LETTERS TO THE EDITOR

Dental enamel defects and coeliac disease

EDITOR,—The association between coeliac disease and dental enamel defects (DED) is already known.[1] These defects range from discolouring to pitting, grooving, and total loss of enamel, and are considered to be coeliac disease specific when distributed symmetrically and chronologically in all four sections of dentition. The Italian Society of Pediatric Gastroenterology and Hepatology (SIGEP) promoted a multicentre study for evaluating the prevalence of DED in a large group of Italian patients with coeliac disease.

A total of 603 children with coeliac disease were studied (327 girls, 276 boys, mean age 17.8 years) in 13 Italian centres for paediatric gastroenterology. All subjects had permanent or mixed (permanent plus primary) dentition. The diagnosis of coeliac disease had been made in all cases using the criteria of the European Society of Paediatric Gastroenterology and Nutrition.[2] The dental enamel inspection was performed in each centre by a paediatric gastroenterologist experienced in identifying enamel defects, with a dentist present. A group of 6949 schoolchildren (mean age 12.4 years) served as healthy controls. When a subject from the control group presented with DED, an antiendomysium antibody assay was performed (indirect immunofluorescence, Endomisium Eurospital, Trieste, Italy), leading to small bowel biopsy in subjects testing positive. Student's *t* test was used for statistical analysis.

Altogether, 195 of our 603 patients with coeliac disease (32 4%) had systematic enamel defects with a fair degree of variation between centres (table 1). Mean age at diagnosis of coeliac disease was significantly higher in the group with DED (8.1 *v* 4.1 years, $p<0.01$). In the group of 6949 healthy controls, specific DED were found in 52 subjects (0.59%) ($p<0.00001$ in respect of the coeliac disease patients). Ten of them tested positive for antiendomysium antibodies, nine underwent intestinal biopsy (one refusal), and four had a flat mucosa.

DED are therefore present in one third of Italian patients with coeliac disease, with a prevalence lower than that found in the Finnish studies[1] but higher than that found in UK.[3] This study also suggests that DED may be connected to late diagnosis of coeliac disease. The mechanism of development of DED in coeliacs is not clear, but it seems more likely that they are the consequence of immune mediated enamel damage rather than related to malnutrition. In fact, similar lesions that appear to be associated with HLA DR3 aplotype,[4] are common in autoimmune disease (such as polyendocrinopathy), but are rare in nutrition disorders such as rickets. Our study also confirms that coeliac disease can be symptomless or can present with an atypical clinical picture. We recommend that subjects with symmetrical DED in permanent teeth undergo serological testing for antiendomysium antibodies and intestinal biopsy when testing positive.

ALESSANDRO VENTURA
STETANO MARTELOSSI
* (COORDINATORS)
SIGEP Study Group on Dental Enamel Defects in Coeliac Disease
*Istituto di Clinica Pediatrica, Universita' di Pisa and * Istituto per l'Infanzia IRCCS (Burlo Garofolo), Trieste, Italy*

Correspondence to: Professor Alessandro Ventura, Istituto di Clinica Pediatrica, Ospedale S Chiara, Via Rome 67, 56100 Pisa, Italy.

Participants to the Italian Society of Paediatric Gastroenterology and Hepatology DED study group: R Lazzari, C Corvaglia (Bologna), S De Virgilis (Cagliari), A Miano, M Marani (Cesena), F Bascietto (Chieti), M Sanfilippo (Fiesole), G Magazzu' (Messina), A M Giunta, I Prampolini (Milano, 'De Marchi'), C Bianchi, G. Barera (Milano 'S Raffaele'), F Balli, P Bertolani (Modena), F Cataldo (Palermo), G Maggiore (Pisa), M Bonamico (Roma), M S Scotta (Varese).

1 Aine L, Maki M, Collin P, Keyrilainen O. Dental enamel defects in coeliac disease. *Oral Pathol Med* 1990;**19**:241-5.
2 Report of Working Group of European Society of Paediatric Gastroentcrology and Nutrition. Revised criteria for diagnosis of coeliac disease. *Arch Dis Child* 1990;**65**:909-11.
3 Ballinger A, Huges C, Kumar P, Hutchinson I, Clark M. Dental enamel defects in coeliac disease. *Lancet* 1994;**343**:230-1.
4 Mariani P, Mazzilli MC, Margutti G, *et al.* Coeliac disease, enamel defects and HLA typing. *Acta Paediatr* 1994;**83**:1272-5.

