LETTERS TO THE EDITOR

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Palivizumab and RSV prevention

EDITOR,—The letters from Drs Deshpande and Nicholl, in relation to the Impact-RSV study and the UK guidance for the use of palivizumab in the prevention of serious RSV infections, raise interesting questions that need an answer.

I believe Dr Deshpande “has got it wrong” in that he fails to realise that the primary objective of the IMpact study was to investigate whether palivizumab reduced RSV hospitalisations in high risk infants. It was never intended that this study would address the severity of RSV infections, the need for paediatric intensive care, the need for mechanical ventilation, or a reduction in death rate. It is unreasonable to suggest that because the study didn’t show these then it is not valid. To show such benefits would require a totally different protocol, the numbers of patients being such that the study could never have been undertaken.

To reiterate the findings of the IMpact study, there was a 55% reduction in hospital admission rate for RSV proven disease—a significant result, however one wishes to rationalise it. Those high risk patients admitted with RSV infection spent fewer days in hospital, had less need for oxygen treatment, and had lower respiratory infection clinical scores if they received palivizumab.

The study was designed in association with and with the approval of the licensing authorities to grant a marketing licence for palivizumab. It was not designed to provide economic data on the cost effectiveness of the product. Both Deshpande and Nicholl fail to realise that if they wish this information then different studies are needed.

Does anyone know the lifelong cost of RSV disease in infancy? What is the relationship between RSV hospitalisation in the first year of life, recurrent wheezing in childhood, or indeed the possible development of chronic obstructive pulmonary disease in later adult life? To develop a relevant, long term, cost effectiveness plan, all these points need to be taken into consideration. In an attempt to help with this there are two ongoing studies that Deshpande, Nicholl, and others, may find helpful. One is taking place in four centres in the UK and the other is a follow up study from the IMpact trial. Both are attempting to identify the health service costs over a three year period following hospitalisation for RSV disease, and it is hoped the results will be available later on this year.

The UK guidance on the use of palivizumab does not advocate universal usage of the product, but makes recommendations on how infants may benefit. It is the role of clinicians in local hospitals to discuss with their managers, the local health authority, and the individual primary care group or trust, which specific patients they feel should receive palivizumab. These decisions may well differ between centres depending on budgets, the morbidity of their patients and interpretations of evidence both research and clinical.

RSV bronchiolitis remains the greatest annual epidemic disease to hit paediatric departments in Europe, the USA, and Australasia.1 The treatment of the symptoms is unsatisfactory in that the only proven benefit is oxygen. Each year, vast amounts of money are wasted on bronchodilators, steroids, ipratropium bromide, and antibiotics. Palivizumab, the first monoclonal antibody to be developed specifically for use in paediatrics, has been shown to be effective in reducing hospital admission in high risk infants. To dismiss it out of hand seems churlish. To rationalise its use in whom it may most benefit seems clinically sensible. All new treatments must to be considered with caution. However, I believe that if clinicians take a back seat view whilst awaiting definitive confirmation of absolute cost effectiveness, we will continue to deny our most vulnerable patients the benefits of scientific advance.

WARREN LENNEY
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EDITOR,—I am writing in reply to the recent correspondence regarding the use of palivizumab (Synagis),1,2 a monoclonal antibody licensed for the prophylaxis of respiratory syncytial virus (RSV) infection in premature infants. RSV is a disease that affects 50% to 70% of all infants within the first year of life, and causes significant morbidity and mortality, particularly in a number of well defined high risk groups.

The major trial demonstrating the safety and efficacy of palivizumab prophylaxis in premature infants was the IMpact-RSV trial,3 a randomised, double blind, placebo controlled, multicentre trial that enrolled 1502 children with prematurity (≤35 weeks gestation) or bronchopulmonary dysplasia (BPD). One hundred and twenty three of the children enrolled were from 11 UK centres. The primary end point of the IMpact-RSV study was hospitalisation due to confirmed RSV disease. The study was not powered to demonstrate a reduction in mortality, neither was it designed as a pharmaco-economic study. The average gestation of all the infants was 29 weeks and the placebo (n=500) and palivizumab (n=1002) groups were well matched for both demographic parameters and RSV risk factors. The study demonstrated a relative reduction in RSV related hospitalisation of 55% (10.6% placebo v 4.8% palivizumab p=0.0004). A significant reduction in RSV hospitalisation was seen irrespective of gestational age, diagnosis of BPD or gender. Of all the children in both groups admitted with RSV infection, 27.7% were admitted to intensive treatment units (this figure was similar in both groups). There was however a significant reduction in the overall incidence of RSV related intensive treatment unit admission in the palivizumab group (3% placebo v 1.3% palivizumab p=0.026).

