inotropes. He developed peripheral gangrene, requiring amputation of the fingers but not thumb of his left hand to the metacarpophalangeal joints, amputation of his right fingers to the proximal interphalangeal joints, amputation of all his toes, and removal of necrotic tissue and bone on his left heel. He had extensive skin grafting to the lower limbs. Good function of his right hand was predicted. He was discharged on prophylactic antibiotics. Polyvalent pneumococcal vaccine and meningococcal vaccine will be given at 24 months and conjugate vaccine against *Haemophilus influenzae* type b as soon as it is available in Australia.

These siblings presumably have recessively inherited congenital asplenia and normal hearts. Hereditary splenic hypoplasia was first described in three of five siblings by Kevy *et al*.[4] In a review of 60 children with asplenia or polysplenia from Toronto, there were two families in which two siblings had isolated asplenia and one family in which two siblings had polysplenia.[5]

It was assumed that our first patient died of meningococcal sepsis, although in retrospect she may have had pneumococcal sepsis, as both organisms may sometimes fail to grow from severe cases. In an ideal world, a necropsy would have been performed, would have revealed asplenia and we would have screened the next child at birth. Prophylactic antibiotics have been shown to be effective in reducing the incidence of bacterial sepsis in children with congenital asplenia,[6] and immunisations as already described could have been given at the appropriate age.

J GILLIS
J HARVEY
D ISAACS
M FREELANDER
B WYETH
The Children's Hospital, Camperdown, NSW 2050, Australia

1 Dyke MP, Martin RP, Berry PJ. Septicaemia and adrenal haemorrhage in congenital asplenia. *Arch Dis Child* 1991;**66**:636–7.
2 Gill DG, Kara M. Septicaemia and adrenal haemorrhage in congenital asplenia. *Arch Dis Child* 1991;**66**:1366.
3 Moore L. Septicaemia and adrenal haemorrhage in congenital asplenia. *Arch Dis Child* 1991;**66**: 1366–7.
4 Kevy SV, Tefft M, Vawter GF, Rosen FS. Hereditary splenic hypoplasia. *Pediatrics* 1968; **42**:752–7.
5 Rose V, Izukawa T, Moes CAF. Syndromes of asplenia and polysplenia. *Br Heart J* 1975;**37**: 840–52.
6 Biggar WD, Ramirez RA, Rose V. Congenital asplenia: immunologic assessment and a clinical review of eight surviving patients. *Pediatrics* 1981;**67**:548–51.

Growth performance of affluent Indian children under 5 years of age

SIR,—Paediatricians interested in the growth patterns of children of families originating from the Indian subcontinent may like to know of a study by the Nutrition Foundation of India on 'Growth performance of affluent Indian children (under fives)'.[1] The study was undertaken to determine whether the international growth standards developed by the National Center for Health Statistics in the USA were applicable to Indian children. A corollary to this was the desirability, or otherwise, of collecting data on Indian children on a national scale.

The authors accepted that socioeconomic and environmental factors and the malnutrition-infection complex rather than genetic factors were the main constraints determining the differences between the growth performance of children in developed and developing countries.[2] In order to eliminate these factors as far as possible, only children from affluent families (they took some pains to define 'affluent') were studied in seven cities: two in the north (Ludhiana and Delhi), two in central India (Kota and Varanasi), one on the west coast (Bombay), one in the south (Bangalore), and one on the east coast (Calcutta).

The results showed that 'affluent' children (0–5 years) in Ludhiana and Delhi in the north had attained a level of growth in height and weight which nearly corresponded to those of the international growth standards and it was therefore concluded that these standards could be used for Indian children. In the remaining cities where growth fell below the American standards it was thought that the explanation lay in the dietary differences between the various parts of India.

The value of this study is that paediatricians in Britain can use (or continue to use) the American or the Tanner-Whitehouse standards for the children of Indian origin in the important 0–5 year period. It must be emphasised, however, that these findings do not apply to the neighbouring countries of Pakistan, Bangladesh, Nepal, and Sri Lanka. Those interested in the details of the study should consult the original report.

JOHN BLACK
54 Ruskin Park House, Champion Hill, London SE5 8TQ

1 Agarwal KN, Agarwal DG, Benakappa SM, *et al*. *Growth performance of affluent Indian children (under fives)*. New Delhi: Nutrition Foundation of India (scientific report 11), 1991. (Available in the Library of the Royal Society of Medicine, London.)
2 Anonymous. A measure of agreement on growth standards [Editorial]. *Lancet* 1984;i:142–3.

