LETTERS TO THE EDITOR

Book review of Physical Signs of Child Abuse

EDITOR,—The medical aspects of child abuse and neglect are undeniably an area of some controversy within the profession. Clearly doctors you invite to review books on the subject are entitled to express their opinions within the context of their reviews. However, even allowing for this we were disconcerted by the tone and content of Dr Sunderland’s review.1 To us it read as an oblique attack on the overall work of Jane Wynne and Chris Hobbs from someone in an opposing camp. Where Dr Sunderland sticks to factual criticism he is on safe ground (paragraphs 4, 5, and 8). However, the other paragraphs consist of personal criticism to an extent that we believe is unacceptable.

Dr Sunderland clearly implies that Drs Hobbs and Wynne are overzealous pioneers who are personally responsible for an era of overdiagnosis of abuse. We would like to differ and believe that they should be congratulated for their dedicated work in protecting children and publishing their findings. If Dr Sunderland wishes to make such allegations he should choose some other forum than a book review, thus allowing the authors a right of reply.

We believe that the vast majority of paediatricians would not wish to see book reviews of such a contentious nature in the journal.

SIMON COURT (Newcastle)
J R JAMES (Northallerton)
CAMILLE LAZARO (Newcastle)
PETER MORRELL (Middlebrough)
JO SIBERT (Cardiff)
NIGEL SPEIGHT (Durham)
TONY WATERSTON (Newcastle)
(Responsibility to Dr Speight, Dryburn Hospital, North Road, Durham DH1 5TW)

Editors’ comments:
We ask reviewers to provide new and informative information about the subject matter covered by the book in question. We enjoin them to be entertaining, readable, and informative; we remind them of the words of Harold Evans, once editor of The Sunday Times. He asks of a book whether it is of particular topical interest? Does it fill a gap in the market? Is the subject changing rapidly? Is the book relevant to the reviewer’s personal experience, and have the authors succeeded in their task?

We believe readers appreciate this approach rather than being presented with what could amount to a dull precis of a book’s contents page. We do not consider Sunderland’s review is a personal attack on the authors; we would not have published it had we thought it so. Moreover, we disagree that the majority of our readers do not wish to see contentious reviews and we will continue to publish them. Authors have an opportunity to reply through the correspondence column if they wish.

Editors of other scientific journals appear to share our view, as can be seen by publication of what is probably a more contentious review of the book in question than that of Sunderland1 and by correspondence elsewhere about a critical review of another book.3


Differential cytology of bronchoalveolar lavage fluid

EDITOR,—Ratjen et al suggest that differential cell counts in bronchoalveolar lavage (BAL) fluid can be of value in the differential diagnosis of pulmonary infiltrates in immunocompromised children. Firstly, I was confused by figure 1 where several legends state that the open bars represented children with bacterial or fungal infections. But this group, according to the figure, had the lowest, not highest, proportion of neutrophils. One or other part of the legend or the figure must be wrong.

Secondly, inspection of the data in table 4 shows that the group with bacterial and fungal infection had the highest blood leucocyte count of 6.3, and neutrophil count of 4.1 × 109/l. It therefore remains possible that most were neither leucopenic nor had clinically important neutropenia. I could not find the median counts, although they state in the methods that medians were reported for all data. Were the counts skewed? It could be argued that their results do not support their claim to have found ‘marked cell proliferation on the bronchoalveolar surface despite systemic leucopenia’. Their results, as presented, suggest that differential counts of BAL fluid may be useful in immunocompromised children without systemic leucopenia/neutropenia, but it would have been interesting to see data from a substantial group of infected children all of whom were demonstrably leucopenic (or neutropenic). Such children usually cause us at least as much concern as those with adequate, normal, or raised counts. Furthermore, since we were not given the individual patient data, it is difficult to see how these results might help us make a diagnosis or institute appropriate therapy in an individual patient. The really important findings are the organisms isolated from the BAL fluid, whatever the cellular composition might be.

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Dr Ratjen comments:
We regret the mistake in the legend of figure 1 where the first column (open bars) represents children without bronchopulmonary disease (who have the lowest percentage of neutrophils) while the second column represents immunocompromised children with bacterial or fungal infection (closed bars).