The placebo RSV hospitalisation rate of 10.6% reported in the IMpact-RSV trial was lower than that seen in previous controlled trials which have reported rates of 13.5%,4, 20%,4, 22.4%,5 and 37%. Further reported rates of hospitalisation vary depending on the risk group studied, and data from the US demonstrate that it is possible to predict subgroups who have considerably higher hospitalisation rates.6 Further data from both Europe7 and the US8 reported RSV readmission rates in large numbers of premature children receiving palivizumab prophylaxis over the 1998/9 RSV season (neither study had a placebo arm). Of the 565 European infants enrolled, 1.2% had confirmed RSV hospitalisation, whilst two US groups of 1839 and 7013 children had RSV hospitalisation rates of 2.3% and 1.5% respectively. Despite the lack of comparator arms these data do suggest that the IMpact-RSV trial may have underestimated the true efficacy of palivizumab.

The generation of pharmaco-economic arguments directly from the IMpact-RSV data very much oversimplifies what is an extremely complex issue. Hospitalisation rates vary considerably between risk groups, and measuring the true economic cost of RSV hospitalisation requires long term follow up, both of hospital, community, and parental costs.

Despite its relatively high costs, modern neonatal care has led to dramatic improvements in the outlook of premature infants. Advances such as surfactant therapy and mechanical ventilation seem expensive on the face of it, but both controlled trials9 and clinical experience have shown the investment to be worthwhile.

Dr Deshpande refers to the guidance document reflecting the outcome of a consensus committee of a number of UK clinicians,10 and issued by ourselves. Many were aware of the guidelines published by the American Academy of Pediatrics regarding RSV prophylaxis and the use of palivizumab,11 and felt that whilst they were very useful, UK guidelines should be formulated at a local level, taking into account local risk groups and epidemiology. For these reasons, the UK guidance document deliberately avoids being too prescriptive and whilst describing the two major risk groups (premature infants, ≤35 weeks gestation, and those with BPD), it emphasises that treatment priorities are likely to vary locally and that decisions regarding which preterm infants to treat will be individualised.

Abbott Laboratories are continuing to work with many in the paediatric community in order to help better define many of the issues. We strongly feel that palivizumab is an important breakthrough in the battle against RSV infection, a disease that continues to
cause high levels of morbidity and significant mortality in high risk infants.

CHRISTINA CARNEGIE
Medical Director, Abbott Laboratories Ltd, UK


The editor comments:

In her letter, Dr Carnegie refers to a guidance document reflecting the outcome of a consensus committee of a number of UK clinicians and issued by Abbott Laboratories Ltd.

Earlier this year, we received as a submission for publication such a document, headed by the names of a number of distinguished paediatricians and neonatologists. I was puzzled because it was addressed from a public relations company. I contacted all those named to the correspondence in the corresponding letter. I was told that they did not know the paper was to be submitted to a peer reviewed journal.

Consequently, I invited the PR company to withdraw the submission, which they did. The paper, itself, was marked as having been produced with the aid of an educational grant from Abbott Laboratories.

In general, statements of ‘Dietary intake of reviewers and patients’ in childhood is reluctant to publish the results of consensus groups, unless the methods by which they are arrived at their conclusions are totally transparent. This case illustrates one reason why we believe it is right to be cautious.

HARVEY MARCOVITCH
Editor in Chief

Dietary products used in infants for treatment and prevention of food allergy

EDITORS.—The joint statement of the European Society for Paediatric Allergology and Clinical Immunology (ESPACI) and the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPghan)1 deserves some comment.

Firstly, on the use of soy based formulas for the treatment, as well as for the prevention of food allergy: I was disappointed that no word about this subject appeared in the conclusions of the statement. Many have claimed that the use of soy bean formulas in infancy is an efficient way of preventing food allergic disorders, but more recent prospective and randomised clinical studies have shown that soy protein is as allergenic as cow’s milk protein. As the matter remains controversial,1 I believe that the conclusions should have been that soy based formulas are not recommended for the treatment or prevention of food allergy until more data are available.

