kg), after excluding glycosuria, mannitol administration, or renal failure.^{7,13} The plasma sodium concentration may be a more reliable guide than osmolality in the acute phase. Thus in adults a total 24 hour output of over 3.5 litres of hypotonic urine together with a plasma sodium concentration of more than 143 mmol/l establishes the diagnosis.

Only once the diagnosis of cranial diabetes insipidus has been confirmed should antidiuretic agents be used as water overload because of inappropriate treatment may cause cerebral oedema and convulsions. Desmopressin (DDAVP), 1-2 µg subcutaneously or intramuscularly (0.4 µg in children), usually gives antidiuresis for 12-24 hours and should be accompanied by sufficient fluid intake to maintain the fluid balance. Occasionally, larger doses of desmopressin are needed during the first day, perhaps to overcome receptor blockade by inactive antidiuretic hormone precursors.¹³ Antidiuretic hormone itself (Pitressin), which has vasoconstrictor effects (not manifested by its selective renal analogue desmopressin), should not be used. Chlorpropamide and carbamazepine are usually ineffective and have unwanted actions.

Both overhydration and dehydration are risks in patients with coma or with defective thirst due to hypothalamic dysfunction. Repeat doses of desmopressin should be administered only if polyuria recurs so that water overload is avoided. Initially, the plasma sodium concentration should be measured at least daily, and then if the cranial diabetes insipidus remits (particularly after suprasellar surgery) it should be monitored over 14 days to detect the syndrome of inappropriate antidiuresis or recurrent cranial diabetes insipidus.^{7,16} The syndrome is managed by water restriction until the likely relapse of cranial diabetes insipidus occurs.

Long term cranial diabetes insipidus is treated with intranasal desmopressin; the metered dose spray is useful, particularly in patients with impaired vision. Patients who do not have thirst sensation require desmopressin in a dose tailored to a fixed water intake, which can be changed to allow for

increased drinking during exercise or exposure to heat. Thus a sensible interpretation of water balance and the electrolyte data is the key to an accurate management of postoperative cranial diabetes insipidus.

JONATHAN SECKL

Lecturer in Medicine,
University of Edinburgh,
Western General Hospital,
Edinburgh EH4 2XU

DAVID DUNGER

Consultant Paediatrician,
John Radcliffe Hospital,
Oxford OX3 9DU

- Robertson GL. The regulation of vasopressin function in health and disease. *Recent Prog Horm Res* 1977;33:333-74.
- Lipsett MB, Maclean JP, West CD, Li MC, Pearson OH. An analysis of the polyuria induced by hypophysectomy in man. *J Clin Endocrinol Metab* 1956;16:183-95.
- Randall MB, Clark EC, Dodge HW, Love JG. Polyuria after operation for tumors in the region of the hypophysis and hypothalamus. *J Clin Endocrinol Metab* 1966;20:1614-21.
- Sharkey PC, Perry JH, Ehgri G. Diabetes insipidus following section of hypophyseal stalk. *J Neurosurg* 1961;18:445-60.
- Ikkos D, Luft R, Olivercrona H. Hypophysectomy in man: effect on water excretion during the first two postoperative months. *J Clin Endocrinol Metab* 1955;15:553-67.
- MacCubbin DA, VanBuren JM. A quantitative evaluation of hypothalamic degeneration and its relation to diabetes insipidus following interruption of the human hypophyseal stalk. *Brain* 1963;86:443-64.
- Verbalis JG, Robinson AG, Moses AM. Postoperative and posttraumatic DI. In: Czernichow P, Robinson AG, eds. *Diabetes insipidus in man. Frontiers of hormone research. Vol 13*. Basle: Karger, 1985:247-65.
- Moses AM. Clinical and laboratory observations in the adults with DI and related syndromes. In: Czernichow P, Robinson AG, eds. *Diabetes insipidus in man. Frontiers of hormone research. Vol 13*. Basle: Karger, 1985:156-75.
- Thomsett MJ, Conte FA, Kaplan SL, Grumbach MM. Endocrine and neurologic outcome in childhood craniopharyngioma: review of effect of treatment in 42 patients. *J Pediatr* 1980;97:728-35.
- Newman CB, Levine LS, New MI. Endocrine function in children with intrasellar and suprasellar neoplasms: before and after therapy. *Am J Dis Child* 1981;135:259-62.
- Lyon KR, Grant DB. Endocrine function, morbidity, and mortality after surgery for craniopharyngioma. *Arch Dis Child* 1982;57:837-41.
- Hans P, Stevenaert A, Albert A. Study of hypotonic polyuria after trans-sphenoidal pituitary adenomectomy. *Intensive Care Med* 1986;12:95-9.
- Seckl JR, Dunger DB, Lightman SL. Neurohypophyseal function during early postoperative diabetes insipidus. *Brain* 1987;110:737-46.
- Hollinshead WH. The interphase of diabetes insipidus. *Mayo Clin Proc* 1964;39:92-100.
- Laszlo FA, De Wied D. Antidiuretic hormone content of the hypothalamo-neurohypophyseal system and urinary excretion of antidiuretic hormone in rats during the development of diabetes insipidus after lesions in the pituitary stalk. *J Endocrinol* 1966;36:125-37.
- Cusick JF, Hagen TC, Findling JW. Inappropriate secretion of antidiuretic hormone after transsphenoidal surgery for pituitary tumors. *N Engl J Med* 1984;311:36-8.

