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Intoxicated by Water

Polydipsia and Water Intoxication in a Mental Handicap Hospital

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A cross-sectional survey of the drinking habits of 877 mentally handicapped in-patients revealed 31 patients (prevalence 3.5%) who, in the opinion of nurses, drank five litres or more daily. Low urine specific gravity was a less useful indicator of polydipsia. Polydipsia appeared to be significantly associated with a borderline level of handicap and with a diagnosis of schizophrenia, autism or severe personality/behaviour disorder. Of five cases of water intoxication associated with polydipsia, one was fatal. In two cases excess drinking improved with increased neuroleptic medication. Lithium and demeclocycline were used in two cases to prevent hyponatraemic episodes.

Primary polydipsia or compulsive water drinking is a syndrome in which a person drinks large amounts of water for obscure reasons unrelated to normal thirst or homeostatic needs (Raskind & Barnes, 1984). The normal adult can excrete 15 litres of water a day, but if intake temporarily exceeds this rate (e.g. 3 or 4 litres drunk over 20 minutes) retention can occur. There may also be abnormal physiological mechanisms which lead to water retention, including the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) (Bartter & Schwarz, 1967). Rapid development of hyponatraemia and hypo-osmolality leads to water intoxication, an encephalopathy presenting as restlessness, confusion, lethargy, nausea, diarrhoea, vomiting, muscle twitching, ataxia, convulsions, seizures, stupor, coma and occasionally death (Rowntree, 1923).

Primary polydipsia has been associated with a variety of psychiatric diagnoses and thought to be relatively benign until the 1970s. It was first recognised in schizophrenia (Sleeper & Jellinek, 1936; Barahal, 1938) and later associated with neurosis and personality disorder (Barlow & De Wardener, 1959) and organic brain syndromes (Bewley, 1964; Stevko *et al*, 1968). Reports were published of the association of water intoxication with acute psychosis, both schizophrenic (Fowler *et al*, 1977; Rosenbaum *et al*, 1979; Kramer & Drake, 1983) and affective (Raskind *et al*, 1975; Zubenko *et al*, 1984).

In recent years evidence was accumulated for a variety of long-term complications of chronic polydipsia. As well as recurrent episodes of water intoxication, these include bowel and bladder dilatation (Viewig *et al*, 1984), hydronephrosis, renal failure, malnutrition, projectile vomiting, cardiac failure and dementia (Blum *et al*, 1983), hernias and incontinence (Delva & Crammer, 1988), and spinal fractures secondary to calcium loss (Delva *et al*, 1989).

Although polydipsia and water intoxication have been studied in psychiatric populations, there have been no systematic surveys in mentally handicapped populations and few case reports (Mendelson & Deza, 1976; Noonan & Anath, 1977; Jose & Perez-Cruet, 1979). We describe a cross-sectional survey of polydipsia in mentally handicapped patients followed by case histories and management of water-intoxicated patients.

Method

The population comprises the 877 in-patients of a large Victorian hospital on the outskirts of north London. All have been diagnosed as mentally handicapped with a range of ability from profound handicap to borderline average intelligence. Included in the population is the regional secure unit for mentally handicapped patients. Patients over 70-years-old comprise 20% of the population and the proportion of males in this group is 37%. Of those aged under 70 years, males comprise 55% of the population.

The sample is defined as those patients who were estimated by staff to drink five litres or more per day (measured in cups) and/or who went to extraordinary lengths to drink fluid. The latter clause is to include those patients who had limited access to water, but who would exploit any opportunity (e.g. drinking avidly from bath or urinal) so that the amount ingested was very hard to estimate. The whole hospital was screened for urinary specific gravity to identify those with a specific gravity of less than 1.008 on three occasions and to determine the usefulness of this measure as an aid to detecting polydipsic patients.

