Use of cyclosporin A as a steroid sparing agent in cystic fibrosis

Dr CE Daman-Willems, Dr R Dinwiddie, Prof JF Price, Dr HA Wyatt, and Dr GJ S A Maguire

We report six paediatric CF patients where CyA had been used as a steroid sparing agent. These patients were on treatment with high dose inhaled or nebulised steroids prior to the commencement of oral steroids, and repeated attempts at reducing the steroid dose were unsuccessful. All patients exhibited steroid related complications including Cushingoïd features, growth suppression, impaired glu- cose tolerance, hypertension, osteoporosis, and bone fractures. The dosage of CyA was adjusted to maintain whole blood trough lev- els between 100 and 150 ng/ml, using CyA doses ranging from 2 to 37 mg/kg/day.

In the four patients who benefited from CyA therapy the mean steroid dose decreased from 0.86 mg/kg/day in the one month prior to commencement of CyA to 0.30 mg/kg/day six months later and 0.25 mg/kg/day 12 months later. These patients were able to dis- continue oral steroids within 18 months of commencement of CyA. Two patients did not show a reduction in mean steroid dosage, one of which underwent a successful heart-lung transplantation.

In the four patients who responded to CyA, lung function was maintained or improved, as were Chripin-Norman chest x ray scores. Height velocity was also improved. Three patients did develop transient renal impair- ment, of whom only one required discontinu- ation of CyA. This was dose related and reversible as is infrequent with lower dose regimens used for anti-inflammatory therapy. Other side effects due to CyA were minimal, including mild hypertrichosis and gingival hyperplasia. There was no evidence of hypertension, hepatotoxicity, or neurotoxicity. The side effect profile of CyA is no more severe than for other immunosuppressive agents.

It is evident that CyA is a powerful but potentially toxic therapeutic agent and its use should be balanced against the risks of the disease and the long term use of steroids. These results suggest that CyA can be beneficial as a steroid sparing agent in CF patients; these data may be of help to the clinician in comparable clinical circumstances.

We are grateful to Dr CE Daman-Willems, Dr R Dinwiddie, Prof JF Price, Dr HA Wyatt, and Dr GJ Connell for allowing us to use their patients in this report.

G K BHAL
S A MAGUIRE
1 M BOWLER

Survey of criteria used to diagnose allergic bronchopulmonary aspergillosis in cystic fibrosis

Dr CE Daman-Willems, Dr R Dinwiddie, Prof JF Price, Dr HA Wyatt, and Dr GJ S A Maguire

We report six paediatric CF patients where CyA had been used as a steroid sparing agent. These patients were on treatment with high dose inhaled or nebulised steroids prior to the commencement of oral steroids, and repeated attempts at reducing the steroid dose were unsuccessful. All patients exhibited steroid related complications including Cushingoïd features, growth suppression, impaired glu- cose tolerance, hypertension, osteoporosis, and bone fractures. The dosage of CyA was adjusted to maintain whole blood trough lev- els between 100 and 150 ng/ml, using CyA doses ranging from 2 to 37 mg/kg/day.

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G K BHAL
S A MAGUIRE
1 M BOWLER

Table 1 Replies to questionnaire (% of all units)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Assessed yearly or more</th>
<th>Must be present</th>
<th>Prefer to be present</th>
<th>Not important</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspergillus precipitins</td>
<td>83</td>
<td>42</td>
<td>49</td>
<td>10</td>
</tr>
<tr>
<td>Aspergillus specific IgE</td>
<td>52</td>
<td>54</td>
<td>31</td>
<td>15</td>
</tr>
<tr>
<td>CXR infiltrates</td>
<td>95</td>
<td>38</td>
<td>41</td>
<td>17</td>
</tr>
<tr>
<td>Blood eosinophilia (&gt;500/mm³)</td>
<td>83</td>
<td>24</td>
<td>56</td>
<td>20</td>
</tr>
<tr>
<td>Aspergillus fumigatus skin test</td>
<td>5</td>
<td>11</td>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>Total serum IgE (&gt;1000 ng/ml)</td>
<td>79</td>
<td>45</td>
<td>45</td>
<td>10</td>
</tr>
<tr>
<td>Bronchiectasis Wheeze/cough</td>
<td>—</td>
<td>46</td>
<td>39</td>
<td>15</td>
</tr>
</tbody>
</table>

*Six major criteria investigations.
rather than H pylori infection alone, might delay pubertal growth. We investigated the frequency of diminished growth in 30 H pylori positive children (21 girls and 9 boys) diagnosed by serology and histology. The mean age was 11.5 (2.0) years (range 8–15). We found 11 (36.7%) H pylori positive patients with height values below the 25th centile. Anaemia was determined in none of the patients. Mean haemoglobin concentration was 130 (6) g/l. H pylori infection is a chronic persistent infection, leading to diminished growth. Chronic gastric inflammation, dyspepsia, decreased nutritional intake, and malnutrition themselves could be due solely to H pylori infection itself, and is not related to iron deficiency anaemia.

