LETTERS TO THE EDITOR

Allicin: a possible answer to antibiotic resistant campylobacter diarrhoeal infection?

EDITOR,—Several *Campylobacter* spp are a frequent cause of gastroenteritis in children, particularly those younger than 2 years in developing countries.1 Recent articles have reported the emergence of campylobacter isolates resistant to quinolones, which are commonly used for the treatment of this infection.2 We have observed the emergence of *Campylobacter* spp resistant to nalidixic acid, a commonly used quinolone. We propose that due consideration be given to the research and development of allicin, as an alternative to antibiotic treatment in cases of campylobacter infection that fail to respond to conventional antibiotic regimens. Allicin, a naturally occurring antibiotic, has been shown in vitro to have a broad spectrum of activity against Gram positive and Gram negative bacteria, yeasts,1 Candida spp, Cryptococcus neoformans, and Helicobacter pylori.2

The antibacterial effect of an aqueous garlic extract was investigated against 38 clinical isolates of campylobacter from children presenting with diarrhoeal infections, and 32 isolates of *H pylori* from adults with peptic ulcers. The campylobacter strains tested were *C jejuni* (n = 20), *C coli* (n = 3), *C upsaliensis* (n = 3), *C jejuni* sub *jejuni* (n = 2), and one strain of each of the following: *C hyointestinalis*, *C urealyticum*, *C mucosalis*, and *C helveticus*. An aqueous garlic extract was prepared according to a modified method of Fromtling and Bulmer.3 Cloves of garlic were dehusked, crushed, and homogenised. Distilled water (100 ml) was added to each 50 g of garlic pulp. The aqueous mixture was mixed for one hour and allowed to stand for a further two hours at 4°C. The resulting aqueous supernatant was decanted to remove the larger tissue particles. The supernatant was centrifuged for 15 minutes to remove smaller particles and then passed through a 0.45 μm acetate membrane filter followed by storage at 4°C for up to seven days before in vitro testing. The Nathan agar well diffusion assay4 was used to test the susceptibility of the 70 clinical isolates of campylobacter and *H pylori* to allicin in the aqueous garlic extract at a concentration of 0.5 g/ml. Blood agar plates were inoculated with the different strains and a well (6 mm diameter) made in the centre of each plate. The well was filled with 100 μl of the aqueous extract and incubated at 37°C in an H, increased atmosphere for 48 hours. Peptones were examined, re-incubated, and examined frequently for an additional four days. The 70 clinical isolates were all susceptible to allicin as seen by zones of inhibition (range 15–32 mm in diameter, mean 21 mm) (table 1). Subcultures from the zones of inhibition produced no growth after 48 hours, suggesting that the mode of action of allicin against campylobacter is bactericidal. These results suggest that allicin should be investigated as a possible enteral treatment for gastroenteritis involving antibiotic resistant campylobacter strains and as a possible cure for *H pylori* infections.

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H RODE
D SIDLER
Department of Paediatric Surgery,
Rondebosch 7700, South Africa

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<thead>
<tr>
<th>Organism</th>
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<td><em>H pylori</em></td>
<td>32</td>
</tr>
<tr>
<td><em>C jejuni</em> sub <em>jejuni</em></td>
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<tr>
<td><em>C fusus</em></td>
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Table 1 Effect of allicin on Campylobacter and Helicobacter spp

ACTH treatment for gelastic seizures

EDITOR,—Gelastic seizures are recurrent attacks of inappropriate laughter not precipitated by external stimulation. Usually, they are poorly responsive to antiepileptic drugs.1 We report a case of tuberous sclerosis with gelastic seizures that disappeared after treatment with adrenocorticotropic hormone (ACTH).

A 6 month old girl had repetitive tonic spasms of her head and arms and was referred to hospital. There was no significant family history. Pregnancy and delivery were uncomplicated and developmental milestones were normal. Physical examination revealed retinal hamartomas and hypomelanotic macules on her chest and back. The EEG showed hypsarrhythmia. Computed tomography and magnetic resonance imaging (MRI) of her brain showed multiple subependymal calcified nodules and cortical tubers. She was diagnosed with tuberous sclerosis and treated with ACTH (synthetic ACTH, 0.015 mg/kg/day).2 Her seizures and hypsarrhythmia on the EEG disappeared soon after ACTH treatment, which was administered every day for two weeks and tapered gradually.

Six months later, she started to have episodes of sudden inappropriate laughter followed by salivation and unconsciousness for a few minutes. These episodes occurred a few times a week. The EEG showed sporadic spikes or polyspikes in the left posterior temporal region. MRI showed no hypothalamic masses. These gelastic seizures were difficult to control and lasted for three months, despite the administration of valproate, zonisamide, and nitrazepam. Repetitive tonic spasms of her head and arms recurred. She was treated with ACTH in the same way as before and gelastic seizures disappeared. Her tonic spasms also diminished but still occurred a few times a week after eight weeks of ACTH treatment. The sudden laughter observed in this case is consistent with the criteria of gelastic seizures.3 ACTH might be useful to treat gelastic seizures in selected cases.