Table 1 Prevalence of DED in 603 Italian children with coeliac disease (CD) in 13 paediatric gastroenterology departments. The correlation with the age of diagnosis of CD is shown

Centre	*No with CD*	*Positive (%) for DED*	*Mean age of diagnosis of CD (years) Total*	*With DED (years)*	*Without DED*
Chieti	31	26 (83)	4.3	6.2	2.3
Milano	40	30 (75)	6.6	8.4	5.0
Varese	33	17 (55)	6.3	6.9	5.1
Trieste	90	48 (53)	10.1	13.1	7.1
Fiesole	4	2 (50)	4.6	6.2	3.5
Modena	25	12 (48)	8.2	9.4	7.1
Milano 2	37	15 (40)	6.7	5.1	7.2
Messina	23	7 (30)	8.4	9.5	7.2
Cagliari	85	14 (16)	10.4	15.5	5.3
Bologna	77	12 (15)	6.1	8.4	3.1
Roma	82	10 (12)	4.5	4.2	5.2
Cesena	46	2 (4)	7.6	11	4.7
Palermo	30	0	1.5	–	1.5
Total	603	195 (32)	6.5	8.1	4.1

Iridoplegia in severe Guillain-Barré syndrome

EDITOR,—We report a child with rapidly progressive and severe acute Guillain-Barré syndrome (GBS) with complete ophthalmoplegia (ptosis, loss of eye movements, and pupillary paresis).

CASE REPORT

A previously well 13 year old boy presented with a seven hour history of increasing breathlessness, aching of his facial and neck muscles, and a rapidly progressive weakness of all four limbs. The patient had complained of a mild coryzal illness for the previous five days. The patient was afebrile and there was no evidence of rash, tick bite, or lymphadenopathy. He was 'alert' and his breathing pattern was shallow. Neurological examination demonstrated marked hypotonia and diffuse muscle weakness (Medical Research Council (MRC) classification grade 2); muscle stretch and abdominal reflexes were absent. Plantar responses were flexor. Extraocular eye movements were absent; there was some movement of the eyelids (MRC grade 1–2) and pupillary responses to accommodation and light (direct and consensual) were normal. Because of significant hypoventilation, the patient required immediate intubation and ventilatory support. Three doses of intravenous immunoglobulin at 1 g/kg/dose were administered over three consecutive days.

By the third day of admission paralysis was total, with no movement seen in any muscle. Autonomic dysfunction was evident from day one through day 15 of admission, manifested by labile blood pressure, a sinus tachycardia, sweating and flushing, urinary incontinence, and constipation. From the third day of admission the patient's pupils were reacting sluggishly to light; by the fourth day the pupils were 4 mm in diameter and unreactive to light and accommodation. Resolution of the iridoplegia began on the 11th day of admission, initially to light and subsequently to accommodation; the pupils had returned to normal (size and reactivity) by day 15.

Recovery from paralysis began on the 14th day in a cephalocaudal progression, beginning with blinking of the eyelids and lateral movements of the head. The patient remained ventilator dependent for 59 days. Ten months after admission, the patient is able to walk unaided. The muscle power in the upper limbs (proximally and distally) is grade 4, and in the proximal and distal lower limbs, grade 4 and 2 respectively.

Magnetic resonance imaging of the head and spine were normal. Protein concentrations in the cerebrospinal fluid were 0.54 and 4.46 g/l on the first and seventh days of admission respectively (upper limit of normal 0.45 g/l); the rest of the cerebrospinal fluid biochemistry and white cell counts were normal. Urine porphyrins were negative. Bacterial and viral culture and viral antibody titres were negative. Electrophysiology, undertaken on day 40 of admission, demonstrated absent sensory action potentials and a markedly delayed M response from the right biceps, consistent with a demyelinating motor (and sensory) neuropathy with severe secondary axonal degeneration.

The clinical features and investigations in this patient were entirely consistent with a diagnosis of acute GBS, and diphtheria or a brainstem lesion were excluded. The progression was extremely rapid and the patient's

recovery has been gradual but sustained, and is not yet complete. Pupillary involvement in GBS appears to be rare as it is not cited in at least one reference text[1]; furthermore, it has been stated that ocular pupillary abnormalities do not occur in GBS.[2] However, pupillary abnormalities have been reported in seven adults with GBS; all except one required ventilatory support and two (the non-ventilated, and one of the ventilated patients) died of a cardiac arrhythmia.[3–8] Although there may be a reporting bias, pupillary involvement would appear to be associated with severe disease and an increased risk of dying. The pathophysiology is unclear but almost certainly represents a manifestation of autonomic dysfunction. Pharmacological testing previously has suggested either simultaneous postganglionic involvement of sympathetic and parasympathetic nerves or isolated parasympathetic involvement.[4] In our patient, pupillary paresis developed at the height of autonomic instability. It appeared to affect both sympathetic and parasympathetic nerves, although this was not confirmed pharmacologically. Bilateral ptosis and failure of full pupillary dilatation in the dark indicated sympathetic involvement, while absence of light and consensual responses suggested parasympathetic involvement. The 'mid-point' dilatation of the pupils was probably due to the presence of intrinsic muscle tone within the pupillary muscles. We believe that the recognition of iridoplegia as an associated feature of GBS is important and its presence warrants intensive monitoring.

