Douglas Gairdner won a scholarship to Oxford before transferring to medicine at the Middlesex Hospital, London, graduating in 1936. After a fellowship at Bellevue Hospital, New York, he served with the Royal Army Medical Corps in the Middle East. After the war he was appointed assistant to Professor Sir James Spence in Newcastle before moving to Cambridge in 1947. His contributions to paediatric research and literature, which his modesty prevents him from enumerating, are too extensive to detail. His approach is exemplified, however, when he says of neonatal research 'It was a joy for us that the extremely simple technology, which was all we had, was yet capable of yielding worthwhile results' and he praises the dedication of his hard working

colleagues. He was one of the many paediatricians of the day who rejoiced in the elevation of babies from being something of second class citizens to fully accepted 'patients' in their own right.

Douglas Gairdner has served all aspects of paediatrics with great distinction and is possibly best known for his 15 years Co-Editorship of the *Archives of Disease in Childhood* and Editorship of the first four editions of *Recent Advances in Paediatrics*. The BPA recognised his many contributions in awarding him the Spence Medal (1976) and electing him to Honorary Membership (1977).

C H M WALKER

LETTERS TO THE EDITOR

Mechanical ventilation and respiratory syncytial virus infection

SIR,—Drs Lebel *et al* report weight and prematurity as risk indicators for the need for ventilation in bronchiolitis.¹ In their case-control study a viral cause was determined in only 32%; infants with underlying cardiopulmonary abnormalities—who are known to be at increased risk for severe respiratory syncytial virus infections and mortality^{2,3}—were excluded. Thus the results from their study may not readily be applied to all infants with bronchiolitis who need hospitalisation.

We would like to present the preliminary results of a prospective study (1987–9) of children admitted with a respiratory syncytial virus infection to the Sophia Children's Hospital, The Netherlands. In all patients the infection was proved by direct immunofluorescent assay. Nineteen patients needed mechanical ventilation and 82 were admitted to the general ward.

Weight and age (corrected for prematurity) on admission were significantly related with mechanical ventilation (by χ^2 , $p < 0.05$). No relation between mechanical ventilation and risk factors for severe respiratory syncytial virus infections (prematurity, congenital heart disease, bronchopulmonary dysplasia, or immunodeficiency states) was found. Nine (47%) of the ventilated patients and 36 (43%) of the non-ventilated patients belonged to the risk group. In a stepwise logistic regression analysis (while controlling for the covariates: age, prematurity, and risk factors for severe respiratory syncytial virus infection) only weight appeared to be significantly related to

the need for mechanical ventilation (coefficient -0.36682 (SE 0.1685), $p = 0.01$), while none of the other variables did. Prematurity and age were not independent risk indicators for mechanical ventilation in this study. In contrast to Lebel *et al*, who report odds ratios as 'relative risk', from the data of our prospective study we are able to estimate the absolute risk of mechanical ventilation related to the weight on admission (figure). A low weight is related to an increased risk for mechanical ventilation. For infants less than 5000 g, a relative risk for mechanical ventilation of 4.3 (95% confidence interval 1.3 to 13.9) was estimated.

H A VAN STEENSEL-MOLL

W J E TISSING

Department of Pediatrics, Sophia Children's Hospital, Erasmus University Rotterdam, Gordelweg 160, 3038 GE Rotterdam, The Netherlands

M OFFRINGA

Centre for Clinical Decision Analysis, Erasmus University Rotterdam, The Netherlands

J A HAZELZET

Department of Pediatrics, Subdivision of Pediatric Intensive Care, Sophia Children's Hospital, Erasmus University Rotterdam, Gordelweg 160, 3038 GE Rotterdam, The Netherlands

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Helicobacter pylori and protein losing enteropathy

SIR,—We have demonstrated a high prevalence of anti-*Helicobacter pylori* (formerly *Campylobacter pylori*) antibodies in children under the age of 3 years in The Gambia, West Africa.¹ Using a serological test validated by histology and microbiology, 41/77 (53%) of children with chronic diarrhoea were shown to have significantly high anti-*H pylori* IgG antibody titres, and indeed, the prevalence of *H pylori* antibodies in healthy asymptomatic children of the same age was also high at 26%.

It was against the background of these findings that we were interested in the description, in 1987, by Hill and colleagues, of transient protein losing enteropathy in association with acute infection with *H pylori*.² This observation might be due to chance but could have important consequences in the nutritional rehabilitation of infants with chronic diarrhoea and severe protein energy malnutrition.

