essential information, to be updated at each reassessment by the multidisciplinary team, and incorporating the contributions of all disciplines.

New members of staff, or those dealing with emergencies, would need to read only a single typed sheet to acquire a grasp of the case.

Forms with headings could be used for recording information, e.g.:

> Name, ward—date of birth—borough of origin—dates of and reasons for admissions—treatments and special precautions, e.g. drug sensitivity—general progress—other medical/surgical conditions. Present mental state (date of examination)—nursing dependency—occupational ability—links with family—social activities—diagnostic formulation. Current treatment/rehabilitation programme and aims.

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CLINICAL ASPECTS OF THE INTERACTION OF LITHIUM AND STIMULANTS

DEAR SIR,

Recently it has been shown that lithium can prevent some aspects of the response to amphetamine (Van Kammen and Murphy, 1975) and methylphenidate (Huey *et al*, 1977). However, others have questioned the uniformity and degree of the lithium block of stimulant effects (Angrist and Gershon, 1979; Wald *et al*, 1978). We present here two cases where lithium did *not* prevent stimulant effects in humans and it is suggested that clinicians should remain open about the nature of lithium interaction or lack of it with other psychoactive drugs in clinical situations.

Case 1. A 47-year-old married man with a 27-year history of bipolar manic-depressive illness was stabilized on lithium therapy with a marked elimination of manic attacks. Mild chronic outpatient depressions persisted and were not responsive to addition of amitriptyline up to 150 mg or nialamid to 125 mg for 4 weeks each. After cessation of previous antidepressants but while remaining on lithium with plasma levels of 0.6–0.7 mEq/L, methylphenidate 10 mg each morning led to rapid remission of depressive symptoms of low mood, lack of energy, loss of sex interest and psychomotor retardation. The methylphenidate was continued for three months and then discontinued with no return of symptoms. Lithium did *not* block the antidepressant effect (Bassik and Schoonover, 1977) of methylphenidate.

Case 2. A 57-year-old divorced man with a 24-year history of bipolar manic-depressive illness was a lithium responder. He suffered from a bucco-facial-lingual masticatory syndrome diagnosed as tardive dyskinesia due to neuroleptic usage before institution of lithium prophylaxis. As part of a research protocol using the concept of receptor sensitivity modification treatment of tardive dyskinesia (Friedhoff, 1977), the patient received 125 mg L dopa and 12.5 mg carbidopa daily for 3 days. On the fourth day the dose was raised to 250 mg L dopa plus 25 mg carbidopa. On the following day the patient developed typical manic syndrome with pressure of speech, aggressiveness, hyperactivity, and absence of need to sleep. The lithium blood level was 1.04–1.1 mEq/L. The effect of L-dopa to induce mania in bipolar patients has been previously noted (Murphy *et al*, 1971) but the phenomenon's occurrence in the presence of therapeutic lithium levels is important. This same patient had previously developed a transient (8 hours) manic attack in a research design employing 30 mg of intravenous methylphenidate, despite therapeutic lithium levels then (Wald *et al*, 1978).

In summary, lithium coverage does not necessarily prevent either positive or negative consequences of arousal-inducing drugs.

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