JEREMY C C HUNG
RICHARD E APPLETON
The Roald Dahl EEG Unit, Royal Liverpool Children's Hospital, Alder Hey, Eaton Road, Liverpool L12 2AP

1 Hughes RAC. *The Guillain-Barré syndrome*. London: Springer-Verlag, 1990.
2 Jones HR Jr. Childhood Guillain-Barré syndrome: clinical presentation, diagnosis, and therapy. *J Child Neurol* 1996;**11**:4-12.
3 Anzai T. Uematsu D, Takahashi K, Katayama T. Guillain-Barré syndrome with bilateral tonic pupils. *Intern Med* 1994;**33**:248-51.
4 Williams D, Brust J, Abrams G, Challenor Y, Devereaux M. Landry-Guillain-Barré syndrome with abnormal pupils and normal eye movements: a case report. *Neurology* 1979;**29**: 1033-6.
5 Fuller GN, Jacobs JM, Lewis PD, Lane RJ. Pseudoaxonal Guillain-Barré syndrome: severe demyelination mimicking axonopathy. A case with pupillary involvement. *J Neurol Neurosurg Psychiatry* 1992;**55**:1079-83.
6 Heafield MT, Gammage MD, Nightingale S. Williams AC. Idiopathic dysautonomia treated with intravenous gammaglobulin. *Lancet* 1996; **347**:28-9.
7 Hassan T, Mumford C. Guillain-Barré syndrome mistaken for brain stem death. *Postgrad Med J* 1991;**67**:280-1.
8 Coad NR, Byrne AJ. Guillain-Barré syndrome mimicking brain stem death. *Anaesthesia* 1990; **45**:456-7.

Predictive value of preschool surveillance in detecting learning difficulties

EDITOR,—In trying to predict learning difficulties using preschool data, Corrigan *et al* have overlooked some basic principles of health surveillance and screening for developmental problems.[1] Detection of learning difficulties is a reasonable goal of health surveillance. However health surveillance does not include using data from one point in time to predict problems seven to eight years later.[2] In fact, the efficacy of preschool developmental screening as a global phenomenon is still not clear and certainly has not been shown to be useful in detecting mild to moderate learning difficulties.[3] There is also generally a poor correlation between perinatal events and subsequent learning outcomes except in extreme cases.

Predicting learning difficulties using data from the preschool or even neonatal period also seems a paradox given that learning difficulties are by definition related to educational problems that occur in schools. The authors have supported this theory by specifically choosing children who had been at school a minimum of two years, thereby allowing teachers time to detect learning problems.

The outcome measures of developmental delay or learning difficulties in the preschool period that are used in this study are vague and both require further definition. The diagnosis of developmental delay using referral to a psychologist or documentation in the child health record would fit best with surveillance methodology rather than an outcome measure. Preschool learning difficulties, once again seem to be an incongruent concept given the previous definition.

The results do not seem to answer any hypotheses of clinical relevance and this is evident by the fact that being a single mother in the neonatal period appeared to be protective against learning disorders, when in fact we know the opposite is true.

In conclusion, the authors have reiterated the supposition that has been part of the literature for a number of years; learning difficulties cannot be assessed until the child is in an educational facility. Mild learning difficulties may be a reflection of maturational variability and the importance of detecting this in the preschool period is still being debated. There is no doubt that it is important to detect learning problems early in a child's schooling and services should be in place to support these children.[4] There seems little point in mounting extensive surveillance programs in an attempt to predict difficulties years later. Outcomes of health surveillance should be rather directed to interventions which can be implemented in the present.

SHARON GOLDFELD
THERESA LAZZARO
FRANK OBERKLAID
Centre for Community Child Health and Ambulatory Paediatrics, Royal Children's Hospital, Flemington Road, Parkville, Victoria 3052, Australia

Drs Corrigan and Stewart comment:
That Dr Goldfield *et al* have missed the basic premise of this paper is evident in their opening comment. Our aim was not to predict learning difficulties using preschool data but rather to challenge the scientific basis of the existing system of child health surveillance which claimed early detection of mild to moderate learning difficulties as a stated goal.[1]

We agree entirely with their concerns regarding the efficacy of preschool developmental screening and the difficulties in correlating perinatal events and subsequent learning outcomes. It was these concerns, and the challenges of Professor Hall's report *Health for all Children*,[5] that prompted our original research. The preschool outcome measures quoted, which Dr Goldfield *et al* correctly refer to as vague, are those actually recorded by the preschool surveillance team. This is the reality of preschool developmental screening that we set out to challenge.