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Less diarrhoea but no change in growth: 15 years’ data from three Gambian villages

EDITOR,—The paper by Poskitt and colleagues from The Gambia4 indicates that there has been a disappointing lack of improvement in the nutritional status and mortality of young African children during the 25 years’ existence of the famous field station. Previous research from Keneba suggested a relation between diarrhoeal diseases and growth faltering, but despite considerable reductions in diarrhoea incidence and duration of attacks over the past 15 years, nutritional status has not improved, implying that these children were malnourished primarily because of a lack of food rather than because of high rates of diarrhoea.

This challenge to the orthodox “diarrhoea—malnutrition vicious cycle” theory, first voiced by Briend in an analysis of morbidity and growth data from Bangladesh,5 is also supported by our findings in rural Zimbabwe.6 Children from commercial farm labour compounds were enrolled in a prospective study of diarrhoea morbidity and growth. Growth faltering in the 2nd year of life was severe but there was little difference in the average rates of growth between children with frequent and infrequent diarrhoea. The results of an interval based data analysis similar to that employed by Briend, were consistent with there being only a transient effect of diarrhoea on weight gain. Estimation of weight faltering following episodes of diarrhoea and the rate of return to the trend in the 9–14 month age group indicated that weight loss associated with diarrhoea was small (around 2% of body weight), and return to the child’s trend was 90% complete within a month. Our observations lend weight to the hypothesis that recurrent episodes of diarrhoea are not a potent cause of growth faltering in early childhood except in a small number of largely catastrophic cases. Inadequate food intake is a more plausible explanation.

Table 1 Ect of diarrhoea on weight gain

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*Table 1: Effect of allicin on Campylobacter and Helicobacter spp*

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The paper points out that catch up growth after acute diarrhoeal disease requires energy
intakes 50% in excess of recommendations. In Gambia micronutrient and macronutrient
supplementation has been tried over 14 years without obtaining significant catch up growth, as quoted in the paper. Mention is made of the role of enteropathy in causing
malnutrition and poor growth. The evidence for this was published in 1991, and
suggested that impaired mucosal integrity, secondary to chronic inflammation, is largely to blame with 40% of poor growth attributable to poor small bowel function. Thus the effect of enteropathy appears to be significantly
greater than any response to dietary supple-
mentation. Enteropathy is widespread in the tropics and has been termed “tropical enteropathy”, the cause of which is uncertain and could include the idea of infection, nutrient deficiency or postenteritis food allergy. Although acute diarrhoeal disease may not contribute significantly towards poor growth, the underlying enteropathy undoubt-
ably does. Clearly, adequate nutrition is crucial for growth, but for many African children this may not be enough.

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BGC and tuberculosis
Editor.—There is no doubt that BCG increases sensitivity to tuberculosis but this cannot be taken to equate with protection against tuberculosis as it is implied by a number of studies including 2 cited in the article by Bannon.

Evidence from the large Medical Research Council (MRC) trial in the UK showed that after BCG vaccination, non-
responders to a 3 tuberculin unit (TU) Mantoux test had the same degree of protection against tuberculosis as responders. The delayed hypersensitivity reaction should not be regarded as evidence of immunity but as evidence of infection. Distinguishing a BCG infection from a false positive test may be difficult and the tuberculin test is only one element in the assessment. In a high risk population in Leicester who had neonatal BCG vaccination, use of a 1 TU Mantoux test (0.1 ml of 1 in 10 000) can significantly and safely reduce the number of children who need chemoprophylaxis.1

The larger tuberculin response that follows later vaccinations is not evidence that neonatal BCG is less effective. The present system of universal vaccination at 13 years is illogical and derives from the original MRC trial design. This age was chosen for operational and statistical reasons that have nothing to do with the clinical effectiveness of the vaccine.2 More a rational approach would be to dispense with school BCG in favour of universal neonatal BCG, which is logistically much easier and protects those most at risk of disease in childhood from its most serious manifestations. Funding for tuberculosis services could then be directed more towards case finding and treatment comple-
nance, which will break the transmission of disease to the next generation.

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Dr Bannon comments:
The tuberculin protein skin test should never be used in isolation in the diagnosis of child-
hood tuberculosis. A more rigorous approach is required that includes good history taking, meticulouis medical consultation, and judi-
cious use of appropriate investigations. A positive tuberculin protein skin test merely indicates that a child’s immune system has been exposed to certain strains of mycobacteria. However, there is now some agreement that a reaction is more likely to be indicative of infection rather than prior BCG immu-
nisation if:

- it is strongly positive
- BCG had been given at least five years previously
- there had been close contact with a sputum positive adult
- the child is a recent immigrant form a part of the world where there is a high prevalence of TB
- All children fulfilling these criteria must be referred for specialist opinion.

