The preparation used by Shield *et al* had the same concentration of anticryptosporidial titres as preparations used and reported by other authors.^{1,4} As the patient died six months after completing the treatment I believe it would be speculative to state that permanent clearance of cryptosporidia had occurred; significant reinfection may have been detected at some point in the future had the patient survived. It was not clear whether serum immunoglobulins had been abnormal during the course of the study and neither whether human serum immunoglobulin had been administered at any point; these are factors which may have had bearing on the clinical course.

It has been observed that colostrum contains significant concentrations of non-antibody immunologically active compounds including glycoconjugates that may have activity against cryptosporidia.⁶ The pathophysiology of cryptosporidiosis is unclear and lack of effective mucosal antibody may be only one part of a complex disease process. This may be why diverse approaches to enteral immunotherapy have all shown promise. There are no data available so far to confirm that one preparation is superior to another in the management of cryptosporidiosis affecting immune deficient patients and I believe that continued single case reports will not clarify the situation. Controlled trials may enable comparisons to be made between different enteral preparations not only in terms of effectiveness but also cost, palatability, dosage, and duration of treatment.

PAUL HEATON
Taranaki Base Hospital,
Private Bag 2012,
New Plymouth 4620,
New Zealand

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Inappropriate prescribing of promethazine in infants

EDITOR,—Several publications have indicated a possible link between phenothiazine administration and some cases of sudden infant death syndrome (SIDS).¹⁻³ Prompted by the observation that four of seven infants presenting to one Belgian hospital with SIDS had received trimeprazine in the days before death,¹ Kahn and Blum prospectively studied 52 SIDS victims, 36 near miss infants, and 175 controls and found 23% of SIDS victims, 22% of near miss infants, and 2% of controls were taking a phenothiazine preparation (with the same proportion of infants in each group suffering from nasopharyngitis).² Furthermore, the same group investigated the influence of phenothiazines on cardiorespiratory

and sleep characteristics in four normal infants.³ In these infants recordings showed an increase of 39% in the number of central apnoeas and short lived obstructive apnoeas on the second night of promethazine administration. These authors suggest that phenothiazines may cause central and obstructive apnoea in infants and reduced arousal and recommend that all central nervous system depressants are avoided in children under 1 year. Alternative mechanisms for phenothiazine induced apnoea have been suggested including an increase in endogenous opioid activity and an alteration in temperature regulation.^{4,5} Reviewing these studies Cantu felt that the data linking phenothiazines and SIDS was inconclusive but advised caution in the use of this class of drugs in infants less than 1 year in view of the risk of central nervous system depression and apnoea.⁶

We are concerned that promethazine is frequently prescribed for children under 2 years despite recommendations to the contrary. On reviewing the notes of the 93 consecutive children under 2 years attending casualty at Birmingham Children's Hospital with respiratory symptoms during the week before Christmas 1992, we found that 10% (six of 59 infants) of those under 1 year and 3% (one of 34 children) of those between 1 and 2 years were taking promethazine.

The manufacturers data sheet for promethazine hydrochloride (Phenergan, Rhône-Poulenc Rorer) states 'not recommended' in children less than 1 year and 'use as recommended by a doctor' in children from 1-2 years. We recognise the ambiguity of data sheet entries for many drugs used in childhood with respect to product licences and are aware that more than 25% of all drugs used at Birmingham Children's Hospital for accepted clinical indications are used outside of assumed product licence regulations (personal communication). However, the potential risks of administration of promethazine to infants outweigh any possible therapeutic benefit and we therefore urge doctors, pharmacists, and parents to avoid its use in infancy.

A J POLLARD
G RYLAND
The Children's Hospital,
Ladywood Middleway,
Birmingham B16 8ET

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Child resistant packaging and accidental child poisoning

EDITOR,—The introduction of child resistant closures for children's aspirin and paracetamol preparations in the UK in 1976 led to a significant fall in the numbers of children admitted to hospital for accidental aspirin and paracetamol poisoning.¹ The Pharmaceutical Society has since recommended that liquid methadone and all solid dose formulations are

issued in bottles with child resistant closures (R Odds, personal communication).

We are carrying out a population based study of children attending accident and emergency departments as a result of injuries and poisoning in a district in south London. On the 73 days studied over a one year period, there were three children between 2 and 3 years of age who presented after paracetamol ingestion. They had all taken liquid paracetamol, dispensed on prescription, in containers without child resistant closures.

