comments thus add further weight to the original observations of Baastrup *et al.*

Doubts regarding the true cause of the clinical events in Cohen & Cohen's patients will never be completely resolved, although the validity of their own postulate of a lithium/haloperidol interaction seems to be diminishing as the full explanation.

M. R. LOWE

Basildon Hospital
Basildon, Essex

D. H. BATCHELOR

Janssen Pharmaceutical Ltd
Wantage

References

COHEN, W. J. & COHEN, N. H. (1974) Lithium carbonate, haloperidol and irreversible brain damage. *Journal of the American Medical Association*, **230**, 1283–1287.

SCHOU, M. (1984) Long-lasting neurological sequelae after lithium intoxication. *Acta Psychiatrica Scandinavica*, **70**, 594–602.

GOLDNEY, R. D. & SPENCE, N. D. (1986) Safety of the combination of lithium and neuroleptic drugs. *American Journal of Psychiatry*, **143**, 882–886.

BAASTRUP, P. C., HOLLNAGEL, P., SORENSEN, R. & SCHOU, M. (1976) Adverse reactions in treatment with lithium carbonate and haloperidol. *Journal of the American Medical Association*, **236**, 2645–2646.

Tuberous Sclerosis and the Autistic Syndrome

SIR: Lawlor & Maurer (*Journal*, March 1987, **150**, 396–397) described tuberous sclerosis presenting with autistic behaviour. Reported cases of this association are rare; however, I believe that it is not that uncommon. I can recall seeing three such cases. The following is an example.

Case Report: A male was born following a delayed labour and assisted delivery but recovered normally. Within 5 days he was found to be a restless baby with much screaming and difficulty in feeding. He walked at 12 months, but at 15 months he started to have temper tantrums. Subsequently, it became apparent that he had delayed speech. Later he developed overactive and disruptive behaviour, which eventually lead to his admission to a child psychiatry unit at the age of 3 years. He was diagnosed as having a profound language disorder with severe autistic traits, but was of potentially normal intelligence. Physical examination at this time showed no neurological abnormalities, but roughened skin on the cheeks and between the shoulder blades was noticed. Over the next few years there were many reports of poor social interaction, delayed speech with reversal of pronouns, mannerisms, inappropriate emotional reactions, resistance to change, and unusually good reading ability. At the age of 6 he developed epilepsy, which remained well controlled with anticonvulsants. At various times during his childhood he attended an ESN (M) day school, several private residential schools, and a school for autistic children.

At the age of 9 he was referred to me by the social worker because of difficult and aggressive behaviour. Physical examination revealed clear facial adenoma sebaceum, several areas of depigmented skin on the trunk and legs, and the previously recorded roughened areas between the shoulder blades. The family was advised of the possible diagnosis of tuberous sclerosis. Both parents were very intelligent and they had a normal daughter. The only significant feature in the family history was that the mother had had a mentally handicapped half brother who died at the age of 22 years with a tumour of the liver. Further physical examination of the family by a consultant geneticist revealed that the mother had a slight skin lesion in keeping with the diagnosis. The father and the sister appeared to be unaffected. The parents were advised of the significance of the findings, but had already decided to have no more children.

The patient has continued to present problems of management, especially aggressive behaviour, and still has many autistic features. He attends an Adult Training Centre and lives in a Social Services hostel.

The above illustrates not only the association of the two conditions but also supports the authors' conclusion that a suspicion of tuberous sclerosis should be aroused if autistic features and epilepsy coexist. It also suggests that history taking and physical examination in child psychiatry should always involve a genuine appreciation of genetic factors.

B. E. OLIVER

Chelmsley Hospital
Marston Green
Birmingham B37 7HL

Delusional Parasitosis

SIR: Macaskill (*Journal*, February 1987, **150**, 261–263) reports the first case of a non-pharmacologically induced remission of delusional parasitosis. Hunt & Blacker (*Journal*, May 1987, **150**, 713–714) suggest that this was a mild form of the disorder, associated with clear precipitants. I report two further cases of spontaneous remission.

Case reports: (i) A 51-year-old housewife with no previous history of medical or psychiatric illness presented to a dermatology clinic with a one-month history of parasites in her hair. She described hearing them moving, and could feel them burying into her scalp. She produced a matchbox containing skin scrapings as proof. There were no obvious precipitants, although she was under chronic strain through coping with her demented mother. The dermatologist found no abnormality, and prescribed no treatment. However, he concurred with her suggestion of a brief holiday in Spain. On her return two weeks later she was seen by the psychiatrist. She said the parasites had now gone, but was certain they had been present. There was no evidence of

psychiatric disorder (CIS score = 7; HAD score = 4). She remained well one month later.

