

for other SSRIs such as fluoxetine (Hwang & Magraw, 1989), sertraline (Thornton & Resch, 1995) and paroxetine (Lisi, 1993), but not for citalopram.

Case report

Mrs. A, a 73-year-old woman suffering from a bipolar affective disorder and from hypertension was medicated with diuretics (hydrochlorothiazide and amiloride) resulting in several episodes of hyponatraemia. The first episode occurred seven years before present hospitalisation, with a Na concentration of 126 mmol/l (normal range: 137–150 mmol/l). After one month of severe depression, the patient was hospitalised due to non-response to amitriptyline and penfluridol. She was depressed, retarded, confused, and complained of extreme fatigue. Na and K serum concentrations were low: 121 mmol/l and 3.21 mmol/l (3.5–5.3 mmol/l), respectively. Medication with amitriptyline was discontinued, but it was only after stopping administration of hydrochlorothiazide and amiloride, and after installing hydrous restriction and providing a supplement of electrolytes that serum Na and K normalised.

One month later, we introduced citalopram (20 mg/day). Serum Na decreased to 133 mmol/l and then to 128 mmol/l, after 7 and 10 days of treatment, respectively, while serum K and urea remained within the normal range. The patient equally became more anxious and retarded. Citalopram treatment was then interrupted, 6 g of NaCl was administered for 3 days, and a hydrous restriction was installed. Within one month, serum Na slowly normalised (3 days after discontinuation of citalopram: 131 mmol/l; 4 days: 133 mmol/l; 24 days: 138 mmol/l). Four days after discontinuation of citalopram treatment, blood osmolality was decreased to 264 mmol/kg (270–295 mmol/kg), while urinary osmolality was 522 mmol/kg (50–1400 mmol/kg). These values, the normalisation of serum Na after discontinuation of citalopram, and the absence of any other treatment suggest the involvement of citalopram in this hyponatraemia. Moreover, it did not reappear after a later treatment with maprotiline and flupenthixol.

Diuretics and fluoxetine represent a risk factor of developing hyponatraemia, but apparently not age (Sieglar *et al.*, 1995). While no report has yet been published about hyponatraemia after citalopram administration, Mrs A first developed it after administration of diuretics. For unknown reasons, this person may belong to that part of the popula-

tion who run a risk of developing hyponatraemia, and which reappeared after treatment with citalopram. Therefore, in patients who have already developed hyponatraemia while being medicated with diuretics, serum electrolytes should be carefully monitored when comedicated with SSRIs.

HWANG, A. S. & MAGRAW, R. M. (1989) Syndrome of inappropriate secretion of antidiuretic hormone due to fluoxetine. *American Journal of Psychiatry*, **146**, 399.

LISI, D. M. (1993) Comment: paroxetine-associated hyponatremia. *Annals of Pharmacotherapy*, **27**, 1547–1548.

SIEGLER, E. L., TAMRES, D., BERLIN, J. A., *et al* (1995) Risk factors for the development of hyponatremia in psychiatric inpatients. *Archives of Internal Medicine*, **155**, 953–957.

THORNTON, S. L. & RESCH, D. S. (1995) SIADH associated with sertraline therapy. *American Journal of Psychiatry*, **152**, 809.

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Seizure length during ECT

SIR: Seizure activity is important to therapeutic success during electroconvulsive therapy (ECT) and Scott & Lock (1995) have recommended that peripheral seizures should be at least 15 seconds (maximum 90 seconds). A number of SSRIs have been reported to increase seizure length during ECT including fluoxetine (Gutierrez-Esteinou & Pope, 1989) and paroxetine (Curran, 1995). In addition, the Special Committee on ECT of the Royal College of Psychiatrists has received a number of reports of increased seizure length in patients taking SSRIs but there have been no reports concerning fluvoxamine (Curran & Freeman, 1995).

A patient recently under the care of the authors with DSM-IV major depression underwent a course of ECT. Physical examination and routine investigations were all normal. The only medication taken by the patient was fluvoxamine 100 mg bd. Seizure length was determined in accordance with Royal College of Psychiatrists guidelines and the patient received a course of six treatments. The average seizure length during treatment was 63.1 seconds (s.d. 28.0, range 26–111). This average seizure duration was approximately double that observed in the same ECT department over the same period; the highest previous seizure length recorded in our department was 75 seconds. In addition, the correlation between seizure length and stimulus dose in mC was low (Spearman Rank

Correlation Coefficient 0.25, $P < 0.9$, $n = 6$, two tailed).

Excessively long seizures should be avoided during ECT, partly because of the short acting nature of anaesthetics and muscle relaxants, but also because of the risk of cerebral hypoxia. It is likely that the increased seizure length seen in this patient was due to fluvoxamine. Although there were no serious complications arising from the combined use of ECT and fluvoxamine, a seizure length of 111 seconds is potentially dangerous (Curran & Freeman, 1995).

CURRAN, S. (1995) Effect of paroxetine on seizure length during electroconvulsive therapy. *Acta Psychiatrica Scandinavica*, **92**, 239–240.

— & FREEMAN, C. P. (1995) ECT and drugs. In *The ECT Handbook*. The Second Report of the Royal College of Psychiatrists' Special Committee on ECT, Council report CR39, (ed. C. P. Freeman), pp. 49–57. London: Royal College of Psychiatrists.

GUTIERREZ-ESTEINOU, R. & POPE, H. G. (1989) Does fluoxetine prolong electrically induced seizures? *Convulsive Therapy*, **5**, 344–348.

SCOTT, A. & LOCK, T. (1995) Monitoring seizure activity. In *The ECT Handbook*. The Second Report of the Royal College of Psychiatrists' Special Committee on ECT, Council report CR39 (ed. C. P. Freeman), pp. 62–66. London: Royal College of Psychiatrists.

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Suicide prevention versus alcohol promotion

SIR: Ohberg *et al* (1996) found that 36% of suicides in Finland were committed under the influence of alcohol. Although they did not estimate the proportion of those who were dependent on alcohol, it is likely to be high. It is well known that the prevalence of alcoholism is dependent on the per

capita consumption. In the UK, the "safe levels" of alcohol consumption were recently increased by 33–50%. It is possible that there will be a proportionate increase in the prevalence of alcohol misuse and dependence and hence the number of suicides. We wonder how this will affect our endeavours to reduce the number of suicides by the year 2000.

OHBERG, A., VUORI, E., OJANPERA, I., *et al* (1996) Alcohol and drugs in suicide. *British Journal of Psychiatry*, **169**, 75–80.

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Quaker belief and child psychiatry

SIR: Professor Graham's article on Quaker belief and child psychiatry makes a most valuable contribution to the consideration of the kind of human values which should inform our approach to children and young people in need of care or treatment. This is especially true of chronic, young offenders whom all efforts to help seem to fail and social attitudes become increasingly punitive.

Quaker beliefs and practice might form the basis from which could emerge a suitable therapeutic community approach, offering a quality of care which would do justice to the problems of such desperate and damaged young people.

I feel sure that we can produce a more appropriate response than a British version of the 'boot camp', or the more usual prison or secure accommodation with only minimal therapeutic means at its disposal.

GRAHAM, P. (1996) Quaker belief and child psychiatry. *British Journal of Psychiatry* **169**, 1–5.

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