HUYLA DEMIR
INCI NUR SALTIK
NURTEN KOCKAR
AYSEL YUCE
HANAN OZEN
FIGEN GURAKAN
Hacettepe Uzman Dogumaci Cocuk Hastanesi, Gastroenteroloji Utesi, 06100 Ankara, Turkey
email: ayuce@hacettepe.edu.tr

Growth monitoring

EDITOR,—Garner and colleagues recently presented a much needed review of growth monitoring.1 This is a component of primary health care on which so much finance and health workers’ time is being expended. No doubt this review will stimulate more necessary trials.

However, they did not touch on one important aspect of growth monitoring—that is, whether health workers using growth charts comprehend the weight for age curve.2 Piaget (1896–1980) considered the line graph to be one of the more difficult subjects to teach. Graphic representation of numbers on a graph comprehends the weight for age graph.3

We did not detect anaemia in H pylori positive patients with diminished growth. We suggest that the development of short stature in H pylori positive patients may be due solely to H pylori infection itself, and is not related to iron deficiency anaemia.


Detecting outbreaks of E coli O157 infection in nurseries

EDITOR,—In their report of a serious outbreak of E coli O157 in a nursery in North Wales, Al-Jader and colleagues recommended that more than one child with more than one bowel motion in a nursery should trigger action including “informing and seeking the advice of public health agencies”.4 Using data on healthy children in nurseries, in the paper we have calculated the additional work that would be generated for the Public Health Department in the district where the outbreak occurred if this policy was implemented.

Of 19 well children on the ground floor of the nursery, six had more than one bowel motion on at least one of the half day sessions attended during the surveillance period. Well children attending for six days during the period, given an approximate total number of sessions attended of 228 (19×6×2), the probability of a well child having more than one bowel motion during any half day session was therefore about 0.026 (6/228). There are 385 nursery days and playgroups in North Wales, with an average of 23 children per nursery.5 In an average nursery the probability that two or more well children would have more than one bowel motion in a session on any one day is 0.12, equivalent to a false alarm every eight days.

Therefore, if the suggested policy was implemented, and incidents were reported to the Public Health Department, this would result in approximately 46 inappropriate calls per day (0.12×385)—that is, 230 per week. Even if the normal background rate was ten times lower than that seen among well children during this outbreak, this would still result in just over three calls a week to the department reporting false alarms. The proposed “early warning system” is therefore almost unworkable, and the claim that it could have prevented 10–12 of the 31 cases in the outbreak needs to be reviewed.

R J ROBERTS
Consultant in Communicable Disease Control, Department of Public Health, North Wales Health Authority, Hendy Road, Mold, Flintshire CH7 1EZ, UK
dr.richard.roberts@nwales-ha.nwales.wales.nhs.uk

3 Megean M, Morley D, et al. Child weighing may overcome this difficulty. With this, the parent sees a large weight for age curve. With a ball pen, they then create the weighing scale, as they release the child’s weight into the weighing trousers below the scale. Spring stretching up their child’s chart, locating in the scale, they are able to understand the weight for age curve.4 In one study among the pastoral Maasai in Kenya, action was taken by the parents to give an additional drink of milk to children whose weight for age was subnormal (Meegan M. Personal communication, 1999).


Dr Salmon comments:

Children who attend out of home care are at increased risk for infectious diseases of which gastrointestinal tract infection is among the most common.1 Numbered among these are VTE Coli O157 infections which, as this outbreak showed, can cause severe disease. The challenge is to identify disease early.

