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 volumes 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 mortality2 —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 $x^2$, $p<0.05$). 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 3 kg, a relative risk for mechanical ventilation of 4.3 (95% confidence interval 1.3 to 13.9) was estimated.

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3 MacDonald NE, Breeze Hall C, Saffin SC, Alexson C, Harris PJ, Manning JA. Respiratory syncytial virus infection in infants.

Heliocobacter pylori and protein losing enteropathy

Sir,—We have demonstrated a high prevalence of anti-Heliocobacter 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 immunobosorbent assay (ELISA) in all subjects. Gastroscopy and antral mucosal biopsy were performed in 20/53 children.

Protein losing enteropathy was estimated by random faecal a1-antitrypsin measurement. Whole single stools were collected ensuring that both the liquid and solid phase were obtained. These were frozen and lyophilised and a1-antitrypsin measured by a single radial immunodiffusion method. Random faecal a1-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 15chromium 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 Guardia lamblia were found in 11 and 38% of the patients respectively. Respiratory syncytial virus occurring in children with S stercoralis was found to be associated with ($r=0.952$, $p<0.001$) with increased faecal a1-antitrypsin excretion (mean (SEM)$=2.47 (0.9)$ mg/g stool) whereas there was no difference between levels of faecal a1-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.

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1 Sullivan PB, Thomas JE, Wight DGJ, et al. Heliocobacter pylori in Gambian children with...
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 irrespective one. The authors including warts, increased susceptibility to infection, improved plasma protein losing enteropathy associated with convulsions and convulsions due to metabolic failure.2

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 follow the 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 cytotoxic changes on tissue culture. During 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.3 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.

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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 convolution. Any individual may convulse if the temperature rises high enough, as in heat stroke, and as the incidence of febrile convolution decreases with age, cerebral maturation appears to be associated with increase in temperature threshold required for convolution. 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 convolution 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 convolution associated with low grade fever. Simple methods of fever control are less likely to prevent a pyrexia of 38–39°C than one of 40–41°C, and it may be better 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 convolution threshold may well have risen to levels where fever control alone is adequate.

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3] 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. 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 ongoing study by Beasley and colleagues. 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. These cards are for personal use by parents or the child. 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 from the National Asthma Campaign, 300 Upper Street, London N1 2XX. I urge you to use them.

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