6 Cooke RPD, Riordan T, Jones DM, Painter MJ. Secondary cases of meningococcal infection among close family and household contacts in England and Wales, 1984-87. *BMJ* 1989;298: 555-8.
7 Olivares R, Hubert B. Clusters of meningococcal disease in France (1987-1988). *Eur J Epidemiol* 1992;8:737-42.
8 Ege PP, Samuelsson SI, Lind I. [Meningococcal disease 1987-1989. Outbreak of serogroup B:15:PI.16 in the county of Frederiksborg]. *Ugeskr Laeger* 1993;155:3456-9.
9 Public Health Laboratory Service Communicable Diseases Surveillance Centre Report. *BMJ* 1986;293:1293-4.
10 PHLS Meningococcal Infections Working Group and Public Health Medicine Environmental Group. Control of Meningococcal disease: guidance for consultants in communicable disease control. *Communicable Disease Report* 1995;5(13):R189-93.

Diphtheria: are we ready for it?

EDITOR,—The article by Begg and Balraj discusses the adequacy of current control and containment measures for diphtheria.[1] We agree with the authors that the diagnosis of *Corynebacterium diphtheriae* and *Corynebacterium ulcerans* has in the past often been delayed or missed altogether as many laboratories have ceased to culture throat swabs routinely for these organisms.

The Public Health Laboratory Service (PHLS) Standardisation of Clinical Bacteriology Methods Working Group recommends in their standard operating procedure (SOP) on the investigation of throat swabs that all throat swabs should be cultured routinely for *C diphtheriae* and *C ulcerans* using Hoyle's tellurite medium.[2]

Reasons for this include:

- Immunisation does not prevent asymptomatic carriage of the organism
- Vaccinated individuals may still be susceptible
- There is a risk of indigenous transmission
- A major outbreak is possible
- Early recognition of a case allows for containment of the patient
- Treatment must be initiated at an early stage to reduce the risk of fatality.

With laboratories returning to this kind of routine screening, isolation of *C diphtheriae* and *C ulcerans* from asymptomatic carriers will be increased thereby minimising the potential for missed or delayed diagnosis. It will also allow for the collection of consistent epidemiological data on the presence of the organism in the population.

The PHLS has recently issued a standard method for the investigation of throat swabs as part of the 50 specimen SOPs to be issued to the PHLS during the next 18 months.[3] These SOPs may also prove useful to microbiology laboratories other than the PHLS.

KURT DJEMAL
VALERIE BEVAN
PHLS, Headquarters Office, 61 Colindale Avenue, London NW9 5DF

1 Begg N, Balraj V. Diphtheria: are we ready for it? *Arch Dis Child* 1995;73:568-72.
2 Public Health Laboratory Service. *PHLS clinical microbiology standard operating procedure for the investigation of throat swabs*. B.SOP9; Issue 1. London: PHLS, 1996.
3 Djemal K, Matthews S, Bevan V. Standardisation of clinical bacteriology methods. *PHLS Microbiology Digest* 1996;13 (1):39-40.

Non-steroidal anti-inflammatory drugs may predispose to invasive group A streptococcal infections

EDITOR,—The suggestion that ibuprofen should be considered as an alternative to paracetamol for the treatment of fever in young children [1] warrants caution. There have been numerous reports suggesting an association between the use of non-steroidal anti-inflammatory drugs (NSAIDs) and the progression to severe invasive group A streptococcal infections, including necrotising fasciitis.[2-6] NSAIDs may also mask important clinical features that may help in the early recognition of invasive group A streptococcal disease.

Prompt diagnosis and treatment of group A streptococcal infection has become increasingly important as there has been a worldwide resurgence in invasive group A streptococcal disease since the mid-1980s with the emergence of strains of increased virulence.[5]

Recently, it has been proposed that the underlying biochemical basis for the possible link between the use of NSAIDs and invasive group A streptococcal infection is the ability of NSAIDs to inhibit neutrophil function and enhance cytokine (particularly tumour necrosis factor) production.[6] In addition, by masking cardinal signs of inflammation, such as myalgia, arthralgia, erythema and localised swelling, these agents may delay the recognition of invasive group A streptococcal infection until signs of shock and multiorgan failure are apparent. This hypothesis may also apply to staphylococcal toxic shock syndrome.

