LETTERS TO THE EDITOR

Long term follow up of children born to mothers with periconceptional multivitamin supplementation

EDITOR,—Before recommending periconceptional folic acid-containing multivitamin supplementation universally for the prevention of neural tube defects one would also want to be sure that it was doing no harm. A UK study showed the over-representation of worries and anxiety among children at age 7–10 years born to mothers with periconceptional multivitamin supplementation.1 In our previous study based on a short term postnatal follow up of 3356 infants (mean age 11 months) born to mothers supplemented with multivitamin or placebo-like trace element1 in the periconceptional period, the results of the tests of mental and behavioural development were similar in the two groups. However, atopic dermatitis was found more often in the multivitamin group. We now summarise the results of long term follow up to the ages of 2 and 6 years with respect to the occurrence of atopic dermatitis (and other allergies) and anxiety.

The children were born to mothers who had been supplemented with multivitamin (including 0.8 mg of folic acid) or trace elements in the Hungarian randomised, double blind, controlled trial.2 Altogether 200 case and 200 control children were randomly selected with equal sex distribution for each age group (2 and 6 years) and they were examined by a double blind method. Of the 800 invited children, 625 (78%) took part in the study, 336 (84%) in the 2 year and 289 (72%) in the 6 year age groups. Parents of 120 children did not want to participate, 44 families had a new unknown address, three children died after the short term follow up study, four had severe congenital abnormalities, and four were unable to cooperate at the psychometric behavioural examinations. The occurrence of atopic dermatitis based on a medically documented case history did not differ significantly between the two nutritional groups at 2 years (32/176 (18.2%) in the multivitamin and 19/160 (11.9%) in the trace element group) and at 6 years (12/147 (8.2%) and 11/142 (7.7%). The occurrence of other allergies including bronchial asthma, obstructive bronchitis, pseudocroup, and food allergies were similar in the two nutritional groups at 2 years (21.0% in the multivitamin and 29.4% in the trace element) and at 6 years (30.6% and 31.8%).

At 2 years of age, developmental quotients were measured by the Bruin-Lezine method, while at 6 years intelligence quotient was measured by the Budapest Binet and Raven tests. In addition, the Goodenough drawing test was used. There was no significant difference in the mental development of children between the multivitamin and trace element groups. Anxiety was measured by the child version of thematic aperception test in 6 year old children. Five levels of anxiety were examined but ‘extremely low’ did not occur, while ‘extremely high’ was found only in two children of the trace element group. The proportion of children scoring ‘above average’ levels of anxiety was not higher in the multivitamin group and we were thus unable to confirm the previous UK finding.

In conclusion, no adverse effect was observed in children whose mothers had received periconceptional multivitamin supplementation.

MARTA DOBÓ ANDREW E CZEIZEL
Department of Human Genetics and Teratology, National Institute of Public Health, WHO Collaborating Centre for the Community Control of Hereditary Diseases, Budapest, Hungary (Correspondence to: Dr M Dobó, H-1096 Budapest OKI, Gyalü st 2-6, Hungary)


Gastro-oesophageal reflux and cows’ milk protein allergy

EDITOR,—In their very stimulating paper, Cavataio et al reported their experience concerning gastro-oesophageal reflux (GOR) associated with cows’ milk protein allergy (CMPA).1 In a series of 47 cases of GOR, they found a very high prevalence of CMPA: 14 cases (30%) and they also stressed the possibility of recognising these cases from characteristic ‘phasic’ pH tracing. We evaluated a series of 112 infants (65 boys and 47 girls; median age 3.7 months) with GOR who had profound reflux 24 hour pH monitoring. Using the results of an elimination diet followed by an open challenge test, we found CMPA dependent GOR in 18 patients (16%), a rate significantly lower than that found by Cavataio et al but similar to that of other authors.2 Moreover, we failed to find any characteristic oesophageal pH-metric pattern distinguishing patients with CMPA from patients with GOR only: one out of 18 cases with CMPA dependent GOR showed the oesophageal ‘phasic’ pH monitoring reported by Cavataio et al, and this pattern was also present in three out of 67 cases with GOR only. It is difficult to explain the difference in these two sets of results. The increase in pH subsequent to feeding found by Cavataio et al (‘phasic’ tracing) may have been an non-specific event, evident for some reason only in those patients in whom CMPA was diagnosed. Besides, such a high prevalence of CMPA in patients with GOR is truly surprising and has never been found by us or other authors in Southern Italy,1 or by others those in Europe.2 Staiano et al showed that in cases with CMPA dependent GOR, there is a significant increase in intestinal permeability3 that suggests an involvement of the entire tract. As a matter of fact, 10/18 of our cases with CMPA dependent GOR showed failure to thrive: a significantly higher number, than in cases with GOR only (55% vs 14/67=20%; p<0.05). Cavataio et al found difference neither in clinical symptoms, nor in the prevalence of failure to thrive between patients with GOR and/or CMPA, and this can cast doubts on whether CMPA was correctly diagnosed. We think that other studies are necessary to further evaluate the prevalence of CMPA in infants with GOR and to investigate the possibility of distinguishing these cases by a characteristic oesophageal pH monitoring pattern.

