

## Summary

Lung mechanics were studied in 8 infants with cystic fibrosis at 6 months of life and radiosotopic lung function was measured in 5 of them at 5 years of age. The children who were initially asymptomatic had normal lung mechanics in infancy but the 2 restudied later had abnormal radioisotopic lung function. The symptomatic children showed abnormalities in infancy and more marked changes later. It is concluded that the lungs in cystic fibrosis are probably normal initially and that damage occurs later even in the absence of symptoms.

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## Hyponatraemia in children with febrile convulsions

In a recent study of investigations routinely performed in children admitted to hospital with a febrile convulsion, we found that mild hyponatraemia was common (Rutter and Smales, 1977). In 163 children, the mean sodium was  $135 \pm 4$  mmol/l ( $\pm$ SD) with a range of 125-145 mmol/l. 31% had plasma Na levels of 132 mmol/l or less. To investigate this further, we

started a prospective study comparing the plasma Na and osmolality with the urine osmolality, looking for evidence of inappropriate secretion of antidiuretic hormone (ADH).

## Method

Urea, electrolytes, sugar, and osmolality were measured on venous blood taken on admission from randomly selected children admitted to Nottingham Children's Hospital with a diagnosis of a febrile convulsion. Osmolality was measured on the first specimen of urine passed after admission. The time of the urine collection was recorded. Where the time of the specimen was uncertain or more than 6 hours after admission, the child was excluded from the study.

## Results

Twenty-three children were studied, one child being admitted on two separate occasions. Their ages ranged from 8 months to 5 years. Plasma Na, osmolality, and urine osmolality are shown in the Fig. The mean Na was  $133 \pm 3$  mmol/l and mild hyponatraemia was present in 8 cases (132 mmol/l or less). Details of these hyponatraemic children are shown in

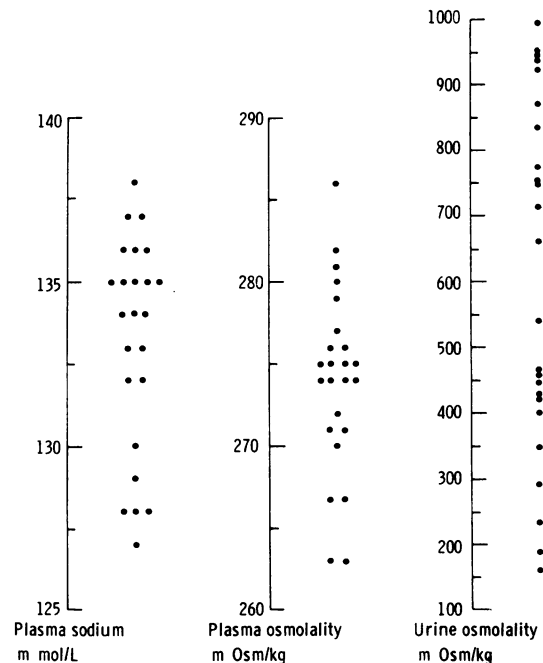


Fig. Plasma sodium, plasma and urine osmolalities of 23 children admitted after a febrile convulsion.

Table *Details of 8 children with hyponatraemia following a febrile convulsion. Those above the line show evidence of inappropriate secretion of antidiuretic hormone (ADH)*

Age	Cause of fever	Duration of fit (min)	Plasma sodium (mmol/l)	Plasma osmolality (mOsm/kg)	Urine osmolality (mOsm/kg)	Plasma urea (mmol/l)	Plasma sugar (mmol/l)
2 yr	Tonsillitis	2	132	274	662	3.6	5.3
3 yr	Tonsillitis	3	128	270	713	2.6	—
10 m	URTI	3	132	271	458	6.0	9.5
2 yr 6 m	Measles	9	130	267	923	2.4	5.9
10 m	URTI	5	129	275	467	2.4	—
1 yr 6 m	Gastroenteritis	6	128	267	937	3.8	5.5
1 yr 6 m	Gastroenteritis	3	127	263	234	4.0	6.5
3 yr	Unknown	3	128	277	293	3.2	5.8

URTI = Upper respiratory tract infection.

the Table. The 10-month-old baby who was admitted twice was hyponatraemic on both occasions. There was no correlation between hyponatraemia and age or length of convulsion. The first 6 children fulfil the criteria for the syndrome of inappropriate ADH secretion, with a low plasma osmolality, high urine osmolality, and normal plasma urea (Mendoza, 1976).