It is apparent from table 4 that a substantial number of children (16 of 28) were not neutropenic at the time of BAL. However, all children were neutropenic (most of them induced by chemotherapy) at the onset of their pulmonary illness. The timing of BAL was guided by the clinical course of the child’s pulmonary disease and varied between children. Of the 12 children with blood neutrophil counts below 1000/nl at the time of BAL, four had bacterial or fungal pathogens in their BAL. As the absolute number of patients in this subgroup is small, we have not described their results in greater detail. A standardised approach performing BAL early in the phase of neutropenia would be necessary to address the question how useful BAL differential cytology will be in this situation. The notion that we have found marked cell proliferation despite systemic neutropenia was included to reflect our observation that individual patients, who did not have any neutrophils in their bloodstream, still had raised neutrophil counts in BAL fluid. Our aim was to assess BAL cytology in all immunocompromised children who developed pulmonary infiltrates in the course of their illness and underwent BAL for clinical indications in a three year period. We agree with Dr Reid that the most important information is to be gained from cultures of BAL fluid. Nevertheless, our data suggest that BAL cytology provides useful additional information as it can show alterations in BAL cytology in response to pathogens that may be helpful in the differential diagnosis of pulmonary infiltrates in these immunocompromised children.


Hypocitraturia in patients with urolithiasis

EDITOR,—Akcay et al observed a significantly lower urinary citrate excretion in children with a previous history of urolithiasis.1 Their findings are comparable with data presented in adult stone forming patients, showing a high incidence of hypocitraturia.2 As citrate is a potent inhibitor of calcium-oxalate or calcium-phosphate crystal aggregation,3 hypocitraturia is one important factor influencing recurrent urolithiasis.

Urinary citrate excretion expressed as a citrate/creatinine ratio, in idiopathic stone forming children (n = 25) was compared with the citrate excretion in healthy boys and girls (n = 24). Unfortunately the authors did not indicate whether they present a molar creatinine ratio (mol/mol), or a ratio expressed in mg/mg. Therefore, the data are of limited value at present.

We examined urinary citrate excretion in 473 healthy infants and children of different age groups, showing that citrate excretion is not only sex but also age related.4 Mean molar citrate/creatinine ratio was higher (p <0.05) in both male and female infants, than in older age groups; in infancy it was higher in females than in males (1.9 ± 0.63 mol/mol, p <0.05). During childhood, girls tended to have slightly lower mean molar ratios than boys (0.27 ± 0.37). This relationship increased in adolescence, when girls again had higher mean molar excretions than boys (0.32 ± 0.28 mol/mol), as observed in healthy adults.5

The absence of a relationship between age, gender, and urine citrate excretion in the study of Akçay et al is likely because of an insufficient power to detect such differences. In conclusion, we look forward to a response from the authors about the unit of the citrate/creatinine ratio. We suggest that
there exist normal age and sex related values for citrate/creatinine ratio in infants and children which are based on adequate population data. This will allow the clinician to evaluate further idiopathic urolithiasis.

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Professor Akçay comments:
We examined the urinary citrate/creatinine ratio in 25 children with idiopathic calcium urolithiasis and in 25 controls. The mean citrate excretion was calculated as citrate/creatinine ratio and we presented a ratio expressed in g/L. The mean (SD) citrate excretion in controls, 0.51 (0.2), was significantly higher in patients with urolithiasis, 0.18 (0.7).

We couldn’t determine a correlation between urinary citrate excretion and age because in the children our study were of about the same age.


Stroke due to arterial disease in congenital heart disease

EDITOR,—A 1 year old girl with pulmonary atresia developed an acute right hemiparesis. She had had an intact ventricular septum at birth. Ventricular septostomy and insertion of a Blalock-Taussig shunt were carried out at 1 week. She had otherwise been well and was developmentally age appropriate. At the time of the hemiparesis, she was only mildly poly-}


Injuries and the risk of disability in teenagers and young adults

EDITOR,—In the paper by Barker et al on injuries and disabilities in teenagers and young adults the information obtained from the patients would appear to have been largely, if not wholly, derived from questionnaires, which is likely to have been subjective and therefore biased. Specifically, ‘disability’ is a highly subjective term and may be perceived and interpreted differently by different individuals. In addition, patients may have had a variety of reasons for responding ‘yes’ to the question, ‘Has this.... accident(s) resulted in any permanent disability?’. The authors do not state whether the ‘patients’ disabilities were independently assessed.