It would be illogical to discontinue with the school BCG programme at this present time. Even if universal neonatal BCG immu-
sisation were to be implemented immediately, there would still be a large cohort of children who would remain unprotected for many years. School attendance is an ideal opportu-
nity for opportunistic health promotional activities, and tuberculin protein testing of adolescents should continue to detect those children who have subsequently become infected with the Mycobacterium tuberculosis bacillus or who, despite receiving neonatal BCG, have developed active tuberculosis.

For the foreseeable future, we should con-
tinue with existing TB prevention and eradi-
cation programmes. In addition, TB must achieve a higher profile in medical education programmes and in future strategic health care planning. Incidentally, how many readers were aware that 24 March 1999 was world TB awareness day?


Varicella: to vaccinate or not to vaccinate?
Editor.—Gershon summarised the rationale for universal varicella immunisation and strongly advocated its implementation in developed countries. While the incidence of varicella associated morbidity is indeed an important public health goal, it seems warranted to draw attention to epidemiologi-
cal, political, economic, and cultural charac-
teristics of Europe that may thwart the launch of a universal varicella vaccination pro-
gramme.

Recent epidemiological data indicate that severe complications of varicella are uncom-
mon in Europe.1 Persuading primary care physicians of the need for universal varicella vaccination will face obstacles similar to those reported from the state of Washington,2 where fewer than 50% of physicians surveyed followed the policy of universal varicella vacci-
nation. One of the main reasons for non-adherence was the impression that varicella is a benign illness. While ongoing monitoring of severe complications is crucial, it is clear that an appreciable burden of the illness must be obvious to those in charge of admin-
istering the vaccine.

The demise of the East–West divide, the Balkan war, and the free employment market in the European Union contribute to an accelerated migration of people across na-
tional boundaries, the directions and flow of which are poorly predictable. Unless a multi-
national varicella vaccination programme can be implemented, such migration will dilute immunisation rates, and the emergence of populations of susceptible adults becomes a realistic scenario.

The cost effectiveness of the varicella vaccine relies heavily on indirect savings resulting from the prevention of parental work loss. In many European countries, most children are cared for by one parent and in almost every family member. Overall cost effectiveness in this socioeconomic environment is unclear.

A considerable proportion of parents object to immunisations against common childhood illnesses. A recent survey1 of vaccine coverage at the age of 36 months among Swiss children found immunisation rates for diphtheria–tetanus, pertussis, and poliomyelitis of 98%, 90%, and 98%, respectively, while the rate for measles-mumps-
rubella (MMR) was as low as 76%. The last rate does not reflect the lack of opportunity, but the lower acceptance of the MMR vaccine. The varicella vaccine is likely to fall into the same category.

Both the safety and efficacy of the varicella vaccine are undisputed, and the prospect of preventing herpetic zoster may become a powerful argument supporting its use. However, strategies for overcoming the issues described must be available before universal vaccination should be recommended. Equally important, new vaccines of high priority, such as the pneumococcal conjugate vaccine, will soon be available. It seems prudent not to waste the cooperation of both paediatricians and parents with a vaccine of lower priority.

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Nurse-led asthma education and childhood asthma readmission rates

EDITOR—Wesselline and colleagues showed a significant decrease in asthma readmission rates in children whose parents received 20 minutes of structured asthma education before their child was discharged from hospital.

In October 1992, a community based, nurse-led, children’s asthma service was established in Central Manchester, UK with the aim of informing and empowering asthmatic children and their parents to undertake day to day management of asthma through a structured education programme. Over a third of the educational encounters take place in the patient’s home. Parents and teenagers have direct access to asthma nurse specialists by telephone during working hours and to the weekly asthma education drop-in clinic.

All children admitted to our inner city general paediatric unit with acute asthma are routinely referred to asthma nurse specialists. We undertook a retrospective case note audit to test the hypothesis that this structured nurse-led education programme would lead to a reduction in the hospital readmission rates for acute asthma. Hospital admission and readmission rates of all patients with acute asthma between the ages of 2 and 15 years were compared during 24 month periods before (January 1990 to December 1991) and after (January 1994 to December 1995) establishment of the nurse-led asthma education service (table 1).

We accept the limitations of using a retrospective case note audit; nevertheless, given the intensity of the educational input provided in the community and in the patient’s home, we were disappointed to observe a small increase in the rate of readmission of children with asthma in the “posteducation” period. The relatively deprived population that we serve (the average Jarman score of families studied was 44), cultural beliefs of some of our patients about asthma, and the content and timing of the education programme might have contributed to the failure of our service in reducing hospital readmissions. Results of the study by Wesselline and colleagues1 and our audit suggest that the delivery of an asthma education programme to the captive population in hospital rather than after discharge may be more effective in preventing readmissions.

