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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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

explained by the hypothesis that steroid increases the permeability (Rowland, 1976) of the defective plasma membrane of the muscle fibres of DMD cases (Mokri and Engel, 1975), resulting in additional release of CPK to blood. Similar but less appreciable CPK release phenomena may occur in other MD cases. The CPK clearance factor probably remains constant in these cases. A possible explanation of the rise in CPK in some carriers was proposed by Hughes *et al.* (1971) that there are two populations of carriers—normal and abnormal. The abnormal carriers might respond to steroid in a similar way provided they had similar plasma membrane defects to DMD cases. Other cases of neuromuscular disorders and normal controls probably have no such plasma membrane defect.

The results indicate that steroid-induced CPK estimation can differentiate MD from other muscle diseases. The test may also help to increase the rate of detection of Duchenne carriers in those cases with 'borderline' basal CPK activity and negative electromyographic and muscle biopsy findings.

SHYAMAL SEN and P. K. DAS
Neurology Unit,
Medical College and Hospitals,
Calcutta-700012, India.

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Comparison between microscopical examination of unstained deposits of urine and quantitative culture

Sir,

The conclusions reached by Littlewood and his colleagues in their article comparing microscopical examination of unstained deposits of urine and quantitative culture (*Archives*, 1977, **52**, 894) are not supported by the data presented. Urine was collected in four different ways, as suprapubic, midstream, clean catch, and bag specimens from children aged 2 weeks to 14 years. A surface bacterial count $\geqslant 10^5$ was taken as indicating infection in all urines however collected and regardless of the child's age. However, this criterion of infection is not applicable to urine collected by bladder puncture in which *any* growth indicates infection (Newman *et al.*, 1967; McFayden and Eykyn, 1968; Paterson *et al.*, 1970; Rubin, 1975) nor to bag urines from neonates which often have a surface viable count $\geqslant 10^5$, but are sterile when a suprapublic specimen is obtained (Edelmann *et al.*, 1973).

By not using these generally accepted criteria of infection and noninfection in suprapubic and bag specimens of urine, the authors will have underdiagnosed infection in some cases of the former, and overdiagnosed infection in some cases of the latter. This vitiates completely the correlation found between urinary infection and the number of bacteria seen on microscopy.

MARTIN MONCRIEFF
Department of Paediatrics,
The Radcliffe Infirmary,
Oxford OX2 6HE.

Dr Littlewood comments:

Our study amounted to a simple comparison between the numbers of organisms visualised in the urine deposit and the quantitative culture, irrespective of the method of collection. Our data confirm that the culture result can usually be predicted from the number of organisms visualised on microscopy. It is unfortunate that we referred to urines growing $>10^5$ organisms per ml as 'infected'. There was no implication that the *patients* had a urinary tract infection. We are well aware that any growth from a suprapubic urine is meaningful and bag urines even from healthy infants contain many bacteria (Littlewood, 1971). Whether or not the patients had bacteria within the urinary tract is not the point of the study. Those experienced in the art of urinary microscopy will realise the great benefits that accrue to all if this simple, cheap, interesting, and informative technique is used in the clinic.

J. M. LITTLEWOOD
Department of Paediatrics,
St James's Hospital, Leeds LS9 7TF.

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Discharge of small babies from hospital

Sir,

Early discharge of very small babies from neonatal units could be valuable in reducing mother-child separation. Nursery occupancy figures could be reduced, making it possible for a limited number of nurses to care more