CRISTINA MILOCCO GUILIANO TORRE ALESSANDRO VENTURA
Clinica Pediatrica
Istituto per l’Infanzia ‘Barolo Garofolo’
Trieste, Italy
(Correspondence to: Professor A Ventura, Istituto di Clinica Pediatrica, Ospedale S Chiara, via Roma 67, 56100 Pisa, Italy)


6 Di Stefano G, Violante M, Traverso G, Oddo S, Marino V, Catataldo F. Usefulness of 24-h
Chromosomal and clinical features in an infant with Hallermann-Streiff syndrome

EDITOR,—Hallermann-Streiff syndrome (HSS) is a disorder of unknown origin rarely diagnosed in neonatal period. The early diagnosis of HSS is important for management because many complications, that are often life threatening, may occur early in this syndrome.1 We report an infant with clinical and radiological findings of HSS, a spontaneous fracture of the left arm, and with chromosome variant 46, XX, 16qh+. She was a girl born at term after a normal pregnancy. Her delivery history was unremarkable. Her birth pregnancy was normal. Birth weight was 2700 g and length was 44 cm. Physical examination showed frontal prominence and cutaneous atrophy of the forehead and over the nose, which was thin, pointed and curved, micrognathia with relative macrogllobus, microphthalmia, and a cataract in the right eye. Arms and legs were short in comparison to the body. Abdomen, thorax, neurological examination, cardiac structure and function were normal. Skeletal radiography showed thin ribs, clavicles, and long bones (ulna and radius). Chromosomes were 46, XX, 16qh+ by R banding and C banding techniques. Because of micrognathia and macrogllobus the infant was fed by gavage in the first weeks. No respiratory problems occurred in the first month of life. At the age of 3 months the patient had a spontaneous fracture of the left radius and at 10 months (fig 1) she returned to our department because of a severe pneumonia; examination revealed growth retardation (her weight was 4050 g), hypotonia, squinting, and a cataract in the left eye.

Few chromosome anomalies have been found in this case and, with only Carones reporting a discrepancy in the size of two members of pair D group chromosomes,2 and Jalbert et al describing a del(Bp)3. In our patient we found a well known polymorphism 16p+. The meaning of this chromo-

somal finding in HSS is not clear and does not explain the features of this syndrome. Slender long bones with fractures have been described in a fetus with facial appearance of HSS.4 We think that fractures of long bones should be considered a possible complication in HSS related to the bone abnormalities described in these patients.

Neutral lipid storage disease—response to dietary intervention

EDITOR,—Neutral lipid storage disease (NLSD) is an autosomal recessive metabolic disorder characterised by a multisystem accumulation of neutral lipids (triglycerides). The patients with NLSD have congenital ichthyosis and variable systemic manifestations.5 The storage of cytoplasmic triglycerides in NLSD patients results either from a severe defect in the degradation of cytoplasmic triacylglycerols containing long chain fatty acids6 or rapid triacylglycerol resynthesis. Based on the biochemical data, one might expect that a low fat diet poor in long chain fatty acids could be beneficial to these patients. We report a boy with NLSD with emphasis on his response to dietary intervention.