In this outbreak, given that the first two cases attended the nursery for two days after the onset of their disease on 21 August and the first case from the nursery was not reported until 1 September by which time 13 further symptomatic cases had occurred, our claim that 10–12 cases could have been prevented by taking further action, at this point, is straightforward. The toileting record might have constituted a reason for such action. We list a range of possible responses, particularly when the bowel motion is loose or offensive (inquiring about symptoms at home, suggesting a visit to the family doctor, arranging a faecal sample, informing and seeking the advice of public health agencies). We were aware of the issue of specificity and did not suggest that all these activities should necessarily occur on every occasion that more than one child with more bowel motion was recorded. Most agree that faecal sampling needs, generally, to be encouraged.2 However, to combine the activities into a workable algorithm was beyond the scope of the report. Constructing an algorithm is worth attempting, however, since, as a starting point, a toileting record constitutes a straightforward record used in a number of care settings.

R L SALMON
Consultant Epidemiologist


Meningococcal disease due to W135: fresh public health concerns

EDITOR,—The paediatric intensive care unit at St Mary’s Hospital in London admits more than 100 cases of meningococcal disease each year from over 50 different hospitals in the south east of England. Since 1992, the unit has treated over 650 patients with the disease,1 but had not treated a single case of serogroup W135 meningococcal infection until April 2000. We would like to report four children treated at our unit for meningococcal infection due to serogroup W135, type 2A, subtype P1.2, P1.5, within a one month period from April 2000. They had been vaccinated recently with meningococcal serogroup C conjugated vaccine, and had all been
in contact with travellers returning from Mecca. The clinical features of these cases are outlined in table 1.

The cases represent four out of 38 cases (with five fatalities) of serogroup W135 Neisseria meningitidis infection in England and Wales within the six week period from March to May 2000 (PHLS Meningococcal Reference Unit, personal communication), with hundreds of cases of the identical subtype being reported throughout Europe. Saudi Arabia has reported over 225 cases, with almost 25% mortality to the end of April 2000. It is thought that this large outbreak of an unusual strain origin in Saudi Arabia, with the pilgrimage of a record 1.3 million people to the Haj between 15–18 March 2000.

A similar outbreak occurred in 1987, due to serogroup A, subgroup III. This also followed the yearly pilgrimage to Mecca, and spread throughout Europe, USA, and Africa over the next two years. Requirements for pilgrims entering Saudi Arabia now include documented vaccination with meningococcal A and C polysaccharide preparation. This public health measure has been effective in irradiating serogroup A disease in these travellers. A quadrivalent vaccine is available for serogroup W135 as well as serogroups A, C, and Y. This vaccine, however, is not licenced in the UK, and is only available on a named patient basis. This raises public health issues, including whether people returning from Mecca to the UK should be screened or given prophylaxis.

Even with the anticipated beneficial effects of the meningococcal C vaccination programme in England and Wales, it is important to remember that other serogroups of meningococci will continue to cause significant disease in the UK.

Until 1950, England was predominantly affected by epidemics of serogroup A meningococcal disease. The switch to serogroup B and C disease occurred after the second world war, and serogroup A disease is now rarely seen in the UK. Neissera meningitidis has the potential to alter its capsular polysaccharide antigen through recombinational exchanges at the capsular locus. In his commentary in the Lancet in 1999, Martin Maiden expressed concern that new hyper-virulent strains of serogroups including B, W135, and Y may emerge as serogroup C disease is eliminated. This recent outbreak of serogroup W135 infection does not seem to represent such selection pressure. However, it highlights the need for continued clinical, laboratory, and epidemiological vigilance for meningococcal infections, particularly now that there may be a theoretical risk of other serogroups becoming more prevalent as meningococcal serogroup C disease is controlled.

Table 1: Clinical presentation, severity and outcome

<table>
<thead>
<tr>
<th>Case sex</th>
<th>Contact with travellers</th>
<th>Presentation</th>
<th>Resuscitation fluid*</th>
<th>Mechanical ventilation (days)</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 10m/F</td>
<td>Grandmother</td>
<td>Petechiae, septicaemia</td>
<td>80 ml/kg fluid</td>
<td>2</td>
<td>Discharged</td>
</tr>
<tr>
<td>2 27n/M</td>
<td>Father</td>
<td>Purpura fulminans, septicaemia</td>
<td>350 ml/kg fluid</td>
<td>11</td>
<td>Peripheral gangrene</td>
</tr>
<tr>
<td>3 4m/F</td>
<td>6 family members</td>
<td>Meningitis, seizures, no rash</td>
<td>2.2 mcg/kg/min</td>
<td>0</td>
<td>Neurological sequelae</td>
</tr>
<tr>
<td>4 19n/M</td>
<td>2 Aunts</td>
<td>Purpura, septicaemia</td>
<td>90 ml/kg fluid</td>
<td>2</td>
<td>Discharged</td>
</tr>
</tbody>
</table>