The second issue concerns the use of partially hydrolysed formulas for preventing food allergy. A recent five year follow up prospective, randomised, and controlled study by Chandra,4 which showed a beneficial preventive effect of a milk hydrolysed formula in high risk infants, was ignored. The only study where the preventive effect of an extensively hydrolysed formula was compared with the effect of a partially hydrolysed one, showed that the former was superior to the second.4 This paper, however, has a possible methodological shortcoming: the manufacturer (Mead Johnson, Evansville, Indiana, USA) provided both a commercially available extensively hydrolysed formula (Nutramigen) and a non-commercially available at least in Sweden where the study was undertaken) partially hydrolysed formula, prepared by mild (how mild?) enzymatic hydrolysis. In future, such studies should only use commercially available formulas of either the same or different brands. I consider that current data are insufficient to allow a firm view.

Therefore, I believe the conclusions should have stated that no clear recommendation can be made for the use of a partially hydrolysed formula to prevent food allergy.

Conclusions of consensus committee statements are generally considered as guidelines for the practitioner. Omissions, as in the case of soy based formulas, or ambiguities, as in the case of partially hydrolysed formulas, do not clarify the issues so should be avoided. I believe that modified conclusions, as referred to above, would have been more in agreement with the literature and more helpful to the reader.

1 Chandra RK. Five year follow-up of high risk infants with family history of allergy who were exclusively breast-fed or fed partial whey hydrolysate, soy, and conventional cow’s milk formula. J Pediatr Gastroenterol Nutr 1997;24:380–8.

Health care needs for travellers

EDITORS.—The article recently published by van Cleemput has made a valuable contribution to the health care needs of travellers and has drawn attention to a very deprived section of our community.1 However, the assertion that childhood asthma is more common in travellers is not based on sound evidence. This suggestion was based on a study by Anderson, who reported on the health concerns and needs of traveller families.2 The selection criterion for Anderson’s study was families with children of less than 5 years of age. The traveller families had a mean of six children aged 1 to 15 years. The control

1 Chandra RK. Five-year follow-up of high risk infants with family history of allergy who were exclusively breast-fed or fed partial whey hydrolysate, soy, and conventional cow’s milk formula. J Pediatr Gastroenterol Nutr 1997;24:380–8.

The editor comments:

We thank Professor Salazar-de-Sousa for his insightful comments on the joint comment of ESPACI and ESPghan.

We kept our conclusions brief and did not repeat all the considerations discussed earlier in the text but, rather, focused on the practically most relevant advisable measures to treat and prevent food allergy. In the text of the comment it is stated that, based on information currently available, we do not recommend the use of soy protein based formulas as a first line choice to prevent food allergy in infants. However, we hope that different views exist on this issue and that further studies may be useful to extend the rather limited database available, in order to clarify the allergenicity of soy formulas in infants with allergy risks.

The data presented in one of the studies by Chandra referred to by Professor Salazar-de-Sousa were not ignored. However, the comment felt that neither this paper nor many similar studies allowed definitive conclusions on all the issues. Since our comment was not intended to be an extensive review of all available publications, we did not cite this particular paper or the many other original papers on this topic, but referred to a recent editorial considering these and other data.2

We agree with Professor Salazar-de-Sousa that currently available data are insufficient to allow a firm conclusion on the relative effects of partially versus extensively hydrolysed formulas for the prevention of food allergy, an important issue for clinical practice. Hence, we concluded that more studies are needed.

BERNHARD KOLETZKO
Professor of Paediatrics, University of Muenchen, Germany Secretary, ESPghan Committee on Hypoallergenic Formulas

ARNE HOST
Professor of Paediatrics, University of Odense, Denmark Chair, ESPACI Committee on Hypoallergenic Formulas
affluent families had a mean of 1.7 children aged 1 to 3 years, and the control inner city families had a mean of 1.9 children aged 1 to 4 years. Anderson reported that asthma was a concern to 30% of travellers compared with 11% of inner city families and 4.5% of affluent families, using a questionnaire that seemed to tackle parental concerns only, and was not validated for asthma incidence. Yet, van Cleeput extrapolated a high incidence of asthma in travellers’ children from this study, and did not comment on questionnaire validation or the confounding factors of age and transient early wheezing.