CASE REPORT

An 8 year old boy was born as a colloidion baby to unrelated parents. Examination at the age of 22 months revealed ichthyosiform erythroderma, hepatomegaly (11 cm below the right costal margin), and diffuse bilateral cataracts. Liver enzymes were raised (aspar-
tate aminotransferase (AST): 177 IU/l, alanine aminotransferase (ALT): 179 IU/l, γ-glutamyltransferase: 42 IU/l). Liver histol-
ogy showed gross fatty infiltration of the hepatocytes with lobular fibrosis. Leucocyte neutral lipid vacuolation was detected in the peripheral blood smear. Lipid thin layer chromatography from skin tissue showed increased accumulation of triglycerides.

The boy was put on a low fat diet (table 1) and at the end of the first year of treatment the size of the liver decreased by 50% and the liver function improved. The skin also became less erythematous and less scaly. At the age of 3.5 years the boy was operated for cataracts. At the age of 8 years, still on the special diet, his skin condition improved fur

The storage of triglycerides in NLSD patients results from a severe defect in their catabolism or rapid triacylglycerol resynthesis. Based on the biochemical data, one might expect that a low fat diet poor in long chain fatty acids could be beneficial to these patients. We report a boy with NLSD with emphasis on his response to dietary intervention.

Table 1 Diet given to the patient with NLSD

<table>
<thead>
<tr>
<th>Frequent meals (every 4-6 hours) to avoid lipolysis</th>
<th>Normal protein (15% of total energy)</th>
<th>High carbohydrate (65-70% of total energy) mainly in the form of starchy foods and one or two glucose 10% polymer drinks during the night (Maxijul, Scientific Hospital Supplies)</th>
<th>Low fat (20% of total energy) 18% derived from medium chain fat (MCT Oil, Mead Johnson) and 2% from essential long chain fatty acids (Cod Liver Oil was added at the end of the cooking into hot milk (optimum cooking temperature &lt; 160°C)</th>
<th>Supplementation of vitamin and mineral mixture (Seravit Paediatric, Scientific Hospital Supplies)</th>
</tr>
</thead>
</table>

3 Jalbert P, Gilbert Y, Leopold P, Mouriquand C, Beaudoing A. Syndrome d’Hallermann-Streiff-Francois, a propos d’une nouvelle observation associee a une anamolye cariotypique 4P. Pedi-

 foram 1989;20(3-5).
4 Dennis NR, Fairhurst J, Moore IE. Lethal syndrome of slender bones, intrarterine fractures, characteristics facial appearance, and
always exceeded 100 ng/ml, while simultaneously the dose of corticosteroids was reduced to 1 mg/day. Eight months later, after a short minor infection of the upper respiratory tract, the boy developed the clinical signs of Henoch-Schönlein purpura with a typical purpuric skin rash and ecchymotic areas particularly on the lower legs and the feet, joint involvement with periarticular pain, tenderness and swelling, and abdominal pain with or without fever and guaiac positive stools. During the subsequent course, renal involvement manifested with one episode of macrohaematuria and intermittent proteinuria, but without impairment of renal function (glomerular filtration rate 144 ml/min/1.73 m²) and no hypertension. As a complication, localised swelling of the left testis developed. The symptoms lasted for four weeks; no urinary abnormalities were noted afterwards.