If these findings are confirmed by further studies, it poses an important challenge in the delivery of a predischARGE asthma education programme in the setting of short stay or ambulatory paediatric units, which are increasingly being proposed as models of care for all but very ill children.

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Kearns Sayre syndrome initially presenting as hypomelanosis of Ito

EDITOR—Hypomelanosis of Ito (HI) is a neurocutaneous syndrome characterised by hypopigmented skin lesions along Blaschko’s lines, frequently associated with neurological, musculoskeletal, ocular, and other extracutaneous manifestations. Chromosomal mosaicism in skin fibroblasts is responsible for approximately 30% of the cases.1 Kearns Sayre syndrome (KSS) is a mitochondrial multisystem disorder, usually associated with a single large scale muscle mtDNA deletion. Its main clinical characteristics are progressive external ophthalmoplegia and pigmentary retinopathy but virtually all organ systems can be involved.2

We report a 13 year old boy who, at the age of 5 years, was diagnosed with HI and who later, at the age of 12 years proved to have KSS. At the age of 5 years the patient had short stature, mild mental retardation, corneal opacities and myopia, irregularly spaced teeth with hypoplastic dental enamel, hypopigmented patchy lesions on the left side of the trunk, and linear streaks on the left limbs. The laboratory investagation, including KSS or other mitochondrial disorders should be suspected in patients fulfilling the criteria of KSS.

At the age of 10, he presented with hypocalcaemic tetany. He had also bilateral ptosis of the eyelids, slurred speech, and limb weakness. Parathyroid ultrasound, serum 25(OH)D vitamin, muscle enzymes, tensin test, and brain magnetic resonance imaging (MRI) were normal. At the age of 12, ophthalmological examination did not reveal a general limitation of horizontal eye movements. Fundoscopy examination was normal but the electroretinogram was compatible with pigmentary retinopathy. Hearing acuity and cardiac function were normal. The laboratory investigations, relevant to mitochondrial cytopathy showed serum lactate, 2.80 mmol/l (normal 0.33–1.33), serum pyruvate, 0.235 mmol/l (normal 0.33–1.85), cerebrospinal fluid lactate, 7.25 mmol/l (normal 0.33–1.85), cerebrospinal fluid pyruvate, 0.209 mmol/l (normal 0.03–0.08), cerebrospinal fluid protein, 300 mg/dl (normal < 20 mg/dl). Brain MRI showed normal brain signals in the lenticular nuclei. Gomez trichrome stain of quadriceps muscle biopsy demonstrated ragged red fibres; biochemical analysis of mitochondrial enzymes in muscle revealed low cytochrome activity. Analysis of muscle mtDNA showed a large scale deletion (5 kb).

The wide spectrum and severity of manifestations of HI as well as its genetic heterogeneity led several authors to believe that HI is not a single clinical entity.3 As far as we know, hypopigmented skin lesions have not been described in KSS but they might have been overlooked. It must be noted that except for external ophthalmoplegia and hypoparathyroidism, all other extracutaneous abnormalities of our patient could be found in KSS as well as in HI.4 Although one case could be a chance association, we suggest that KSS or other mitochondrial disorders should be suspected in patients fulfilling the criteria of HI.

KAKOUROU TALIA
GAROUFI ANASTASIA
NIKOLAIDOU POLYXена
DAFINI EVMORFIA
TSAMOURI MGMGDALI
A PAPADIMITRIOU
T KARPATHIOS
2nd Department of Pediatrics, “A & P Kyriakou” Children’s Hospital, Athens 11527, Greece

<table>
<thead>
<tr>
<th>Table 1 Hospital admission and readmission rates of patients aged between 2 and 15 years with acute asthma before (January 1990 to December 1991) and after (January 1994 to December 1995) establishment of a nurse-led asthma education service</th>
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<tr>
<td><strong>Before</strong></td>
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<td>Total paediatric medical admissions</td>
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<td>Total acute asthma admissions</td>
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<td>Patients readmitted with acute asthma</td>
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<td>Asthma readmission rate</td>
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<tr>
<td>Patients referred to asthma nurse specialists</td>
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<td>Nurse-led educational sessions/patient (median; range)</td>
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<td>Overall</td>
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*Percentage of total acute general paediatric hospital admissions.


The vitamin K dekalbe and infants with cholestatic liver disease

Editor—We write in support of Tripp and McNinch’s paper recommending daily oral administration of 25 µg phytomenadione to all breast fed infants up to the age of six months. We remain concerned that high risk infants with cholestatic jaundice (up to 1 in 500 births) are still liable to develop bleeding secondary to vitamin K deficiency. An audit of all infants younger than 3 months admitted between 1 January 1998 and 30 June 1998 was carried out by retrospective review of case notes. The aim of the review was to evaluate vitamin K prophylaxis and the prevention of coagulopathy in jaundiced neonates. The setting was a supraregional centre for paediatric liver disease in Birmingham, UK.

