assumed that the antidepressant effect of flupenthixol is manifested at lower doses than the anti-psychotic effect – although this has never been proven by case studies or clinical trials. The most likely explanation for her response is an interaction between clomipramine and flupenthixol, not involving the anti-psychotic effects of the latter, the rapidity of the response to the drugs in combination suggesting synergism between the two compounds.

Illness episodes of this chronicity and severity are uncommon and we think that the fact that she responded to the combination is encouraging. More case reports of such efficacy in resistant depression would be necessary to assess the feasibility of this regime being used in a clinical trial in this condition.

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Water Intoxication in Congenital Neurosyphilis

SIR: Water intoxication may occur in almost any psychiatric disorder (Ferrier, 1985), but this is the first report of it in congenital neurosyphilis. In about 80% of cases of water intoxication the patients are psychotic (usually schizophrenic). Although this patient was psychotic, mentally retarded, and had some abnormal neurological signs, the precise diagnosis of congenital neurosyphilis was delayed, as by the time she was seen VDRL and TPHA were negative and Hutchinson's teeth had been extracted.

Case report: A 48-year-old mentally retarded woman was admitted to a psychiatric ward after she violently attacked her 75-year-old mother, with whom she still shared a bed at night. Prior to admission she was doing little apart from smoking heavily (80 cigarettes a day) and drinking fluids excessively. Each day she would consume 3 pots of tea, 6 pints of milk, 2.5 litres of orange juice, and an unknown quantity of water directly from the taps. The patient had three previous admissions to the psychiatric unit. The first time, at the age of 45, diagnoses of mental retardation, alcoholism, and depression with obsessional and hypochondriacal features were made. The second admission (aged 46) followed her attempt to set fire to her home. She was still abusing alcohol (8–10 pints of beer per day) and was depressed. While in hospital she drank water to such an extent that she developed hyponatraemia, hypokalaemia, and had *grand mal* seizures. During the third admission (at the age of 47) she was still drinking alcohol and water excessively, and was thought to be depressed. Following discharge she did not abuse alcohol, but continued to drink water as before. On this fourth occasion she presented as a slim, dishevelled, perplexed woman who was incontinent of urine. She was agitated, incoherent at times, mumbled, and drew a cross on my forehead with her finger and prayed, but was unable to explain why. She was disorientated in time and place, described herself as being frightened and everything around her as being larger (macropsia). There were no auditory hallucinations or systematised delusions. Physical examination of cardiovascular and respiratory systems was normal. There was an abdominal scar from a duodenal ulcer operation. Central nervous system examination showed decreased visual acuity and two small aneurysms in the right eye. The right knee jerk was absent and the right plantar response was up; the left plantar response was normal. The ankle jerks were absent. Proprioception was impaired, but more on the right side. Pin prick showed glove and stocking peripheral neuropathy. She had a tremor of both hands. Her gait was slightly ataxic. Musculoskeletal system was normal apart from swelling over right knee and left ankle.

Investigations: FBC, thyroid, liver function tests, serum glucose, midstream urine culture, ECG, and EEG were normal. VDRL, TPHA, FTA were negative. CT scan showed minimal degree of general cerebral atrophy. Urea, calcium, sodium, and potassium were below the lower end of the reference range, in keeping with excessive water intake. Urinary osmolality was 116 mosmol/kg (reference range 40–1400) and plasma osmolality was 277 mosmol/kg (reference range 275–295). Old case notes were obtained from the Rheumatology Department; those stated that at the age of 19 (when she presented with backache) her Hutchinson's teeth were noticed. WR was found to be negative, TPI positive, and CSF gave a typical Lange curve. She was treated with a full course of penicillin and Hutchinson's teeth were extracted.

Progress: following administration of trifluoperazine (15 mg nocte, orally) her fluid intake decreased, she became continent, and her mental state improved, but the serum electrolytes were still abnormal, so demeclocycline (250 mg q.d.s.) was added. Further restrictions on water intake and cigarette smoking were made. On discharge her mental state was normal apart from mental retardation (IQ 63) and slight memory defect. Her fluid intake was 1.9 litres daily, and all electrolytes were normal. Two weeks later she stopped taking her medication and soon had a relapse. The same treatment was attempted on an out-patient basis, and she again improved.