During the acute stage, laboratory data showed the following results: normal values for haemoglobin, red and white blood cell count, platelet count, blood coagulation parameters (partial thromboplastin time, prothrombin time, fibrinogen, factor XIII, bleeding time), C3, C4 according to the age of a 4 year old boy; negative anti-DNA and P/C-anti-neutrophil cytoplasmic antibodies (ANCA) antibody serology; raised plasma concentration of IgG (18.1 g/l), and IgM (2.7 g/l). Three weeks later, Epstein-Barr virus (EBV) serology showed raised titres of IgG antibodies to virus capsid antigen (VCA 1:320) and EBV early antigen (1:160), but negative VCA-IgM serology and positive titre for anti-EBV nuclear antigen-1 IgG. In addition, EBV-DNA was detected in peripheral blood leucocytes, presumably reflecting a reactivated EBV infection. Henoch-Schönlein purpura is considered to be a vasculitic disorder with immune pathogenesis. Raised serum IgA concentration, circulating IgA immune complexes, IgA rheumatoid factor, an increased percentage of IgA bearing peripheral blood lymphocytes, etc., were demonstrated.1 Triggering infectious mucosal stimuli are responsible for development of Henoch-Schönlein purpura and circulating dimeric IgA are distinctly involved in this process.2 Whereas isolated cases of recurrent Henoch-Schönlein purpura nephritis in adults after renal transplantation have been observed,3 this is our knowledge the first report on a patient, who, despite of receiving regular immunosuppressive treatment after liver transplantation, developed a primary manifestation of Henoch-Schönlein purpura eight months later. This event confirms the assumption of an initial infectious triggering agent for Henoch-Schönlein purpura (for example EBV), rather than a primary immunological process. This is supported by the fact of the absence of autoantibodies (for example anti-DNA, ANCA).4

MATTHIAS SCHWAB
ROLF BEHRENS
HANS RUDER
KLAS KORN
University Hospital for Children and Youths and Institute of Clinical and Molecular Virology
University Erlangen-Nuernberg
D-91054 Erlangen, Germany
*Correspondence to: Dr Matthias Schew, Dr Margareta Fischer-Bosch Institut fuer Klinische Pharmacologie, Auerbachstrasse 112 D-70739, Stuttgart, Germany


Thyroid morphological findings in the mothers of infants with congenital hypothyroidism

EDITOR,—Little information is available on the aetiology of primary not transient congenital hypothyroidism due to thyroid malformations. Usually it is considered to be a sporadic disease; however, to date, there are no reliable studies to identify some possible risk factors for the disease.4 Therefore between April and September 1996, during therapeutic follow up of affected children, we examined the mothers of 19 consecutive infants with congenital hypothyroidism (group A) and 18 controls (group B). At the time of this examination the clinical status of the infants with congenital hypothyroidism was 5.5±3.8 years; in all cases the diagnosis of congenital hypothyroidism was confirmed and all had malformations (75% in girls, 37% in boys, and 1% other abnormalities). All the mothers examined were resident in our region, clinically euthyroid, and had not previously had any thyroid investigations. In all subjects a thyroid ultrasound was performed by the same blind operator using a 10 MHz probe and blood samples were taken to determine serum concentrations of thyroid stimulating hormone, free triiodothyronine, and free thyroxine and thyroid autoantibodies. At the ultrasound examination three mothers in group A (15.8%) showed congenital thyroid abnormalities that were pathological asymmetries of the gland (agenesis of the left lobe in one case and severe hypoplasia of the left lobe in the other two cases). Their incidence was 35.4% (1/3 cases) in the mothers of athyrotic children and 13.3% (2/15 cases) in the mothers of children with ectopic glands. None of the mothers in group B showed the same abnormalities. Thyroid enlargement on ultrasound examination was found in 8/19 cases in group A and in 7/17 cases in group B; the percentage of nodular goitres was 75.0% and 85.7% respectively. All subjects examined showed thyroid hormone values within the normal range; positive antithyroid antibody titres were found in 2/19 subjects of group A and in 1/17 subjects of group B. Congenital thyroid hemiagenesis is thought to be a rare developmental anomaly of the gland and its exact incidence is unknown as the data are usually skewed toward selected patients.5 The only data in literature from a large necropsy series reported a prevalence of 0.34%.6 The preliminary results of our study seem to indicate that the ultrasound findings of a pathological thyroid asymmetry represent a possible risk factor for fetal congenital hypothyroidism. Further family studies are needed to evaluate the thyroid transcription factor-1 gene which is probably responsible for thyroid development. In our region (Emilia-Romagna, Italy) we have observed an elevated incidence of ‘asymptomatic’ goitres in women of fertile age. This disease, however, does not appear to be related to a higher risk for thyroid malformation in the offspring.

