explain this. Normally, fronto-striatal pathways would suppress some aspects of striatal outflow. Animal models of dyskinesia suggest that neuroleptic drugs may sensitise the striatum to producing dyskinetic movements. However, in humans these movements may not become evident in many cases until fronto-striatal suppression is lost. This would not only help to explain the increasing prevalence with age but also a variety of other observations such as: (a) greater cognitive impairment in dyskinetic versus non-dyskinetic patients may be due to cortical atrophy; (b) association of dyskinesia with soft neurological signs (King *et al*, 1991); (c) the lack of consistency in demonstrating a clear relationship of dyskinesia to prior neuroleptic treatment might be expected if another pathological process is necessary for full expression of the drug effect. Such a hypothesis would also be consistent with suggested frontal lobe deficits in schizophrenia itself (Morihisa & Weinberger, 1986). A few patients display dyskinesias at an early stage of illness, even in the absence of drug treatment. They may be those with more marked frontal impairment.

It would be interesting to know if further examination of the CT scans from the study of McClelland *et al*, would lend support to this hypothesis.

COOPER, S. J., DOHERTY, M. M., FOSTER, J. *et al* (1991) The relationship of involuntary movements to treatment variables, symptomatology and CT scan variables in chronic schizophrenia. *Biological Psychiatry*, **29** (suppl 115), 408.

KING, D. J., WILSON, A., COOPER, S. J. *et al* (1991) The clinical correlates of neurological soft signs in chronic schizophrenia. *British Journal of Psychiatry*, **158**, 770–775.

MORIHISA, J. M. & WEINBERGER, D. R. (1986) Is schizophrenia a frontal lobe disease? An organising theory of relevant anatomy and physiology. In *Can Schizophrenia be Localized in the Brain* (ed. N. C. Andreasen), pp. 17–36. Washington, DC: American Psychiatric Press.

STEPHEN J. COOPER
Department of Mental Health
The Queen's University of Belfast
The Whitla Medical Building
97 Lisburn Road
Belfast, Northern Ireland

MICHAEL M. DOHERTY
Belfast City Hospital

JOHN WADDINGTON
Royal College of Surgeons in Ireland

Verapamil in major depression?

SIR: Jacques & Cox (*Journal*, January 1991, **158**, 124–125) reported the case of an 82-year-old woman who suffered from what they diagnosed as a major (psychotic) depression, who dramatically and accidentally responded to treatment with verapamil. As a consequence, they suggest a possible aetiological role for calcium in affective disorders.

However, if the case is carefully examined *as described in the article*, the diagnosis must be in question. It is more likely to have been an organic affective syndrome of vascular aetiology since:

(a) The patient had no personal or family history of psychiatric disorders. The depressive syndrome appeared at an advanced age which means that an organic aetiology should be considered.
(b) The patient suffered hypertension of 20 years duration which lately was poorly controlled (210/100 mmHg).
(c) The onset of symptoms coincided with a stressful life event, but psychiatric symptoms of vascular aetiology can be precipitated by stressful events.
(d) The development of a supraventricular tachycardia and cardiac failure two days after the second electroconvulsive therapy (ECT) confirms the previous poor cardiovascular state.
(e) Mental symptoms worsened with an antidepressant medication (fluvoxamine, 300 mg daily) which would not be expected in a primary depression.
(f) The quick, complete and simultaneous recovery of her cardiac and psychiatric symptoms after treatment with verapamil and frusemide.
(g) The absence of depressive relapse in the four-month follow-up on no antidepressant medication.

All these suggest that the diagnosis of an affective organic disorder of vascular aetiology should be considered before thinking of major depression. In

principle, the fact that a medication with a cardiovascular therapeutic action should prove effective in a cardiovascular pathology with secondary psychiatric symptoms should not surprise anybody; on the contrary, what surprises us is the fact that the authors did not even mention that possibility in the discussion of their case.

