

of digoxin makes DC cardioversion more hazardous, the diving reflex may be a useful additional manoeuvre to restore sinus rhythm.

M. F. M. BAMFORD and DOUGLAS PICKERING
*Department of Paediatrics,
 The Radcliffe Infirmary,
 Oxford OX2 6HE.*

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Hyponatraemia in children with febrile convulsions

Sir,

Rutter and O'Callaghan (*Archives*, 1978, **53**, 85) have drawn attention to the common finding of mild hyponatraemia in children with febrile convulsions. They define hyponatraemia as plasma Na levels of 132 mmol/l and below but do not indicate their reasons for choosing this particular concentration. In our study of 50 healthy babies aged between 18 and 120 days (Dale *et al.*, 1975) we found a mean plasma Na concentration of 135 mmol/l (SD 2.3)—similar to the mean value of 135 mmol/l reported by Rutter and Smales (1977) for 163 children with febrile convulsions. It would seem to be wise, in infancy at least, to take 130 mmol/l (mean -2SD) as the level below which hyponatraemia is said to exist.

GORDON DALE and J. R. SIBERT
*Department of Clinical Biochemistry,
 Royal Victoria Infirmary,
 Newcastle upon Tyne;
 and Department of Paediatrics,
 University Hospital of Wales, Cardiff.*

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- Dale, G., Goldfinch, M. E., Sibert, J. R., and Webb, J. K. G. (1975). Plasma osmolality, sodium, and urea in healthy breast-fed and bottle-fed infants in Newcastle upon Tyne. *Archives of Disease in Childhood*, **50**, 731-734.
 Rutter, N., and Smales, O. R. C. (1977). Role of routine investigations in children presenting with their first febrile convulsion. *Archives of Disease in Childhood*, **52**, 188-191.

Drs Rutter and O'Callaghan comment:

We have no information about plasma Na levels in healthy Nottingham children in the febrile convulsion age group. We therefore defined hyponatraemia as a plasma Na below the normal ranges quoted by three large

children's hospitals: The Hospital for Sick Children, Great Ormond Street (normal range 135-143 mmol/l), Birmingham Children's Hospital (136-143 mmol/l), and Melbourne Children's Hospital (133-143 mmol/l). Our measurements were made on venous blood by flame photometry. Plasma osmolalities, measured by a different method, were also low, suggesting that our low Na levels were not simply a reflection of our laboratory method.

It may well be that commonly quoted normal ranges for plasma Na in children are too high, as Dale and Sibert suggest, but we used them in the absence of any other data. We did not use their own values for healthy babies under the age of 4 months because the children in our study were all older, with a mean age of just under 2 years.

N. RUTTER and M. J. O'CALLAGHAN
*Department of Child Health,
 Nottingham University, and
 Nottingham Children's Hospital.*

Steroid-induced CPK estimation—a new diagnostic test for human muscular dystrophy and its carrier state

Sir,

Increase of serum creatine phosphokinase (CPK) has been regarded as the most sensitive index in the detection of early cases of muscular dystrophy, especially in Duchenne type (DMD). Increased CPK is also found in other neuromuscular diseases such as spinal muscular atrophy, motor neurone disease, chronic polymyositis, as well as in 70-75% of 'definite' carriers of DMD. Takahasi *et al.* (1975) observed a marked rise of CPK after IV prednisolone in 14 DMD cases and suggested this to be specific for muscular dystrophy (MD), but put forward no explanation for such phenomena.

We have observed the change of CPK in response to IV hydrocortisone in different neuromuscular diseases in Indian patients, so as to confirm or deny Takahasi's findings. Similar testing was also carried out on Duchenne carriers and relatives of other MD cases. 44 cases of MD (23 cases of DMD and 21 cases of other MD), 11 cases of other muscle diseases, 6 known Duchenne carriers and 32 suspected carriers were studied. 40 normal controls and 36 healthy relatives of MD cases were also studied.

CPK activity increased significantly 4 hours after 1 mg/kg IV hydrocortisone in MD cases but not in patients with other neuromuscular disorders. Post-steroid rise in CPK (as % basal level) was highest in DMD cases (119 ± 1.78 SD). 50% of known (definite and probable) Duchenne carriers and 18.7% of unknown (possible) carriers also showed positive results with the test. No such increase was observed in relatives of MD cases. An inverse correlation was observed between the grade of disability and the % poststeroid increase of CPK in DMD cases. Such inverse correlation was also found between the duration of disease and poststeroid increase of CPK in DMD cases. The increased CPK may be