A CASSIO
E CACCIAMARO
M BAL
C COLLI
1st Pediatric Clinic
University of Bologna
B BELANOVA
Nuclear Medicine Service,
St Orsola-Malpighi Hospital,
40138 Bologna, Italy

Investigating children with mild to moderate learning difficulties

EDITOR,—Dr Corrigan and colleagues ask important questions about the value of diagnostic screening for children with mild to moderate developmental delay.1 Unfortunately, their study has many of the methodological weaknesses highlighted in a recent review on this field.2 The vital issue of clear case definitions is not adequately addressed mainly because no standardised cognitive or developmental assessment tools were employed. Furthermore to meaningfully evaluate the performance of the diagnostic tests in this study requires not only accurate prevalence data, but also a knowledge of the individual tests’ sensitivities and specificities.

Most of the known organo-specific causes of mild to moderate developmental delay are of a low prevalence, limiting the usefulness of small studies. In the specific case of fragile X screening it is worrying that the authors conclude ‘The failure of this study to detect any cases of the fragile X gene would suggest that until larger studies are completed on similar populations fragile X screening of children with mild to moderate learning difficulties should not be recommended’. The prevalence of fragile X syndrome in the general population is currently estimated at around 0.2/1000 and in boys with learning difficulties nearer 3/1000.3 It is not therefore surprising that no cases of fragile X were detected in this study’s sample of 95 children. Perhaps a more appropriate conclusion would be that although they have not yet found any affected children, they should keep looking. DNA testing for fragile X has a high sensitivity and specificity, and testing is not only for the child’s benefit but also for the mother and extended family.

Choosing medical investigations in developmentally delayed children remains a diffi-
Increased incidence and prevalence of diabetes mellitus in Down’s syndrome

Editor,—In 1968 Milunsky and Neurath reported an increased prevalence of diabetes mellitus in patients with Down’s syndrome.1 However, the response rate in their survey was low (55%) and the authors did not distinguish between insulin and non-insulin dependent diabetes mellitus. In 1973, Jeremiah et al also reported an increased prevalence of diabetes mellitus in Down’s syndrome patients.2 Their observations, however, were based on the presence of glucosuria.

In the second Dutch nationwide study on the incidence of diabetes mellitus type I in children,3 five cases of Down’s syndrome in the age range of 0–14 years were spontaneously reported. Taken into account a yearly incidence of 250 newborns with Down’s syndrome, this results in an incidence of diabetes mellitus in Down’s syndrome of 50/100 000/year (95% confidence interval CI 16 to 116/100 000/year), whereas in the general population the incidence during the same period was 12.4/100 000/year (95% CI 12.1 to 12.7/100 000/year). These data suggest a threefold increased risk of diabetes mellitus in children with Down’s syndrome. We also estimated the prevalence in a population based study among 893 children with Down’s syndrome aged 0–9 years born between 1986 and 1994. Three of them had diabetes mellitus, giving a prevalence rate of 335/100 000 (95% CI 87 to 980/100 000), which is higher than the prevalence of diabetes mellitus type I in the age matched general population (40/100 000; 95% CI 35 to 45/100 000).

Our data confirm the suspicion that children with Down’s syndrome are more prone to develop diabetes mellitus. All children were insulin dependent. However, it remains to be investigated whether these patients have type I or type II diabetes mellitus as both autoimmunity and signs of premature ageing in Down’s syndrome could predispose towards type I and type II diabetes mellitus.4 As the symptoms of diabetes mellitus in Down’s syndrome children may be more difficult to detect and there is an increased risk of complications such as hyperosmolar coma, special attention has to be taken to symptoms as polydipsia and polyuria.

J C VAN GOOR
G G MASSA
Department of Paediatrics,
University of Leiden,
POB 9690, 2300 RC Leiden,
The Netherlands

R HIRASING
TNO Prevention and Health,
2301 CE Leiden,
The Netherlands

Clinical lecturer posts under threat

Editor,—Should anyone decide to scrap secondary school education, one effect would be to make it near impossible for anyone to go to university. To axe clinical lecturer posts, as discussed in the alarming January 1997 editorial in the journal,1 would be an equally insane act, and would at a stroke virtually abolish recruitment into academic medicine.