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Letters, Book reviews

The role of the Glasgow meningococcal septicaemia prognostic score in the emergency management of meningococcal disease

Editor,—Pollard et al give an excellent review of the emergency management of meningococcal disease. We strongly support the involvement of the paediatric intensive care unit (PICU) at an early stage; however, only a third of all admissions with meningococcal disease are admitted to a PICU. Deciding which children need PICU admission can be a difficult clinical challenge. It would be inappropriate to transfer all children—they would occupy beds better used for other patients. Although the algorithm in fig 1 of Pollard et al’s article is useful in assessing children who may need PICU admission, it does not cover most children needing PICU admission as an adjunct to Pollard et al’s algorithm.

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Dr Pollard et al comment:

We are grateful that Carrol et al have raised the important issue of scoring systems in the evaluation of children with meningococcal disease. Such scores were designed and validated to predict death in cohorts of patients. In contrast, the emphasis of our algorithm is on the emergency management of meningococcal disease and prevention of death in the individual child. However, we recognise that the GMSPS may be useful in alerting the clinician to the important signs of critical illness in children with meningococcal disease and, when used for repeated review of such children, could aid the monitoring of stability or deterioration of the patient. GMSPS is also an important research tool allowing cohorts of patients to be categorised and compared between groups in audit, drug trials, and health planning.

Unfortunately, GMSPS is of only limited value when making decisions about the management of individual patients and could be misleading. False reassurance may be provided when the score is low. For example,
infancy it will be difficult to accept that there is any association between QT interval and sudden infant death syndrome.

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No strings attached: preventing deaths from children's clothing

EDITOR.—Drawstrings on children's clothing are a hidden hazard that can lead to death and injury when they catch on cribs, playground equipment, vehicles, and escalators.1–3 Although rare, 17 deaths and 42 non-fatal accidents involving the entanglement of drawstrings on children's outerwear were reported from 1985–95 in the United States. At least 12 of these incidents involved the entanglement of drawstrings in the doors of school buses.4 The recent unrelated deaths of two young boys in Ireland, following entanglement of part of the body of the child in the door of a school bus and rotating power-shaft of a tractor,5 prompted us to evaluate the safety of children's outerwear.

We evaluated the safety of different designs of children's winter outerwear, carrying drawstrings in nine department stores and boutiques in Cork City, Ireland. In addition, we examined the outerwear worn by children (younger than 12 years) in two local primary schools. Outerwear was defined as "safe" according to the following guidelines for children's outerwear established by the United States consumer product safety commission:6

- outerwear should not carry hood or neck drawstrings
- it should not have waist strings that extended beyond 7.4 cm
- the waist strings should be sewn down at the midpoint
- it should not be toggles or knots on the ends of strings.

Of the 77 different designs of outerwear examined from nine department stores and boutiques, only 28% (34 of 120) were "safe" according to the following guidelines for children's outerwear established by the United States consumer product safety commission:6

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The Irish Industrial Research and Standards Order 1976 states that "it is unlawful to manufacture, assemble or sell a child's outerwear if the hood is designed to be secured by means of a cord drawn through the material." This study confirms that this law is not
being enforced and that consumers are making unsafe choices in selecting outerwear. As the clothing stores we studied are common to most areas in the UK, we have no reason to believe that the design of children’s clothing in the UK is safer than in Ireland, despite the recommendations of the British Standards Institution.

The effectivenes of properly enforced legislatve and regulatory interventions in the children’s clothing industry has been well illustrated by the reduction in childhood burns from loose and flammable nightclothes. However, the fact that less than a quarter of children’s outerwear styles in shops were considered safe in terms of drawstrings indicates a lack of enforcement of recognised safety standards. Although some clothing hazards are difficult to correct without altering the function or aesthetic appeal, this is not the case for outerwear. Metal snaps, buttons, velcro or elastic can replace drawstrings, the main source of danger in outerwear.

Deaths due to children’s clothing are uncommon but prevenable. It is essential that care professional, the government, and clothing industry work together to ensure that established safety standards for the design, sale, and importation of children’s outerwear are properly enforced.

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Where should paediatric surgery be performed?

EDITOR,—The figures quoted for surgical activity in East Anglia by Arul and Spicer in their response to the letter by Wilkinson and Crowle are many years out of date. Over the four year period between 1995 and 1998 two accredited general paediatric surgeons working in Cambridge dealt with 504 neonates. Surgery requiring anaesthesia was carried out in 76% of these (table 1). However, we do support Arul and Spicer’s view that specialist paediatric surgical services should be centralised to concentrate experience and so maintain high standards.

