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 to be addressed.

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 interpret it. Those high risk patients admitted with RSV infection spent fewer days in hospital, had less need for oxygen treatment, and with RSV infection spent fewer days in hospital, had less need for oxygen treatment, and with RSV infected patients the benefits of scientific advances will be denied to our most vulnerable patients the benefits of scientific advance.

**WARREN LENNEY**
Academic Department of Child Health,
City General Hospital, Newcastle Road,
Stockton-on-Tees TS1 4SG, UK


**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 (Synagis)1 was 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%,10 20%,12 22.4%,13 and 37%.14 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.

Further data from both Europe12,15 and the US16 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 658 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 trials13 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,1 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,15 but 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 individual.

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, and 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

10 Cohen A, Hirsch RL, Sorrentino M et al. A prospective, randomised, and controlled study by Chandra, which showed a beneficial preventive effect of a partially 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. 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 (heat 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.

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

EDITOR,—The joint statement of the European Society for Paediatric Allergology and Clinical Immunology (ESCAPCI) and the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPghan) describes 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, 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, which showed a beneficial preventive effect of partially 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 formula, showed that the former was superior to the second. 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 (heat 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 this joint statement are generally considered as guidelines for the practitioners. 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.

J SALAZAR-DE-SOUZA
Professor of Paediatrics,
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1050.197 Lisbon, Portugal

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 formulas. J Pediatr Gastroenterol Nutr 1997;24:380–8.

Drs Koletzko and Host comment:

We thank Professor Salazar-de-Souza for his insightful comments on the joint comment of ESCAPCI 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 also point out 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-Souza were not ignored. However, the comments of both felt that neither this nor any 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.

We agree with Professor Salazar-de-Souza 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.

BERNARD KOLETZKO
Professor of Paediatrics,
University of München, Germany
Chair, ESPACI Committee on Hypoallergenic Formulas

Health care needs for travellers

EDITOR,—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. However, the assertion that childhood asthma is more common in travellers is not based on sound evidence. This suggestion was made on a study by Anderson, who reported on the health concerns and needs of traveller families. 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.

Letters, Book reviews

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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 Cleemput extrapolated a high incidence of diagnosed 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% ± 14.8%, OR 5.6, p=0.025), and for doctor diagnosed asthma (25.6% ± 11.1%, OR 4.1, p=0.04). We concluded that the experience of the travelling lifestyle may be a protective factor in the development of asthma.

Fits, pyridoxine, and hyperprolinaemia type II

Editor,—There are currently two types of fits, pyridoxine, and hyperprolinaemia type II. 

Van Cleemput 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.

Letters, Book reviews

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

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

Editor,—We believe that intravenous access to the circulation in infant resuscitation is undervalued and therefore under utilised.

Intraosseous access in infant resuscitation

Editor,—We compared the average flow rates through a range of intravenous canulae 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.

In a laboratory study, we compared the average flow rates through a range of intravenous canulae 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.
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 than that of the intravenous devices. The resistance to flow in situ has not been calculated, but one can 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, leads 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
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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.2 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 FURNESSE
Department of Paediatrics, Sunderland Royal Hospital, Keay Road, Sunderland SR4 7TF, UK


Dr’s 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.

CARLRO CALAFFARELLI
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

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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>
</tr>
</thead>
<tbody>
<tr>
<td>Yellow venflon*</td>
<td>24</td>
<td>35.6</td>
<td>2.81</td>
</tr>
<tr>
<td>Blue venflon*</td>
<td>22</td>
<td>60.6</td>
<td>1.65</td>
</tr>
<tr>
<td>Pink venflon*</td>
<td>20</td>
<td>126.8</td>
<td>0.79</td>
</tr>
<tr>
<td>Green venflon*</td>
<td>16</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>
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 educa-
tion, 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 grate-
ful 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 gastro-
chisis, 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 annoys with drawn out expla-
nations set up to implement those recommen-
dations, and the national organisa-
tions. The book does not set out to duplicate
previous edition which also featured a graph
of normal PEFR values related to height.