We demonstrated that not only did the system fail to identify children as at risk of later learning difficulties in the preschool period but it was impossible to use the data recorded to develop a useful predictive model. This challenges a basic premise of a system that is enormously expensive in terms of both health care resources and parental time and energy. In a world of financial constraints and evidence-based practice we would suggest that the clinical relevance of such a finding is self evident.

Finally, we would agree that there seems little point in mounting an extensive surveillance programme to predict later learning difficulties if this cannot be shown to be both sensitive and specific and offer proved interventions to aid the children identified as at risk by it. Far from disagreeing with our conclusions we would consider these points integral to them.

1 Corrigan N, Stewart M, Scott M, Fee F. Predictive value of preschool surveillance in detecting learning difficulties. *Arch Dis Child* 1996;**74**:517-21.
2 Keogh BK, Becker LD. Early detection of learning problems: questions, cautions and guidelines. *Exceptional Children* 1973;41(1):5-11.
3 Dworkin P. Developmental screening: (still) expecting the impossible. *Pediatrics* 1992;**89**: 1253-5.
4 Oberklaid F. It's time: the future of school health in Australia. *J Paediatr Child Health* 1990;26:244-251.
5 Hall DMB. *Health for all children*. 2nd Ed. Oxford: Oxford University Press, 1991.

Bisphosphonates in osteogenesis imperfecta

EDITOR,—I was interested to read the recent articles by Allgrove[1] and Williams *et al*[2] on the use of bisphosphonates in children and would like to report my experience of their use in a child with osteogenesis imperfecta. The patient was referred to me age 9 years with a history of recurrent fractures since infancy and a family history consistent with osteogenesis imperfecta. She had a nine month history of low back pain, evidence of a thoracolumbar kyphosis, and tenderness over the thoracolumbar spine. She had become wheelchair bound following a fractured femur three months previously and a recent hospital admission with urinary and faecal incontinence was associated with upper motor neurone signs in her legs. Radiography of her spine showed collapse of numerous vertebrae in the thoracic and lumbar spine with marked osteoporosis. Routine biochemistry showed no abnormality of serum calcium, phosphate, alkaline phosphatase, parathyroid hormone, or 25-hydroxyvitamin D and a normal urine calcium/creatinine ratio. Bone density of the lumbar spine (L_2–L_4), using a Lunar DPX-L DXA scanner with the paediatric software, was 0.395 g/cm^2 with a Z score for age of −4.0. After discussion with her parents it was decided to treat her with bisphosphonates. This was initially with pamidronate 0.5 mg/kg given intravenously every three months for six months which was then increased to 1 mg/kg/day for two consecutive days for a further six months. Because of difficulties with venous access this was then changed to etidronate given orally for a period of two weeks in every three month period in a dose of 600 mg/day (9 mg/kg) which has continued for the past nine months. No adverse effects were seen during the period of treatment.

Change in bone mineral density (BMD)

	Lumbar spine		*Whole body*	
Time (months)	*BMD (g/cm²)*	*Z score*	*BMD (g/cm²)*	*Z score*
0	0.395	−4.0	0.731	−1.78
6	0.509	−2.83	0.791	−1.02
18	0.709	−1.51	0.895	−0.25

The change in bone density during this period is indicated in table 1. There was a 44% increase in the bone density of the lumbar spine with no further fractures occurring during this time. This has been accompanied by a progressive improvement in her mobility such that she is now walking with the aid of a rollator and uses the wheelchair only for long distances. She now has no evidence of spinal deformity. Although it could be argued that some of this improvement was due to the onset of puberty, the improving Z score indicates an effect independent of changes in body size. Thus this case supports other reports indicating the potential benefits of bisphosphonates in osteogenesis imperfecta, although it is important that there is careful selection of cases, and as indicated by Allgrove that monitoring of bone biochemistry and density is undertaken. The need for appropriate informed consent is also essential.

N J SHAW
Metabolic Bone Disease Clinic, Department of Endocrinology, Birmingham Children's Hospital, Ladywood Middleway, Ladywood, Birmingham B16 8ET

1 Allgrove J. Biphosphonates. *Arch Dis Child* 1997;**76**:73-5.
2 Williams CJC, Smith RA, Ball RJ, Wilkinson H. Hypercalcaemia in osteogenesis imperfecta treated with pamidronate. *Arch Dis Child* 1997; **76**:169-70.

