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 does 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, at新时期 of the age range 2 and 6 years with respect to the occurrence of atopic dermatitis (and other allergies) and anxiety.

The children were born to women 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, 629 (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%) vs 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 (30.4% vs 38.8%).

At 2 years of age, developmental quotient was measured by the Brunet-Lezine method, while at 6 years intelligence quotient was measured by the Budapest Binet and Raven tests. In addition, the Goodenough man 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 apperception test1 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.

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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%)—they also assessed 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 during a 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 in patients in whom CMPA was diagnosed.2 However, we found 60% of our cases with CMPA dependent GOR associated with cows’ milk protein allergy (CMPPA).3 In our cases with GOR, we also assessed the possibility of recognising these cases from characteristic ‘phasic’ pH tracing.

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6 Di Stefano G, Violante M, Traverso G, Oddo S, Marino V, Catalfo 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 a 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 family history was unremarkable. During pregnancy there was no exposure to known teratogens. 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 macroglossia, microphthalmia, and a cataract in the right eye. Arms and legs were short in comparison to the body. Abdomen, thorax, genitalia, 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 macroglossia 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, squatting, and a cataract in the left eye.

Few chromosomal anomalies have been found in past cases, with only Carones reporting some discrepancy in the size of two members of pair of D group chromosomes,2 and Jalbert et al describing a del(Bp).3 In our patient we found a well known polymorphym, 16qh+. The meaning of this chromosomal 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.

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4. Dennis NR, Fairhurst J, Moore IE. Lethal syndrome of slender bones, intrasternal fractures, characteristics facial appearance, and

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% polymers 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 fat. The MCT Oil was added at the end of the cooking into meal (optional, cooking temperature &lt; 160°C)</th>
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Figure 1  Infant at 10 months of age.


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.7 Based on these 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 (aspartate 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 smears. 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- ther and the liver size was normal (AST: 60 IU/l, ALT: 70 IU/l). There was no hearing or muscle power impairment and he made good progress in school.

In our patient a low fat diet, poor in long chain and enriched with medium chain fatty acids, led to regression of liver size, improvement of his skin condition, and possibly preven- tion of other organ involvement. In 1980 Angelini et al also reported improvement in liver size with a medium chain triglyceride diet in a 5 year old girl with NLSD.8 As in other metabolic disorders the special diet did not restrain the progress of the patient’s cataracts.9 It is conceivable that by starting the special diet before cataract surgery, this complication might be totally prevented. This observation indicates that in cases of NLSD, an early initiation of a diet poor in long chain fatty acids might improve the skin condition and prevent systemic disturbances.

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Primary manifestation of Henoch-Schönlein purpura during immunosuppressive treatment

Editor.—Although numerous investigations suggest an immunological process involving a disturbance of the regulatory mechanism for IgA synthesis,1 the autoimmunity of Henoch-Schönlein purpura are still unresolved.

We report a 4 year old boy who received a liver transplant at the age of 3 years after developing a fulminant liver failure due to intoxication with α-aminopterin. After transplantation, immunosuppressive treatment with cyclosporin (Neoral, Sandoz) and prednisone was carried out. During later care the cyclosporin trough serum concentrations

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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 nausea, but no positive stools. During the subsequent course, renal involvement manifested with one episode of macrohematuria and intermittent proteinuria, but without impairment of renal function (glomerular filtration rate 144 ml/min/1.73 m^2) 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-anitneutrophilic cytoplasmatic (ANCa) antibody serology; raised plasma concentration of IgG (14.8 g/l), IgA (15 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 1:80) 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 leukocytes, presumably reflecting a recent 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.1 Whereas isolated cases of recurrent Henoch-Schönlein purpura nephritis in adults after renal transplantation have been observed,4 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 failure to prove the presence of autoantibodies (for example anti-DNA, ANCA).1


**Thyroid morphological findings in the mothers of infants with congenital hypothyroidism**

Environ.—Little information is available on the aetiology of primary not transitory 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 family risk factors for the disease.2 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 17 age matched 'asymptomatic' goitres in women of fertile age. This event confirms the assumption of a familial predisposition in the pathogenesis of congenital hypothyroidism (group B). At the time of this examination the prevalence of goitres in group A (15.8%) showed congenital hypothyroidism was 5.5/1000; in all cases the diagnosis of congenital hypothyroidism was confirmed and all had malformations (15 with cervical, three with atrioventricular defects, one hypospadias). 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 antithyroid antibodies. 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 and of the right lobe in case number one 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.4 The only data in literature from a large necropsy series reported a prevalence of 0.34%.5 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 number 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.


**Investigating children with mild to moderate learning difficulties**

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cult area, but there are well described protocols for critically appraising medical investigations which should be followed before making recommendations on the performance of diagnostic investigations.