Proprietary brands of paracetamol elixir are supplied with child resistant closures but hospital and private pharmacies dispense prescriptions of generic paracetamol elixir in bottles with plain tops. The reason given for this practice is that a standard child resistant closure design for use with the bottles used by pharmacists for liquid prescriptions has not yet been finalised and made generally available.

Although the number of children reported here is too small to allow the calculation of risk in a population, they did account for nearly 10% of all accidental ingestions in this sample. This suggests that there may be a significant number of children at risk from an easily avoidable hazard. The Department of Health should be encouraged to ensure that adequate supplies of child resistant closures are produced and that their use for children's liquid formulations is recommended. Payment for dispensing should include reimbursement of any extra cost involved in using these lids.

GABRIELLE LAING
Child Health Unit,
West Lambeth Community Care Trust,
South Western Hospital, Puttoss Road,
London SW9 8EA

MARY THOMPSON
Optimum Health Care,
St Giles Hospital,
St Giles Road,
London SE5 7RN

STUART LOGAN
Department of Paediatric Epidemiology,
Institute of Child Health,
30 Guilford Street,
London WC1N 1EH

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Nasal instillation of 'Olbas Oil' in an infant

EDITOR,—Proprietary formulations of essential oils are readily available to the public for inhalation and are enjoying an increased popularity as natural remedies. Their toxicity when taken inappropriately by ingestion, ocular or nasal instillation is not generally appreciated. We report a case of nasal instillation.

Case history

A 4 month old boy had had four days of upper respiratory tract symptoms affecting feeding, and a relative had given his mother, a 30 year old woman with three other children, some 'Olbas Oil' without the box or instructions. She did not notice the warning against use in infants and put several drops in his right nostril. He immediately coughed, became tachypnoeic, and his colour deteriorated. An ambulance was summoned and he was brought into casualty.

On arrival he was peripherally cold with

poor perfusion. His respiratory rate was 80–90/min with wheeze, inspiratory stridor, central cyanosis, and an oxygen saturation of 85% in air. His heart rate was 160/min and there were profuse oral and nasal secretions requiring repeated suction. The pupils were constricted but the child was too agitated for formal neurological assessment. The cyanosis responded readily to facemask oxygen. Over a period of 24 hours his respiratory rate settled and he remained well saturated in air. However, two hours after admission the boy's eyes became inflamed and an ophthalmological examination revealed bilateral superficial corneal scarring. There was also a chemical conjunctivitis making it impossible for the infant to open his eyes. The child was treated with regular saline (0.9%) irrigation and chloramphenicol prophylaxis, the inflammation settled over the next four days. Fortunately there was no residual scarring.

We have been unable to find any reports of nasal instillation of Olbas Oil in infants. Olbas Oil contains oils of peppermint, clove, eucalyptus, menthol, and cajup but it is the latter three that cause the respiratory symptoms. Menthol and eucalyptus are terpenes that if ingested may cause epigastric burning, nausea, vomiting, dizziness, miosis, tachycardia, and a feeling of suffocation. In infants cyanosis, delirium, and convulsions may occur.¹ The respiratory symptoms associated with instillation, although dramatic, resolve eventually and treatment is supportive.

Oral poisoning with menthol and eucalyptus oils has occurred in the UK^{2,3} but nasal instillation is more common in Europe where bottles may be confused with normal saline.⁴ However, ocular involvement has been described only after direct application of camphor and eucalyptus.⁵

In young children nasal instillation causes immediate respiratory distress and agitation. This can lead, as in this case, to ocular administration. In patients with severe respiratory symptoms, needing oxygen or ventilation it is important to get an ophthalmic opinion. Untreated the oils can cause local burning and permanent corneal scarring.

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JONATHAN P WYLLIE
Department of Paediatric Cardiology,
Freeman Hospital, Heaton,
Newcastle upon Tyne NE7 7DN

FRASER W ALEXANDER
Department of Paediatrics,
Newcastle General Hospital,
West Road,
Newcastle upon Tyne NE4 6BE

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Pseudomonas cepacia

EDITOR.—The spread of *Pseudomonas cepacia* through previously stable populations of cystic fibrosis clinics is an increasing cause for concern. Anxiety has arisen after observations that some patients with previously mild

disease experience an accelerated and fatal deterioration in lung function after colonisation with the organism.¹ Early surveillance studies in the UK suggested a maximum prevalence of 7%,² however, this has risen in recent reports to approach the 40% described in contemporary studies from North America.³ The organism, which is an environmentally ubiquitous plant pathogen, is nevertheless surprisingly difficult to isolate from the environment; a recent American study only obtained 15 isolates from 900 cultures.