*Total resuscitation fluid required in first 24 hours

Preparation and treatment of cow's milk allergy

EDITOR,—Divergences in existing guidelines on the preparation and treatment of cow's milk allergy (CMA) in infants seemed settled when a joint statement by the committees of ESPACI/ESPGHAN appeared in ADC. However, we take exception to some of the assumptions, which have been left open to challenge from both nutritional and allergological points of view. Our concern is that lactose free diets from birth may cause neurological problems in healthy children. Galactose is a functionally important component of myelin galactolipids, but it is unclear whether a lactose free diet plays a role in the clinical neurological abnormalities of children with galactosaemia. However, lactose is essential for patients with UDP-galactose-4-epimerase deficiency. Though rare, this disorder should be considered in the evaluation of the risk/benefit ratio and the costs of planning a prevention strategy for which the benefits are still unclear. In this context, issues of colonic ecology and malabsorption take second place. The use of screening tests for errors of lactose metabolism as interpreted in the statement may also be misleading. The claim that “feeding lactose-free diets from birth . . . will cause false negative results in most neonatal screening tests for galactosaemia” overlooks the fact that these tests do not establish blood galactose levels but the presence/deficiency of the enzymes responsible for galactosaemia. The assertion that “…formulas based on intact soy protein isolates are not recommended for the initial treatment of food allergy in infants, although a proportion of infants with cow's milk protein allergy tolerate soy formula” is based on the ESPGAN Committee on nutrition and on the AAP recommendations. While the former concerns itself with clinical gastrointestinal manifestations, the latter recommends state in conclusion (point 8): “Most infants with documented IgE-mediated allergy to cow milk protein will do well on isolated soy protein-based formula”. Initial treatment for allergic disease is avoidance of the incriminated allergen. Soy formula has been recommended in treatment of CMA on grounds of efficacy, adequate nutrient intake, and cost. In the absence of prospective studies comparing the allergenicity of cow's milk hydrolysates against soy formulas in children with CMA, the rationale to alter this indication appears to be lacking.

Pyridoxine dependent and pyridoxine responsive seizures

EDITOR,—Seizures in infancy and early childhood responsive to pyridoxine are well recognised but rare. Baxter has recently observed that almost a third of neonatal cases of pyridoxine dependency present with apparent birth asphyxia and/or suspected hypoxic-ischaemic encephalopathy, and recommended that, because of the high proportion of atypical cases, all children with early onset (younger than 3 years old) intractable seizures or status should receive a trial of pyridoxine whatever the suspected cause. Following this recommendation can be of remarkable benefit. We report a case of a caucasian boy, born at term who presented at delivery in a state of unexpected collapse requiring intubation and resuscitation. He developed tonic seizures within hours of birth and was treated with phenobarbitone, phenytoin, and clonazepam. At 48 hours, an EEG showed a burst

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E RIVA A FIOCCHI L FIORI M GIOVANNINI Department of Paediatrics, University of Milan Medical School, San Paolo Biomedical Institute, Via Di Ridini 8, 20142 Milan, Italy
suppression pattern. There was biochemical evidence of multi-organ damage. He was extubated on day 5 and discharged on day 16 on phenobarbitone. He continued to have frequent myoclonic seizures. At 6 months, phenobarbitone was replaced by sodium valproate with some initial benefit. By 7 months, he was having focal motor seizures affecting his right arm up to 40 times a day and additional atypical absences and tonic seizures. He also showed signs of an emerging spastic quadraparesis. EEG showed right sided spike and wave discharge with a frontal emphasis. At 8 months a trial of oral pyridoxine (30 mg/kg/day) was given. No seizures have been seen since this dose was increased to 50 mg/kg/day. He is now 16 months old. He is maintained on pyridoxine 15 mg/kg/day; valproate has been discontinued. The EEG no longer shows spike and wave activity. The signs of spastic quadraparesis remain.

We have reviewed the notes of children attending The David Lewis Centre, a residential school for children with severe epilepsy. Children at The David Lewis Centre are referred from all over the UK and their early epilepsy management has been undertaken at many different centres. All children with intractable cryptogenic epilepsies, which started before they were 3 years old, were identified (dates of birth–1979–1992). Only one of these children was recorded as having received a trial of pyridoxine early in the evolution of their epilepsies. The true prevalence of pyridoxine responsive epilepsy is difficult to assess if the recommendations of Baxter are seldom applied. Giving pyridoxine can be diagnostic and therapeutic—not giving a trial of pyridoxine is common and can lead to a treatable cause of difficult epilepsy unrecognised and inadequately treated.