The problem is that it is near impossible to get clinical lecturers to perform world class research (a) early in their research careers and (b) while needing to continue with clinical training. The situation is bizarre: it is like expecting an 11 year old to play cricket for England. The poor child needs to get further experience in the sport, as well as needing to continue with a general education. The solution is quite simply to forget about 11 year olds as being eligible for the national team, and to concentrate on their further development. By agreement with the Higher Education Funding Council for England (HEFCE), who make the rules, clinical lecturers should be excluded from the quinquennial research assessment exercise.

In commenting on the irrationality of this decision, an editorial in the Lancet has summed up the situation as ‘a research culture that asks too much’.2 Radical alternatives have been proposed, but are unlikely to work. One option, for which there is considerable support, would be to make clinical lecturers wholly supernumerary to the clinical service, for approximately three years—sufficient time for a PhD. They would be engaged 100% of the time on research, and could have to spend additional years undergoing clinical training. However such an arrangement would require aspiring academics to take a very large salary drop, hardly a practical way to tackle the existing combined difficulties of recruiting into academic medicine.

2 Turnberg L. The place of research in the training of NHS consultant physicians. London: Royal College of Physicians, 5 April 1993.

186
Letters, Notice
and into academic medicine. Another option would be for NHS trusts to take over the funding of HEFCE funded clinical lecturer posts, but this seems little more than a pipe dream given the existing difficulties of getting NHS trusts to fund ordinary training grade posts.

T J DAVID
University Department of Child Health,
Booth Hall Children’s Hospital,
Manchester M9 7AA


Fashion victim: infective endocarditis after nasal piercing

EDITOR,—We report a case of infective endocarditis occurring after nasal piercing.

A previously well 14 year old girl presented three weeks after nasal piercing and metal stud insertion. After insertion she developed progressive ‘flu’ like symptoms with fever, myalgia, headache, nausea, and vomiting. Examination revealed a normally developed girl who was pyrexial (39.4°C) and uncom- fortable, with suprapubic and epigastric tenderness.

Examination of her cardiovascular system demonstrated no abnormality. Investigations showed a neutrophil leuco- cytosis, a raised C reactive protein and eryth- rocyte sedimentation rate. Blood and urine cultures were taken. Because of her persisting abnormalities a laparoscopy and appendic- ectomy were performed and showed no abnormalities.

Despite treatment with flucloxacillin, cefox- tamine, and metronidazole she proceeded to rigor. Nasal swabs and blood and urine cultures repeatedly grew Staphylococcus aureus despite appropriate antibiotic treatment. Subsequently she developed signs of focal sepsis and an echocardiogram was performed, revealing a large vegetation on the anterior leaflet of the mitral valve with no evidence of valvular incompetence. The size of the vegetation and the blood culture results indicated the likely infecting organ- ism to be S aureus. A diagnosis of infective endocarditis was made and she was treated with high dose flucloxacillin and vancomycin.

Subsequently she developed clinical signs of mitral regurgitation confirmed on echocardio- graphy. She developed an allergic rash to flu- cloxacillin and treatment was changed to vancomycin alone for the last three weeks of her six week treatment. The mitral valve vegetaion decreased in size after treatment but she was left with minor degree of mitral incompetence evident clinically and ecchae- diographically.

Infective endocarditis due to S aureus is the absence of an underlying cardiac defect is uncommon. In vitro studies have demon- strated the ability of S aureus to induce a tissue factor promoting adherence to valve endothelium, altering host responses and partially protecting from antimicrobial treat- ment resulting in a prolonged bacteremia.

Initial treatment of infective endocarditis comprises intravenous benzylpenicillin and gentamicin. If staphylococcal infection is confirmed then flucloxacillin is added and treatment continued for six weeks, using van- comycin in cases of penicillin allergy.

Nasal carriage of S aureus renders piercing of this area more likely to result in infective endocarditis, however piercing of any mucous membrane may result in bacteremia and infective endocarditis.

We report for the first time infective endocar- ditis arising after nasal piercing in a person with a structurally normal heart, and empha- size the importance in excluding this diagno- sis in patients with a persisting pyrexia after recent invasive adornment.