Varicella is an important predisposing factor for both invasive group A streptococcal and staphylococcal infection in immunocompetent children.[7] NSAIDs may be particularly dangerous in this condition: their use has been associated with the progression to necrotising fasciitis and toxic shock syndrome.[4]

Antipyretics play an important part in the management of febrile young children with non-specific signs in whom the diagnosis is unclear. However, the possibility that NSAIDs may facilitate the invasion of group A streptococci should limit the use of these agents in patients with varicella or in those in whom the cause of fever is not known.

NIGEL CURTIS
Paediatric Infectious Diseases Unit, Department of Paediatrics, Imperial College School of Medicine at St Mary's, Queen Elizabeth the Queen Mother Wing, South Wharf Road, London W2 1NY

1 McIntyre J, Hull D. Comparing efficacy and tolerability of ibuprofen and paracetamol in fever. *Arch Dis Child* 1996;74:164-7.
2 Rimailho A, Riou B, Richard C, Auzepy P. Fulminant necrotizing fasciitis and nonsteroidal anti-inflammatory drugs. *J Infect Dis* 1987;155: 143-6.
3 Smith RJ, Berk SL. Necrotizing fasciitis and nonsteroidal anti-inflammatory drugs. *South Med J* 1991;84:785-7.
4 Brogan TV, Nizet V, Waldhausen JH, Rubens CE, Clarke WR. Group A streptococcal necrotizing fasciitis complicating primary varicella: a series of fourteen patients. *Pediatr Infect Dis J* 1995;14:588-94.
5 Curtis N. Invasive group A streptococcal infections. *Current Opinion in Infectious Diseases* 1996; 9:191-202.
6 Stevens DL. Could nonsteroidal antiinflammatory drugs (NSAIDs) enhance the progression of bacterial infections to toxic shock syndrome? *Clin Infect Dis* 1995;21:977-80.
7 Pollard AJ, Isaacs A, Lyall EGH, *et al.* Life threatening bacterial infection associated with varicella. *BMJ* 1996;313:283-5.

HIV related Kaposi's sarcoma

EDITOR,—McCarthy *et al* report a Zambian mother and her son with HIV related Kaposi's sarcoma and suggest the possibility of vertical transmission of a Kaposi's sarcoma agent.[1] They comment on the rarity of Kaposi's sarcoma in the United States and Europe and in their abstract say that it is 'extremely rare in children'.

In Kampala Kaposi's sarcoma is not rare. In a study of children presenting with lymphadenopathy to the paediatric wards of Mulago Hospital, Kampala, between September 1992 and April 1993, 15 cases were diagnosed. The sex ratio was M:F 10:3. Since 1993 about two to three cases are being diagnosed each month. Apart from lymphadenopathy, which can be differentiated from tuberculosis only by histology, other clinical features include pigmented nodular skin lesions, hepatosplenomegaly, pleural effusion, and oral mucosal lesions. We do not have facilities for bronchoscopy, lung puncture, or intestinal biopsy to detect internal lesions. As mothers are not routinely examined in cases of childhood Kaposi's sarcoma we do not know how many might have it. However, it would not be surprising, considering the not uncommon occurrence of Kaposi's sarcoma in women, that both mother and child might have it if they originate from areas of high HIV prevalence such as Uganda or Zambia.

Nevertheless, a transmissible Kaposi's sarcoma agent (whether vertically transmitted or not) is a possibility, and we are also looking for an association with a herpes-like virus.