We used the ISAAC (International Study of Asthma and Allergies in Childhood) questionnaire to compare the prevalence of asthma in schoolboys, aged 6 to 12 years, from travellers’ families with settled controls. The parent reported prevalence of wheezing and related symptoms were all more common in schoolboys from the control group than in traveller schoolboys. The values were significant for wheeze in the last year (31.3% vs 14.8%, OR 5.6, p=0.025), and for doctor diagnosed asthma (29.6% vs 11.1%, OR 2.1, p=0.04). We concluded that the experience of B6 deficiency is not common in man.

B6 deficiency. We have a suboptimal intake with or without dietary supplements, or early in life. In infants, the nutritional intake and the removal of vitamin B6 from the diet. (direct chemical index) within one week of birth. We conclude that this treatment is effective.

We used LHRH for the evaluation of height measurements and also to achieve and to compare paediatric endocrinologists. Although a number of possibilities have been raised for failure in attainment of desired height in the patient, the early medical attention would have been expected to lead to the possible prevention of short stature.

Fits, pyridoxine, and hyperprolinaemia type II

Editor.—We report an 11 years 8 months old girl with juvenile hypothyroidism and precocious puberty accompanied by precocious puberty.

Editor.—It would be interesting to know if and how the authors had measured the transaminases. Their results could indicate if this is a cost and clinically effective screening test.

S VIVEKANANDAN
Clinical Biochemist,
Chemical Pathology, Guy’s and St Thomas’s NHS Hospital Trust, London, UK


LHRH analogue and growth hormone did not improve the final height of a patient with juvenile hypothyroidism accompanied by precocious puberty

Editor.—We report an 11 years 8 months old girl with juvenile hypothyroidism and precocious puberty who failed to respond to thyroxine, growth hormone, and luteinising hormone releasing hormone (LHRH) analogue. The patient was considered to be hypothyroid for about two years before the therapy was started. She had a very low serum thyroxine concentration, a height SD score of −3 SD, and a bone age of 10 years 3 months. Her puberty development was graded as Tanner stage IV of breasts and Tanner stage II of pubic hair. Her menarche occurred at the age of 10 years 3 months. The enlarged pituitary gland reduced in size with the thyroxine treatment (100 µg/day).

In addition to thyroxine, she was treated for 31 months with an LHRH analogue (30 µg/kg, once a month) and growth hormone (0.5 µg/kg divided into six doses) to avoid the progression of puberty and improve the final height. She reached the final height at the age of 15 years 1 month (−2.8 SD), which was the same as before the treatment (fig 1).

Minamitani et al reported that treatment with LHRH analogue and growth hormone in addition to thyroxine was successful in improving the growth and avoiding pubertal growth of patients with juvenile hypothyroidism in the prepubertal stage.

Difference between the report of Minamitani et al and our case is that our patient already had the advanced bone age relative to height age and the progression of puberty at the start of treatment, to which our failure to improve the final height with the combination therapy might have been ascribed. To improve the final height, we should have increased the dose of LHRH analogue and growth hormone. During the combination therapy, peak serum insulin like growth factor 1 was 710 ng/ml (normal: 370–896 ng/ml), and peak concentrations of LH and FSH were completely suppressed in response to gonadotropin-releasing hormone. Although her menstruation was successfully suppressed, bone maturation was not inhibited.

We concluded that patients with juvenile hypothyroidism who are often found to be in progressive pubertal development may have failure to respond to LHRH analogue and growth hormone. An early diagnosis may therefore be of utmost importance in improving the final height. In Japan, schoolchildren are biannually measured for height and weight. It is therefore strongly urged to educate school nurses to direct their attention to the evaluation of height measurements and also to achieve and to compare paediatric endocrinologists. Although a number of possibilities have been raised for failure in attainment of desired height in the patient, the early medical attention would have been expected to lead to the possible prevention of short stature.

Intraosseous access in infant resuscitation

Editor.—We believe that intraosseous access to the circulation in infant resuscitation is underevaluated and therefore under utilised.