Exclusion criteria were renal disease, diabetes mellitus, diabetes insipidus, and use of diuretics or lithium. An early-morning urinary osmolality of greater than 300 mosm/kg

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with a serum sodium of less than 148 mmol/l was taken to exclude diabetes insipidus. Patient consent or written consent of the nearest relative was obtained for the following investigations: serum urea and electrolytes, glucose, creatinine, osmolality, early morning urinary sodium and osmolality and diurnal weighing (08.00 and 20.00 h).

A group of non-polydipsic patients (non-drinkers) were matched with drinkers for ward, age, sex, weight and level of handicap, then had daily fluid intake assessed and were also weighed diurnally; more invasive investigations were not performed in this group for ethical reasons. We interviewed each polydipsic patient and a staff member who knew the patient well over a long period. We examined case notes to determine level of handicap, psychiatric, medical and family history, the course of the person's development or illness, the response to medication and current medication. We inquired about smoking, eating and behavioural habits. We made an assessment psychiatric diagnosis based on all information available. We assessed personality guided by a standardised personality assessment (Mann *et al*, 1981) which has been used previously for mentally handicapped patients (Ballinger & Reid, 1987). Where possible patients were also interviewed for their views on the reasons for their drinking so much. Statistical analysis was performed using the fourfold χ^2 test with Yates' correction for continuity.

With respect to water intoxication, 'cases' were defined by neurological symptoms in association with low serum sodium and/or osmolality, and a history of recent polydipsia, with no other cause for the neurological symptoms or hyponatraemia being apparent.

Results

Of 877 patients we found that 31 (3.5%) frequently drank over five litres a day without an apparent physical cause, or went to extraordinary lengths to obtain water. Eight drinkers had been excluded on physical grounds (two with diabetes mellitus, five taking lithium, and one taking diuretics). Of the 31 patients, six had repeated low urinary specific gravity, but only one patient was identified by specific gravity alone and subsequently discovered to be drinking excessively. It was impossible to test the urine of 50 (6%) patients because of difficulties with patient co-operation or incontinence. Another 33 (4%) patients had a low specific gravity as a result of age (29), renal disease (2) or lithium (2).

There were 21 men and 10 women, whereas the sex ratio is almost 1:1 in the total hospital population ($\chi^2 = 3.02$, $P < 0.1$). The average age of drinkers was 42 (range 24–84 years), compared with an average age of the whole hospital population of 49 years. A significant association with borderline handicap was found, whereas absence of polydipsia was found to be associated with severe mental handicap ($\chi^2 =$ 29.94 and 4.16, $P < 0.001$ and 0.05 respectively) (Table 1).

There was no drinker newly diagnosed as having diabetes mellitus or diabetes insipidus. Serum sodium of drinkers ranged from 127 to 140 mmol/l with a median of 135 mmol/l (normal range was 136–148 mmol/l). However, it should be borne in mind that blood samples were taken in the early morning when drinking behaviour would have least effect (although this enabled us to use the results to exclude diabetes insipidus). Serum creatinine of drinkers ranged from 35 to 78 μmol/l (normal range 60–120 μmol/l).

Table 1
Degree of mental handicap in polydipsic sample compared with the general hospital population

Level of handicap (ICD-9)	IQ	Drinkers ($n = 31$) no.	(%)	Hospital population ($n = 877$) no.	(%)
Borderline[1]	70–85	7	(22)	30	(3)
Mild	50–69	9	(29)	149	(17)
Moderate	35–49	6	(19)	201	(23)
Severe[2]	20–34	7	(22)	368	(42)
Profound	<20	2	(6)	75	(9)
Unspecified		0	(0)	54	(6)

1. $\chi^2 = 29.94$, d.f. = 1, $P < 0.001$.
2. $\chi^2 = 4.16$, d.f. = 1, $P < 0.05$.

