sodium concentration reaches 130 mmol/l. In a study of seven patients Ayus *et al* used 3% hypertonic saline to increase the serum sodium concentration by 2.4 mmol/l/h without morbidity or mortality.¹⁴ In patients with hyponatraemic seizures, however, we thought that an acute osmolar gradient at the beginning of treatment, to reduce cerebral oedema and control seizures rapidly, may also be necessary to reduce the incidence of irreversible neuronal damage. In our study all the patients survived, comparing favourably with the 50% mortality previously recorded,³ but only four patients recovered completely. Although we thought that the prolonged episode of status epilepticus and hypotension that had occurred before treatment was responsible for the persistent neurological defect in the patient in case 2, we could not exclude an adverse effect of treatment as a possible cause.

In the five cases reported we thought that water intoxication from excess intravenous and oral fluids was the main cause of the hyponatraemia. With the initial infusion of 50 ml of 29.2% saline the mean serum sodium concentration increased from 105 (SD 4.8) mmol/l to 112 (4.64) mmol/l (table I). The increases in serum sodium concentration and osmolality were 7.4 (1.14) mmol/l and 11.8 (2.77) mmol/kg, respectively, and the reduction in serum potassium concentration was 0.62 (0.5) mmol/l. The fall in serum potassium concentration may have been caused by an increase in intracellular sodium, increasing the activity of the sodium-potassium adenosine triphosphatase.¹⁸ The total amount of sodium administered was 790 (139) mmol, which increased the serum sodium concentration to 133 (2.35) mmol/l over 10 (2) hours. The rise in serum sodium concentration after the initial bolus of 29.2% saline was given was 2.14 mmol/l/h (table II), which was only slightly lower than the 2.4 mmol/l/h reported by Ayus *et al*.¹⁴ The rapid administration of 500 mmol did not produce any discernible untoward clinical effects, which was not unexpected as similar

osmotic amounts of mannitol and radiological contrast media have been rapidly infused in patients without complication.^{8,19}

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SHORT REPORTS

Dietary practices of Asian diabetics

There is a widespread belief that Asian diabetics in Britain already follow a diet with a high carbohydrate, high fibre, and low fat content, which might be expected to help protect against diabetes and vascular disease. We investigated the dietary practices of a group of Asian diabetics living in the London Borough of Brent.

Patients, methods, and results

About 30% of patients attending this hospital's diabetic clinic are of Asian origin. About 70% of these are Gujarati, who have come either directly from India or from east Africa and are mostly Hindus. The remainder come from the Punjab and Pakistan, and many are Moslems. The Asian population covers all socioeconomic groups; 62% of men and 32% of women speak some English, usually as a second language. We randomly selected 48 patients (29 men and 19 women). Nine were treated with insulin and 39 by diet alone or with oral hypoglycaemic agents. Their mean age (range) was 53 (20-74) years, duration of diabetes 8 (2-23) years, and time in the United Kingdom 16 (2-36) years; in most cases the diagnosis had been made in the United Kingdom. The mean (SEM) body mass index was 25.1 (0.6) kg/m² in the men and 27.4 (0.6) kg/m² in the women.

A detailed dietary questionnaire was administered in the mother tongue by a nutritionist (JTD) during a home visit to each patient. Questions covered frequency of food consumption, methods of preparing and cooking food, and a 24 hour recall. Sizes of portions and daily intakes were calculated with the aid of food models and samples, graduated containers, and weighing scales. Any missing data were collected during a second interview in the clinic. Nutrient intakes were calculated from food composition tables.^{1,2}

The basic traditional diet followed by most patients consisted of chapatis (made with 85% wheat flour), white rice, vegetables, pulses, beans, and fruit. Six men and 12 women were vegetarian, all being Hindus. The table gives an analysis of the diets. The high fat intake was accounted for mainly by ghee (clarified butter) and cooking oil. Most patients regularly ate fried snack foods such as chevda, ganthia, crisps, and nuts. Thirteen consumed more than one pint of whole milk daily. Intake of fibre is not reported as not all the relevant figures for Asian dishes are available in food tables, but it was unlikely to have been much higher than the national average for the United Kingdom. Rich sources of soluble fibre

such as beans and lentils were consumed in only small amounts and were eaten daily by 20 patients. Chapati flour was the major source of cereal fibre; other sources such as bread and breakfast cereals were not popular.