I AN J RAMAGE 
NEIL WILSON
Royal Hospital for Sick Children,
Yokhill,
Gloucester G3 8SJ

RUTH H THOMSON
Dunmoyes and Gallowsay Royal Infirmary,
Dumfries DG4 4AP


Needle injuries as a cause of non-accidental injury

EDITOR,—Carers of children need to be aware of the wide range of possible non-accidental injuries so that they may be recognised and managed appropriately. The insertion of sharp needles into body cavities or soft tissues is an uncommon form of child abuse and we wish to report three cases presenting to us during a four year period.

Case 1—A 4 week old infant of Asian par- ents died before being admitted to the emer- gency department. A necropsy confirmed the cause of death as bacterial pneumonia. Examination of the scalp revealed a healed 1 cm laceration posterior to the anterior fontanelle. Tracts led to three broken sewing needles embedded within the occipital lobes.

Case 2—A female sibling to case 1 had previously presented at the emergency depart- ment with bruises, abrasions, and burns. A skeletal survey demonstrated four needles in the soft tissues of the head, neck, and forearm. These were removed surgically and found to be sewing needles.

Case 3—A baby boy was born at 25 weeks’ gestation after a concealed pregnancy in a young African mother. An abdominal radio- graphy after 10 days showed two further opaque objects in the abdomen. Repeat radio- graphy after 10 days showed two further similar objects in the pelvis. At laparotomy four needles were removed.

The insertion of sharp needles through the skin or mucous membranes of young children is a rare form of non-accidental injury with only seven other cases having been reported. We are not aware that this is a recognised part of African or Indian culture and this must be considered and managed as child abuse.

C FEARNE
J KELLY
J ISABEL
D P DRAKE
Great Ormond Street Hospital for Children NHS Trust,
Great Ormond Street,
London WC1N 3JH


Acknowledgment and authors

EDITOR,—I share Dr Moncrieff’s irritation at the number of requests for information which I receive and replying to the first request for information often invites further trouble. I agree with him that researchers should do their own research.

I also agree with him about authorship but I would point out that in a department which supports a serious number of research fellows, the head of department is quite likely to have had the idea and written the grant application, as well as having a substantial hand in writing the abstracts and reports.

This is hardly guest authorship and five or six research fellows can easily generate 10 to 20 papers in a year if they are correct towards the end of their period of research.

C G D BROOK
Gobbold Laboratories, Paediatric Endocrinology, Middlesex Hospital, Montmore Street, London W1N 8AA


Height and weight achievement in cleft lip and palate

EDITOR,—We were interested to read the recent article by Lee et al.1 Of course, it is not surprising that children with cleft lip and palate have growth failure in infancy, especially with their feeding difficulties. However, follow up in this study was only to a mean of 25.5 months.2 Growth failure is common in children with cleft lip and palate and is related to the type of cleft, as well as age and sex.3 Some children with cleft palate and especially those combined with an additional midline cleft lip may be part of holoprosen- cephalic complex and have hypothalamic pituitary deficiency, there being no other dys- morphic features.4 However, growth hor- mone secretion has little influence on growth in early childhood.5 Growth may be normal in children with pituitary deficiency until approx- imately 2 years of age. In addition, children with midline defects may have evolving endocrinopathy with pituitary defi- ciencies only appearing in later childhood.6 Moreover, growth hormone deficiency has been described as 40 times more common in children with cleft lip and palate.7 We believe that although early catch-up growth is undoubtedly important, growth of children with cleft palate should continue to be mon- itored throughout childhood.

C AZCONA
R STANHOPE
Nutrition, Metabolism, Endocrinology and Dermatology Unit,
Great Ormond Street Hospital for Children NHS Trust,
Great Ormond Street, London WC1N 3JH

Aetiology of asthma

EDITOR,—Austin and Russell suggest that mobility of families may be more important in the aetiology of asthma then exposure to any one individual allergen or pollutant. This ties in with the finding of Hughes and Baumer,1 except that they failed to ask whether there is an emotional component to this. Most of us know that moving house is second only to divorce as a traumatic event in life, and considering the greater attachment that children have to their home (they after all spend more time there than adults as a rule), it is no surprise that this could be the mechanism of the effect these authors describe. In some cases the move may be due to family break up.

SEBASTIAN KRAEMER
Child and Family Psychiatry Service, Whittington Hospital NHS Trust, Highgate Hill, London N19 5NF

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 WC1H 9JR.