On the whole, Essential paediatrics can be
described as user friendly, with numerous
relevant line drawings and important infor-
mation in the margin and in highlighted boxes.
Interesting and useful x rays have also been
included in this edition.

Yet why does one get the feeling that this
may not be the first choice textbook for many
medical students? One reason is that the
is the limited number of colour photographs
compared with some other books on the
market. Another reason, I would suggest, is
the lack of adequate definitions of some of
the common disorders—for example, coeliac
disease and ulcerative colitis.

Despite some drawbacks, I find that Essen-
tial paediatrics is invaluable and have no
qualms about recommending it to medical
students as essential reading.

MINI MARGARET NELSON
Staff Paediatrician


Their children's eating disorders pose serious
problems for parents. They may seek profes-
sional help, but services in the United
Kingdom are fragmented and under devel-
oped; therefore, any book that is designed
specifically for parents is to be welcomed.

My clinical experience is that parents
appear bemused and shocked by the realisa-
tion that their daughter or son has an eating
problem. They are often confused and may
be angry or in denial. Parents may turn to the
popular press, in which articles are some-
times sensible, sometimes sensationalist, wor-
rying, or misleading. High profile cases, such
as those of Princess Diana or Lena Zavoroni
tend to dominate.

The authors have obviously recognised the
lack of sensible self help and advice for
parents of younger children and adolescents.
This book, therefore, is timely and fills an
important gap. A lot of the information is

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 neurological 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


In his chapter in this book entitled “Neuronal migration disorder and epilepsy in infancy”, Vigeveno 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 opened up exciting new fields in the understanding of the pathophysiology of epilepsy and its treatment. This book is a compilation of papers presented at a meeting on epileptogenic cortical developmental abnormalities, organised by the editors. As with books produced in this way there are strengths and weaknesses, with a bias towards specific topics of interest.

The book starts with a short introduction by Frederick Andermann, followed by several chapters on cortical development and animal models. These early chapters are not easy reading but persistence is rewarded by information of direct clinical relevance from the dry basic scientific details—for example, I learnt that work with animal models has shown that pathological changes continue for years after the initial insult, explaining the delay in the development of clinical epilepsy. Furthermore, the progressive maturation of the neurotransmitter pathways could explain why neonatal encephalopathies are often catastrophic, and why children can grow out of their epileptic tendency, even with lesional epilepsy.

The later chapters on electroclinical imaging, neuropathological studies, genetics, and surgery are more relevant for the clinician. In this section, several of the authors emphasise the importance of using the term “neuronal migration disorders” for all dysplasias, when the disturbance can be of neuronal proliferation or organisation and not always an arrest of neuronal migration. Of particular interest to me were the chapters on neuroradiology of malformations, neuronal migration disorders and epilepsy in infancy, and genetic findings, and periventricular nodular heterotopia, especially the genetic implications of recognising these various malformations. I also enjoyed Guerrini’s excellent chapter on the development of polymicrogyria. As in his other publications, he points out that polymicrogyria is the only cortical developmental abnormality which can produce ESES with eventual spontaneous remission, and when this pathology is identified on neuroimaging, surgery should be avoided. This leads us to the two chapters on the problems of resective surgery in focal developmental abnormalities and epilepsy; the first by the Montreal group and the second outlining the Italian/French experience. Both emphasise the specific difficulties of deciding the demarcation of surgical resection in these patients. I was particularly interested in the approach of Munari et al to two step surgery, reoperating with more invasive electrocorticography if the seizures do not stop with lesionectomy alone. While acknowledging that cortical dysplasias can be intrinsically epileptogenic, Munari et al state that, in practice, the epileptogenic zone is often wider than the MRI limits of the lesion, suggesting that either 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 and the reader would need considerable prior knowledge of the subject to find the book useful.

ZENOBIA ZAIWALLA
Consultant Paediatric Neurophysiologist