Delirium tremens

Try to spot it early

When he coined the term delirium tremens in 1813 Thomas Sutton cannot have known that it would enter the vernacular.¹ But lay usage dilutes meaning, and for some people today the "DTs" is merely the shakiness and transient perceptual illusions that may be experienced by a person who suddenly stops heavy drinking. But delirium tremens proper is a dangerous state of disorientation with hallucinations in which terrified patients have leapt from windows and severe autonomic overactivity has led to fatal cardiac arrhythmias.

At present deaths among series of patients with delirium tremens from hospital have fallen to less than 5%—for example, Cushman²—and almost always concurrent illnesses are implicated. A recent report from Sweden of a 15 year follow up of 44 patients who had had an episode of delirium tremens found that 11 had made an apparently stable recovery from their alcoholism.³

The studies of Isbell *et al* and Mendelson *et al* established that the syndrome is caused by withdrawal from sustained heavy drinking.^{4,6} Thiamine deficiency and malnutrition are not necessary factors, though the confusional state of Wernicke's syndrome may be provoked by alcohol withdrawal and perhaps by the first meal and compound the withdrawal

syndrome. Littleton suggests that the neuronal disturbance underlying the syndrome may be in the cell membrane⁷: the number of calcium channels may be increased by exposure to ethanol, leaving the cell hyperexcitable.⁸ Calcium channel blockers seem to reduce the withdrawal symptoms from alcohol.^{8,9}

The key to treatment is anticipation. The risk of delirium tremens begins at admitted alcohol consumptions as low as 80 g ethanol a day and upwards—that is, 10 units—but becomes appreciable only at 120 g a day and upwards—that is, half a 750 ml bottle of spirits or more a day, or the equivalent.¹⁰ Doctors should ask patients about their alcohol consumption and symptoms related to alcohol intake more often than they do at the moment and should seek corroboration from relatives. Alcohol on the breath, a raised mean cell volume, or raised serum activity of γ-glutamyltransferase are pointers to regular heavy drinking.⁹ A low serum concentration of potassium sometimes heralds the development of delirium tremens.^{11,12} A tense person, unable to sleep, with tremor of the tongue or fingers, sweaty palms, and a tachycardia may be in the prodromal phase: delirium may develop over the next day or so.¹³ In the stage before confusion develops the patient will readily accept sedation, and

admission to hospital may be avoided. If disorientation is allowed to develop or if the patient has not presented soon enough to prevent it referral to hospital will be necessary. Other diagnoses should be considered in the disoriented alcoholic patient, particularly subdural haematoma, pneumonia, and meningitis.

Nursing the patient in a well illuminated uncluttered environment helps to reduce disorientation. If associated physical illnesses permit delirious patients are best managed in psychiatric wards where they may be out of bed. Sometimes it helps to have a relative present. Sedation must be given quickly, with an adequate, sufficient loading dose: diazepam 50 mg or chlorthalidopoxide 100 mg might be started, followed by smaller hourly doses until agitation is controlled. Dosage is tailed off to zero over about the next four days. Patients in hospital have died from hypostatic pneumonia when cumulative sedatives are continued for too long; this is particularly likely to occur when sedatives such as chlor-methiazole are given by intravenous infusion, a method that may also cause death from respiratory depression.¹⁴ (Ethanol itself is less cumulative and therefore perhaps safer, and has been used in intensive care units in a dose of 30 ml initially followed by 10 ml every hour in 5% dextrose.) For severe agitation haloperidol or droperidol 10 mg intramuscularly may be needed, but the benzodiazepine should be continued for its anticonvulsant effects. A parenteral vitamin preparation containing thiamine should be given daily for about five days. Fluid should be replaced sparingly because alcoholics may have impaired ability to excrete water¹⁵ and a tendency to cerebral oedema.¹⁶

As the mental state improves the doctor has the opportunity to explain in a non-judgmental manner the link between the illness and the patient's drinking. In the Swedish study five of the 11 who made a good long term recovery from their alcoholism reported that the episode of delirium tremens had been a turning point.² Four of those who did well reported at

follow up that they were now drinking beer occasionally but never to excess, and their claims were supported by an informant and blood tests. They were all men who had sustained a close relationship. Most importantly, however, the period between moving out of problem drinking and resuming limited drinking was punctuated by some three to five years of abstinence; two had achieved this with disulfiram. The doctor's advice to patients recovering from delirium tremens must be abstinence.