Patterns of food consumption

	Mean (range) energy intake (MJ)	% Of daily energy intake obtained from:		
		Carbohydrate*	Fat†	Protein
Men	10.7 (5.1-17.3)	41	46	12
Women	6.8 (4.0-11.4)	43	45	11
Both	9.2	42	46	12

*Numbers of diabetics who regularly (that is, more than five times a week) ate various carbohydrates were: Asian sweets 11; confectionery 19; sweet biscuits and cakes 33; fizzy drinks 20; gur (unrefined sugar) 16; bread 11; and breakfast cereals 10.

†Numbers of diabetics who regularly ate various fats were: ghee 46 (those from Gujarat ate an average (range) of 245 (40-500) g/week; those from Kutch, a part of Gujarat, 424 (140-950) g/week; and others 188 (0-780) g/week; butter 36 (13 ate >120 g/week, 23 ate <120 g/week); unsaturated oil (<120 g/week) 15; and corn oil (<500 g/week) 21 and groundnut oil (<500 g/week) 13.

Conversion: SI to traditional units—Energy: 1 MJ = 239 kcal.

Comment

This is the first detailed study of the diets of Asian diabetics in this country. It highlights the discrepancies between their dietary practices and current recommendations for diabetics.³ Previous studies in India suggested that Asian diabetics there follow a diet close to these recommendations.^{4,5} We found, however, that most Asian diabetics followed a diet with a low carbohydrate and high fat content and consumed sugar and refined carbohydrates regularly. There was widespread use of dairy products such as milk, ghee, and yoghurt, which are rich in saturated fats; this applied regardless of the patients' regional origins.

Diet has a central role in the management of diabetes. Many health workers lack the basic knowledge about the diets of Asians on which to base practical advice, and little educational material is available. There is a

pressing need for culturally acceptable educational aids that recognise the regional differences between Asian communities. Such aids should encourage a reduced consumption of refined carbohydrates and saturated fats and an increased consumption of unrefined carbohydrates rich in fibre.

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Neuroleptic malignant syndrome associated with inappropriate antidiuresis and psychogenic polydipsia

The neuroleptic malignant syndrome is a life threatening complication of major tranquillisers that is probably underdiagnosed. The factors that precipitate it are not clear. I report a case in which on two occasions hyponatraemia was initially thought to be responsible for the neurological symptoms and signs. The hyponatraemia was later shown to be due to inappropriate antidiuresis, probably associated with psychogenic polydipsia, and this may have precipitated the syndrome.

Case report

The patient was a 41 year old man who for many years had been treated with depot injections of flupenthixol and oral orphenadrine for chronic schizophrenia. He did not take any other drugs but smoked 60 cigarettes daily and drank at least 15 litres of fluid daily, mostly in the form of instant coffee. In August 1984 he was found unconscious at home. He was admitted to hospital semiconscious and found to have greatly increased muscle tone. He was clinically normovolaemic and feverish (39°C), and blood pressure fluctuated between 100/60 and 170/120 mm Hg. Dipstick testing of the urine gave a ++ reaction for blood. White cell count was $28.3 \times 10^9/l$; serum sodium concentration 115 mmol(mEq)/l; serum calcium 1.97 mmol/l (7.9 mg/100 ml); serum aspartate transaminase 830 IU/l; serum creatine phosphokinase 2438 U/l; measured serum osmolality 251 mosmol/kg; calculated serum osmolality 250 mmol/l; urine osmolality 317 mosmol/kg; and urine sodium 35 mmol/l. His condition was complicated by hypoxia (thought to be due to decreased compliance of the chest wall), oesophagitis, and acute renal failure (thought to be due to acute rhabdomyolysis) that necessitated temporary peritoneal dialysis. After recovery of normal renal function the serum sodium concentration remained normal, even when he resumed his previous intake of fluids on discharge. Depot injections of flupenthixol were continued.

