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Reye's syndrome: assessment of intracranial monitoring

SIR,—We agree with Dr J G Jenkins and colleagues (7 February, p 337) that the maintenance of an adequate cerebral perfusion pressure is important in managing patients with Reye's syndrome, but this may not be the only factor determining outcome, as a recent case shows.

A 7 year old girl was admitted as a precaution shortly after her 4 year old brother developed Reye's syndrome. She was well on admission, but her clinical condition deteriorated rapidly, and within four hours she was unconscious. Reye's syndrome was diagnosed (table), and early intensive management was started, but she deteriorated further to Lovejoy stage 4. Though her intracranial pressure was not raised when a Ladd epidural monitor was inserted, she subsequently had several sustained rises in pressure with peaks >50 mm Hg. On at least two occasions her cerebral perfusion pressure fell below 25 mm Hg for periods of more than an hour. Though continuous electroencephalographic monitoring showed seizure activity and many adverse features, full intensive care was not withdrawn as her brain stem auditory evoked potentials were normal. Active treatment for raised intracranial pressure was needed for 21 days, and she was eventually weaned off the ventilator on day 28. She was discharged on day 59, and at review nearly a year later she had no neurological deficit and had returned to school and full activities.

In the series reported by Dr Jenkins and coworkers all 10 patients with a cerebral perfusion pressure <40 mm Hg either died or sustained severe neurological handicap. In spite of her cerebral perfusion pressure falling to <40 mm Hg our patient recovered. We suggest that this might be because our patient was treated very early as she had been admitted as a precaution after the onset of Reye's syndrome in her brother. Delay in diagnosis of Reye's syndrome is commonly associated with a poor outcome, particularly in patients with rapidly advancing coma.¹ Other reviewers have suggested that the likelihood of recovery is related to the stage of the illness at which intensive management or raised intracranial pressure is started.²

Our case illustrates, firstly, the need for a high index of suspicion for siblings of children with Reye's syndrome and, secondly, that even in a case

of rapidly progressing coma with profound reduction in cerebral perfusion pressure a full functional recovery is still possible.

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Dangers in treating hyponatraemia

SIR,—Professor J D Swales (31 January, p 261) seems to support the recommendation of Stern *et al* that hypernatraemia should not be corrected by more than 12 mmol/day.¹ We consider this advice to be too cautious, particularly in patients with acute hyponatraemia. In addition, we find hypertonic, not isotonic, sodium to be effective, as the following two cases illustrate.

Case 1—A 73 year old man became confused, restless, and disorientated after transurethral prostatectomy. He was hypotensive, in respiratory distress, and anuric. Massive absorption of the irrigating 1.5% glycine caused the transurethral reaction syndrome. He was thought to be hypovolaemic, and a further 2 units of blood and 4 litres of isotonic solution were given. After 24 hours he went into coma, with fixed dilated pupils; his blood pressure fell to 80/40 mm Hg, his pulse to 30 beats/min, and his central venous pressure to 1 cm H₂O. The amount of sodium required to raise serum osmolality was calculated,^{2,3} and 500 ml 5% sodium chloride was given in one hour, then repeated. He regained consciousness during the infusion and passed 5 litres of urine. He made a full recovery.

Case 2—A woman aged 74 was admitted with myocardial infarction confirmed by raised cardiac enzyme activities. She had been receiving nifedipine and Moduretic, which were withdrawn as she became persistently hypotensive (90/50 mm Hg). She suffered a cardiac arrest on the sixth day. Two days later she became confused and had visual hallucinations. She had been receiving 2.5 litres 5% glucose intravenously daily, but her urine output was gradually declining. A normal serum sodium concentration of 140 mmol/l on admission dropped to 114 mmol/l, and osmolality fell to 241 mmol/kg over eight days. Treatment with fluid restriction and frusemide 160 mg for two days was ineffective. She continued to deteriorate; serum sodium concentration dropped to 108 mmol/l and osmolality to 236 mmol/kg, with urine osmolality of 294 mmol/kg and undetectable sodium. She underwent an infusion of 500 ml 1.8% sodium chloride over four hours, which raised her serum sodium concentration to 122 mmol/l. A further dose given over the next eight hours raised the serum sodium concentration to 132 mmol/l and osmolality to 294 mmol/kg. She passed 3 litres of urine, and her neurological signs disappeared. She made a full recovery.

These cases are anecdotal but provide useful guidance. Any clearly definable, acute reduction in serum sodium concentration (>20 mmol/l) associated with severe neurological signs should be corrected rapidly. Hypertonic sodium relieves brain oedema and induces diuresis when given early and in a concentration that corrects osmolality without aggravating volume overload.^{2,3} Rapid correction may be dangerous in patients with chronic hyponatraemia in whom osmotic adaptation has occurred. A delay in treating acute hyponatraemia, however, may result in permanent damage of the oedematous brain. An isotonic solution can also be dangerous as it achieves little osmotic correction and worsens volume overload.³

We conclude that acute hyponatraemia of iatrogenic origin, such as the transurethral reaction syndrome² and medical and postsurgical syndromes,^{3,5} necessitates immediate action. Hyper-

tonic sodium is lifesaving and deserves scientific evaluation.

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AUTHOR'S REPLY.—There are already numerous case reports presenting conflicting evidence for or against the safety of rapid correction of hyponatraemia.¹⁻³ Though I agree that anecdotal evidence may be helpful when controlled trials are difficult, there are also grave dangers in using very selected case reports as a basis for general advice. Even in the study by Sterns *et al*, which reported on a consecutive group of patients, neurological damage was observed in only a minority of the patients (five out of 43) when hyponatraemia was corrected rapidly.³ The absence of neurological damage in two isolated cases is not particularly persuasive in such circumstances. Apparent improvement caused by a rise in serum sodium concentration must be distinguished from spontaneous recovery and the effects of volume expansion in a sodium and fluid depleted patient; the second patient described by Mr Ghanem and coworkers, who had been exposed to large doses of diuretics and water restriction, may well have benefited from such volume effects. It may be possible to obtain a clearer picture with experimental hyponatraemia induced by three days of vasopressin treatment in rats. In one study rapid correction of hypertonic saline produced pontine demyelination.⁴

Rapid rises in serum sodium concentration may not be harmful in the correction of acute hyponatraemia that may occur, for example, postoperatively: osmotic shifts between intracellular and extracellular fluid may then not have had time to occur. Interestingly, in the review reported by Sterns *et al* most of the reports of neurological sequelae associated with rapid correction occurred in hyponatraemia induced by diuretics, which was presumably fairly chronic. The most important lesson in such reports (and of the first case reported by Mr Ghanem and colleagues), however, is surely that postoperative water intoxication caused by infusion or absorption of sodium free fluids should be prevented. The dangers of such mishaps are well recognised and may be irreversible whatever corrective regimen is used.²

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Biochemical details of patient with Reye's syndrome

	On admission	Maximum	Normal range
Alanine transaminase activity (IU/l)	1215	1215	0-45
Aspartate transaminase activity (IU/l)	25	690	6-51
Ammonia concentration (mmol/l)	210	396	10-40
Prothrombin time (index/control)	18/13	19/13	
Blood glucose concentration (mmol/l)	7.3	7.3	3-9
Bilirubin concentration (mmol/l)	13	20	<20