JONATHAN CHICK

Consultant Psychiatrist,
University Department of Psychiatry,
Royal Edinburgh Hospital,
Edinburgh EH10 5HF

- 1 Sutton T. *Tracts on delirium tremens, on peritonitis and on some other internal inflammatory affections, and on the gout*. London: Thomas Underwood, 1813.
- 2 Cushman P. Delirium tremens—update on an old disorder. *Postgrad Med* 1987;82:117-22.
- 3 Nordstrom G, Berglund M. Delirium tremens: a prospective long-term follow-up study. *J Stud Alcohol* 1988;49:178-85.
- 4 Isbell H, Fraser HF, Wikler A, Belleville RE, Eisenman AJ. An experimental study of the etiology of "rum fits" and delirium tremens. *Quarterly Journal of Studies on Alcohol* 1955;16:1-33.
- 5 Mendelson J, ed. Experimentally induced chronic intoxication and withdrawal in alcoholics. *Quarterly Journal of Studies on Alcohol* 1964;25:suppl 2.
- 6 Mendelson JH, Stein S, McGuire MT. Comparative psychophysiological studies of alcoholic and nonalcoholic subjects undergoing experimentally induced ethanol intoxication. *Psychosom Med* 1966;28:1-12.
- 7 Littleton J. Biochemical pharmacology of ethanol tolerance and dependence. In: Edwards G, Littleton J, eds. *Pharmacological treatments for alcoholism*. London: Croom Helm, 1984:119-44.
- 8 Little HJ, Dolin SJ, Halsey MJ. Calcium channel antagonists decrease the ethanol withdrawal syndrome. *Life Sciences* 1986;39:2059-65.
- 9 Koppi S, Eberhardt G, Haller R, Konig P. Calcium-channel-blocking agent in the treatment of acute alcohol withdrawal—caroverine versus meprobamate in a randomised double-blind study. *Neuropsychobiology* 1987;17:49-52.
- 10 Pequignot G, Tuyns AJ. Compared toxicity of ethanol on various organs. *Les Colloques de l'INSERM (INSERM Symposia)* 1980;95:17-32.
- 11 Wadstein J, Skude G. Does hypokalaemia precede delirium tremens? *Lancet* 1978;ii:549-50.
- 12 Nanji AA, Blank DW. Usefulness of serum potassium levels in the diagnosis of delirium tremens. *Intensive Care Medicine* 1984;10:112.
- 13 Chick J. Early intervention in the general hospital. In: Stockwell T, Clement S, eds. *Helping the problem drinker: new initiatives in community care*. London: Croom Helm, 1987:105-20.
- 14 Scheid HW, Braunschweiger M, Schupmann A. Treatment of delirium tremens in German psychiatric hospitals: results of a recent survey. *Acta Psychiatr Scand [Suppl]* 1986;73:153-6.
- 15 Emsley RA, Potgleiter A, Taljaard JJF, Coetzee D, Joubert G, Gledhill RF. Impaired water excretion and elevated plasma vasopressin in patients with alcohol-withdrawal symptoms. *Q J Med* 1987;244:671-8.
- 16 Mander HJ, Weppner GJ, Chick JD, Morton JJ, Best JJK. An NMR study of cerebral oedema and its biological correlates during withdrawal from alcohol. *Alcohol Alcohol* 1988;23:97-102.

Assessing clinical competence

Need for improvement

Licensing agencies and the public are much concerned with assessing the competence of doctors.¹ But the methods that are widely used to assess competence are largely outdated. Better methods are available, and bodies responsible for both undergraduate and postgraduate education need to use them.

The essential skills required for a doctor are generally agreed and include establishing a rapport with patients; eliciting accurate information about patients' problems and establishing the patients' reactions to them; conducting an examination of physical and mental state; selecting and interpreting investigations; showing diagnostic ability; undertaking education, reassurance, and counselling of patients; and managing patients in the immediate and long term. The problem is how best to determine the required level of competence in each area and assess it.

The notion of how well a newly qualified doctor should be able to perform constitutes minimum competence. This can be determined by asking experienced and recently qualified doctors to identify the problems they meet commonly and those they meet less often but must manage effectively if disasters are to be avoided. The required skills can then be made explicit.² An analysis of critical incidents (in which skills are needed to their fullest extent) and of examples of good and

bad practice as judged by both doctors and patients may also help identify skills.³ With checklists of essential skills assessors would know what they are looking for.

Traditionally assessment has relied on written tests and clinical examinations, but doubts have been cast on their validity and reliability. Performance on multiple choice questions and patient management problems correlates poorly with ratings of ability by clinical instructors. Scores on standard clinical examinations relate poorly to interviewing and observational skills,⁴ and measures of psychosocial attitudes and perceived competence are no substitute for proved ability.⁵ These methods provide indirect measures of performance and probably tap different skills.

The objective structured clinical examination has been introduced to assess essential clinical skills.^{6,7} Typically clinical students must attend a series of stations. At each station they are given an explicit task such as eliciting key information, examining a system of the body, suggesting and interpreting investigations, educating the patient about his or her condition and treatment, and advising on further management.

Examiners use a previously agreed checklist to judge performance. Concurrent written exercises using multiple