LETTERS TO THE EDITOR

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Palivizumab and RSV prevention

EDITOR,—The letters from Dr 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 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 the medication. 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 want 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 whether 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, intrapulmonary broiliolitis, 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 those whom it may most benefit seems clinically sensible. All new treatments need 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
Academic Department of Child Health, City General Hospital, Newcastle Road, Stukeley-Morse ST4 6BG, UK


EDITOR,—I am writing in reply to the recent correspondence on 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 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 pharmacoeconomic 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 vs 4.8% palivizumab p=0.0004). A significant reduction in RSV hospitalisation was seen irrespective of gestational age, diagnosis of bronchopulmonary dysplasia, 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 whether 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.

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WARREN LENNEY
Academic Department of Child Health, City General Hospital, Newcastle Road, Stukeley-Morse ST4 6BG, UK

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 conference 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 “The European Society for Paediatric Allergy and Clinical Immunology (ESPACI) and the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN)”, and was published in the March issue of Archives of Disease in Childhood. However, the assertion that childhood asthma is more common in travellers is not based on sound evidence. That suggestion is based 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 formulas. J Pediatr Gastroenterol Nutr 1997;24:380–8.

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

EDITOR,—The joint statement of the European Society for Paediatric Allergy 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 to prevent developing allergic disorders, but more recent prospective and randomised clinical studies have shown that soy protein is as allergenic as cow’s milk protein.2 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,3 which showed a beneficial preventive effect of extensively hydrolysed formula in high risk infants, was ignored. The only study where the preventive effect of an extensively hydrolysed formula was compared with the extensively 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 (but 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 do not 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 the joint statement 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

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.1 However, the assertion that childhood asthma is more common in travellers is not based on sound evidence. That suggestion is based 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

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 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.1 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% v 14.8%, OR 5.6, p=0.025), and for doctor diagnosed asthma (29.6% v 11.1%, OR 2.5, p=0.04). We concluded that the experience of the travelling lifestyle may be a protective factor in the development of asthma.

PF KEARNEY
PM KEARNEY
Department of Paediatrics and Child Health, Cork University Hospital, Cork, Ireland
email: p.kearney@ucc.ie

Fits, pyridoxine, and hyperprolinaeemia type II

EDITOR.—There are currently two types of measurements that are used to assess vitamin B6 status. These are measurements of vitamin B6 and its metabolites, and activation of vitamin B6 dependent enzymes and associated amino acids. Tryptophan loading test is also used to reveal the subtle defects by stressing the B6 metabolic pathway. None of them is ideal, and a combination of them is recommended.

Additionally, there is no concordance between these indices. Transaminase activity in serum and red blood cells (functional index) decreases along with plasma pyridoxal phosphate, urine B6, and pyridoxic acid (direct chemical index) within one week of the removal of vitamin B6 from the diet. Electromyographic abnormalities appear within three weeks.1 Some population groups have a suboptimal intake with or without excess protein intake, although severe vitamin B6 deficiency is not common in man.3

Epileptiform convulsions are a common finding in young vitamin B6 deficient subjects.1 These (sub)clinical deficiencies can be routinely screened by a clinical laboratory if simple tests like transaminases are used. Vitamin B6 deficiency in a well nourished child with an autosomal recessively inherited A1-pyrolin-5-carboxylate with vitamin B6, as reported by Walker et al.5 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 pubertal 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/wk 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 hormone and avoiding pubertal growth of patients with juvenile hypothyroidism in the prepubertal stage.1 Difference between the report of Minamitani et al and our case is that our patient already had the advanced bone age relative to height 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 are indicated for treatment with 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 consult 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.

This work was supported by grants from the Ministry of Health and Welfare of Japan, the Ministry of Education, Science, and Culture, the Japan Private School Promotion Foundation, and the Mami Mizutani Foundation.

Rika Miyazaki
Nagano K. Hypothalamic–pituitary轴と
Hirohiko Higashino
Yohosuke Kobayashi
Department of Paediatrics, Kanazawa Medical University, 10–15 Fumiwakacho, Moriyoshi, Osaka 570–8506, Japan

Intraosseous access in infant resuscitation

EDITOR.—We believe that intraosseous access to the circulation in infant resuscitation is undervalued and therefore underutilised. Intraosseous cannulation is a safe and effective technique that can be performed both quickly and safely in resuscitation.4,5 There have been relatively few complications reported with this technique.3

In a laboratory study, we compared the average flow rates through a range of intravenous cannulae with that of an 18 gauge intraosseous cannula. We purged intravenous Hartmann’s solution through the various devices, at a constant pressure of 300 mm Hg, recording the average volume 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 intravenous 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 intravenous
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, leads us to advocate that greater emphasis is placed on the value of intraosseous cannulae (22 and 20 gauge) can be di

Family 3—This family are Irish travellers and they have had three affected children. The first died with a severe movement disorder and the third, although he was known to be at risk, had an episode of decompensation at 6 weeks. He developed a severe movement disorder and died suddenly and unexpectedly at the age of 13 months. The second child has had some mild problems and attends a normal school.

None of these children were receiving any specific dietary treatment or medication. While we would agree that early diagnosis is essential, the diet is a significant imposition and all that may be needed is intensive treatment during intercurrent infections.