Intraosseous cannulation is an simple and effective technique that can be performed both quickly and safely in resuscitation.1,4 There have been relatively few complications reported with this technique.1

In a laboratory study, we compared the average flow rates through a range of intraosseous cannulae with that of an 18 gauge intravenous cannula. We purged intravenous Hartmann’s solution through the various devices, at a constant pressure of 300 mm Hg, recording the average volumes over one minute intervals. The results and calculated infusion time for a 20 ml/kg bolus in a 5 kg baby are shown in table 1.

Administration of intraosseous fluid is an essential component of infant resuscitation. Fluid boluses have to be infused under pressure through an intravenous cannula placed in a peripheral vein. Successful cannulation can be a technical challenge in collapsed infants. Small veins are prone to damage when fluids are rapidly purged through them. Central venous access is not usually established in infants in the immediate resuscitation period and larger intraosseous cannulae are impractical. An alternative easily available route is through an Astra intraosseous cannula (Astra Technika, Sweden) placed in the proximal tibia. Flushing the cannula after insertion is necessary to clear the lumen and to enable the cannula to function properly. The size of the cannula (16 gauge) is suitable for an average 5 kg baby. There is a limit to the amount of fluid that can be infused through the cannula and severe hypovolaemia can be expected to occur. Therefore, the cannula should be used only as a temporary measure to facilitate the establishment of central venous access if necessary.
cannulae (22 and 20 gauge) can be difficult to site in small infants presenting with circulatory failure.

Our simple experiment has shown that fluids can be infused through an intraosseous cannula at a significantly higher rate to that of the intravenous devices. The resistance to flow in situ has not been calculated, but one could reasonably expect the capacitance of the marrow cavity to be greater than that of an infant’s peripheral vein. These factors, in addition to the ease and success of placement of intraosseous over intravenous cannulae, lead us to advocate that greater emphasis is placed on the value of intraosseous cannulation during the early phase of resuscitation in infants.

This is an important issue that should be addressed both locally and nationally, as well as through advanced life support provider courses (APLS/PALS).

ROSS FISHER
Specialist Registrar, Paediatric Surgery

DYLAN PROSSER
Consultant Paediatric Anaesthetist, Royal Bristol Hospital for Sick Children, St Michael’s Hill, Bristol BS2 8BJ, UK

Table 1 Results and calculated infusion rate for a bolus in a 5 kg baby

<table>
<thead>
<tr>
<th>Access device</th>
<th>Gauge</th>
<th>Flow rate (ml/min)</th>
<th>Infusion time for 100 ml bolus (minutes)</th>
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<tr>
<td>Yellow venflon*</td>
<td>24</td>
<td>35.6</td>
<td>2.81</td>
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<tr>
<td>Blue venflon*</td>
<td>22</td>
<td>60.6</td>
<td>1.65</td>
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<td>Pink venflon*</td>
<td>20</td>
<td>126.8</td>
<td>0.79</td>
</tr>
<tr>
<td>Green venflon*</td>
<td>18</td>
<td>161.2</td>
<td>0.62</td>
</tr>
<tr>
<td>Intraosseous needle</td>
<td>18</td>
<td>248</td>
<td>0.40</td>
</tr>
</tbody>
</table>

* BOC Ohmeda AB, SE-25106 Helsingborg, Sweden.

Gastrointestinal symptoms in asthmatic patients

EDITOR,—Caffarelli et al comment on several immunological mechanisms by which gastrointestinal symptoms could occur in asthma.1 They do not comment on whether they excluded cystic fibrosis (CF). This is relevant as there are an increasing number of mild phenotypes of CF presenting as asthma. CF could be a unifying diagnosis in the “asthmatic” with gastrointestinal symptoms.

The important clinical message is to consider a diagnosis of CF in difficult cases of asthma.

JOHN FURNES
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DrS Caffarelli and Atherton comment:

We appreciate the comments made by Dr Furness, and we would certainly concur with his view that one must consider a diagnosis of CF in any child presenting with the combination of asthma and gastrointestinal symptoms.