White matter attenuation and megalencephaly

EDITOR,—In 1985 one of us (ROR) published a case of unusual self resolving leukodystrophy.[1] We now describe a second case. As with the first, neuroimaging was prompted by a large head size. At 14 months, she had an occipitofrontal circumference of 53.5 cm some 4 SDs above the mean. Her father's head circumference was similarly increased being 63 cm. His father's head was also said to have been large. Unfortunately the patient's head circumference at birth was not available. Apart from a slight delay in acquisition of head control and sitting attributable to the mechanical disadvantage of her large head, her development up to that time was age appropriate. Computed tomography showed pronounced low attenuation throughout the cerebral white matter (see fig 1A). Investigations for progressive leukodystrophy including relevant lysosomal enzyme studies, very long chain fatty acids, peripheral neurophysiology, and urine for *N*-acetyl aspartate were all normal. A second computed tomogram at 2.2 years showed partial resolution of the white matter changes in all areas, but to a lesser extent in the frontal lobes (see fig 1B).

Her head growth continued parallel with the normal growth trajectory at 3.5 to 4 SDs above the mean. When seen aged 8.6 years, her occipitofrontal circumference was 57 cm. She was otherwise normal neurologically and academically functioning around the mean. Magnetic resonance imaging of her brain at this stage showed slight increase in signal in the terminal myelination areas and a small discreet high signal area in the anterior frontal lobe of uncertain aetiology and unlikely to be of clinical significance.

This second case confirms that occasionally children with familial macrocephaly initially have pronounced white matter changes which appear alarming but are nevertheless benign.

R O ROBINSON
T COX
Newcomen Centre, Guy's Hospital, St Thomas' Street, London SE1 9RT

1 Robinson RO. White matter attenuation and megalencephaly. *Arch Dis Child* 1985;**60**:1079-81.

Salmonella meningitis acquired from pet snakes

EDITOR,—Salmonellosis associated with reptiles is well documented[1–3] but a proved link between salmonella meningitis and reptilian carriage has not been reported.

Salmonella uzaramo was isolated from the cerebrospinal fluid, blood, and stool cultures of a 5 month old white boy, admitted with symptoms, signs, and typical findings of bacterial meningitis. Treatment with cefotaxime 200 mg/kg daily for three weeks appeared to provide a rapid clinical response but the child was readmitted three days after discharge with vomiting and irritability. *S uzaramo* was reisolated from the cerebrospinal fluid. Ciprofloxacin was added to cefotaxime and this time the infection was successfully eradicated, the child being discharged after a further three weeks with normal cerebrospinal fluid and no apparent neurological deficit.

The family owned three snakes: an Indian python and two Royal pythons, which lived in two separate tanks in the dining room. The Indian python was frequently handled and roamed freely around the house. Faeces samples from the Indian python grew *S uzaramo*; faeces from the Royal pythons grew *S uzaramo*, *S arizonae*, *S lome* and an unnamed salmonella species. None of the family members had suffered significant diarrhoeal episodes and stool samples were negative.

Import regulations on reptiles have been significantly eased in recent years and a large market in reptile purchase and exchange exists in the UK. Most reptile owners are probably unaware of the likelihood of salmonella carriage in their pets, even though carriage of salmonella species in reptiles is almost universal (as high as 94%).[4] Adequate information is not provided to the potential purchaser on possible health risks and the importance of hand washing: young children are at particular risk of serious infection, the majority of salmonella meningitis cases occurring in patients under 1 year. Investigators of cases of salmonella infection should be aware of the possible significance of reptiles or exotic pets and liaise with local microbiology departments; faeces samples can be obtained relatively easily from lizards and snakes.

J H PATON
M B MIRFATTAHI*
*Department of Microbiology and Department of Paediatrics**, *Queen's Hospital, Belvedere Road, Burton upon Trent, Staffordshire DE13 0RB*

1 Paton JH. A young man who liked lizards and lost his job. *Lancet* 1996;**347**:1376.
2 Makin G, Abu-Harb M, Finn A, *et al*. Salmonella durban in an infant. *Lancet* 1996; **348**:200.
3 Fonseca RJ, Dubey LM. Salmonella montevideo sepsis from a pet snake. *Pediatr Infect Dis J* 1994;**13**:550.
4 Chiodini RJ, Sundberg JH. Salmonellosis in reptiles: a review. *Am J Epidemiol* 1981;**113**: 494-9.

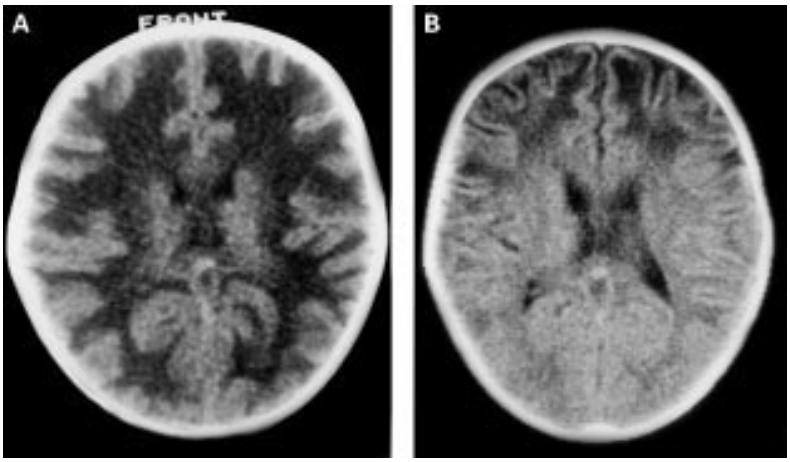

Figure 1 Computed tomograms showing (A) pronounced low attenuation throughout the cerebral white matter and (B) partial resolution of white matter changes but to a lesser extent in the frontal lobes.