Therefore we undertook a study to establish whether or not *H pylori* infection was associated with protein losing enteropathy in Gambian children with chronic diarrhoea.

Fifty three subjects (25 boys, 28 girls; mean age 19 months) were studied and all had chronic diarrhoea (more than three loose stools per day for more than two weeks) and severe protein energy malnutrition (32 marasmus, 21 marasmic-kwashiorkor).

After admission, three consecutive fresh stool samples were collected for virological, parasitological, and bacteriological investigation. Specific anti-*H pylori* IgG antibody was measured by enzyme linked immunosorbent assay (ELISA) in all subjects. Gastroscopy and antral mucosal biopsy were performed in 20/53 children.

Protein losing enteropathy was estimated by random faecal α_1 -antitrypsin measurement. Whole single stools were collected ensuring that both the liquid and solid phase were obtained. These were frozen and lyophilised and α_1 -antitrypsin measured by a single radial immunodiffusion method. Random faecal α_1 -antitrypsin measurement (normal mean (SD) value in healthy Gambian children = 1.54 (0.23) mg/g stool) has been shown to be a reproducible screening test for excessive enteric protein loss and has been validated against ^{51}Cr chromium labelled albumin excretion in the stool.³

Fifty six percent of this group of children had significantly raised anti-*H pylori* antibody titres and in 11/20 gastroscopied this was associated with recovery of the organism and histological gastritis. *Strongyloides stercoralis*, and *Giardia lamblia* were found in 11 and 38% of the patients respectively. Hypoalbuminaemia occurring in children with *S stercoralis* was found to be associated ($r = 0.952$, $p < 0.001$) with increased faecal α_1 -antitrypsin excretion (mean (SEM) = 2.47 (0.9) mg/g stool) whereas there was no difference between levels of faecal α_1 -antitrypsin found in children with (1.57 (0.2) mg/g stool) or without (1.62 (0.3) mg/g stool) evidence of *H pylori* infection.

Therefore, in this study we failed to show that chronic infection with *H pylori* is associated with protein losing enteropathy.

P B SULLIVAN

Department of Child Health, Westminster Children's Hospital, London SW1P 2NS

J E THOMAS

E J EASTHAM

Department of Child Health, University of Newcastle upon Tyne

P G LUNN

G NEALE

Dunn Nutritional Laboratory, Cambridge

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Probability of need for mechanical ventilation. Dotted lines show 90% confidence interval based on logistic model.

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Monitoring treatment in congenital adrenal hyperplasia

SIR,—Appan *et al* are to be congratulated on achieving normal growth in so many of their patients with congenital adrenal hyperplasia.¹ I would not wish paediatricians to conclude, however, that the management of this condition merely requires a standard dose of glucocorticoid and mineralocorticoid calculated from surface area to be chosen at birth and growth monitored thereafter until adulthood. The decision to adjust treatment on the basis of an abnormal change in growth velocity or skeletal maturation is inevitably a retrospective one. The authors concede that an advanced bone age can rarely be recovered. Why not try to predict such an occurrence before it is too late?

The biochemical indices to monitor control are by no means entirely reliable alone. They are certainly useful to detect problems with treatment compliance, particularly plasma testosterone values which are less influenced by good short term compliance. Appan *et al* conclude their paper by stating that frequent adjustment of medication is needed during periods of rapid growth in infancy and puberty. Periods of short term monitoring of biochemical indices along the lines recently recommended,² together with the use of standard auxological techniques in the longer term, is surely a sensible way to achieve this.

I A HUGHES

Department of Paediatrics,
Level 8, Addenbrooke's Hospital,
Hills Road, Cambridge
CB2 2QQ

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Herpes simplex infections in atopic eczema

SIR,—Patients with atopic eczema have an increased susceptibility to cutaneous viral infections, including herpes simplex, common warts, vaccinia, and molluscum contagiosum. This is thought to be due partly to a mild but definite depression of T cell function.¹

I was struck, however, by the apparent high incidence of the herpes simplex infection in children admitted to the Birmingham Children's Hospital with an acute exacerbation of atopic eczema. To test my observations, I did a retrospective study to find this incidence in those admitted over a period of two years and a prospective study over a period of one year.

Swabs were taken if herpes simplex was clinically suspected—that is, there were vesicles or pustules in a child with atopic eczema. The presence of the herpes simplex virus was tested by indirect immunofluorescence and cytopathic changes on tissue culture.