We accept that a diagnosis of CF may not always be obvious on clinical criteria alone, but it remains the case that there is no simple cheap screening test for CF, and we must therefore continue to test only those children in whom there is at least some clinical suspicion for suspecting this diagnosis. We believe that we did adequately consider CF in the children that participated in our study according to clinical criteria, but sweat testing was not undertaken routinely, nor did we

screen for CF mutations. While it is possible that we may have missed a child in whom the combination of asthma and respiratory symptoms was due to CF, we consider it exceedingly improbable that such omission would have substantially prejudiced our results.

The finding that gastrointestinal symptoms, for most of which there was no simple explanation, are common both in children with atopic eczema and in children with asthma, suggests that these symptoms are a reflection of the patients’ atopic status itself, and undiagnosed CF is unlikely to be a significant contributory factor. Neither do we believe that these symptoms can merely be dismissed as being due to food allergy, any more than one could dismiss either atopic eczema or asthma themselves as being caused exclusively by food allergies. The precise aetiologies of these conditions remain to be clarified.

CARLO CAFFARELLI
DAVID J ATHERTON

BOOKS


The youth of today are not what they were: they are bigger. Rona and Chinn, in their long and meticulous study of the health and growth of some 87 000 children, have documented the continuing trend to increasing height for age in primary school children over a 20 year period. This is generally thought to be a good thing and indicative of improving health and nutrition. The trend has been rumoured to be at an end many times, but in fact continues. Similarly, poverty was thought to be at an end in the 1970s when this study had its beginnings, only to be reluctantly rediscovered after the Black report. The two clearly go hand in hand: when there is no more poverty and perfect health and nutrition have been achieved, there will be no further gain in height. The effect of poverty is illustrated in this study, as in many others, by the social class gradient in height. Yet the exact mechanism of the relationship is mysterious as most of the gradient disappears after adjustment for parental height. The authors argue that most of the variation must therefore be genetic, others argue that there has been overadjustment.

The other secular trend observed has been of increasing obesity: a worrying trend in light of the much larger epidemic in adult obesity. But then again all is not what it seems. Mean weight for height is referred to throughout as “obesity”. Yet, as this is the age when children pass through the thinnest phase of their growth, few if any will be actually obese and presumably a proportion were actually underweight. When does less undernutrition become too much overnutrition, and how do we tell? So a paradox: the secular trend to increasing height is good and is due to improved overall nutrition. The parallel trend
to increasing weight for height is bad and is due to improved overall nutrition. No dataset can provide all the answers. By collating their long work and summarising all their analyses in this well structured and admirably slim volume, the authors make it possible for the idle and speculative like myself to argue with their conclusions. The range of the work is vast: from heart disease risk factors and asthma prevalence, to the prevalence of enuresis and food intolerance. It may come as no surprise that the last has a strong inverse relation with level of education, but the adverse impact of food exclusion on height certainly surprised me. No doubt future generations will dip into this rich data set and pick out many more plums to inform both research and practice. We can be grateful to Rona and Chinn for making it possible. CHARLOTTE WRIGHT Honorary Consultant in Community Child Health


Good, I thought, as these books dropped through the letterbox. The day before I’d been party to a family receiving an antenatal diagnosis of gastrochisis, and the father had commented on “looking it up on the Internet”. I wanted to learn more about the condition myself, and reckoned I’d follow the man’s example.

Using the Internet in Healthcare sounded an ideal title; disappointingly it wasn’t. It’s a book about the basics of the Internet, which isn’t bad, but is presented better in other books (for example, Internet for dummies). It’s “medical” legitimacy comes from a good summary of NHSnet and a crumb of information about healthcare searches on the Web. (Embarrassingly, it was MedLine: a guide to effective searching that contained the nicest www resources.)

MedLine: a guide to effective searching was also a let down. It’s beautifully written, starts with a lovely summary of the history of MedLine, but annouys with drawn out explanations of Boolean logic and historical access systems. In explaining PubMed, it doesn’t even mention the excellent “Clinical queries” search page (www.ncbi.nlm.nih.gov:PubMed/clinical.Btw. I was aware on the work of Brian Haynes and colleagues.

For clinicians, there are better summaries of framing questions and effective database searching in Sackett’s book.1 For researchers, there are better databases for citation searching than MedLine.


One of the main premises of the authors is that parents are the best people to help their child, and they set about informing them how to do it in a better position to do so. There are seven main chapters, which cover subjects such as “What are eating disorders?”, “Causes”, “What to do”, “General principles of treatment”, and “Collaboration with professionals”. There are case vignettes, outlines of overall care, therapeutic treatments, and a very sensible dietary plan. A glossary of the terms commonly used in the treatment of eating disorders is provided.

