Serum lithium concentrations in relation to treatment.

Conversion: SI to traditional units—Lithium: 1 mmol/l $\approx$ 0.69 mg/100 ml.

four times daily. Serum lithium concentrations were monitored over the next few weeks (see figure). In the three days after starting the diuretic the concentrations rose steadily to 1.7 mmol/l (1.18 mg/100 ml), at which level her lithium intake was halved. After this the concentrations showed a slow decline and by day 28 she was back to her original dose of lithium carbonate. On day 36 she was discharged home and since then over a four-month follow-up her lithium concentrations have remained within the therapeutic range. She has improved symptomatically in that her urinary frequency has decreased to day/night = 3-4/0-2 and her mood has remained stable.

Comment

In a patient who is well-controlled on long-term lithium the development of a state requiring diuretic treatment presents a problem. In the case reported here bendroflumazide was successfully combined with lithium to control this patient's diabetes-insipidus-like state. Nevertheless, a clinician prescribing a diuretic to a patient on long-term lithium should bear in mind the following: (1) fairly close laboratory control of the serum lithium concentration is required, possibly as an inpatient; (2) an immediate increase in the serum lithium concentration should be anticipated, thus avoiding toxic effects which might make the patient lose confidence in the treatment³; (3) restabilisation may be at a lower lithium dose,⁵ or, as in this case, at the original dose level.

¹ Kerry, R J, in *Lithium Research and Therapy*, ed F N Johnson, p 162. London, Academic Press, 1975.

² Weinstein, M R, and Goldfield, MD, in *Lithium Research and Therapy*, ed F N Johnson, p 259. London, Academic Press, 1975.

³ Macfie, A C, *British Medical Journal*, 1975, **1**, 516.

⁴ MacNeil, S, et al, *Lancet*, 1975, **1**, 1295.

⁵ Levy, S T, Forrest, J N, Heninger, MD, *American Journal of Psychiatry*, 1973, **130**, 1014.

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Assessment of sympathetic overactivity in tetanus

Overactivity of the sympathetic nervous system is thought to contribute to the still considerable mortality from severe tetanus,¹ and improved results have been reported after the use of adrenergic blocking agents.² Unless these agents are to be given to all patients with tetanus prophylactically, however, sympathetic activity will have to be monitored so that adrenergic block can be induced at an appropriate time. Plasma and urinary catecholamine concentrations, if measured at all, are usually measured in retrospect so that the results are not available to guide treatment on the hour-to-hour basis necessary in severe tetanus.

Various clinical indicators of sympathetic activity have been proposed, including increases in (a) the level and variability of the blood pressure, (b) the blood pressure response to aspiration of secretions from the trachea (tracheal suction),³ and (c) heart rate.² These clinical indices were compared with serial measurements of plasma catecholamine concentrations during severe tetanus in a 29-year-old farmer.

Methods and results

Blood pressure was measured every half hour by nursing staff using a standard cuff and mercury sphygmomanometer. The mean blood pressure was calculated from all the observations obtained within 24 hours and the blood pressure variability was calculated by averaging the absolute difference between successive readings of systolic pressure.³ A mean heart rate was calculated from corresponding readings. The increase in blood pressure during tracheal suction was the difference between the presuction systolic pressure and the highest systolic level recorded during or within one minute of the procedure.³ Blood samples were collected at least once a day before suction and within 30 seconds of the recommencement of artificial ventilation. Plasma adrenaline and noradrenaline concentrations were estimated using the radioenzymatic method of Hörtnagl et al.⁴

Until the eighth day after the onset of trismus, plasma catecholamine concentrations were relatively normal and the cardiovascular system fairly stable (see table). On the eighth day, despite unchanged treatment and in the absence of any indication of infection, there were significant increases in heart rate, mean blood pressure (largely due to an increase in diastolic pressure), and plasma catecholamine concentrations. Both adrenaline and noradrenaline concentrations reached abnormally high values. Blood pressure variability was slight throughout the illness and changes in pressure and catecholamine concentrations induced by tracheal suction were minimal. Treatment with the beta-adrenergic blocking agent propranolol (40 mg/8 h) was started because of the tachycardia, and no further plasma catecholamine estimations were carried out. The patient's subsequent course was uneventful and he recovered fully after three weeks of artificial ventilation.

Assessment of sympathetic activity in a 29-year-old man with severe tetanus

Day after onset of trismus:	3	4	5	6	7	8
Curare dosage (mg/24 h)	330	270	300	330	360	330
Average mean blood pressure (mm Hg)	85	86	96	88	90	102
Blood pressure variability (average absolute systolic difference) (mm Hg)	9	12	14	8	7	8
Average heart rate	71	84	82	83		
Resting plasma concentrations:						
Adrenaline (nmol/l)	0.71	0.44	1.2	1.8	2.46	9.01
Noradrenaline (nmol/l)	1.71	1.3	2.72	2.01	1.83	5.50
Responses to tracheal suction:						
Systolic blood pressure (mm Hg)	+10	+10	+20	+13	+11	+13
Adrenaline (nmol/l)	-0.05	+0.93	+0.87	+0.33	+0.33	0.00
Noradrenaline (nmol/l)	+0.19	+0.65	+0.12	+0.18	+0.71	+1.24

Conversion: SI to traditional units—Adrenaline: 1 nmol/l $\approx$ 183 ng/l. Noradrenaline: 1 nmol/l $\approx$ 169 ng/l.

Comment

Our experience with this patient suggests that the appearance of an unexplained tachycardia in a patient with tetanus shows that there is an increase in sympathetic activity and that careful monitoring of the heart rate may indicate when adrenergic blocking agents can be introduced to lessen the possibility of catecholamine-induced myocardial damage.⁵

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³ Kerr, J H, in *Third International Conference on Tetanus*, p 128. Special Scientific Publication No 253. Washington, Pan American Health Organisation, 1972.

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