Eight months later he was readmitted after collapsing at home. On admission he was agitated, dysarthric, and feverish with increased muscle tone, hyperreflexia, and a decreasing level of consciousness. He was clinically normovolaemic, but blood pressure again fluctuated between 120/80 and 240/140 mm Hg. On admission serum sodium concentration was 118 mmol/l; serum osmolality 249 mosmol/kg; urine osmolality 421 mosmol/kg; and urine sodium 123 mmol/l. There was no evidence of rhabdomyolysis, and renal function remained normal. His serum sodium concentration and conscious level returned to normal over three days. One week later excretion of a water load was normal. Thyroid function tests gave normal results, as did a Synacthen test and insulin stress test.

Comment

The neuroleptic malignant syndrome is an idiosyncratic reaction to neuroleptic drugs characterised by muscular rigidity, hyperthermia, auto-

nomic instability, and altered consciousness and is often complicated by aspiration pneumonia, oesophagitis, and acute renal failure. The syndrome bears some similarities to malignant hyperthermia but is probably underdiagnosed. Treatment is supportive, although there is limited evidence of benefit from dantrolene and dopaminergic agents.¹ The syndrome may develop after years of uneventful treatment with a neuroleptic, and the factors that precipitate it are not known but are thought to include infection and dehydration. Hyponatraemia was reported as the possible precipitant in one case.² The fact that the neuroleptic drug may be reintroduced after recovery from the syndrome without causing a further episode indicates that such trigger factors are important.

In our patient the syndrome of inappropriate antidiuresis may have acted as the trigger factor on two occasions. This in turn may have been caused by psychogenic polydipsia,³ although a direct effect of fluphenazine on release of antidiuretic hormone cannot be discounted.⁴ Antidiuresis of rapid onset with rapid recovery after a period of enforced fluid deprivation has been reported in patients with psychogenic polydipsia,⁵ although the mechanism remains unknown. Having seen another patient with hyponatraemia and the neuroleptic malignant syndrome previously, I believe that the syndrome goes unrecognised because the presenting features are attributed to hyponatraemia alone. Accurate diagnosis is important as it would allow appropriate supportive measures to be undertaken and complications to be anticipated.

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Exceptional case of survival in cold water

People immersed after shipwreck in water colder than 6°C usually die of hypothermia within 75 minutes.¹ We know of no first hand account of survival for many hours in such water, though there is an anecdotal account.² In laboratory experiments thick subcutaneous fat allowed volunteers to maintain body temperature for a time in water at 5°C, but after 30 minutes cold vasodilatation caused them to cool progressively.³ We report investigations on a man who survived prolonged immersion in water at 5-6°C.

Case history and investigations

The subject, a 23 year old Iclander, had been fishing off Iceland when his boat capsized at about 10 pm on 11 March 1984. Water temperature was 5-6°C and air temperature -2°C. He and two out of four companions climbed on to the keel but after about 45 minutes swam towards shore, about 5 km (3 miles) away. His two remaining companions disappeared within 10 minutes. Wearing shirt, sweater, and jeans, he swam five to six hours to shore. He remained clear headed throughout.

EXPERIMENTAL IMMERSION

The following experiment was carried out 17 months after the accident. The subject was immersed to the neck for 83 minutes in thermostatically controlled water at 5.3-5.4°C,⁴ air 21°C, clothed as during his swim. He exercised arms and legs to mimic the swim, with water driven backwards past him at 500 mm/s. Metabolic rate, temperatures, heat flows, and fat thickness were measured as described.⁴

Results—The subject was a large man, with a height of 1.93 m, weight 125 kg, and surface area 2.54 m². Subcutaneous fat thicknesses were biceps 9 mm, thigh 10 mm, abdomen 26 mm, and subcostal 25 mm, giving an overall mean subcutaneous fat thickness of 14 mm²; at maximum width of forearm (flexor