J B S COULTER
Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA

C M NDUGWA
Department of Paediatrics and Child Health, Makerere University, PO Box 7072, Kampala, Uganda

1 McCarthy GA, Kampmann B, Novelli V, Miller RF, Mercey DE, Gibb D. Vertical transmission of Kaposi's sarcoma. *Arch Dis Child* 1996;74:455-7.

BOOK REVIEWS

Symptoms of Disease in Childhood. By T J David. (Pp 258; £15.95 paperback.) Blackwell Science, 1995. ISBN 0-632-03635-4.

Updating *Common Symptoms of Disease in Children* by R S Illingworth, this book follows a symptomatic as opposed to a system approach. For each of the 185 symptom (sign) headings a list of causes is followed by a text, giving a brief account of the conditions listed.

In an attempt to be thorough, many of the lists are lengthy and daunting not only to the medical student but also to the experienced paediatrician. Some attempt has been made to subdivide the causes but further subdivision would have supported the problem solv-

ing theme: bronchiolitis, cystic fibrosis and bronchopulmonary dysplasia, among others, are listed together as causes of wheezing but would rarely be considered together as possible diagnoses.

In the text I would have welcomed more emphasis on differential diagnosis and more information about the relative incidence of the conditions mentioned under each heading. There are some excellent concise accounts. These are particularly useful where information in textbooks is lacking, for example Munchausen disease by proxy, and where an overview directs further reading, for example headaches and convulsions. However, combining an attempt to say something about every condition with a need for brevity often results in simplistic accounts, for example, 'Crohn's disease is a rare though important cause of abdominal pain in childhood'.

This book, despite its limitations, usefully supplements standard texts. Paediatricians may be helped when encountering less common symptoms and signs and when there is a need to consider the rarer causes of common symptoms. I have found it helpful when considering the differential diagnosis of such diverse symptoms and signs as livedo reticularis, excessive sweating, and the rarer causes of apnoea in infancy. The enthusiastic student may also usefully refer to the book when starting to face diagnostic dilemmas.

CAROLYN J ADCOCK
Senior registrar

Paediatric Pathology. 3rd Ed. Edited by Sir Colin Berry. (Pp 942; £195 hardback.) Springer-Verlag, 1996. ISBN 3-540-19936-5.

Professor Berry's *Paediatric Pathology* now enters its third edition, only six years after its second. Over the three editions the size has increased significantly with a smaller text size on more pages. As a paediatric book, it largely avoids those diseases and conditions associated more with the process of birth or prematurity, although of course congenital malformation features large. While some overlap is inevitable, it is a stablemate of, and largely complementary to, Keeling's *Fetal and Neonatal Pathology*. The book is directed primarily towards the general rather than the specialist paediatric pathologist and, as there is no direct competition in paediatric pathology, should find a receptive audience.

As a paediatric pathologist, I have found previous editions a little too thin to be of very much practical help when faced with a problem. Exception are those chapters that describe a very practical approach to a problem whether of description (cardiac) or of specimen handling and diagnostic requirements (metabolic).

If described at all, conditions have been covered too briefly with little discussion of differential diagnosis. As I doubt a general pathologist will need less information and explanation to understand a problem than a specialist, I suspect my experience is true for the target readership.

But that is the past, what of the third edition? There is no significant change to the overall format. As before, chapters cover organ and system pathology in a conventional manner but also with chapters on sudden unexpected infant death, embryonal tumours, and theoretical aspects of congenital malformation. There are some changes in authorship and new chapters on the pathology of AIDS and bone marrow pathology. The text is well set out and the illustrations generally of good quality.

However, the most significant alteration since the first edition is a cumulative one. The modification of chapter titles together with a gradual expansion of some chapter lengths, not necessarily extensive, has led to a text that will be a better resource to general and specialist pathologists needing an introduction to less familiar areas. It may be premature to look forward to the fourth edition, but I hope this trend towards expansion continues.

STEVEN GOULD
Consultant paediatric pathologist

Gellis and Kagan's Current Pediatric Therapy. 15th Ed. Edited by F Burg, J Ingelfinger, E Wald, and R Colin. (Pp 894; £55 hardback.) WB Saunders, 1996. ISBN 0-7216-5016-3.