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Dr Corrigan and Steuart comment: As has been correctly pointed out research into children with mild to moderate learning difficulties is plagued by difficulties in case definition. In our study we used operational criteria based on school placement and provision of remedial assistance to define learning difficulties. This reflects the clinical reality and outlines the extent of the problem. The majority of these children were maintained in mainstream education and did not have formal educational assessment. They represent a large number of children (16% of population in our study) that are becoming an increasing focus for paediatricians with a remit in educational medicine. At present there are no formal recommendations regarding medical assessment or investigation in this group.

Any decision to institute a regular screening programme for these children would have significant implications for both the child and health resources. We feel therefore that the benefits of a programme such as screening for fragile X would first have to be proved. Studies should show positive results in an identifiable group of children using easily translatable operational criteria before recommending their inclusion.


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 (550%) 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 1 in children, 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.

UK paediatric clinical research under threat

EDITOR—The message of your contributor(s) concerning prospects for clinical research in paediatrics was disturbing.1 Two aspects cause me concern.

First, that the author(s) requested anonymity. Was this through reticence or self effacement? There seemed little in the article to cause offence. If the author(s) thought that they or their departments might be censured by their universities or grant giving bodies, this would be a serious enough matter to be taken up by national academic and representative bodies. Academic paediatricians need visible, resolute leadership and the unequivocal support of non-university paediatricians.

Second, your contributor(s) offered no proposals to deal with the threat. It will not recede spontaneously and neither academic medicine nor clinical practice is immune from the laws of natural selection—adapt or perish. Those who lead our specialty and promote its academic underpinnings should be making plans individually and collectively (including other specialties) to meet and overcome the threat. These include your contributors, presumably, and our newly elected college Vice President who has declared his commitment to promoting paediatric research.

One tactic would be to expect that all trainees in paediatrics should undertake a period of research—not a series of audit projects. Some European countries specify that a number of papers in peer reviewed journals is required for the final certification process. The arguments for promoting research in the training of NHS consultants were set out by the President of the Royal College of Physicians a few years ago.3 Practising evidence-based medicine without knowing how the evidence was assembled is as absurd as managing asthma without knowledge of its respiratory physiology.


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 performers 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 need for rational, 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 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 paediatrics

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.

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Fashion victim: infective endocarditis after nasal piercing

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

Thirty years ago a 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 uncomfortable, with suprapubic and epigastric tenderness. Examination of her cardiovascular system demonstrated no abnormality. Investigations showed a neutrophil leukocytosis, a raised C reactive protein and erythrocyte sedimentation rate. Blood and urine cultures were taken. Because of her persisting abdominal pain a laparoscopy and appendicectomy were performed and showed no abnormalities.

Despite treatment with flucloxacillin, cefotaxime, and metronidazole she proceeded to rigour. Nasal swabs and blood and urine cultures repeatedly grew Staphylococcus aureus despite appropriate antibiotic treatment. Subsequently she developed signs of focal septal perforation 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 organism 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 echocardiography. She developed an allergic rash to flucloxacillin and treatment was changed to vancomycin alone for the last three weeks of her six week treatment. The mitral valve vegetation decreased in size after treatment but she was left with minor degree of mitral incompetence evident clinically and echocardiographically.

Infective endocarditis due to S aureus in the absence of an underlying cardiac defect is uncommon.1 In vitro studies have demonstrated the ability of S aureus to induce a tissue factor promoting adherence to valve endothelium, altering host responses and partially protecting from antimicrobial treatment resulting in a prolonged bacteraemia.

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 vancomycin in cases of penicillin allergy.2

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 bacteraemia and infective endocarditis. We report for the first time infective endocarditis arising after nasal piercing in a person with a structurally normal heart, and emphasise the importance in excluding this diagnosis in patients with a persisting pyrexia after recent invasive adornment.

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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 parents died before being admitted to the emergency 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 department 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 non-accidental injury in a young African mother. An abdominal radiograph at 3 weeks demonstrated two linear opaque objects in the abdomen. Repeat radiography 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.3 4 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.

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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 conscious of the end of their period of research.

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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.2 Some children with cleft palate and especially those combined with an additional midline cleft lip may be part of holoproscephaly complex and have hypothalamic pituitary deficiency, there being no other dysmorphic features.3 However, growth hormone secretion has little influence on growth in early childhood.4 Growth may be normal in children with pituitary deficiency until approximately 2 years of age. In addition, children with midline defects may have an evolving endocrinopathy with pituitary deficiencies only appearing in later childhood.5 Moreover, growth hormone deficiency has been described as 40 times more common in children with cleft lip and palate.6 We believe that although early catch-up growth is undoubtedly important, growth of children with cleft palate should continue to be monitored throughout childhood.

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Aetiology of asthma

EDITOR,—Austin and Russell suggest that mobility of families may be more important in the aetiology of asthma than exposure to any one individual allergen or pollutant. 1 This ties in with the finding of Hughes and Baumer,2 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.

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Long term follow up of children born to mothers with periconceptional multivitamin supplementation
MÁRTA DOBÓ and ANDREW E CZEIZEL

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