The severe side effects reported by others—for example, peripheral neuropathy and psychosis—were not seen in our patients. This could theoretically have been because of the reduced chloroform content of the mixture. Previous reports, however, were published before the current analytical techniques for detecting the presence of various drugs in the urine were available and it may well be that the patients in previously reported cases of chlorodyne abuse were taking other drugs such as amphetamines, barbiturates, or alcohol without investigator knowing. The question arises whether chlorodyne dependence is a widespread urban problem. The manufacturers claim to sell 1½m bottles a year (equivalent to 9.0 kg of morphine) and a Glasgow pharmacist was recently struck off the *Register* for overselling chlorodyne (Finer, 1973). The proportion of the total amount of the drug manufactured which is used in bona fide therapeutics is unknown but our evidence suggests that abuse is commoner than has been suspected. Probably only a small proportion of chlorodyne-dependent people reach medical supervision.

Conclusions

Chlorodyne dependence affects the young as well as the middle-aged and its extent has been underestimated. Doctors and pharmacists need alerting to this problem. The prognosis in terms of giving-up the drug seems to be very poor and further work needs to be done to establish a satisfactory withdrawal regimen. In the light of our findings we recommend placing the supply of chlorodyne under some form of control.

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MEDICAL MEMORANDA

Idiopathic Hypoparathyroidism Presenting as Dementia

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Idiopathic hypoparathyroidism is a relatively rare condition and is believed to occur mainly in children and young adults (Steinberg and Waldron, 1952; de Mowbray *et al.*, 1954; Bronsky *et al.*, 1958; Kopin and Rosenberg, 1960). The present case is of particular interest as the patient, an 80-year-old man, was completely demented on admission but was discharged after treatment well and with normal, rational behaviour.

Case Report

An 80-year-old-man was admitted to hospital in December 1972 because he had been falling about at home. On admission he was unable to give any history or account of himself. His wife said that he had been deteriorating for many months and had been very confused and forgetful for a long time.

Previously he had been well until April 1971, when he complained of a deterioration in vision for two years. He was found to have bilateral cataracts, which were extracted in April (right) and October (left) 1971. He was admitted for a short period in February 1972 complaining of malaise and frequent belching and was noted to have early dementia; no cause was found for his gastrointestinal symptoms on barium-meal examination or gastroscopy.

On examination he was found to be completely disorientated in time and space. He was unable to respond to commands though could answer simple questions. There were no abnormal neurological signs but he had a raised temperature and signs of a chest infection and there were scattered bronchopneumonic changes on the chest x-ray film. The chest infection responded to Septin

(co-trimoxazole) but he remained demented. His answer to any question was "not bad," and he spent a large part of the day walking up and down the ward, colliding with objects and people.

Three weeks after admission he had a grand mal convulsion. There were no further abnormal neurological signs and the skull x-ray picture was reported as normal apart from some calcification in the carotid syphon. He was started on phenytoin 100 mg thrice daily. Seven days later he was noted to be totally unresponsive, and was lying in bed with a Parkinsonian tremor of the hands. This was followed by a period of tetanic spasm of the left hand accompanied by a degree of stridor. The combination of tetany, cataracts, convulsions, and Parkinsonian features suggested hypocalcaemia and possibly hypoparathyroidism. He had no skeletal malformations of pseudohypoparathyroidism; he was a tall man with a thin face.

Investigations were: calcium 5.9 mg/100 ml; phosphate 5.6 mg/100 ml; alkaline phosphatase 23 K.A. units; protein 7.1 g/100 ml (albumin 3.5 g, globulin 3.6 g/100 ml); electrophoresis, normal pattern; bilirubin 0.4 mg/100 ml; serum aspartate aminotransferase 15 IU/l; magnesium 1.8 mg/100 ml; sodium 133 mmol/l; potassium 4.2 mmol/l; 24-hour urinary calcium 2.1 mg/100 ml.

Electrocardiograms showed a prolonged Q-T interval (0.5 sec) in lead aVL, where no U wave was present. The Q-Tc was 0.51 sec and is not usually more than 0.38 sec at rates of 60-80. In the absence of heart disease, drugs, and hypokalaemia this was thought to be an indication of hypocalcaemia. These changes were present on the electrocardiogram taken in February 1972.

X-ray examination showed no evidence of rickets or osteomalacia. A three-day faecal fat estimation was 8.3 g/day, but there seemed no clinical cause for malabsorption; he did not have diarrhoea and a jejunal biopsy specimen was normal. A repeat collection showed 4.1 g/day (normal). Tests for autoantibodies including parathyroid antibody all gave negative results. An assay for parathyroid hormone failed to detect the presence of any hormone.

Treatment.—He was given intravenous calcium followed by dihydrotachysterol 1 mg twice daily and Calcium Sandoz one tablet three times a day. After a few days he showed considerable mental improvement and his serum calcium started to rise. After three weeks the serum calcium was 8.0 mg/100 ml, phosphate 4.8 mg/100 ml, and alkaline phosphatase 17 K.A. units. He was stabilized on calciferol 1.25 mg daily and Calcium Sandoz one tablet three times a day. At the beginning of March 1973 the serum calcium was 9.4 mg/100 ml, phosphate 4.2 mg/100 ml, and alkaline phosphatase 11 K.A. units; the 24-hour urinary calcium was 5.5 mg/100 ml. He was discharged home in March well recovered mentally able to walk steadily and to carry out a lucid conversation, and with reasonable memory for recent and past events; he showed a keen interest in his calcium metabolism.