Drinkers had an estimated daily fluid intake ranging from 5 to 16 litres (median 6 litres) whereas matched non-drinkers had a range of 1–3.5 litres (median 2 litres). The weight gain of drinkers over 12 hours ranged from none to 7.5 kg (median 1.5 kg) and of matched non-drinkers from none to 2.5 kg (median 0 kg). Twelve of 31 drinkers had a weight gain of over 2 kg compared with 3 of 31 non-drinkers ($\chi^2 = 5.63$, d.f. = 1, $P < 0.02$).

In comparing the psychiatric diagnoses on the hospital register of drinkers with non-drinkers, significant associations with polydipsia were found for schizophrenia ($\chi^2 = 7.32$, $P < 0.01$), severe personality/behaviour disorder ($\chi^2 = 6.73$, $P < 0.01$) and autism ($\chi^2 = 18.94$, $P < 0.001$). The

Table 2
Psychiatric diagnosis of the polydipsic sample compared with the rest of the hospital population

Psychiatric diagnosis	Drinkers ($n = 31$) Assessment diagnosis no.	(%)	Register diagnosis no.	(%)	Hospital population ($n = 877$) Register diagnosis no.	(%)
Schizophrenia[1]	10	(32)	8	(26)	87	(10)
Manic-depressive psychosis	1	(3)	1	(3)	7	(1)
Past psychosis	2	(6)	2	(6)	27	(3)
Neurosis	2	(6)	1	(3)	11	(1)
Personality/ behaviour disorder (severe)[2]	6	(19)	7	(23)	73	(8)
Autism[3]	6	(19)	5	(16)	22	(2)
Dementia	0	(0)	0	(0)	35	(4)
No psychiatric[4] diagnosis	4	(13)	7	(23)	615	(70)

1. $\chi^2 = 7.32$, d.f. = 1, $P < 0.01$.
2. $\chi^2 = 6.73$, d.f. = 1, $P < 0.01$.
3. $\chi^2 = 18.94$, d.f. = 1, $P < 0.001$.
4. $\chi^2 = 23.42$, d.f. = 1, $P < 0.001$.

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Table 3
Personality assessment in polydipsic patients

Personality type	Grade I[1]	Grade II[2]	Total (grade I/II) ($n=31$)
Self-conscious	1	0	1
Schizoid	2	0	2
Paranoid	0	1	1
Cyclothymic	4	0	4
Obsessional	5	0	5
Anxious	6	0	6
Asthenic	2	0	2
Sociopathic	0	1	1
Explosive	3	1	4
Hysterical	1	1	2
'Stable'	3	0	3

1. Grade I, personality can be matched by one of the types.
2. Grade II, personality unusual or handicapping.

validity of the register diagnosis was largely supported by our assessment diagnosis (Table 2). Three men had a history of sexual deviation and one woman had a history of bulimia and anorexia.

Personality types were variable, although cyclothymic, obsessional and anxious personalities were most common, and together accounted for almost half the sample. In those four patients with personality disorder who showed several personality types, the dominant type was recorded (grade II) (Table 3).

Of those 18 patients able to discuss their drinking, 12 admitted they drank a lot; only six said they were thirsty and six said they had a dry mouth. Of those 11 patients who could discuss a reason for drinking excessively, eight did not know why, two said they had a dry mouth, and one said it was a habit.

Of the 31 cases, 15 smoked cigarettes, 11 tended to bolt food quickly, five had pica, nine had repeated self-injury, eight had marked stereotypic movements, and six were generally overactive. Of medical diagnoses recorded, epilepsy was by far the most common and was noted in seven of the drinkers. It is interesting that none had Down's syndrome, although these comprise 8% of the hospital population.

Case histories of water intoxication

In a retrospective survey of the mental handicap population of 877 patients over three years, we knew of nine people who apparently developed water intoxication. Two were not polydipsic and had a clear underlying medical cause (carbamazepine in one case and cardiac failure with thiazide diuretics in the other). The other seven patients were polydipsic at the time of apparent water intoxication. Of these seven, three had chronic polydipsia and had their first episode of water intoxication when being treated with either carbamazepine or chlorpropamide. In the other four patients with episodic polydipsia, water intoxication was in each case associated with psychotic or disturbed episodes and all four were heavy smokers. Of the seven patients, two did not have sodium levels recorded. The five with documented hyponatraemia, satisfying the criteria of 'caseness', are described.