Birthweight ratio in public health

SIR,—A similar birthweight ratio to that used for guidance in sophisticated neonatal intensive care discussed by Dr Lucas *et al*[1] has been suggested as a public health indicator. This was termed the 'socioeconomic birth weight [b wt] quotient' (or ratio).[2] It was defined as:

$$\frac{\text{average b wt low socioeconomic population}}{\text{average b wt upper socioeconomic population}} \times 1000$$

It was considered as a rough potential cumulative measure of 'social development', especially of prenatal care, maternal nutrition, and infections (notably placental malaria[3 4]).

This approach is only valid when the community concerned is, in general, genetically uniform. Also, as always with such comparisons, problems occur in defining the two groups. Generally well nourished, genetically homogeneous communities with good prenatal services, as in Denmark, should have an index of 1000. In a study in an area in India, results ranged from 872 to 885.[5]

DERRICK JELLIFFE
PATRICE JELLIFFE
School of Public Health, University of California (UCLA), 10833 Le Conte Avenue, Los Angeles, California 90024–1772, USA

1 Lucas A, Morley R. Birthweight ratio. *Arch Dis Child* 1991;**66**:1099.
2 Jelliffe DB, Jelliffe EFP. The socio-economic birth weight quotient. In: Sterky G, Mellander L, eds. *Birthweight distribution: an indicator of social development*. Report No 8. Stockholm: SAREC, 1978:41–3.
3 Jelliffe EFP. Low birth weight and malarial infection of the placenta. *Bull World Health Organ* 1968;**38**:69–73.
4 Jelliffe EFP. *Materno-fetal malaria: multiple dyadic dilemmas*. Geneva: World Health Organisation (in press).
5 Prema K. Pregnancy and lactation: some nutritional aspects. *Indian J Med Res* 1978;**68**: 78–82.

Toxic shock-like syndrome due to *Streptococcus pyogenes*

SIR,—Streptococcal toxic shock syndrome has been recently reported in both adults[1–3] and children.[4] Torres-Martinez *et al* described four children all of whom survived but required intensive treatment and aggressive cardiovascular resuscitation.[5] Reports in adults have often demonstrated less favourable outcomes despite appropriate management. Our recent experience with one child would agree with this.

A previously well, 5 year old boy was admitted to Crawley Hospital with a two day history of cough and sore throat. He developed inspiratory and expiratory stridor three hours after admission becoming distressed and requiring nasotracheal intubation. The epiglottis was normal but there was evidence of a tracheitis; a tracheal aspirate grew group A β haemolytic stretococcus but blood cultures were negative. He was given intravenous benzylpenicillin, flucloxacillin, and cefotaxime. Thirty six hours later he was extubated but after 12 hours he suddenly deteriorated and required reintubation. At that time a chest radiograph showed partial collapse of the left lower lobe. He was transferred to the Royal Alexandra Hospital for Sick Children at Brighton for further management and on arrival 72 hours after his original hospital admission his condition was stable. His full blood count, haemoglobin, urea, and electrolyte concentrations, and blood gases were all normal. His blood pressure was 100/50 mm Hg. Intravenous benzylpenicillin was continued in a dose of 150 mg/kg/day in four divided doses and after discussions with the microbiology laboratory gentamicin 40 mg every eight hours and chloramphenicol 500 mg every six hour were added. A chest radiograph shortly after admission showed considerable improvement. Twelve hours after admission, while fully ventilated, he suddenly became hypotensive, bradycardic, and hypoxic, the deterioration being associated with evacuation of his bowels. The heart rate fell from 120 to 40 per minute and the oxygen saturation from 96 to 40%. Despite 80 ml/kg of colloid followed by a continuous infusion, intravenous adrenaline 2 ml of 1:10 000×3, a dopamine infusion, and intravenous atropine 200 μg×2, he deteriorated further. His liver enlarged to 5 cm below the right costal margin and he developed disseminated intravascular coagulation and renal failure. He died 17 hours later. Postmortem examination revealed only a hyperaemic trachea and bronchi. There were small bilateral pleural effusions. The National Reference Laboratory (Colindale) reported an antistreptolysin-O titre of 400 units/ml, an anti-DNAse B titre of 180 units/ml, and an antihyaluronidase activity of 1024 units/ml. The streptococcus isolated from the