Vietnamese origin who was admitted to the South Western Hospital in January 1982 following a period of "strange behaviour" at school, which included an attempt to jump out of a window, emotional lability, anorexia and insomnia. There was no relevant family history. There was however a previous history of a similar episode during a period of emotional stress three years previously, which was said to have been cured by a Chinese healer using water. This episode again occurred during a period of severe emotional stress associated with bullying at school.

On admission the patient was almost mute, giggled inappropriately, and was observed to talk to herself, often expressing suicidal ideas. She was treated with an injection of haloperidol 10 mg intramuscularly, and the following day she suddenly developed acute catatonic symptoms including posturing and waxy flexibility, which were accompanied by oculogyric crises and torticollis. She continued to appear unperturbed and to smile inappropriately. These symptoms disappeared spontaneously approximately fifteen minutes later, but returned on a further occasion later that day. On this occasion she was given an injection of diazepam 7.5 mg intravenously which quickly abolished both the catatonic and schizophrenic symptoms. Since her behaviour continued to be disturbed, she was observed carefully and her medication changed to oral haloperidol 5 mg qds and oral procyclidine 5 mg tds. Her behaviour gradually returned to normal over the next few days, all physical investigations were normal, and haloperidol was discontinued five days later. She was discharged from hospital eighteen days after admission, on a small dose of amitriptyline, and followed up in the out-patient clinic; she has remained well for almost nine months. A diagnosis of depression with hysterical dissociative reaction under stressful conditions was made, together with an uncommon catatonic reaction to neuroleptic agents.

ANNE CREMONA-BARBARO

St Thomas' Hospital,
London SE1

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HAZARDS OF LITHIUM AND NEUROLEPTICS IN SCHIZO-AFFECTIVE DISORDER

DEAR SIR,

I read with interest Delva and Letemendia's review of lithium treatment in schizophrenia and schizo-affective disorder (*Journal,* October 1982, **141,** 387–400), and I share their concern about the combining of lithium and neuroleptics in treating these conditions. As the authors point out this is a topic which so far has received little attention. This combination of lithium and neuroleptics, especially when both are used in high doses, can sometimes have unexpected results as the following two brief case histories illustrate.

Case 1. A 22-year-old female patient on her first admission to hospital showed paranoid delusions, feelings of being possessed by the Devil, thought disorder, bizarre posturing, along with great hyperactivity, disinhibition, and total insomnia for the previous week. A provisional diagnosis of schizo-affective illness, schizomanic type, was made and she was commenced on lithium carbonate and chlorpromazine. She reached a peak daily dose of 2000 mg lithium and 800 mg chlorpromazine after eight days. Serum lithium levels were done every two days and these were 0.54, 1.24 and 1.52 respectively. On the tenth day when the serum lithium was 1.52 there was a dramatic change in the clinical picture. The patient developed extra-pyramidal side-effects (EPSE: pseudo-parkinsonism and dystonia) along with confusion, disorientation for time and place, and an ataxia which caused her to fall several times. Simultaneous with this the original psychotic symptoms disappeared. All drugs were stopped and benztropine added. Rapid improvement then followed with no long term sequelae.

Case 2. A 35-year-old female was admitted for the fourth time for schizo-affective illness, schizomanic type. She showed paranoid delusions, auditory hallucinations but also great hyperactivity, pressure of speech and nearly total insomnia. She was treated with a peak dosage of lithium carbonate (Priadel) 1600 mg and chlorpromazine 800 mg. Serum lithium levels were done every two days and showed levels of 0.59, 1.28, and 1.42. At the 1.42 level, on the eighth day of treatment, a similar sudden clinical change took place

with severe EPSE (pseudo-parkinsonism) combined with confusion, dysarthria, double orientation for place, poorly formed delusions which were different and unrelated to the original ones, and ataxia. All medication was stopped, benztropine was prescribed, fluids pushed, and again recovery was rapid and uneventful.

In these two cases there seems to have been a pattern: at a high serum lithium level the illness suddenly "broke" much as Jefferson and Griest (1977) have described when lithium is used to treat acute mania, the original psychotic symptoms disappeared and the patients developed severe EPSE and toxic confusional symptoms. The EPSE could have been due to a direct dopamine blocking effect of lithium (Tyrer *et al*, 1980) which would have been enhanced by chlorpromazine. It is possible that the toxic confusional symptoms were caused by the high serum lithium levels reached. These cases show the value of frequent serum lithium levels when one is trying to sort out a mixed clinical picture where EPSE and toxic confusional symptoms co-exist.

A. J. McGennis

St Brendan's Hospital,
Rathdown Road,
Dublin 7

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FAILURE TO MOURN AND MELANCHOLIA

Dear Sir,

I should like to compliment Dr Pedder on his article dealing with pathological mourning (*Journal*, October 1982, **141**, 329–37).

One point that I would wish to make is that the dichotomy between the behavioural psychotherapist and the dynamic psychotherapist, in regard to the treatment of pathological mourning, can only be an unfortunate and damaging hindrance to patients requiring treatment. In my article—"Nineteen Cases of Morbid Grief"—the description of the therapy, I believe, clearly indicates the therapist conscientiously combining behavioural principles of systematic desensitization and implosion with psychodynamic principles in which the therapist remains warm and empathic. I further state that the therapist must prepare the patient for the eventual loss of the patient-therapist relationship and that failure to do so could block the successful conclusion of therapy.

I also feel that it has been insufficiently stated that the effects of morbid grief can mimic an entire range of psychiatric disorders, from neurotic disorders such as agoraphobia through to the precipitation of relapses in schizophrenic illnesses.

Stuart Lieberman

St George's Hospital Medical School,
Cranmer Terrace,
Tooting, London SW17 0RE

MANIA ASSOCIATED WITH WEANING: A HYPOTHESIS

Dear Sir,

Dr Abou-Saleh's letter (*Journal*, May, 1982, **140**, 547,) postulating that a sharp decrease in blood prolactin level that followed weaning was involved in the pathogenesis of post-partum mania requires qualification. I have personally treated 286 patients with puerperal psychosis and the vast majority were affective or schizo-affective but relatively few suffered from mania. Those with acute mania were generally referred while still in the maternity hospital or had been recently discharged. Abrupt weaning was not a factor in these cases.

I had a patient with an acute and severe post-partum mania who, several years later, had a carbon copy psychotic episode following appendectomy and it is doubtful whether prolactin levels were involved in the second episode. This does not mean that in very susceptible people a sharp fall in prolactin level could not be the insult which triggers the psychosis. What is required is a more detailed study of puerperal mania and the variety of insults that can precipitate it.

Dr Abou-Saleh's suggestion of hormonal treatment in these cases is an interesting one but over the years haloperidol has taken over from chlorpromazine as a tranquillizer of choice in puerperal mania and it is the latter which is more potent in raising prolactin levels.

M. Sim

Forensic Psychiatric Clinic,
320, 1175 Cook Street,
Victoria, British Columbia V8V 4A1

EPIDEMIC PSYCHOSES, OR EPIDEMIC KORO?

Dear Sir,

I read with interest Dr Harrington's account of three outbreaks of widespread psychological reactions in Thailand (*Journal*, 1982, **141**, 98–99) and wish to make the following comments.

The symptoms of the first outbreak of Rok-Joo or the genital shrinking disease seems to be remarkably similar to koro, a culture bound psychogenic syndrome in which a subjective experience of penile shrinkage occurs in association with acute anxiety. Whether the victims of the Thailand epidemic had the fear of their genitalia shrinking into their abdomens with a fatal