Case 1

A 58-year-old woman with a moderate mental handicap has a 20-year history of schizophrenia as evidenced by auditory hallucinations, incongruous affect, bizarre mannerisms and regression. Her excessive water drinking was noted at the age of 44 years and thought to be due to mild diabetes, which was managed by diet alone. Over the last 12 years she has had several episodes of water intoxication, once with a serum sodium as low as 116 mmol/l, but on each occasion the vomiting, fits and stupor were initially thought to be due to hypoglycaemia or a cerebrovascular accident. She was prescribed phenytoin following one episode described as atypical epilepsy. It is possible that she drinks in response to hallucinations, as nurses have noticed that in periods when she drinks excessively, she talks to an imaginary person and looks upwards for no obvious reason. Furthermore, this excessive drinking and mental disturbance decline with an increase in her maintenance oral haloperidol.

Case 2

A 53-year-old man has an IQ of 38 following brain damage from meningitis in infancy. There is a family history of affective disorder. Over 18 years he has had several clearcut episodes when he is apathetic, anorectic, sleepless, loses weight and wants to die. At other times he has periods when he is restless, garrulous, oblivious to all concerns, hoards rubbish and wants to wander off to sleep out of doors. He drinks tea copiously, smokes compulsively and self-inflicts skin injuries.

He has had two periods of acute confusion with hyponatraemia. The first episode (serum sodium 125 mmol/l) was attributed to thiazide diuretics, which were discontinued. The second occurred during a hypomanic episode, when he was also drinking tea excessively and had been treated with chlorpromazine (300–400 mg/day) over three weeks. On this occasion he wandered off and when found the next day, he was clinically dehydrated with the following biochemistry: serum sodium 122 mmol/l, potassium 3.3 mmol/l (normal range 3.5–5.5), bicarbonate 25 mmol/l (normal range 22–32), urea 10.6 mmol/l (normal range 2.5–6.5), creatinine 105 μmol/l (normal range 60–100), serum osmolality 264 mosm/kg (normal range 270–285), urine osmolality 581 mosm/kg (normal range 40–1400), urine sodium 268 mmol/l, urine potassium 50 mmol/l.

After hospital care his urea returned to normal but he remained hyponatraemic with normal chest X-ray, glucose tolerance test, adrenal-stimulating hormone test, porphyrins, thyroid function and computerised tomography (CT) scan. Chlorpromazine was initially assumed to be responsible for his hyponatraemia, and haloperidol (5 mg/day) was substituted, but his serum sodium remained at 125 mmol/l and he continued drinking excessively. When demeclocycline (300 mg q.d.s.) was added his sodium rose to 130 mmol/l.

With early treatment of his psychotic episodes and monitoring of his fluid intake, there have been no further

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episodes of confusion and his serum sodium is within normal limits.

Case 3

A 51-year-old menopausal woman with an IQ of 47 was deserted in early childhood by an unstable mother. She has a brother who is itinerant, aggressive and had repeated psychiatric admissions for unspecified psychosis and self-neglect. She is described as the ward bully and has always been touchy, disgruntled and explosive, and has a diagnosis of personality disorder. She has periods of self-injury and is always greedy for sweets and cigarettes. She has had several brief depressive episodes, one with marked hysterical features. Depot flupenthixol decanoate had been prescribed in increasing dosage for years with little behavioural change. As she had never been actively psychotic, the fortnightly intramuscular dose of flupenthixol decanoate was reduced over three months from 100 mg to 20 mg. The antidepressant lofepramine (140 mg/day) was continued over this time.

