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Tranquillizers Causing Aggression

SIR,—Your important leading article (18 January, p. 113) has major implications in the prevention and management of child abuse.

We are engaged in research and service in this field. A high proportion of parents in families referred for actual or threatened child abuse are taking drugs at those times that the crises occur. Benzodiazepines and tricyclic antidepressants have been prescribed in the puerperium on the complaints by the mother of "depression" and anxiety about her capacities as mother and spouse. If she is then isolated with an inconsolably crying infant outbursts of aggression occur.

The interaction between drug effects and social stimulation was, as you rightly note, presciently described by Chance[1] some 29 years ago. The clinical implications have been slow to dawn on us because we have tended to treat diseases rather than persons living in specific circumstances.

Many mothers describe how, while taking these drugs, instead of feeling less anxious and depressed they have become more hostile and openly aggressive towards the child, and often to other members of the family. These disturbed mothers have gone to their doctors with complaints which signalled grossly abnormal interpersonal feelings. Their symptoms are frequently treated as isolated anxiety states or depressions, but the frustrating stimuli continue. Thus the stage is set for the mother to react in a paradoxical way with hostility and aggression.

We advocate extreme caution when prescribing tranquillizers and antidepressants for mothers of young children, especially when the complaints include inability to cope with a baby's demanding and frustrating behaviour. It could well be that such complaints are in fact a warning that a child is at risk of abuse, and well-meant prescribing of tranquillizers might help to precipitate just such an event.—We are, etc.,

MARGARET A. LYNCH
JANET LINDSAY
C. OUNSTED

Human Development Research Unit,
Park Hospital for Children,
Headington, Oxford

1 Chance, M. R. A., *Journal of Pharmacology*, 1946, 87, 214.

SIR,—Your leading article (18 January, p. 113) is a reminder of the dangers of the widespread unnecessary prescribing of psychotropic drugs.

I suspect that not only is the aggression produced by benzodiazepines directed outwardly to others but the instability that they produce may not infrequently lead to self-poisoning in an impulsive act undertaken with uncertain intent. This is a common finding which was confirmed in an unpublished study undertaken at Walton Hospital, Liverpool, and designed by Dr. Eric Birchall. In approximately 550 consecutive cases of self-poisoning 30% of the individuals had a diagnosis of depression and 5% some other treatable psychiatric condition; 25% had a diagnosis of personality disorder and 40% had no detectable psychiatric abnormality, the act of self-poisoning being a reaction to stress. Quite clearly, had 65% of these people not had access to tablets the workload of the accident and emergency department with its attending psychiatrists would have been considerably reduced. Roughly 63% of the people reviewed had been taking psychotropic drugs, in many instances unwisely prescribed in circumstances where brief psychotherapy was the treatment indicated.—I am, etc.,

I. B. COOKSON
Priory Day Hospital, Birkenhead

Paraquat Poisoning Treated with Immunosuppressants and Potassium Aminobenzoate

SIR,—I would like to report an interesting clinical result in a case of paraquat poisoning.

The patient, a highly intelligent schizophrenic man aged 32 years, was admitted to this hospital on 21 November 1974 having swallowed seven days previously a quantity of Gramoxone (paraquat) estimated at between one-quarter and one-third of a cupful (>20 ml) diluted with orange juice in a suicide attempt. He did not inform his general practitioner for six days, though during this time he had severe inflammation of the tongue, palate, and pharynx.

On admission he was moderately dyspnoeic at rest with bilateral but minimal left-sided crepitations. Chest *x*-ray showed non-specific consolidation in the left lower lung fields and left basal segment, considered to be in keeping with paraquat poisoning. Renal function tests at this time showed intermittent albuminuria and haematuria. Blood urea was 35·8 mmol/l (216 mg/100 ml) and creatinine 928 mmol/l (10·5 mg/100 ml). A qualitative test for paraquat in the urine with sodium dithionite was negative, but seven days had elapsed since its ingestion. Treatment was initially on standard lines with high dosage of steroids, prednisolone 100 mg daily, and prophylactic antibiotics.

His condition steadily deteriorated and on 26 November left lobe consolidation had greatly increased and the lateral aspect of the right lung was showing involvement, with overlying pleural reaction. The changes were now considered compatible with severe paraquat poisoning. He was now very dyspnoeic at rest and his pulmonary function tests showed progressive deterioration with Po_2 falling to 6·65 kPa (50 mm Hg), Pco_2 remaining at 4·79 kPa (36 mm Hg), and pH 7·48. Liver function tests were unremarkable.