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Thyroid dysfunction in Down’s syndrome: relation to age and thyroid autoimmunity

EDITOR,—The paper by Karlsson et al describes a longitudinal study of monitoring for thyroid disease in children and young people with Down’s syndrome. Although the authors conclude that “annual screening is important”, they do not provide any data to justify the interval being a year. We need to know the interval from changes in thyroid stimulating hormone concentrations and autoantibody titres to a subnormal thyroid level. If the authors have such data, it would be most helpful if they could supply it.

This matter is important in the UK where many districts are introducing an enhanced surveillance programme for children with Down’s syndrome, which includes thyroid function tests. The optimal screening interval for thyroid status is not known and needs to be studied unless Karlsson et al can provide such data.

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BOOK REVIEWS


How much information should children conceived from donor sperm be given about their origin? This is not a small problem now that 2500 children are born every year using donated gametes or embryos. I must admit that this was a problem that had never crossed my mind despite having an adopted son and being pretty much in favour of openness between parents and children on “secrets of the past”. Yet, if asked, I think I would have said it is different for children born as a result of donor insemination. Why? I suppose because I feel that the relationship a child might have with “a gamete” is different from that with a real father, and the same goes for the genetic father (or mother).

However, this book convinced me that openness in this field is desirable. It is clearly a situation that will become more common, and the present regulations—genetic parents who are donors have the right not to be identified to children—will need to change. I was particularly impressed by the point of view of “donor children” in this book, and the volume, which brings together anthropology, genetics, psychiatry, and the law in a robust attack on the culture of secrecy.

Christine Whipp did not learn until she was 41 that she had been conceived by donor insemination, but she had always had anxieties about her origins. This was partly because of a lack of resemblance to her father, and it is interesting that Lauren, the other donor insemination child who writes in the book says that “when secrets are kept the children often grow up sensing that something is different in their family”. There is another interesting quote from Lauren: “When I was younger I had a particularly nasty fight with my brother and afterwards I went up to mum and said, ‘His donor must have been a terrible man’. Mum asked, ‘why, what makes you say that?’ I replied, ‘Because his nature is nothing like yours!’ ”

I suspect that these views may not be typical, and that it is unusual for children to observe physical differences from their parents, unless someone draws attention to it. However, I can believe that children sense something unusual about their family, in the same way as they sense disagreement between parents, or a hidden secret about cancer. Another personal chapter in this book, which is elsewhere rather dry and repetitive, is by the mother of surrogate twins, born to another woman (a friend) but genetically her own. She describes her conflict with both the children—in what for them would be a very strange and unnerving experience “we never contemplated telling our children anything but the truth”. One daughter’s comment was “it was a good job we had Kim as your friend mummy, otherwise you wouldn’t have us.” The children grew up not only knowing Kim but also her family. This mother went on to donate an egg which was used for in vitro fertilisation.

Table 1  Surgical neonatal caseload at Addenbrooke’s Hospital 1995–98

<table>
<thead>
<tr>
<th>Year</th>
<th>Referrals</th>
<th>Surgery (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995</td>
<td>112</td>
<td>92 (83)</td>
</tr>
<tr>
<td>1996</td>
<td>135</td>
<td>96 (70)</td>
</tr>
<tr>
<td>1997</td>
<td>146</td>
<td>107 (73)</td>
</tr>
<tr>
<td>1998</td>
<td>109</td>
<td>88 (81)</td>
</tr>
<tr>
<td>Total</td>
<td>504</td>
<td>383 (76)</td>
</tr>
</tbody>
</table>


5 Grief for boy killed in freak accident. The Examiner, 9 September 1998.

6 Child dies after coat gets caught in tractor. The Examiner, 1 January 1999.

7 The British Standards Institution. The design, manufacture and manufacture of children’s clothing to promote mechanical safety, 1997;BS 7907.

Essentially, *Truth and the child* is a one-sided argument for telling children about their background. I support this, but I went away thinking I should know more about the other side of the argument, which does not appear in this book.

**TONY WATERSON**
Consultant Paediatrician (Community Child Health)

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Brian Hurwitz is a general practitioner and in this small book he has linked literature on clinical guidelines with views on how this might be applied in a legal context. He contrasts the views of the British Medical Association in 1946, which were that individual doctors retain full responsibility for the care of the patient, practising medicine according to its traditions, standards, and knowledge but with freedom of judgment and without interference in professional work, with patterns of practice that persist today.

In discussing the nature and context of clinical guidance, Hurwitz briefly reviews the large variety of terms in use ranging from protocols to codes of practice, and concludes that there is no single definition or guideline and, indeed, that guidelines vary in their quality and in the standards of health care they seek to establish.

He offers helpful criteria on how the authority of guidelines can be assessed and then goes on to discuss their developing legal status. He quotes examples of interaction between statute law and clinical guidelines in the UK, Europe, and the USA, emphasizing the higher degree of development and sophistication of this interaction in the USA compared to Europe.