She was declared fit to go on holiday and that afternoon became confused, vomited, and had a swollen face and a distended abdomen. She then became rigid, cyanosed and comatose and was thought to have a myocardial infarct or a cerebrovascular accident. Investigations showed: serum sodium 118, potassium 3.3, bicarbonate 27, urea 3 mmol/l. Plasma osmolality rose from 252 to 298 mosm/kg, and urine osmolality rose from 153 to 467 mosm/kg with water restriction over 24 hours. She had normal lumbar puncture, electrocardiography (ECG), full blood count, calcium, thyroid function, liver-function test, skull X-ray and CT scan.

When questioned in detail later, staff recalled that she was always at the tap. The depot flupenthixol and lofepramine were suspended. Over the next ten months she had four further episodes of water intoxication with serum sodium levels ranging from 113 to 123 mmol/l. After fluid restriction and a return to normal serum sodium levels she would become depressed and suicidal and did not eat or smoke. She would then drink large volumes rapidly from the toilet and if prevented, became tense and persisted in anal poking and self-mutilation. The extent of her drinking was monitored by daily weighing and she often needed close supervision. With no response to oral antipsychotics and antidepressants, electroconvulsive therapy (ECT) was considered but her water seeking reduced dramatically when depot flupenthixol (40 mg i.m. fortnightly) was restored. Lithium (800 mg *nocte*) was also prescribed primarily for the effect of increasing free water clearance. She remains on lithium and flupenthixol decanoate with no further episodes of intoxication.

Case 4

A 29-year-old man had a normal birth and early milestones. At 18 months infantile convulsions developed and were followed by regression and loss of speech. The subsequent diagnosis of disintegrative psychosis and childhood autism was made. In adolescence he adopted rituals which centred on touching the breasts of women followed by repeated self-mutilation of his eyes. Cataracts developed and despite a series of operations he remained blind in one eye. He continued to produce striking but stereotyped paintings of eyes or severed heads but his speech remained at the four-year level.

At 23 he developed hypertension which was treated with nifedipine. Because of his high anxiety level he remained on antipsychotics with antidepressants when required. At 26 he developed his first episode of water intoxication when he drank large amounts from unusual places, and had hyponatraemia (121 mmol/l) which was thought to be exacerbated by thiazide diuretics and these were discontinued.

His second episode of hyponatraemia (116 mmol/l) was three years later and resolved within 24 hours with fluid restriction. Ten days later he was observed to be drinking water excessively again, then became abusive and tense for several days with the sudden onset of vomiting, diarrhoea and coma. His medication at the time was fluvoxamine (100 mg *nocte*), chlorpromazine (100 mg t.d.s.), atenolol (50 mg *mane*), and prazosin (2 mg t.d.s.). As several empty aftershave bottles were found nearby, self-poisoning was suspected but alcohol was not detected. Serum sodium was 126, potassium 3.2, bicarbonate 22, urea 2.2, and glucose 9.4 mmol/l. Soon after he had a respiratory arrest and became hypotensive. He was resuscitated and given 50 mmol of intravenous sodium bicarbonate and a two-litre infusion of normal saline overnight, but became hypernatraemic (151 mmol/l) with a flaccid tetraplegia. Central pontine myelinolysis (Laureno, 1983) was suspected. A CT scan showed diffuse cerebral oedema and obliteration of the ventricles. He died two weeks later and the post-mortem examination showed total infarction of the cerebral cortices, pons, medulla and cerebral hemispheres, thought to be due to anoxia following his respiratory arrest.

Case 5

A 41-year-old man has an IQ of 41 and a history of epilepsy from the age of two years, with aggressive outbursts and petty thieving from adolescence. When his mother died he was admitted to a centre for epilepsy, where he had confused episodes but no seizures. He began setting fires, usually under the influence of alcohol, and was detained in a mental handicap hospital for close supervision. With each attempt at a hostel rehabilitation programme, he became temperamental, threatened arson, chain smoked and carried a jar of coffee and powdered milk in his pocket to make drinks every five minutes. He had what appeared to be recurrent cerebrovascular episodes until it was clear that he was drinking water to the stage of unsteady gait and slurred speech.