### Discussion

We found that hyponatraemia is a common finding in children admitted to hospital with a febrile convulsion. In the majority of these there was evidence of mild inappropriate secretion of ADH. This syndrome is well known in childhood in association with bacterial meningitis (Nyhan and Cooke, 1956), other cerebral lesions (Bartter and Schwartz, 1967), pneumonia (Mor *et al.*, 1975), vincristine therapy (Suskind *et al.*, 1972), and after surgery. This is the first time, however, that it has been reported in children with febrile convulsions. Hayward (1976) has shown that there is a rise in plasma ADH after stimulation of the amygdala, and Ounsted (1976) has postulated that this mechanism might be important in the child who develops febrile status.

There is no evidence that the hyponatraemia plays any role in causing the convulsion. In this study and in our retrospective study (Rutter and Smales, 1977), the hyponatraemic and normonatraemic children were similar in age, cause of fever, and length of convulsion. However, it is known that hyponatraemia lowers the threshold for febrile convulsions in animals (Millichap, 1968) and therefore a child who has had a febrile convulsion and is hyponatraemic might be more likely to have a subsequent convulsion during the same illness. To see if this was so, we re-examined the data of children in the retrospective study to see who had had a further convulsion after admission to hospital. A higher proportion of hyponatraemic children (5 out of 50) had had a

further convulsion compared with normonatraemic children (2 out of 113); this is significant at the 5% level (Fisher's exact probability test). Although the numbers of children who had subsequent convulsions were small and the data were collected retrospectively, it is likely that hyponatraemia following a febrile convulsion might predispose a child to a further convulsion during the same illness. Hyponatraemia in association with inappropriate ADH secretion is best treated by fluid restriction. It seems reasonable therefore that if a child is admitted to hospital after a febrile convulsion and is not dehydrated, then an excessive fluid intake should be avoided. Such a policy, however, might be dangerous in a child treated at home.

### Summary

In a study of 23 children admitted to hospital with a febrile convulsion, mild hyponatraemia was found on 8 occasions. In 6 of these cases there was evidence of inappropriate secretion of antidiuretic hormone. The hyponatraemia is unlikely to be the cause of the convulsion, but probably predisposes the child to a subsequent convulsion during the same febrile illness.

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### Diagnosis and treatment of *Pneumocystis carinii* pneumonia

Sir,

Lipson *et al.* (1977) report success in all 7 of their patients with *Pneumocystis carinii* pneumonia treated with cotrimoxazole alone. We would like to make some cautionary observations based on a successfully treated child.

A 4-year-old girl with acute lymphoblastic leukaemia in remission for 15 months was seen at a routine follow-up clinic. Although symptom free, she appeared slightly cyanosed and tachypnoeic and a chest x-ray showed diffuse changes. A diagnostic needle aspiration of the lung was performed and *Pn. carinii* was identified (by Dr John Lever). Cultures of the remainder of the aspirate for viruses, fungi, and bacteria were sterile. Despite early treatment with high-dose cotrimoxazole, the child's condition deteriorated steadily over 3 days. Pentamidine isoethionate was added and her condition thereafter improved; there were no complications.

We would like to make 3 points: (1) The child was symptom free on the day of diagnosis. (2) Needle aspiration was a safe, rapid means of diagnosis. Of the cases reported by Lipson *et al.* (1977), 2 out of 14 had serious complications from open lung biopsy. This contrasts with one mild episode of transient haemoptysis complicating 8 needle aspirations in children with pneumonia at Bristol Children's Hospital (personal observations). Open biopsy may of course be indicated after a negative needle aspiration when *Pn. carinii* infection is strongly suspected (Hughes, 1977). (3) Cotrimoxazole appeared to be ineffective in the first 3 days of treatment, in contrast to the observation by Lipson *et al.* that the drug 'seemed to have

a rapid onset of action'. Hughes (1977) reported failure of cotrimoxazole therapy in 3 out of 14 children with *Pn. carinii* pneumonia.

We conclude that needle aspirate is the investigation of choice when *Pn. carinii* pneumonia is suspected, and that cotrimoxazole is not necessarily effective in all cases.

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Sir,

The report by Lipson *et al.* (1977) prompts us to report our recent experience with 2 cases of *Pneumocystis carinii* pneumonia, one aged 9 years and one 3 months, who were under treatment for systemic lupus erythematosus and acute lymphatic leukaemia respectively. Clinical diagnosis of *Pn. carinii* pneumonia was made on the basis of fever, increased respiration rate, and chest x-rays. The agent was shown in tracheal washings (2 ml saline solution under anaesthesia using N<sub>2</sub>O-O<sub>2</sub>-halothane), but was absent from sputa and gastric juice (Chan *et al.*, 1977). The patients' general conditions did not permit lung biopsy or lung aspiration (Cohen and Weiss, 1971). Both were treated first with pentamidine isoethionate, further