Mounting evidence of person to person transmission⁴ has led the Cystic Fibrosis Trust to issue guidelines for the management of colonised patients. This advocates segregation for both inpatients and outpatients, with restrictions on socialising and group activities. *P cepacia* positive patients, therefore, feel frightened and victimised. To address the concerns within our own clinic we have looked for evidence to support person-to-person transmission of the organism.

Sputum samples from all 118 patients attending both the adult and paediatric cystic fibrosis clinics in Sheffield were routinely cultured for the detection of *P cepacia* on Mast *P cepacia* selective agar (Mast Diagnostics Ltd, Bootle). Suspect colonies were further identified by the API 20NE system (BioMerieux, Basingstoke). However, as the API 20NE system cannot differentiate between isolates of *P cepacia*, ribotyping techniques were employed. Traditional ribotyping detects genomic restriction fragment length polymorphisms by probing the extracted chromosomal DNA with ribosomal RNA. The analysis of DNA rather than phenotypic characters provides a more stable determination of isolate identity.

Ribotyping using the restriction endonuclease enzyme *ECO* R1 (kindly performed by Dr Ty Pitt; Central PHLS, Colindale, London) was performed on all strains of *P cepacia* isolated in 1992/3. The five patients positive for *P cepacia* within this period (three of whom were previous positives) had strains with differing ribotype patterns. None of the patients exhibited the 'epidemic' S3/PO: ribotype A strain, nor was there evidence of a rapid deterioration in pulmonary function in this group of patients with forced expiratory volume in one second (FEV₁) essentially unchanged compared with values 12 months before and 12 months after isolation of the organism (table).

Although the modes of transmission of *P cepacia* remain unclear, epidemiological evidence from this study suggests person-to-person spread has not occurred in this cystic fibrosis population. In the absence of the epidemic type A strain, strict segregation may not be necessary.

C J TAYLOR
Department of Paediatrics,
University of Sheffield,
Sheffield Children's Hospital,
Sheffield S10 2TH

Change in FEV₁ after acquisition of *P cepacia*

Patient No	Age first positive (years)	Date	FEV ₁ (% predicted)			Genotype	<i>Pseudomonas aeruginosa</i>	<i>Aspergillus</i>
			Previous	At isolation	After 12 months			
1	14	14.7.87	41	29	36	508/508	+	+
2	4.1	25.8.91	N/A	N/A	N/A	508/N	+	
3	25	15.4.93	58	56	52*	508/508	+	
4	14	17.6.92	49	40	45	508/N	+	+
5	12	3.8.92	57	50	65	508/508	+/-	+

*FEV₁ seven months after isolation. N/A=not available.

R HOWDEN
T SMITH
R C SPENCER
Department of Microbiology,
Royal Hallamshire Hospital,
Sheffield S10 2JF

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BOOK REVIEWS

Childhood Onset Anorexia Nervosa and Related Eating Disorders. Edited by Bryan Lask and Rachel Bryant-Waugh. (Pp 263; £19.95 hardback.) Lawrence Erlbaum Associated Ltd, 1993. ISBN 0-86377-294-3.

This book emanates from the professionals working in the children's inpatient unit in the Department of Psychological Medicine at the Hospital for Sick Children in Great Ormond Street. It draws on relevant literature and the professionals' own experience working with young people below the age of 15 who have anorexia nervosa or related eating disorders. The intention is to fill a gap. Although there are many books about anorexia nervosa, nothing focuses specifically on this age group. The intention appears to be very practical, namely to provide the appropriate background and guidance to those professionals concerned with the direct treatment of children with these disorders.

The book falls roughly into two halves apart from a challenging initial chapter from a parent. The next five chapters review relevant literature concerning the description and classification of these eating disorders, physical aspects, epidemiology, aetiology, and prognosis and outcome. A chapter on assessment is then followed by nine chapters about overall management and the various contributions to treatment including nursing, physical treatment, behavioural and cognitive approaches, individual psychodynamic psychotherapy, group therapy, and finally schooling.

This book is very practical in its orientation and easy to access. Chapters are very clearly structured with a liberal use of headings.