LUIS M. IRUELA
VICENTE IBAÑEZ-ROJO

Department of Psychiatry
Universidad Autonoma de Madrid
Clinic Puerta de Hierro
San Martin de Porres, 4
28035 Madrid, Spain

Psychogenic amnesia?

SIR: Domb & Beaman (*Journal*, March 1991, **158**, 423–425) reported the case of "Mr X", whom they consider to be a psychogenic amnesic.

I agree with their assertion that Mr X's loss of personal identity and the circumstances surrounding his admission to hospital suggest a psychogenic amnesia, possibly representing a flight from suicide. There is, however, another rather puzzling aspect to this case, namely, a persisting and selective failure to learn the difficult paired associates of the Wechsler Memory Scale (WMS), despite improvements in performance on other subtests of the WMS. In the experience of our neuropsychological group, this pattern is frequently seen in patients with organic amnesias of bilateral hippocampal origin (Walsh, 1985, 1987). It is also seen in the chronic phase of recovery after left posterior cerebral infarction (Ishikawa *et al*, 1988), and in patients with left temporal lobe epilepsy in the presence of hippocampal sclerosis (Saling *et al*, unpublished).

In view of the neuropsychological findings in this case, it would be interesting to know the nature of Mr X's two previous suicide attempts. Is it possible that some cases of functional retrograde amnesia represent massive psychological elaboration around a kernel of organic involvement?

ISHIKAWA, K., DARBY, D. G., SALING, M. M. *et al* (1988) Verbal and visual recognition memory improvement in left posterior cerebral artery infarction with general amnesic syndrome. In *Proceedings of the Thirteenth Annual Brain Impairment Conference* (eds M. Matheson & H. Newman), pp. 128–162. Sydney.

WALSH, K. W. (1985) *Understanding Brain Damage: A Primer of Neuropsychological Evaluation*. Edinburgh: Churchill Livingstone.

—— (1987) *Neuropsychology: A Clinical Approach*. Edinburgh: Churchill Livingstone.

MICHAEL M. SALING

The University of Melbourne
Parkville 3052
Australia

Clinical interviews with children and adolescents

SIR: It is not customary for authors to write to editors commenting upon reviews of their books and indeed until now I have never done so. However I feel that some comment is needed about Dr Dora Black's review of *Clinical Interviews with Children and Adolescents*.

Dr Black says that "the author seems never to have got clear in his mind what his aims are". I believe that the aims of the book were stated clearly in the introduction. Re-reading this confirms my belief.

She goes on to say that I seem "to be considering therapeutic interviews as well as diagnostic ones" but that I miss them off the list of reasons for interviewing children. This statement is just wrong. Treatment interviews, defined as aiming to promote rather than impose change, are clearly listed on page 12. Dr Black seems to have missed the point I make, also on page 12, contrasting 'manipulative' interviews – which attempt to impose change – with 'treatment' interviews, which aim to promote it.

Earlier in the review Dr Black says that the idea of my book "is an original one". This is not the case. A book entitled *Interviewing Children and Adolescents* was published by John Rich in 1968 and is cited by me. Indeed, the classification of interviews upon which Dr Black comments is derived, with acknowledgement, from Rich's book.

I find it difficult to avoid the conclusion that Dr Black's review was written after only a cursory look through the book.

PHILIP BARKER

Department of Psychiatry
Alberta Children's Hospital
1820 Richmond Road S.W.
Calgary, Alberta
Canada T2T 5C7

AUTHOR'S REPLY: I certainly read Professor Barker's book from cover to cover. Our differences hinge on the fact that the whole book is devoted to what Rich called fact-finding interviews, and the author states that "psychotherapy techniques themselves fall outside the scope of this book" (p. 12) yet in the chapter on "Termination" there is advice about resolving attachment to the therapist. It is this that led me to state that the aims were not clear. Perhaps it would have been better to say that the aims were stated but a trainee would not emerge from reading the book with a clear idea of whether the interviews described were for assessment or therapy. As I said originally, the book is of value as the distillation of the experience of a sympathetic and skilled