(ii) A 58-year-old shop assistant attended the dermatology clinic with a three-month history of delusional parasitosis. She claimed she had been infected by her sexual partner, and that she could now feel insects crawling under her skin, stinging her, and living in her anus. She also felt worms moving in her stomach. She felt dirty, and thought her body odour was offensive. She had attended her GP, a private dermatologist, and several alternative practitioners, but without benefit. She had had a genuine hook worm infestation as a teenager. She had also received antidepressants from her GP when she was 32 and 42, during times of marital stress.

When seen by the psychiatrist she had been ill for four months. Her beliefs were delusional in nature, and she recorded maximal conviction on visual analogue scales of intensity of belief. Other than fatigue there was little to suggest depression (CIS score = 21). She refused treatment. One month later she reported that she had passed a "worm" in her stools, and described a three inch long creature with horns. Since then the rest of the parasites had died, and she no longer felt infested. She still felt she had a dirty tongue and skin, and still felt self-conscious of her body odour (CIS score = 12).

Previous reports in the psychiatric literature have emphasised the intractable nature of untreated delusional parasitosis (Monro, 1980; Sheppard *et al*, 1986), unless as part of a depressive illness. In a questionnaire survey of dermatologists, Lyell (1983) identified 282 cases, and confirmed the generally poor prognosis, but claimed 13 had remitted. He also described one case history of a spontaneous remission after three months. Batchelor & Reilly (1986) used a similar method to obtain details of 55 patients. Although again confirming the poor prognosis, "a third had a duration less than a year" while one had "improved slowly on dermatological treatment only". The gloomy natural history reported by psychiatrists may partly reflect patterns of referral.

SIMON WESSELY

Department of Psychological Medicine
King's College Hospital
Denmark Hill
London SE5

References

BATCHELOR, D. H. & REILLY, T. M. (1986) Epidemiological aspects of delusional parasitosis in the United Kingdom. In *Proceedings of the 15th European Conference on Psychosomatic Research* (eds J. H. Lacey and D. A. Sturgeon). London: John Libbey.

LYELL, A. (1983) Delusional parasitosis. *British Journal of Dermatology*, **108**, 485–499.

MONRO, A. (1980) Monosymptomatic hypochondriacal psychosis. *British Journal of Hospital Medicine*, **24**, 34–38.

SHEPPARD, W. P., O'LOUGHLIN, S. & MALONE, J. P. (1986) Psychogenic skin disease. A review of 35 cases. *British Journal of Psychiatry*, **149**, 636–643.

Lucid Intervals in Catatonia: A Neuropsychiatric Snare for the Unwary

SIR:

Case report: A 42-year-old woman with a recent history of treatment of a major depressive disorder with dothiepin (150 mg/day) suddenly became agitated, complaining of being unable to walk and that her mind was going blank. She was admitted in a state of retarded depression, but despite an increase in dothiepin (225 mg/day) continued to deteriorate. Her mental state fluctuated between mute immobility and terror-stricken agitation, for which she was given chlorpromazine (100 mg i.m.) on three occasions over a one-week period. She resisted all nursing care and had to be fed and toileted, at times exhibiting verbigeration, bizarre and stereotypical behaviour, and catalepsy. Diazepam (20 mg i.m.) prior to CT scanning produced a dramatic improvement, lasting several hours. She spoke coherently and started eating, before relapsing. Investigations, including CT scan and EEG, were normal. Full recovery followed after three bilateral ECTs, and she remained well at six-month follow-up.

Amylobarbitone sodium interview continues to be advocated as a diagnostic aid in distinguishing catatonia secondary to idiopathic psychiatric ('functional') disorders from those that are toxic-metabolic or neurological ('organic') in origin (Altshuler *et al*, 1986). Our patient's response would be a case in point were it not for the fact that the catatonic signs became apparent only after the introduction of neuroleptic treatment. Ainsworth (*Journal*, January 1987, **150**, 110–112) and Chick *et al* (*Journal*, July 1987, **151**, 130–131) describe similar results with benzodiazepine and barbiturate infusions in catatonia, due to viral encephalitis and structural brain damage respectively. Further doubt is thrown on the diagnostic value of these techniques by reports of temporary or permanent relief obtained in a case associated with pituitary adenoma (Sheline & Miller, 1986), neuroleptic-induced catatonia (Fricchione *et al*, 1983) and the neuroleptic malignant syndrome (NMS) (Lew & Tollefson, 1983), which clinically resembles an iatrogenic variety of Stauder's "lethal catatonia". In addition, Lim *et al* (1986) have reported dramatic responses to i.v. phenytoin in catatonia as a manifestation of non-convulsive status epilepticus.

Several lines of evidence suggest that catatonia is the product of disturbed central neuroregulatory-vegetative functioning. The similarity in symptomatology between the neuroleptic-induced and other