Randomised controlled trials

EDITOR,—We read with interest the recent editorial on evidence-based child health.[1] We were struck by how easy it is to overlook a randomised controlled trial (RCT) in the *Archives* (or any other journal) if this is not indicated in the title or abstract. We wondered if this was the case in commonly used bibliographic databases, such as Medline.

With the help of the Cochrane Collaboration Cystic Fibrosis Group, we retrieved the results of a handsearch for RCTs from the *Archives* from 1986–90. Each RCT (a trial in which participants were allocated to groups by random allocation, excluding pseudorandom methods such as date of birth or case number allocation) was assessed as to whether or not it contained the word

'random' or some variant in the title or abstract/summary section.

These results were compared with the simple (and easily remembered) Medline search strategy of 'random*'—where * is the truncation character.

The simple search strategy found two thirds (59/90) of the handsearch defined RCTs, of which 78% (46/59) had the word 'random' or variants in the title or abstract. However, of the 31 RCTs which failed to be retrieved by this strategy, none (0/31) had 'random' or variants in the title or abstract ($p<0.001$).

We propose that the *Archives* adopts some of the CONSORT recommendations for the reporting of RCTs,[2] by encouraging authors to include the word 'randomised' in the title and abstract of all these papers, enabling easier retrieval of useful clinical studies.

ROBERT PHILLIPS
TAMAR ROTHENBERG
Oxford Medical School, John Radcliffe Hospital, Oxford OX3 9DU

1 Gilbert R, Logan S. Future prospects for evidence-based child health. *Arch Dis Child* 1996;**75**:465-8.
2 Begg C, Cho M, Eastwood S, *et al*. Improving the quality of reporting of randomized controlled trials. The CONSORT statement. *JAMA* 1996;**276**:637-9.

The editors comment:
We agree and we are currently adopting this proposal.

Plasma E-selectin and ICAM-1 in acute Henoch-Schönlein purpura

EDITOR,—We were interested in the paper by Söylemezoğlu *et al* concerning circulating soluble adhesion molecule expression in 20 children with acute Henoch-Schönlein purpura and 12 normal controls.[1]

We recently performed enzyme linked immunoassays (ELISAs) for ICAM-1 and E-selectin in the plasma of 41 children presenting to the Royal Hospital for Sick Children in Glasgow with acute Henoch-Schönlein purpura. Samples were analysed using commercially available ELISA kits for ICAM-1 (Predicta, Genzyme Diagnostics, Cambridge, MA, USA) and E-selectin (Parameter, R&D Systems Europe, Abingdon). Results were compared with reference ranges provided by the manufacturers.

Our results broadly support the findings of Söylemezoğlu *et al.* Of 41 paired measurements, ICAM-1 was raised in one case only, in keeping with the results reported by Söylemezoğlu where acute and control levels did not differ (though there was a difference between acute and convalescent samples). E-selectin however, was raised in 12 (29%) of our cases. In those reported by Söylemezoğlu there was no significant difference in E-selectin between active or inactive Henoch-Schönlein purpura and normal controls.

These data therefore confirm the findings of Söylemezoğlu *et al* in that we are unable to demonstrate increases in soluble ICAM-1 or E-selectin in the majority of paediatric patients with acute Henoch-Schönlein purpura. It is our clinical impression that the raised E-selectin levels found in a minority of our subjects may reflect the presence of more severe systemic and renal vasculitis (seven of the 12 having significant proteinuria and/or haematuria). This however requires confirmation by a prospective study comparing inflammatory markers and clinical status.

F R WILLIS
Princess Margaret Hospital for Children, Perth, Western Australia
G C SMITH
Cardiff Royal Infirmary, Cardiff
T J BEATTIE
Renal Unit, Royal Hospital for Sick Children, Yorkbill, Glasgow G3 8SJ

1 Söylemezoğlu O, Sultan N, Gursel T, Buyan N, Hasanoğlu H. Circulating adhesion molecules ICAM-1, E-selectin, and von Willibrand factor in Henoch-Schönlein purpura. *Arch Dis Child* 1996;**75**:507-11.

