LETTERS TO THE EDITOR

Rapid responses

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Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read rapid responses” on our homepage.

The editors will decide, as before, whether to also publish it in a future paper issue.

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 trial 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 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)1,2, 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 placebo RSV hospitalisation rate of 22.4%, 6 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 546 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 pharma-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 trials 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, 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,4,5 and felt that whilst they were very useful, UK guidelines should be formulated at a local level, taking into account local risk groups and epidemiology. For these reasons, the UK guidance document deliberately avoids being too prescriptive and whilst describing the two major risk groups (premature infants, <35 weeks gestation, and those with BPD), it emphasises that treatment priorities are likely to vary locally and that decisions regarding which preterm infants to treat will be individualised.

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


cause high levels of morbidity and significant mortality in high-risk infants.

CHRISTINA CARNEGIE
Medical Director, Abbott Laboratories Ltd, UK


The editor comments:

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

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

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

In general, statements of Disease in Childhood is reluctant to publish the results of consensus groups, unless the methods by which they arrived at their conclusions are totally transparent. This case illustrates one reason why we believe it is right to be cautious.

HARVEY MARCOVITCH
Editor in Chief

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

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

Firstly, on the use of soy based formulas for the treatment, as well as for the prevention of food allergy: I was disappointed that no word about this subject appeared in the conclusions of the statement. Many have claimed that the use of soy bean formulas in infancy is an efficient way to prevent food allergic disorders, but more recent prospective and randomised clinical studies have shown that soy protein is as allergenic as cow's milk protein.1 As the matter remains controversial,2 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 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 formula, showed that the former was superior to the second.3 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 are insufficient to allow a firm view. Therefore, I believe the conclusions should have stated that no clear recommendation can be made for the use of a partially hydrolysed formula to prevent food allergy.

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

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.

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. This assertion is based on a study by Anderson, who reported on 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
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. 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 4.5, p=0.025), and for doctor diagnosed asthma (25.6% vs 11.1%, OR 4.4, 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 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 binding of the proline metabolite, pyrroline-5-carboxylate with vitamin B6, as reported by Walker et al. 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 U/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 height and avoiding pubertal progression 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 were not 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 thearche and 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**

Nagoya University

Hirohiko Higashino

Yokoosuke Kobayashi

Department of Paediatrics, Kansai Medical University


**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 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 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 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 the immediate resuscitation period and larger intravenous

![Figure 1](http://adc.bmj.com/first-published-as-10.1136/adc.83.1.87f-on-1-july-2000/downloaded-from-http://adc.bmj.com.on Apr 10, 2022 by guest. Protected by copyright. Arch Dis Child: first published as 10.1136/adc.83.1.87f on 1 July 2000. Downloaded from http://adc.bmj.com/ on Apr 10, 2022 by guest. Protected by copyright.)
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 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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DYLAN PROSSEER
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Royal Bristol Hospital for Sick Children,
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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.

<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>

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

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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. They do not comment on whether they excluded cystic fibrosis (CF). This is relevant as there are an increasing number of mild phenotypes of CF presenting as asthma. CF could be a unifying diagnosis in the “asthmatic” with gastrointestinal symptoms.

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

**JOHN FURNESS**
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Kazl Road, Sunderland SR4 7TJ, UK


**Drs Caffarelli and Atherton comment:**

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

We accept that a diagnosis of CF may not always be obvious on clinical criteria alone, but it remains the case that there is no simple cheap screening test for CF, and we must therefore continue to test only those children in whom there is at least some clinical suspicion for suspecting this diagnosis. We believe that we did adequately consider CF in the children that participated in our study according to clinical criteria, but sweat testing was not undertaken routinely, nor did we screen for CF mutations. While it is possible that we may have missed a child in whom the combination of asthma and respiratory symptoms was due to CF, we consider it exceedingly improbable that such omission would have substantially prejudiced our results.

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

**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

Using the Internet in Healthcare. Tyrrell
S. (Pp 168, paperback; £17.95.) Radcliffe
MedLine: a guide to Effective
Searching Katcher BS. (Pp 148,
paperback; $29.) Ashbury Press. ISBN 01
96734 450 6

Good, I thought, as these books dropped
to the letterbox.
The day before I’d been party to a family
receiving an antenatal diagnosis of gastro-
schisis, 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.
On 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 in better 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 containing 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 of Boolean logic and historical access
systems. In explaining PubMed, it doesn’t
even mention the excellent “Clinical queries”
clinicalquery), on the work of Brian
Haynes and colleagues.
For clinicians, there are better summaries
of framing questions and effective database
searching in Sackett’s book. For researchers,
there are better data databases for citation search-
ing than MedLine.

My own searches found a wonderful
paediatric patient information site (http://
www.birthdefects.org/MAIN.HTM), a site
telling the story of a young lad with
gastrochisis (http://www.geocities.com/
Heartland/Flats/1558/), and an excellent
study of outcome (the PubMed/Med/Inn
filters). I wonder how the father of our latest
pregnancy patients fared.

BOB PHILLIPS
Paediatric Senior House Officer

Information for evidence-based care. By
Roberts R. (Pp 79, paperback; £17.95.)
ISBN 1 85775 356 9

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
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 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 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 empha-
sizes 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 that “I can do this”.
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 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 Paediatric
Association 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 included 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,
ISBN 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
specifically for parents must be welcome.

My clinical experience is that parents
appear bemused and shocked by the realiza-
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,
worthy, or misleading. High profile cases, such
as those of Princess Diana or Lena Zavaroni
have led to media attention.

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
Immediate care of the critically ill child.


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

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

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

Areas that stand out include the management of fluid and nutritional problems, toxicological and metabolic emergencies, and the diagnostic investigation of children with cardiac and respiratory problems. It is 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.

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Abnormal cortical development and epilepsy: from basic to clinical science.


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, neuropathological studies, genetics, and surgery are more relevant for the clinician. In this section, several of the authors emphasise the importance 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, schizophrenia and autism, and metabolic and genetic findings, and peryventricular 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 dysplasia be is 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 literature, but the reader would need considerable prior knowledge of the subject to find the book useful.

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