Palivizumab and RSV prevention

The study was designed in association with 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 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 those with 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 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 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
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LETTERS TO THE EDITOR

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.

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Stockton-on-Tees ST4 6GJ, 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) was the IMpact-RSV trial,1 which 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 trial 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 v 4.8% palivizumab; p=0.0004). A significant reduction in RSV hospitalisation was seen irrespective of gestational age, diagnosis of BPD, and 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%,2,3 20%,4,5 22.4%,6 and 37%.7 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.7

Further data from both Europe8 and the US9 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 654 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 arm 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 trials10 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,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 emphasised 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 company. I contacted all those named to ask for the corresponding author. 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. The paper was to be submitted to a peer reviewed journal and Newborn. Prevention of respiratory syncytial virus infection in children 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.

Dr Koletzko and Comment:

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 also noted 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 comments 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.

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.

BERNARD KOLETZKO
Professor of Paediatrics,
University of München, Germany
Secretary, ESPGHAN Committee on Hypoallergenic Formulas

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 travelers

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. This suggestion is 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

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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 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% vs 14.8%, OR 5.6, p=0.025), and for doctor diagnosis asthma (29.6% vs 11.1%, OR 2.4, p=0.04). We concluded that the experience of travel is a contributory factor in the development of asthma.

Fits, pyridoxine, and hyperprolinaemia type II

EDITOR,—There are currently two types of hyperprolinaemia type II, pyridoxine, and hyperprolinaemia. Minamitani et al reported that treatment with 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 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 height and avoiding pubertal development 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 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 menarche 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 were those suggested 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 consider 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.

Intraosseous access in infant resuscitation

EDITOR,—We believe that intraosseous access to the circulation in infant resuscitation is undervalued and therefore under utilised. Intraosseous cannulation is a simple and effective technique that can be performed both quickly and safely in resuscitation.1,2 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 cannulas with that of an 18 gauge intraosseous cannula. We purged intraosseous 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 intraosseous 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 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


Letters, Book reviews


Fits, pyridoxine, and hyperprolinaemia 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.1

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–pyrroline–5–carboxylate with vitamin B6, as reported by Walker et al.1 It would be interesting to know if and how the authors had measured the growth velocity of the patient are shown. F, father’s height; M, mother’s height.

Figure 1 Treatment, bone age, and height of the patient, plotted on a cross sectional growth chart for girls (0–19 y). Height, bone age, and growth velocity of the patient are shown. F, father’s height; M, mother’s height.

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

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We concluded that patients with juvenile hypothyroidism who are often found to be in progressive pubertal development were those suggested 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 consider 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.

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In a laboratory study, we compared the average flow rates through a range of intraosseous cannulas with that of an 18 gauge intraosseous cannula. We purged intraosseous 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.

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...
canulæ (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 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 the 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).

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Natural history of glutaric aciduria type 1

EDITOR,—In their retrospective study, Monavari and Naughten (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.


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.

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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 ever 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 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

Letters, Book reviews

Information for evidence-based care. By
Roberts R. (Pp 79, paperback; £17.95.) Oxford:
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 infor-
mation series”, and summarises how evi-
dence based care has evolved into main-
stream 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 organi-
sations set up to implement evidence based
care, and the different policies in each of the
countries of the United Kingdom are de-
scribed. Many useful resources are high-
lighted, 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 disadvan-
tages for answering different sorts of ques-
tions. 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 continu-
ously pointed in the direction of other useful
information, without it being duplicated in
this book. Patient information has 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
drawn into the picture, and it all makes sense.
Ruth Roberts is a nurse, and she empha-
sises the importance of multidisciplinary
working. This is an easy book to digest, mak-
ing 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 that.”
This will be a useful resource for managers,
nurses, doctors, and clinical quality coordina-
tors. 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 organisa-
tions set up to implement those recommen-
dations. It can be read in a couple of hours,
and will no doubt become pre-interview
reading for would be consultants and special-
ist registrars.

MAUD MEATES
North Middlesex Hospital

Essential paediatrics. Edited by Hull D,
Johnston DL. (Pp 400, paperback; £24.95.)
958 6
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 might provide an introduc-
tion 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 textbook 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
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 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 allowed 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 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

Eating disorders: a parents’ guide.
Bryant-Waugh R, Lask B. (Pp 222,
0 14026 371 3
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
especially for parents is welcome.
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 Zavaroni
tend to dominate.
The authors have obviously recognised the
lack of sensible self help advice and help 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 respiratory 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”, 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 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, neuropsychological studies, genetics, and surgery are more relevant for the clinician. In this section, several of the authors emphasise the failure of 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, schizencephaly: clinical 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 both epileptogenic and non- epileptogenic, Munari et al state that, in practice, the epileptogenic zone is often wider than the MRI limits of the lesion, suggesting 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 of the topic, and the reader would need considerable prior knowledge of the subject to find the book useful.

ZENOBIA ZAIWALLA
Consultant Paediatric Neurophysiologist

Letters, Book reviews