He then applies this interaction to negligence case law, focusing particularly on UK practice. He concludes that guidelines are recognised by the courts as hearsay evidence only and are not a substitute for expert testimony. He nevertheless very reasonably raises the issue that doctors acting outside guidelines are exposed to the possibility of being found to be negligent in their practice.

There is a particularly interesting discussion on the potential liability of authors or sponsors, such as Royal Colleges, which recommends that the legal status of recommendations from bodies such as these should be made clearer to doctors. In contrast, he goes on to make the reasonable points that autonomous clinical thought is undermined by uniformity and that the pressure from purchasers, rigid adherence to guidelines cannot and should not be a formal managerial or legal expectation in the National Health Service.

While the themes presented and discussed by Hurwitz are of particular relevance to doctors with an interest in medicolegal practice, no paediatrician can be immune from an involvement with the issues he details.

This is a relevant and lucid exposition of the current status of clinical guidelines and how they can be, and are, applied in the legal situation. Its content is wholly applicable to paediatric practice and the book deserves to be widely read.

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**Educational video: Primary Ciliary Dyskinesia—How to Treat it and Live with it.** Primary Ciliary Dyskinesia Family Support Group.

The last time my Godsons came to stay, their father brought a video player along. He believed (wrongly) that they would not cope with the boys for the weekend without video diversion. Videos have become the mainstay of family life and are potentially a great educational resource. The Primary Ciliary Dyskinesia (PCD) Family Support Group have recently produced a video aimed primarily at families with PCD sufferers.

The video aims to promote better understanding of the disease, its management, and, most importantly, how to live with it. In this final objective it succeeds best. The individuals on the video give a clear cut, positive, and robust description of how they deal with their disorder, and this should be enormously helpful to newly diagnosed families. Another highlight is the beautiful video pictures of ciliary motion (produced I suspect by Chris O’Callaghan’s group).

I was slightly concerned that the narrator did not use more simple language. This was a missed educational opportunity, although, in practical terms, it could be overcome by a member of the team going through the video with some families.

I have two other minor quibbles. A throwaway line suggested that children aged 2–3 years could learn the active cycle of breathing technique. This is incorrect although they can certainly be taught how to huff and to blow. It is unfortunate that alternative remedies such as reflexology are given some credence towards the end of the video. Patients with PCD should be taught to put up without being exposed to cranks and charlatans. However, grind your teeth and put up with it, for on the whole your patients and your staff will be better off for having seen this video.

**ANNE THOMSON**
Consultant Paediatrician

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The relation between social disadvantage and infant health is of great topical and comprehensive book comprising three extensive literature reviews. It is timely in view of the recent publication of the Acheson report into health inequalities (*Inequalities in health: report of an independent inquiry. London: HMSO 1998*).

Parental smoking is a known risk factor for a range of adverse infancy and childhood outcomes. Socioeconomic status is a risk factor for the same adverse outcomes. The review presents unequivocal evidence for the link between increasing social disadvantage and parental smoking. The problems of disentangling the influence of these related factors on sudden unexpected death of infants (SUDI) and other adverse outcomes are considered, and the authors conclude that the extent to which smoking acts as a marker for socially disadvantaged factors and the extent to which it is a contributory or causal factor in the aetiology of SUDI is uncertain.

Parenting has been correlated with infant health outcomes. There is less clarity, however, in the association between specific aspects of parenting and specific health outcomes, with the possible exception of child abuse and neglect, and in the association between parenting and social disadvantage. The review concludes that, while there are significant differences in behaviour towards children and attitudes to child care associated with social variables, there is less evidence about differences in parenting knowledge and health beliefs. Further, the authors conclude that, with the exception of response to risk reduction recommendations, the mechanism by which any of the differences highlighted in the review may be related to SUDI is not clear.

The parenting review is helpful in that it focuses on the association of social disadvantage with specific aspects of parenting, and makes useful recommendations for future research; however, there are some glaring omissions. The major research programme into cycles of disadvantage in the 1970s by Brown and Madge is missing as is the work in the USA by Elder et al and others, which documented the direct effects of economic hardship on parenting. Brown and Madge concluded that multiple deprivation and its effect on children has been overemphasised, and material factors and not on family psychopathology.

Within the narrow context which the authors set themselves, their methodological review will be of great value to future researchers. They address the problems associated with studying parenting among families living in socially disadvantaged environments with a particular focus on the multiply disadvantaged. They consider in detail the complex, innovative, and challenging research strategies required to overcome the difficulties of engaging such families in research, and ensure their representation in population based studies.