Investigations showed a serum sodium level of 126 mmol/l with normal urea, creatinine, calcium, urinary sodium, fasting lipids, thyroid function, glucose tolerance test and chest X-ray. As he has had no epileptic fits for five years and no other physical cause has been found for his hyponatraemia, carbamazepine is being gradually suspended, and his fluid and cigarette consumption is supervised.

Discussion

Our findings suggest that polydipsia and water intoxication are not uncommon among mentally handicapped patients in long-term hospital care. The

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prevalence rate for polydipsia of 3.5% compares with a range of 6.6% in 239 patients (Jose & Perez-Cruet, 1979) to 17.5% in 241 patients (Blum *et al*, 1983) found in surveys of American state psychiatric institutions. Our lower rate may reflect an association with mental illness and the fact that not all mentally handicapped patients are mentally ill, or it may reflect the higher level of drinking required to be included as polydipsic in our study. Jose & Perez-Cruet defined polydipsia as simply excessive drinking based on nurses' and patients' estimates, and did not state whether medical causes had been excluded. Blum used a subjective assessment to detect those drinking more than four litres a day with or without low urine specific gravity and excluded physical causes.

In view of the difficulties of obtaining satisfactory measures of drinking, the difference in prevalence rates is not surprising. We found that screening patients for a low specific gravity was not a sufficiently valid method for detecting polydipsia and added little to ordinary nursing observation. For those patients needing close supervision of their drinking, weighing appeared a useful non-invasive guide to water retention and hyponatraemia (Delva & Crammer, 1988), as in our case 3.

The group found to be polydipsic were heterogeneous in terms of psychiatric diagnosis and personality. Individually significant associations were found with schizophrenia, autism, and personality disorder. These associations were not predicted, appearing only in a *post hoc* analysis, therefore repetition of these findings is necessary before they can be accepted as proven. Despite this qualification, the association with autism appears highly significant. It should also be noted that having excluded patients taking lithium from our study on the grounds of its effects on water excretion, we have reduced the likelihood of finding polydipsic patients with affective disorder. In psychiatric patients, water intoxication has been found to be associated with psychosis in 80–85% of cases (Ferrier, 1985; Zubenko, 1987) whereas case descriptions of patients found by physicians to have compulsive polydipsia suggest polydipsia is more evenly spread among psychiatric diagnostic groups (Barlow & De Wardener, 1959; Chinn, 1974; Singh *et al*, 1985).

The sex ratio is the inverse of that expected from previous studies in psychiatric patients: 68% of drinkers were found to be male in our study, as opposed to 78% female (Barlow & De Wardener, 1959) and 67% female (Jose & Perez-Cruet, 1979). In our total hospital population, 55% of in-patients with psychosis are male and 66% of in-patients with autism are male. As polydipsia appears to be associated with autism and psychosis, this may explain the reversed gender trend.

The findings relating to pica and bolting of food were unfortunately uncontrolled, but in themselves are suggestive that, at least in some patients, the polydipsia being observed may be part of a general tendency to ingest.

The clinical significance of polydipsia is highlighted by the case histories of patients with water intoxication. One of the five cases was fatal. This compares with a finding in psychiatric patients that water intoxication is associated with a mortality of 10% over two years (Hariprasad *et al*, 1980). Each of our five cases involved initial misdiagnosis. Diagnosis may be especially difficult in mentally handicapped patients among whom epilepsy is common, as a frequent presentation of water intoxication is a generalised seizure (Singh *et al*, 1985). In the event of late-onset seizures, especially if the patient has recently been more disturbed, it is advisable to obtain a drinking history, and check electrolytes and osmolalities. Anticonvulsants may not be needed, and carbamazepine may compound the problem.