It is difficult to judge at what level to set a book like this. There is a danger of being either too complicated or simplistic and patronising. This book is probably set correctly, but the text is a little heavy, and with only seven figures, mostly graphs, can appear a little dense. At 222 pages, it is not too long and can be read in sections, which is helpful for parents going through a particular stage of treatment or assessment. Naturally enough, the book concentrates upon the treatment plans the authors have used with good effect at Great Ormond Street Hospital.

Overall, I can recommend the sympathetic and caring approach taken by the authors, which will reassure parents.

ANDREW JAMES
Warnford Hospital

Immediate care of the critically ill child.

Few would disagree that in the past two decades, world leaders in the relatively young specialty of paediatric intensive care have emerged in Australia, Canada, and the United Kingdom. It is a welcome pleasure, therefore, that the exceptional talents of many of the individuals working in these centres have been brought together to create a much needed practical text encompassing the principles and practice of caring for critically ill and injured children.

The major strength of this book is that it takes into account one of the most important aspects of paediatric critical care, namely that the initial management of these children takes place in a wide diversity of settings. For many children ultimately admitted to a paediatric intensive care unit (PICU), the first few hours of care may have the most significant impact on their clinical course and outcome. This book targets the practitioners most likely to be involved in these situations, and provides key information and a problem based approach that is difficult to achieve in standard texts.

Like most multidisciplinary texts, the bulk of the book is divided into systems, and by and large system *disease and failure* are addressed separately. This distinction doesn’t always work, and the inevitable repetition and need for cross referencing can be distracting. Some sections seem to assume no prior knowledge of paediatrics, and others appear to be aimed at the experienced paediatrician. In spite of this, there is a reasonable and logical flow to the text, and many extremely useful tables and diagrams. Key learning points and common errors are highlighted in most chapters, and there is a list of useful tips based on the considerable collective experience of the authors. This sort of approach is as close to bedside teaching that you can get in a textbook, and will be appreciated by trainees in particular.

Areas that stand out include the management of fluid and nutritional problems, toxicological and metabolic emergencies, and the diagnostic investigation of children with cardiac and pulmonary problems. It is always difficult to do justice to non-clinical topics like the ethical and psychosocial aspects of critical care, but, at least by including them, the emphasis on the whole patient remains intact. Due attention is given to non-accidental injury and the challenges of transporting patients, the latter reflecting modern, increasingly centralised paediatric intensive care.

In a subspecialty defined by rapid intervention and practical procedures, it is especially difficult to strike the appropriate balance between background detail and clinical practice. On the whole, this book accomplishes this very well. It is not a comprehensive reference text for tertiary care paediatric intensivists, but covers first line treatment to optimise the transition from emergency patient to PICU patient. Until recently, this was mainly undertaken by specialist registrars and consultant anaesthetists, but, in the United Kingdom at least, the next generation of consultant paediatricians will increasingly be called upon to manage critically ill children in those crucial first hours. That group, however reluctantly, will particularly benefit from this useful text.

ALISON SHEFLER
Consultant in Paediatric Intensive Care

Abnormal cortical development and epilepsy: from basic to clinical science.
Edited by Spreafico R, Avanzini G, Andermann F. (Pp 328, hardback; £39)

In his chapter in this book entitled “Neuronal migration disorder and epilepsy in infancy”, Vigevano emphasises that brain malformations represent a causal factor in 3–4% of all epilepsies, although this percentage increases to 18–20% in drug resistant epilepsies. With every new generation of MRI scanner, more and more patients with epilepsy are recognised to have a cortical developmental abnormality, and the aetiological significance of these to the development of epilepsy has been increasingly appreciated. The epileptogenic zone is often wider than the MRI limits of the lesion, suggesting that the adjacent cortex is also epileptogenic or that microscopic pathology extends further than that seen on MRI images.

The book is a useful addition to the literature on cortical dysplasias. It does not aim to be a comprehensive review of all epilepsy but the reader would need considerable prior knowledge of the subject to find the book useful.

ZENOBIA ZAIWALLA
Consultant Paediatric Neurophysiologist