Over the years successive editions of Gellis and Kagan's classic work, just like Topsy, have 'growed and growed'. Thirty years on and now in its 15th edition, this magnum opus has four editors and 435 contributors. It seemed a little incongruous (and more than a little overwhelming) for a single reader to comment on such a body of scholarship and, in an attempt to redress the numerical imbalance, I enlisted a handful of willing colleagues to help me undertake the task! These included a couple of general paediatricians, one with an interest in rheumatology, a paediatric oncologist, and a senior registrar. We each chose relevant sections of the book to read. I then collected comments, allowed them to simmer for several weeks and finally tried to prepare a distillate which was representative of our views.

Firstly, the design characteristics of the book were appreciated; printing was clear, subheadings stood out, tables were easily assimilated, and key references were appended after each author's contribution. Inevitably the style of the text was a little uneven with such a huge authorship but it was easy to find one's way around the volume. The book is truly comprehensive with sections on fetal and adolescent medicine, behavioural and social medicine, and balanced consideration is given to emergency management of acute disorders as well as long term management of chronic conditions. I failed to find guidance on one topic only—pain relief in the dying child.

The consensus view is that Gellis and Kagan is a good reference book for providing the historic perspective on treatment as well as current concepts, although precise practical advice on challenging problems is sometimes lacking.

It should be noted that despite our reservations, my willing helpers have extracted a promise that the copy of this book that we are allowed to keep as a reward for our labours is generally available !

GAYNOR F COLE
Consultant paediatric neurologist

Correction

Byler-like familial cholestasis in an extended kindred

An error unfortunately occurred in this paper by Bourke *et al* (1996;75:223-7). A vertical line indicating descent of the father of the larger sibship and his sister, the mother of the smaller affected sibship, from the second consanguineous grandparental marriage was inadvertently omitted from figure 1. The correct depiction of the figure is shown below.

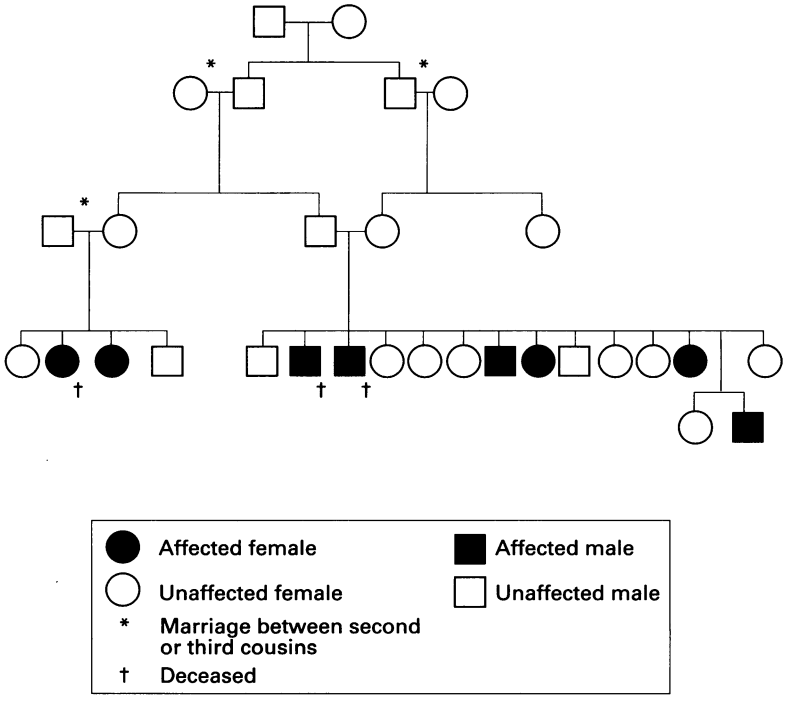

Figure 1 Pedigree of Irish Byler kindred illustrating high degree of intermarriage.