Are sleep studies worth doing?

Editor,—If sleep studies are worth doing, they are worth doing well. The study of sleep in children is an expanding discipline, and with a prevalence of sleep related issues in children of 31% of children aged 5, it is more important than ever to have a robust approach to the study of sleep in children.1 We are concerned that the UK is not making the most of the research that is available.

Sleep studies are used to diagnose a variety of sleep disorders, including obstructive sleep apnoea (OSA), which can lead to significant morbidity and mortality in children.2 The most common cause of OSA is obesity, which is becoming more prevalent in children and adolescents in the UK.3 Sleep studies can be used to determine if a child has OSA and if so, how severe it is. There are different types of sleep studies, such as polysomnography (PSG), which involves monitoring a range of physiological parameters, and simpler tests such as oximetry, which measures oxygen saturation.4 PSG is considered the gold standard for diagnosing OSA, as it can provide detailed information about the sleep architecture and respiratory events. However, PSG is not always feasible, especially in children, as it may require sedation or general anaesthesia.

In this study, we evaluated the effectiveness of different sleep studies in diagnosing OSA in children. We compared the results of PSG with simpler tests such as oximetry and questionnaires. We found that oximetry and questionnaires were less accurate than PSG in identifying children with OSA, as they may miss mild cases of OSA.

We believe that sleep studies should be used more effectively in the UK. Sleep studies are important for the diagnosis and management of children with sleep related disorders. They can help identify children who need specialist intervention and guide the treatment plan. It is important that sleep studies are performed accurately and interpreted correctly to ensure the best possible care for children with sleep related disorders.

S CUNNINGHAM
M HARRIS
Department of Paediatric Respiratory and Sleep Medicine, Mater Children’s Hospital, Brisbane, Australia
email: steve.cunningham@talk21.com


7 S CUNNINGHAM
M HARRIS
Department of Paediatric Respiratory and Sleep Medicine, Mater Children’s Hospital, Brisbane, Australia
email: steve.cunningham@talk21.com

Information on the use of inhaler systems in childhood asthma.

Spacers and holding chambers: Not the last word, we hope

Editor,—We have reviewed the notes of children attending The David Lewis Centre, a residential school for children with severe epilepsy. Children at The David Lewis Centre are referred from all over the UK and their early epilepsy management has been undertaken at many different centres. All children with intractable cryptogenic epilepsies, which started before they were 3 years old, were identified (dates of birth–1979–1992). Only one of these children was recorded as having received a trial of pyridoxine early in the evolution of their epilepsies. The true prevalence of pyridoxine responsive epilepsy is difficult to assess if the recommendations of Baxter are seldom applied. Giving pyridoxine can be diagnostic and therapeutic—not giving a trial of pyridoxine is common and can lead to a treatable cause of difficult epilepsy unrecognised and inadequately treated.

D HINDLEY
Consultant Paediatrician,
Fairfield General Hospital,
Reedsall Old Road,
Bury BL9 7TD, UK

M HUYTON
Associate Specialist,
The David Lewis Centre,
Alderley Edge, Cheshire, UK


Letters, Book reviews, Corrections
cycles greater than about five microns aerodynamic diameter) from the pMDI. A spacer (homemade or otherwise) will not perform this function effectively. Rather, it will momentarily contain the aerosol and then deliver particles of all sizes to the well coordinated patient who is able to time inhalation with actuation of the pMDI. In the case of corticosteroids, the emitted coarser particles can promote local topical infections—such as, oral candidiasis, as well as increases in overall systemic absorption.

The inhalation valve, which distinguishes a VHC from a spacer, needs to be a carefully designed component whose function is to retain the aerosol once created following actuation of the pMDI, then release it during the inspiratory cycle. Many children, particularly those with an acute exacerbation of asthmatic symptoms, have poor coordination, and are therefore likely to mistime inhalation with pMDI actuation. These patients, who are at greatest risk, are thus likely to derive least benefit from the use of homemade spacers.

Although we have other observations of a technical nature, the information given here should be sufficient to provide the message that this study should not be taken as the final word but rather as a finding concerning the debate about the efficacy of hom made vs. manufactured add on delivery devices for use in pMDI based treatment.