During the retrospective two year period, 10 out of 119 children were confirmed to have a herpes simplex skin infection. The prospective study, over a period of one year, showed eight out of 74 children with atopic eczema to

have a confirmed herpes simplex skin swab. Thus during the combined three year period, the incidence of the virus was approximately 10%. Two children had one recurrence and one child had two recurrences.

All children confirmed to have a herpes simplex skin infection improved after seven to 10 days even though two, initially, had developed severe systemic upset. They were all treated with a five day course of oral acyclovir. Herpes simplex was confirmed in all children who were clinically suspected of having the virus, reflecting the ease of diagnosis and of culture of the virus. The incidence of about 10% of herpes simplex infection in acutely exacerbated atopic eczema was also found by David and Longson.² This incidence may have been an underestimate as swabs were only taken on clinical suspicion that the virus was present. There may have been similar underestimations in our study. When infected eczema fails to respond to antibiotics within two days this is thought to suggest a herpes simplex infection.² It would be interesting to determine the incidence of the herpes simplex virus in acute eczema when it is not clinically obvious.

This study confirms my observation of the high incidence of herpes simplex in atopic eczema. It should always be considered in an acute exacerbation of atopic eczema as it can cause considerable associated systemic disease.

B J LIDDLE

The General Hospital,
Steelhouse Lane,
Birmingham B4 6NH

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Self management plans for asthma

SIR,—Comics and booklets, training courses for patients, asthma nurses—do they help to reduce morbidity in asthma? The evidence is at worst negative and at best contradictory. A consensus is emerging that self management is the key to good control and that a clearly written set of instructions—a self management plan—is the means to achieve this goal.

The article by Fletcher *et al* suggests that clear advice might have helped to prevent some deaths from asthma.¹ Two recent articles from *Thorax* come to similar conclusions.

Sibbald concluded that psychological factors were not major determinants of the responses of her adult asthmatic subjects to two hypothetical attacks of asthma.² Simple messages, teaching patients how to cope rather than trying to improve their knowledge of disease, would be most likely to reduce morbidity. A self management scheme of this type was assessed in an open study by Beasley and colleagues.³ A written plan and a peak flow meter were provided for each of the subjects. Patients improved over a seven month period, suggesting that the plan was successful. A controlled study, with an assessment of the (non-standard) advice given and an analysis of the important features which were responsible for improved control, will be essential sequels to this study.

Of immediate practical help, the National Asthma Campaign has recently introduced two children's asthma cards, based largely on a similar design used successfully at

Hammersmith Hospital for over 10 years.⁴ One card is for personal use by parents or child. The other is to be taken to school. There are sections for regular treatment, relief treatment, and emergency management. Brief guidelines are printed for the emergency doctor and for the parents or school teachers.

The cards are available at no charge from The National Asthma Campaign, 300 Upper Street, London N1 2XX. I urge you to use them.

M SILVERMAN

Royal Postgraduate Medical School,
Hammersmith Hospital,
London W12 0HS

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Effect of fever on recurrence rate of febrile convulsions

SIR,—The paper by Drs El-Radhi and Banajeh may be criticised on the grounds that they have combined data from retrospective and prospective studies for joint analysis.¹ Even if their results were to be accepted at face value, I suspect that they have drawn the wrong conclusion from their data. Rather than suggesting that the height of the initial fever provides stimulation of non-specific immunity, thereby reducing the chance of future infections (are we therefore misguided in trying to lower fever?), it is surely more reasonable to interpret their data by assuming that there is natural variability in the temperature threshold required for convulsion. Any individual may convulse if the temperature rises high enough, as in heat stroke, and as the incidence of febrile convulsion decreases with age, cerebral maturation appears to be associated with increase in temperature threshold required for convulsion. Their groups are clearly self selecting: those infants convulsing with temperatures below 39°C have a low temperature threshold and would be expected to have more attacks, as more infections cause pyrexias of 38-39°C than 40-41°C. Those requiring temperatures above 40°C will have less, both because high pyrexia is less common, and because their convulsion threshold will increase naturally with age to levels above those caused by infectious illness.

Perhaps we should be directing our attention to those infants who present with convulsion associated with low grade fever. Simple methods of fever control are less likely to prevent a pyrexia of 38.5°C than one of 40-41°C, and it may be appropriate to consider early introduction of anticonvulsant treatment in this group. This could be withdrawn after a relatively short period of six months to one year, as the infant's convulsion threshold may well have risen to levels where fever control alone is adequate.

S MURCH

Department of Child Health,
St Bartholomew's Hospital,
West Smithfield, London EC1A 7BE

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