Acquired rectovaginal fistulae in South Africa

EDITOR,—Acquired rectovaginal fistula associated with HIV positive children is a new and rather curious entity.[1] The underlying pathology has been reported to be chronic diarrhoea with abscess formation in the anterior rectal wall; after breakdown of the abscess, a persistent fistula appears.[2] We have seen seven such infants with fistulae in the past three years. We treated all these eases with diversion sigmoid colostomy. Biopsy of the fistula confirmed the presence of chronic inflammatory cells only. Our result with colostomy has been disappointing, with no spontaneous healing so far.

We would like to point out that in all these cases the fistula tract was from the upper anal canal to the vestibule just posterior to the introitus, thus making this condition a rectovestibular fistula, rather than rectovaginal.

Furthermore although HIV is implicated in five of our cases, we also have had two cases of infant girls with this condition who were HIV negative on enzyme linked immunosorbent assay (ELISA) test. Their respective mothers also tested negative on ELISA testing. Curiously both of these children suffered from acute onset of diarrhoea one week before the fistula appearance. No causative organisms were grown from their stool cultures. If one is to take account of these two cases, rectovestibular fistula may be caused by a specific but as yet undescribed organism, which is more prevalent in HIV positive infants.

B BANIEGHBAL
J FONSECA
Department of Paediatric Surgery, Baragwanath Hospital, Soweto, Witwaterstrand University, Johannesburg, Republic of South Africa

1 Hyde GA, Sarabah S. Acquired rectovaginal fistula in human immunodeficiency virus-positive children. *Pediatrics* 1994;**84**:940-1.
2 Borgstein ES, Broadhead RL. Acquired rectovaginal fistula. *Arch Dis Child* 1994;**70**:165-6.

WESTMINSTER BRIEFING

The following items are from *Children & Parliament*, Winter 1996–7. *Children & Parliament* is an abstracting service based on *Hansard* and published fortnightly by the National Children's Bureau while parliament is sitting. It covers all parliamentary business affecting children and is produced in either printed or CD-ROM form. Both are available on subscription from the Library and Information Service, National Children's Bureau, 8 Wakley Street, London EC1V 7QE (tel: +44(0)171 843 6035). (The *Hansard* reference is given first followed by the date of *Children & Parliament*.)

Government replies to questions about attention deficit hyperactivity disorder (ADHD) placed responsibility on local education authorities and professionals. Asked whether pupils suspended from school more than three times would be assessed for ADHD the Under Secretary of State replied that the decision about assessment should be made by the local education authority after appropriate referral. The performance of paediatricians and child psychiatrists in diagnosing ADHD was a matter for the royal colleges and for local audit.
(20, 21, 22 Jan 97, Cols 463-464, 516, 663-664; 4.2.97)

The average annual cost to the NHS of family planning services is £40 per patient.
(13 Jan 97, Col 174; 4.2.97)

In a 1993 survey of 14–25 year olds some 17% said that they had been victims of non-sexual violence in the past year.
(18 Dec 96 Col 714-715; 4.2.97)

The labelling of baby milks is controlled by two regulations, the Food Labelling Regulations 1996 and the Infant Formula and Follow-on Formula Regulations 1995. Infant formula labelling must include a statement about the superiority of breast feeding, a recommendation for use only on professional advice, advice about preparing feeds, and a warning about the risks of wrongly preparing them.
(18 Dec 96, Col 647; 4.2.97)

There were 654 confirmed meningococcal infections between 1 July and 22 December 1995 and 585 during the same period in 1996. The Health Education Authority (HEA) has produced a leaflet about meningitis aimed at students and the HEA guide to childhood immunisations includes advice on how to recognise the disease.
(17 Jan 97, Col 410-411; 4.2.97)

In 1995 in England and Wales 364 girls and young women of 18 and under were cautioned or convicted of loitering or soliciting for prostitution; 48 were under 16.
(20 Jan 97, Col, 45-46; 4.2.97)

Just over 2500 people each year are convicted of sexual offences against children under 16 in English and Welsh courts. The number was 2682 in 1991 and 2580 in 1995.
(21 Jan 97, Col 52-56; 4.2.97)

Of people convicted of a sexual offence against a child just over half have a previous conviction for any indictable offence, around 15% for any sexual offence, and around 10% for child sexual abuse.
(22 Jan 97, Col 595-596; 4.2.97)

The number of notified cases of tuberculosis in people aged 19 or under in England and Wales was 730 in 1992, 785 in 1993, 615 in 1994, 631 in 1995, and 429 in the first 39 weeks of 1996.
(17 Jan 97, Col 411; 4.2.97)

About 177 000 legal abortions are performed annually in Great Britain.
(30 Jan 97, Col 333-334; 18.2.97)

The mean number of decayed, missing, or filled teeth in 5 year olds in England in 1987–8 was 1.73 and in 1993–4 it was 1.74. The corresponding figure for 12 year olds was 1.49 in 1988–9 and 1.15 in 1992–3.
(28 Jan 97, Col 161-162; 18.2.97)