A fundamental difficulty with methodological review is the narrow focus on multiply disadvantaged families as the main “problem.” This arises from the findings of the Confidential Enquiry into Sudden Infant Death (CESDI) study that, following the “Back to sleep” campaign, SUDI deaths are concentrated in the most disadvantaged families. Although an important observation, SUDI is common with other adverse infant, child, and adult health outcomes, shows a finely graded social patterning, also shown in the CESDI study. Focusing on the extreme group fails to address the true nature of the social gradient in infant and child health. Why, for example, are infants born to parents with incomes above £200 per week at less risk than those with incomes between £100 and £199 per week? These fundamental questions cannot be addressed by studying the most disadvantaged.

These reviews are refreshing and rewarding in that they treat social disadvantage as a serious risk factor rather than focusing exclusively on the attributes and behaviour of individual parents outside the social context of that behaviour; they should be of particular value to those with a serious commitment to promoting infant and child health.

**NICK SPENCER**
Professor of Community Paediatrics

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Copious information about the efficacy of growth promoting treatment for short stature of varying aetiology has been accumulating in recent years. Substantial insights are also developing into whether being taller (if achievable) is necessarily desirable or beneficial.

In the context of paediatric practice, children are commonly referred for short stature or pubertal delay and often demonstrate (apparently) short stature related psychosocial stress and distress. Is short stature psychologically stressful in itself? Do children with short stature, whether those with underlying pathology or those at an extreme of the normal height distribution, have clinically significant behavioural, emotional, or educational problems? Do children and adolescents with short stature or pubertal delay who are not referred have the same psychological problems as some of those who are? Do the psychological stresses of being short contribute to the development of psychological problems? How do we measure the emotional cost of coping? Does increased growth, in the short or long term, reduce or eliminate the “at risk” psychological status of such children? What are the effects of emotional disorders on growth?

Some of these questions were addressed and discussed at a symposium in Zürich, Switzerland entitled “Growth and psyche” and this book contains the substance of the papers presented. There are 17 contributions from psychologists, psychiatrists, clinicians, and auxologists from Europe, Scandinavia, and the United States with contributions from a number of well known names in the field. The intention of the symposium was to examine the quality of life of small children, and thus to discuss why results in this area have been contradictory, to develop insights into the problems of psychometric methods, and to examine the effects that psychological factors may have on growth.

This book reflects the strengths and deficiencies that would be expected given its origin in a symposium, with chapters that are respectively too superficial and out of date (at one point referring to “an excellent review on recent research” dated 1989) and totally un referenced apart from the author’s own previous review from 1991.

Helpful insights can be gained from a number of chapters, for example, Lindemann’s comment from an evolutionary perspective that “the power of height stereotypes should not be over emphasized—we are not at the mercy of our evolution and stereotypes are often short lived and narrow.” Some chapters are of particular interest and can be read with profit by anyone involved in this area (including all paediatricians). Nevertheless, the book fails to deliver the “clear overview of the current state of knowledge” promised in the foreword.

C J H KELNAR
Consultant Paediatric Endocrinologist


Paediatric oncology textbooks often open with a chapter on epidemiology, usually rather brief and tending to reflect the particular interests of the authors or editors. Numerous review articles have dealt with a few putative risk factors, but until now there has been no comprehensive review of the evidence on childhood cancer aetiology.

Epidemiology of Childhood Cancer represents a Herculean feat of almost single handed compilation and synthesis (only one chapter has any authorship). The book has its main strengths and weaknesses. The introductory chapter contains a lucid discussion of study design, including methods of control selection and the principal sources of bias. This underpins the review of analytic studies that occupies most of the book and could be read with profit by anyone contemplating setting up an epidemiological study. Chapter 2, on descriptive epidemiology, sets a pattern by considering all relevant studies published by early 1997 relating to each major type of cancer in turn. The next eight chapters form the core of the book, each covering a group of possible risk factors. The best of these provide detailed reviews of the evidence on environmental factors such as ionising radiation, electromagnetic fields (on one of each), and exposure to chemicals and dusts. Where a factor has been the subject of several studies, their characteristics and results are tabulated. The tables do not usually give confidence intervals for relative risks, and some are very long and hard to follow; the one relating to birth weight occupies five pages and has 41 footnotes.

The book could on occasion be more critical. For example, the author remarks that smoking in pregnancy is the most important single determinant of low birth weight but does not comment on how few analyses of birth weight and childhood cancer have allowed for maternal smoking or social class.

The estimates of risks for siblings of children with cancer appear not to take into account the need to allow for method of ascertainment, and are of limited value for genetic counselling. A useful concluding chapter summarises factors investigated in relation to each diagnostic group, in each case ranging from those generally accepted to be associated with the specific cancer to those not generally associated. The bibliography of over 1000 references includes virtually every epidemiological study since 1990, but is less complete for earlier publications and relevant clinical and laboratory work. The index is not generally associated. The bibliography of over 1000 references includes virtually every epidemiological study since 1990, but is less complete for earlier publications and relevant clinical and laboratory work. The index is not generally associated. 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