That said, if a VHC is unavailable for whatever reason, an empty drinking bottle may be better than nothing at all.

J P MITCHELL
Scientific Director,
Medical Aerosol Research Laboratory,
Trudell Medical International,
725 Third St, London,
Ontario NSV 5G4,Canada


BOOK REVIEWS


Given the wide prevalence of feeding problems in children and their potential impact on health, it is important for all health professionals working with children to gain an understanding of feeding difficulties. In several chapters of this book there is a refreshing focus on the role of organic factors in feeding problems, which may highlight the wide range of subtle organic features that can contribute to and exacerbate feeding difficulties in children. The impact of other factors on feeding is also covered—for example, the effect of temperament, appetite, growth, developmental stage, prior experience with foods, and cognitive development, all of which are critical in understanding each child’s feeding difficulty and creating appropriate intervention strategies. The various theories of feeding difficulties from physiological (oral motor, regulatory, neurological), psychological (behavioural, cognitive, and psychoanalytical) and cultural perspectives are covered. These are discussed with reference to multidisciplinary teamwork and the development of both hospital and community feeding services. The chapter covering the psychoanalytical perspective sits somewhat oddly within the context of the book. Less helpful advice and practical intervention techniques stem from this chapter than the others, but perhaps those with an interest in psychoanalysis will find it an appealing diversion.

It is vital that health professionals in this field develop an understanding of the impact of cultural factors, from the effect of cultural feeding practices on feeding difficulties, to the perception and importance of food and feeding within cultures. This is critical in understanding the factors that contribute to the development and maintenance of feeding problems in children, and is also essential to facilitate culturally sensitive intervention strategies. The perspectives of Indian culture are discussed and whilst one text alone cannot cover the breadth of multicultural issues that are relevant to the UK population, there is useful information on issues which are specifically related to cultural practices and those which are related to social disadvantage and poverty in general.

Whilst some chapters focus on clinical practice and opinion that may not appeal to an academic audience, practical advice, such as special issues in tube feeding, neurological impairment, and chronic illness, combined with generally sound theoretical discussion, makes this book a useful resource for health professionals involved in the assessment or treatment of feeding difficulties.

JACKIE BLISSETT
School of Psychology, University of Birmingham


Share prices of dot.com companies have plummeted because, we are told, there are too many players in the market place for them all to be viable. The dot.com bubble has burst. This may also be true of paediatric textbooks. Such thoughts might trouble the authors and publisher of the fourth edition of the ABC of One to Seven, were it not for the pictures it contains. Is there really demand for another general paperback text covering well trodden ground, with predictable text and liberal use of blue boxes to convey the impression that there is a lot more colour than is really the case? Perhaps not, but for those pictures. This book isn’t cheap, maybe that’s because of the pictures. In short, this book is worth the investment for the pictures alone.

Medical students like to console themselves with thick books because many of us still hold fast to the well known belief that you can learn a lot about a subject by buying a “good book”, even without opening it. Perhaps the same is true of GPs; fat books with hardcovers are much more impressive shelf-fillers than paperbacks with pictures.

But what about when the time comes to learn paediatrics? We need something on which to hang the facts of any textbook, and we all know the daunting effect of long paragraphs of plain text on page after page. This is where pictures and diagrams come into their own, and the ABC of One to Seven has them in spades. They are almost always helpful and relevant—if not adding to the explanation, then proving the useful peg on which to hang a particular fact. Captions though, are few and far between. The reader can sometimes be left confused as to the purpose of a particular illustration. Several of the pictures appear two or three times and others are decidedly outdated. Ambulances and toys seem to be used as space fillers, but others, particularly the dermatological pictures, are excellent.

This is no reference bible, and the text is simple and narrative. Facts are not flung at the reader, and the practical is emphasised over the theoretical. This is a book to demystify infancy and early childhood—the fear of the unknown can quickly be replaced with enthusiasm for such a fun subject area. The Colour Atlas of Kids: this bubble definitely remains intact.

NICK JENKINS

CORRECTION

In a recent letter by Russell and Gillett (Arch Dis Child 2000;85:436), the sentence: “The in house assays used for AGA and EmA were performed on 10–20 ml of serum or plasma; thus capillary samples were more than adequate.” should have read: “The in house assays used for AGA and EmA were performed on 10–20 microlitres of serum or plasma; thus capillary samples were more than adequate.” We apologise for this error.