The government's estimate of the proportion of UK gross domestic product spent on health is 4.7% for 1979–80, 5.2% for 1990–1, and 5.8% for 1995–6.
(6 Feb 97, Col 722; 18.2.97)

There were 53 UK deaths from abuse of butane gas lighters and refills in 1990, 37 in 1991, 39 in 1992, 36 in 1993, and 28 in 1994. Most of the deaths were concentrated in the 15–19 age group for which the corresponding figures were 39, 29, 20, 21, and 14.
(5 Feb 97, Col 641; 18.2.97)

The Adoptive Mothers (Maternity Leave) Bill which had its first reading in February seeks to give adoptive mothers the same maternity benefits and employment rights as biological mothers.
(11 Feb 97, Col 143-145; 4.3.97)

Published perinatal mortality rates in European Union countries in 1993 varied from 5.1 per 1000 births in Finland to 9.1 in the UK but the figures are not comparable because the minimum gestation defining stillbirth varies from 22 to 28 weeks in different countries.
(10 Feb 97, Col 13; 4.3.97)

Just under 2000 children in the UK were treated with human pituitary growth hormone between 1959 and 1985. A judge has found the Department of Health to have been negligent 'in certain aspects' beginning on 1 July 1977. Appeal against the judge's decision is possible for those who had treatment before that date.
(24 Feb 97, Col 123-130; 4.3.97)

At the Institute of Child Health, London 20 former patients are known to have died of Creutzfeldt-Jakob disease and two have symptoms of the disease.
(25 Feb 97; Col 183, 234; 4.3.97)

Around 2% of the abortions performed in England and Wales are on girls under 16. The number of girls having an abortion fell from 4075 in 1987 to 3167 in 1992 but rose each year after that to reach 3401 in 1995.
(18 Mar 97, Col 543-544; 1.4.97)

In 1995 about one in three births in England, Wales, and Scotland was to an unmarried mother. In Northern Ireland it was less than one in four.
(20 Mar 97, Col 793-794; 1.4.97)

In January 1996 the proportion of pupils in each local education authority in England who were statemented for special educational needs varied from 1.97 to 4.59%.
(10 Mar 97, Col 2-4; 1.4.97)

The number of tonsil and adenoid removals in children in each health authority in England in 1994-95 varied from 9.4 to 156.8 per 10 000 children under 16. The health authority with a rate of 9.4 was the only one less than 34 per 10 000. The mean rate was 71.6 with 71717 operations performed nationally.
(20 Mar 97, Col 798-800; 1.4.97)

MORE MEETINGS IN 1997

ESPGAN Summer School: Paediatric Gastroenterology and Nutrition
30 August–6 September, Stockholm, Sweden
Further details: Dr Yigael Finkel, Paediatric Gastroenterology, St Görans Children's Hospital, S-112 81 Stockholm, Sweden

Global Strategies for the Prevention of HIV Transmission of Mothers to Infants
3-6 September, Washington, DC, USA
Further details: Conference on Global Strategies, 7101 Wisconsin Avenue, Suite 1300, Bethesda, MD 20814, USA

British Society of Endocrinology and Diabetes
23–24 October, London
Further details: Dr R Stanhope, Department of Endocrinology, Great Ormond Street Hospital for Children NHS Trust, Great Ormond Street, London WC1N 3EH

Commonwealth Congress on Diarrhoea and Malnutrition
21–24 November, Karachi, Pakistan
Further details: Dr Z A Bhutta, Department of Paediatrics, Aga Khan University Hospital, PO Box 3500, Karachi 74800, Pakistan

National Symposium on Angelman Syndrome
29 November 1997, Brussels, Belgium
Further details: Dr B Can, 147 Avenue du Parc, 1190 Brussels, Belgium

Hot Topics '97 in Neonatology
7–9 December, Washington, DC, USA
Further details: A Lynn Stillman, Registrar, 52 Overlake Park, Burlington, VT 05401, USA

Eighth Annual Course in Paediatric Gastroenterology
8–10 December, London
Further details: Professor J A Walker-Smith, University Department of Paediatric Gastroenterology, Royal Free Hospital, London NW3 2QG

Correction

Several errors occurred in the review of *Behavioural Phenotypes*, edited by Gregory O'Brien and William Yule, published in the April issue of the journal. The correct details are 250 pages; price £37.50 hardback; ISBN 1-898-68306-9. The book is published by Cambridge University Press/MacKeith Press Publications.

Notice
There are a limited number of indexes for the Fetal and Neonatal Edition available. If you would like a copy please write to: Ms Sue Heels, *Archives of Disease in Childhood,* BMA House, Tavistock Square, London WC1 9JR.