response". Another possible explanation of her findings is that pharmacokinetic factors were responsible for the failure of chronic neuroleptic treatment to produce either a clinical response or maximal prolactin elevation in many of her patients.

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CAPGRAS SYNDROME IN A 14-YEAR-OLD

DEAR SIR,

A mildly mentally handicapped girl was admitted to this unit in September 1981 suffering from a psychosis, with auditory hallucinations and paranoid ideas. Her affect was depressed and she was overactive and aggressive, with flight of ideas. Her eating and sleeping patterns were disturbed. She was treated with Haldol, Melleril and Cogentin.

One morning in October shortly after her fourteenth birthday she said it was not her mother that had visited her the previous day. It had been a man in disguise, because although this person had been wearing a skirt, the hair was a wig. She had noticed this when she looked at the collar edge of the hair. Also this person drove much faster than her mother—like a man, in fact. This idea subsided within a few days but she remained frightened, and felt that people in cars were watching her. Her psychosis resolved and her mother took her home in November 1981.

A search of the recent literature has not revealed the Capgras syndrome in one so young.

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THYROTOXICOSIS PRESENTING AS DEPRESSION

DEAR SIR,

We would like to call attention to a case in which a 23-year-old woman was admitted to our unit following an amitriptyline overdose. She had been prescribed amitriptyline and chlordiazepoxide two weeks before, at a psychiatric clinic where she presented with depressed mood, insomnia, early morning awakening, a 5 kg weight loss, and 'edginess'. Following drug therapy, tearfulness, decreased productivity, and poor concentration ensued and she attempted suicide.

On evaluation following the overdose, she complained of tremulousness, palpitations, and flushing. She gave no past history of psychiatric or medical illness. Physical examination revealed a thin (40 kg) female with tachycardia, mild tremor and the absence of thyromegaly or eye signs. On mental status exam she was sad and tearful with psychomotor retardation and depressed mood. Memory and cognitive functions were intact. Laboratory results indicated elevated thyroid function.

Endocrine consultation was arranged and Graves' disease confirmed. A treatment plan including propylthiouracil, propranolol and radioactive Iodine was followed. Depressive symptoms resolved and follow-up at six months revealed euthymia, normal weight and return to work.

Thyrotoxicosis uncommonly presents with depressive symptoms. Apathetic hyperthyroidism as described by Lahey presents with symptoms of depression, apathy, and intellectual stupor (Lahey, 1931). These 'thyroid melancholics' are generally older, appear ill with weight loss and do not demonstrate the usual signs and symptoms of Graves' disease (Taylor, 1975; Thomas *et al*, 1970). Our patient was only 23 and her deterioration after initial treatment with psychotropic agents serves as a warning that tricyclic antidepressants may exacerbate psychiatric symptoms accompanying hyperthyroidism. She not only did not respond, but noted diminished concentration, palpitations, and worsened depression-anxiety culminating in a suicide attempt. We speculated that her increased levels of thyroid hormone may have made her more sensitive to the anticholinergic and adrenergic effects of amitriptyline. This would be consistent with Whybrow and Prange's hypothesis of thyroid-catecholamine receptor interaction (Whybrow and Prange, 1981). In addition, other signs of thyrotoxicosis developed and persisted until antithyroid treatment took effect.

Initial attention to review of systems and avoidance of one-system dominance would have led to a correct differential diagnosis. The danger of erroneous diagnosis and placement on psychotropic medications is a reminder of the possible associations between depression and thyrotoxicosis, and its responsiveness to antithyroid treatment.

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PIMOZIDE: ADVERSE REACTION AND PROLONGED HALF-LIFE

DEAR SIR,

Extrapyramidal reactions to neuroleptic drugs are so frequently encountered as to be considered an inevitable concomitant of appropriate treatment in many patients. We describe an individual case where a severe and life-threatening reaction appears to have been the result of delayed drug elimination. The patient is a 28-year-old woman with minimal brain damage who was admitted on this occasion because of a hypomanic episode. The drug history is shown in Fig 1. Pimozide was chosen because of its reputedly low incidence of extrapyramidal side effects. Following an initial brief exposure to pimozide the patient was commenced on 4 mg/day and this was maintained for nearly six weeks. Towards the end of this time both thioridazine and benztropine were withdrawn because of drowsiness and of blurred vision respectively. At the time indicated by the arrow in Fig 1 the patient developed laboured breathing and intermittent sweating and the pimozide was abruptly withdrawn. However, over the next 48 hours she developed rigidity of such severity that she was unable to move, eat or speak. Benztropine and diazepam produced only limited and transient improvement and there was still marked rigidity two weeks after stopping the pimozide. Slow improvement did, however, occur and the patient was eventually discharged with no residual extrapyramidal signs.

The severity and duration of this dystonic reaction with a drug initially thought to be associated with mild and readily reversible side effects (Pinder, Brogden, Sawyer, Speight, Spencer and Avery, 1976) suggested that impaired metabolic clearance might be

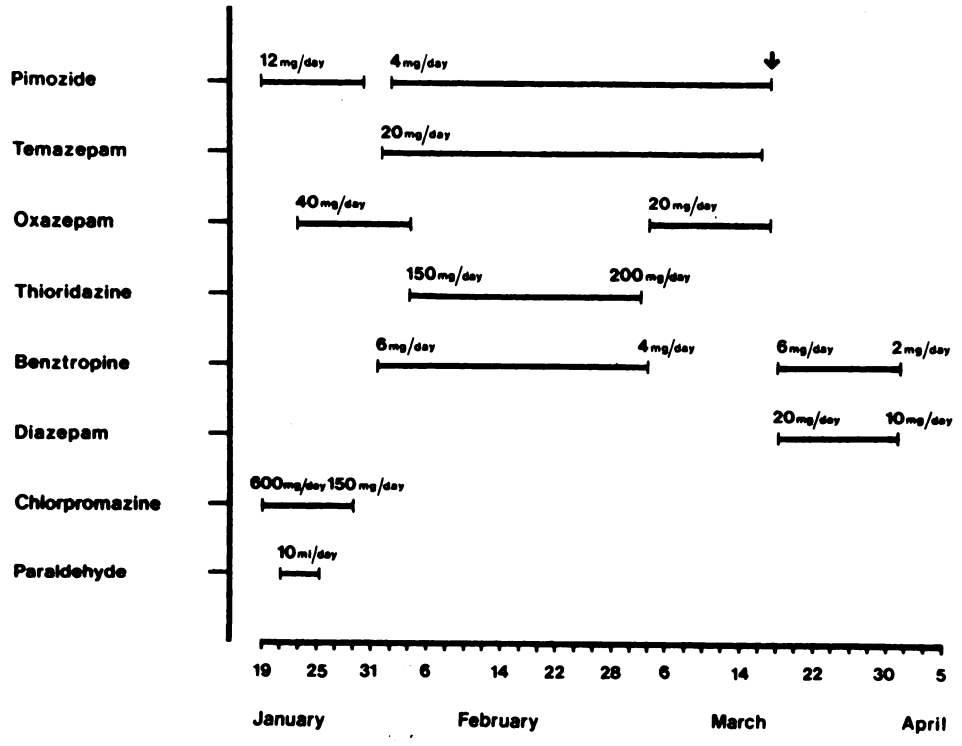

FIG.—Time course of drug exposure in this patient. The onset of severe dystonic symptoms is indicated by an arrow.