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Comment

The clinical features of tetany, convulsions, laryngeal spasm, Parkinsonian tremor, mental changes, irritability, and cataracts all suggested the diagnosis of hypoparathyroidism. This was confirmed by the low calcium and raised phosphate levels. The alkaline phosphatase was mildly raised initially but was subsequently normal, and x-ray examination showed no evidence of rickets or osteomalacia. The blood urea was 62 mg/100 ml but there was no evidence of chronic renal failure. The initial faecal fat collection was a little high but hypocalcaemia may cause steatorrhoea (Harrison, 1970), and after the serum calcium had returned to normal the faecal fat was well within physiological limits. The patient was therefore thought to be suffering from idiopathic hypoparathyroidism. Additional evidence in support of this was the failure to detect any parathyroid hormone.

The hypocalcaemic type of cataract is said to be slightly different from the usual senile type (Kopin and Rosenberg, 1960). In most reports, however, as in this case, this was not recognized at the time of cataract extraction, and it is suggested that routine estimation of the serum calcium on all patients with cataract would disclose a larger number of hypocalcaemic patients in the older age groups. A similar search among irritable or demented patients might also show a greater incidence of hypocalcaemia. Mental changes are recorded in up to 25% of patients with idiopathic hypoparathyroidism (Bronsky *et al.*, 1958; Aurbach and Potts, 1964). These changes include anxiety, depression, increased irritability, psychoses, hallucinations, delusions, mental dulling, and dementia (de Mowbray *et al.*, 1954; Robinson *et al.*, 1954; Treusch, 1957).

The aetiology of the hypoparathyroidism in this case was not clear. There were no grounds for suspecting an autoimmune

process and no signs of Addison's disease or pernicious anaemia were evident (Hung *et al.*, 1963; Blizzard *et al.*, 1966). Antibodies to parathyroid, thyroid, adrenal, and gastric mucosa were not detected.

It is suggested that in the elderly idiopathic hypoparathyroidism may be either a primary degenerative process or secondary to arteriosclerosis and diminished blood supply and that it is more common than is at present suspected.

I am grateful to Dr. Kenneth MacLean, under whose care the patient was admitted, for permission to make this report. I should like to thank him and also Professor Morris Lessof for their help in the preparation of this paper, and Dr. R. Buckle for the parathyroid hormone assay.

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Cerebrovascular Moyamoya Disease

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"Moyamoya" disease was first described in the Japanese literature between 1956 and 1963 and later in the Western literature by Kudo (1965, 1968) and Nishimoto and Takeuchi (1968). It is characterized by bilateral intracranial internal carotid artery occlusion of unknown aetiology, non-filling of the circle of Willis, and development of collaterals including a carotid rete (Kudo, 1968). The first cases described were all in Japanese patients resident in Japan or America (Leeds and Abbott, 1965). It has now been reported in patients from America (Taveras, 1969; Solomon *et al.*, 1970) and Europe (Simon *et al.*, 1968; Lepoire *et al.*, 1969; Galligioni *et al.*, 1971). In 1967 Hawkins and Scott described a bilateral carotid rete and hypoplasia of the internal carotid arteries in an English patient. The report on the cerebral angiograms suggested that this patient might have had moyamoya disease. This condition has been recognized in British patients (Bickerstaff, 1972) but to our knowledge no case histories have been published.

We present two cases in which air encephalography showed gross cerebral atrophy; this is a new finding.

Case 1

A 23-year-old white British woman was well until the age of 13, when she developed numbness of the left side of her body and occasional weakness of the left hand. This persisted intermittently for two years. She was able to attend a normal school, however, and after leaving at the age of 15 worked as a punch-card operator, machine operator, and shipping clerk.

In December 1970, when 10 weeks pregnant, she fell downstairs without injury to her head, and two days later her first convulsion occurred. The fits were repeated and characterized by jerky movements of the left arm and occasionally the left leg. These attacks were thought to be focal in origin. They settled in six weeks after treatment with diazepam but recurred four weeks after the birth of her child and again in October 1971, associated this time with left-sided weakness. For the first time she developed headaches; these were right-sided, intermittent, and very severe, causing her to lie down until they improved, which was usually within one to six hours.

In December 1971 she was admitted for investigation. Examination showed a left homonymous hemionopia, slight left-sided pyramidal weakness, left-sided astereognosis, and loss of two-point discrimination. Wechsler adult intelligence tests showed full-scale I.Q. 87, verbal scale I.Q. 102, and performance scale I.Q. 70. Brachial artery pressure as measured by Doppler was 116 mm Hg, and supraorbital artery pressure was reduced at 44 mm Hg for both right and left arteries. Lumbar puncture showed nothing abnormal. E.E.G. showed flattening of all activity over the right hemisphere, and a brain scan showed increased uptake in the right posterior parietal and occipital regions. Four-vessel cerebral angiography was performed (figs. 1 and 2). There was bilateral occlusion of the internal carotid artery at the level of the syphon and of the middle, anterior, and posterior cerebral arteries. The anterior cere-

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