Diabetes insipidus has been reported as a rare manifestation of congenital neurosyphylis, but no previous reports of water intoxication were found in the literature. While in both conditions polydipsia and polyuria occur, in diabetes insipidus urinary osmolality is well below plasma osmolality, and serum urea and sodium tend to be raised. In water intoxication, both plasma and urinary osmolality are low and serum urea and sodium are below reference values. The syndrome of inappropriate antidiuretic hormone secretion in which hyponatraemia occurs can be excluded by the absence of raised urinary osmolality. This patient has been treated with trifluoperazine, and although psychotic symptoms were effectively treated the water intake remained excessive. Dopamine has been shown to inhibit vasoprotein release in man (Lightman & Forsling, 1980) and it is suggested that in this case its antagonism by trifluoperazine could have led to increased ADH. Demeclocycline, an antidiuretic hormone antagonist, was therefore added to the treatment, and this was followed by the return of serum electrolytes to reference values. However, the effects of neuroleptics on ADH secretion are inconsistent and I suggest that the personal habits of the patient such as smoking and tea and alcohol intake were not considered. This patient drank large quantities of tea and smoked heavily prior to this admission. Theophylline increases cAMP and diuresis. The effect of nicotine is to increase ADH and decrease diuresis, while alcohol has the opposite effect (Laurence, 1977). Therefore, smoking and tea intake should be controlled as part of treatment for water intoxication.

I am grateful to Dr D. Hailstone for his permission to report this patient.

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Neuroleptic Malignant Syndrome and Compulsive Water Drinking

SIR: The neuroleptic malignant syndrome (NMS) is thought to represent an idiosyncratic reaction to dopamine blockade, probably mediated via basal ganglia and hypothalamic pathways (Szabadi, 1984). Compulsive water drinking has been associated with a variety of organic and psychiatric disorders, particularly psychosis (Ferrier, 1985). Although the biochemical control of thirst remains uncertain, it seems likely to involve dopaminergic pathways in the hypothalamus. Both these conditions carry a significant morbidity and mortality and are probably underdiagnosed. I report a patient who exhibited compulsive drinking while suffering NMS.

Case report: A 27-year-old woman was admitted, mute, with generalised muscle rigidity, coarse Parkinsonian tremor, sialorrhoea, difficulty swallowing, and sweating. She was indiscriminately incontinent of urine, and had episodic attacks of tachycardia and profuse sweating. She was never pyrexial. This condition had developed immediately after starting haloperidol (one week previously), and failed to respond to oral or intravenous anti-cholinergic drugs. A diagnosis of NMS was made, and all neuroleptic drugs were stopped. One week later she was observed to be drinking copious amounts of fluid; drinking from taps, water jugs, undiluted bottles of squash, and continually requesting drinks on top of those provided with her diet. She was still rigid and incontinent. The excessive drinking lasted five days, and proved impossible to quantify. Urea and electrolytes had been normal prior to excessive drinking (blood urea slightly reduced at 2.7 mmol/litre; reference range 3.0–7.0 mmol/litre). Two weeks after discontinuation of neuroleptic drugs her condition had completely recovered and there was no evidence of psychosis.

Although I am not aware of any reported association between excessive drinking and NMS, Wedzicha & Hoffbrand (1984) described a case of recurrent NMS associated with hyponatraemia. The authors suggested that the hyponatraemia may have precipitated NMS, and they noted that the patient was observed to be drinking excessive water on the ward, which raises the possibility that the excessive drinking and hyponatraemia could have been secondary to NMS. The association between neuroleptic drugs and drinking behaviour is unclear. Smith & Clarke (1980) concluded that increased thirst is likely to be related to the hyperdopaminergic state, but speculate that neuroleptic drugs may induce thirst by a mechanism similar to that proposed for tardive dyskinesia, i.e. denervation supersensitivity, occurring at hypothalamic dopamine receptors.

Once recovered, our patient was unable to recall drinking excessively or to explain it. Unfortunately, we do not have the data to comment on whether water intoxication occurred, or if there was any disturbance of antidiuretic hormone production, although this would be of great interest. It is not possible to say whether the two conditions were