We found no case of water intoxication to be attributable to antipsychotic or antidepressant medication and in two cases (1 and 3), increasing antipsychotic medication appeared to lead to a reduction in water drinking and an end to episodes of water intoxication. It is interesting that Shen & Sata (1983) also observed that a number of episodes of water intoxication occurred among polydipsic patients when their neuroleptic dosage was decreased. Although psychotropic medication is associated with SIADH, so is psychosis, and as Sandifer (1983) points out, psychotropic medication is often falsely blamed for SIADH. He recommends that the causative role of medication should be documented by a rechallenge or by a water-loading test. If a patient develops water intoxication on a newly prescribed (within one month) psychotropic medication, advice is to check sodium, osmolality and ADH levels, and if other physical causes are excluded, to change the drug with a later rechallenge. However, if such a person is drinking excessively, the most probable cause of both water intoxication and polydipsia is inadequately treated psychosis.

If the hyponatraemia cannot be controlled, we would consider adding demeclocycline (Forrest *et al*, 1978; Nixon & Chin, 1983; Sandifer, 1983; Goldman & Luchins, 1985) or lithium (White & Fetner, 1975) to promote free water clearance (cases 2 and 3). Both demeclocycline and lithium act as ADH antagonists at the level of the renal tubule, causing nephrogenic diabetes insipidus and free water excretion (Shrier, 1985). On the other hand, carbamazepine (Perucca *et al*, 1978), thiazide

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diuretics (Raskind, 1974; Fowler *et al*, 1977) and chlorpropamide (Smith & Clark, 1980) all potentiate ADH by renal or central mechanisms (Shrier, 1986) and can exacerbate hyponatraemia (cases 2, 4 and 5). Hence it seems wise to avoid these medications in polydipsic patients. In addition, nicotine, and thus smoking, induce inappropriate ADH secretion: we therefore followed the advice of Chin *et al* (1976) in restricting cigarettes in those patients with repeated low sodium levels.

In chronic and episodic drinkers, close chaperoning and water restriction should be carried out if serum sodium levels fall below 127 mmol/l, or when the person gains 7% of his morning body weight by the evening (Delva & Crammer, 1988).

There have been many hypotheses advanced to explain the occurrence of polydipsia and the current study does no more than many others to support or refute these explanations. However, our clinical observation in conducting this study suggests that many of the most polydipsic patients were particularly anxious while exhibiting their polydipsic behaviour. If polydipsia is an idiosyncratic response to anxiety, this may explain its association with a variety of psychiatric illnesses. Lee *et al* (1989) have proffered the hypothesis that it is secondary hyponatraemia that is anxiolytic and described a drinker who regularly induced a pleasurable state of altered consciousness. However true this may be of some patients, it cannot explain the occurrence of polydipsia in patients who do not become hyponatraemic. Other explanations have been in terms of family modelling (Kulkarnia *et al*, 1985), and various neurochemical explanations of water drinking to the point of intoxication, including a possible hypothalamic defect (Hobson & English, 1963), a primary inappropriate secretion of ADH (Khamnei, 1984), a reset osmostat (Hariprasad *et al*, 1980; Zerbe *et al*, 1980; Goldman *et al*, 1988) and a hyperdopaminergic state (Smith & Clark, 1980; Illowsky & Kirch, 1988).

We suggest a general explanation of these phenomena might run as follows:

(a) Polydipsia may be viewed as a learned behaviour, which depends on:
  (i) availability of fluid to drink
  (ii) a persistent anxiety, perhaps relating to feelings of emptiness
  (iii) a sense of partial and temporary relief gained from the physical sensation of drinking
  (iv) a lack of other, more effective methods (available to the patient) of relieving this anxiety.

This explanation is not necessarily incompatible with a neurochemical theory of polydipsia.

(b) Water intoxication may occur either when polydipsia is so severe that renal water excretion is overwhelmed, or in conjunction with SIADH, reset osmostat, or drug effects, all of which may be associated with psychosis.

Clearly, further work is required to unravel the basis of these troubling phenomena which may then lead to more effective ways of managing polydipsia and water intoxication.

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