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


Natural history of glutaric aciduria type 1

EDITOR—In their retrospective study, Mounier-Vaudon and Haughton (Arch Dis Child 2000;82:67-70) suggest that early intensive management can alter the natural history of glutaric aciduria type 1. However, the pathogenesis of this disorder is poorly understood and just what is responsible for the better outcome is not clear. In several families in which the first child has the classical phenotype, we have noted a marked difference in outcome of siblings without any specific treatment.

Family 1—In this Jordanian family the first child had a severe movement disorder and died. The second has macrocephaly and mild gait disturbance but is attending normal school.

Family 2—This first child of Nigerian and West Indian parents has a severe dyskinetic cerebral palsy. Her sister has minimal symptoms and attends a normal school.

Intraosseous needle* 18 248 0.40

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

Table 1 Results and calculated infusion time 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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</thead>
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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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<tr>
<td>Pink venflon*</td>
<td>20</td>
<td>126.8</td>
<td>0.79</td>
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<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>
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Evidence based care is upon us, whether we like it or not. There is a multitude of books on the subject, so how is this one different? This is the first in the “Harnessing health information series”, and summarises how evidence based care has evolved into mainstream NHS policy. It does appear to achieve what the series supports to do, as it harnesses health information on the subject. The reader is gently guided around the different organisations set up to implement evidence based care, and is given a guide to how each of the countries of the United Kingdom are described. Many useful resources are highlighted, and the reader feels that he or she can make sense of all the jargon in current usage. There is a brief introduction to the practice of evidence based care, with an overview of the types of research, including qualitative research, and their advantages and disadvantages for answering different sorts of questions. The book does not set out to duplicate the many “How to...” books, but, rather, points the reader in the right direction. There is a useful chapter on information sources on the Internet, and a comprehensive chapter on guidelines, describing most of the arguments for and against. Again, the reader is continuously pointed in the direction of other useful information, without it being duplicated in this book. Patient information is covered in another chapter, and this is interesting and thought provoking reading. Audit, and where it fits into the system, is also included. Finally, clinical quality and clinical governance are brought into the picture, and it all makes sense.

Ruth Roberts is a nurse, and she emphasises the importance of multidisciplinary working. This is an easy book to digest, making common sense of what sometimes seems a complex system. It gives a “warts and all” description of evidence based care. The reader is not put off, but, rather, is left with the feeling, “I can do this”.

This will be a useful resource for managers, nurses, doctors, and clinical quality coordinators. It will be useful for senior staff with a good understanding of the health service and its current requirements, as well as being a good starting point for more junior staff who are trying to make sense of white paper recommendations, and the national organisations set up to implement those recommendations. It can be read in a couple of hours, and will no doubt become pre-interview reading for would-be consultants and specialist registrars.

MAUD MEATES
North Middlesex Hospital


After coming to this country some years ago, I decided to take up paediatrics. I remember asking a senior colleague for advice regarding any textbook that comprised an introduction to the subject. She gave me a choice, but recommended that Essential paediatrics, then in its third edition, would make easy reading. I must say I found this sound advice. Of course, as a postgraduate, one had to progress rapidly on to other textbooks considered the bibles of paediatrics. Hence, when I was asked to review the fourth edition, I was overwhelmed as it brought back memories of my first few months in paediatrics.

As the editors have noted in their preface, this book is meant for medical students. I find that this has been maintained with regard to the manner in which different subjects have been handled with easy to understand language and diagrams. I continue to find the first chapter, “The ill child”, the most impressive and compelling to read, and would not hesitate to recommend this to postgraduate doctors intending to take up a first paediatric post. A similar chapter that needs special mention is that on emotions and behaviour, which, in a brief but concise manner, describes children that we meet daily. It teaches us the importance of careful history taking, including social and family histories.

The book has been updated in many areas, especially in terms of management, in keeping with an evidence based approach. The addition of the British Standards guidelines on the management of chronic asthma is commendable. However, I cannot understand why the importance of the peak flow meter has been downplayed, unlike the previous edition which also had 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 information 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 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 Essential 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 professional help, but services in the United Kingdom are fragmented and under developed; therefore, any book that is designed specifically for parents remains important.

My clinical experience is that parents appear bemused and shocked by the realisation 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 sometimes sensible, sometimes sensationalist, worrying, or misleading. High profile cases, such as those of Princess Diana or Lena Zavaroni 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 initial management of these children 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 pathologi cal 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 error of using the term “neuronal migration disorders” for all dysplasias, when the disturbance can be of neuronal migration 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, schizophrenia, and clinical and genetic findings, and periventricular nodular heterotopia, especially the genetic implications of recognising these various malformations. I also enjoyed Guerini’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 a two step surgery, reoperating with more invasive electrocorticography if the seizures do not stop with lesionectomy alone. While acknowledging that cortical dysplasias can be intracranially epileptogenic, Munari et al state that, in practice, 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 the topic, but the reader would need considerable prior knowledge of the subject to find the book useful.

ZENOBIA ZAIWALLA
Consultant Paediatric Neuropyschologist
Dietary products used in infants for treatment and prevention of food allergy

J SALAZAR-DE-SOUS

Arch Dis Child 2000 83: 87
doi: 10.1136/adc.83.1.87b

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