Secondly, although their data may be correct, implicit within their conclusion is the suggestion, perhaps unintentionally, that for economic reasons preventative measures, and therefore resources should be directed at reducing only the less serious (though more common) injuries, potentially at the expense of ignoring those equally preventable factors that predispose to a more serious injury—and therefore disability. A more serious disability, though less common, does not make it less of a disability—for the patient, their family, or the community.

Finally, a significant reduction in the frequency of the more common (but less serious) injuries may be extremely difficult, if not impossible, to achieve because of the obvious and marked heterogeneity of the causes of these injuries. As a consequence this could, theoretically, result in an inefficient and uneconomic use of available resources in attempting to prevent what may be largely preventable.

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The brain in muscular dystrophy

EDITOR,—Lucina wonders why some boys with Duchenne muscular dystrophy (DMD) also have cognitive impairment, and whether it could be related to brain dystrophy. Most muscles in DMD show signs of repeated necrosis and repair. Fadic et al, reporting a dystrophic variant recently, were puzzled not to find these signs in the myocardium which showed other signs of the disease. I suspect that this is because the heart is not subjected to the disruptive forces that most muscles meet during exercise. I have long wondered why boys with DMD do not have diplopia, and whether the extraocular muscles, which are not subject to disruptive forces, might also lack signs of injury. An analogous situation may be found in the brain lacking dystrophin which might have a low resistance to shear stress at a subcellular level. Insignificant bumps might then cause cumulative damage and loss of intelligence. It would be interesting to know whether affected boys have had more bumps on the head than those whose intelligence is preserved.

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BOOK REVIEWS


I am a busy child psychiatrist and I suspect, like many others in my position, find the time for reading squeezed by ‘post NHS fatigue syndrome’. Depression in children, as the editor of the book comments, is not easily evaluated and difficult to treat, as these read-
I am left with the impression that the book is not wholly successful in its stated purpose. This is because some chapters were confusing to the clinician and probably too brief to interest researchers in development and clinical neuroscience to whom the book is partly directed.

I did come away with new ideas and information from the chapters of a more directly clinical orientation. I was even able to be one step ahead of some colleagues in a recent audit meeting looking at the management of childhood depression.

JOHN HIGGS
Consultant child psychiatrist


Paediatrics is a broad discipline. There is always the possibility that the most experienced of us will meet a problem or a diagnosis that we have never met before. Advances in the understanding of disease or developments in treatment and management are often published in specialist journals which we can only read if they fall within our own field. When we know that we are puzzled we can only read if they fall within our own field. When we know that we are puzzled we can only read if they fall within our own field. When we know that we are puzzled we can only read if they fall within our own field.

Our biggest mistakes occur when we do not undertake an appropriate literature review. To guard against this we must read and study widely and when we do this we must pay attention to all branches of paediatrics. The book brings together the influences of abuse at different stages in the child's life, influencing intervention. Perhaps they should have referred to the work of Moses Laufer who has also looked at the individual's thinking about this series is its emphasis on classification, genetics, social and psychological aspects of morbidity and suicidal behaviour. The book offers a constructive approach to assessment. The chapter on drug treatment adds new ideas in an area where previous work has suggested it is of little help.

The review on suicidal behaviour, from the department of paediatrics at Utrecht, gave a constructive approach to assessment. The chapter brings together the influences of abuse at different stages in the child's life, influencing intervention. Perhaps they should have referred to the work of Moses Laufer for a diagnostic approach to antidepressant drugs in a specialist field, and two in tropical paediatrics. I was helped particularly by Baxter and Rittey's article on epilepsy with its up to date classification of syndromes.

The best bit is kept till the end: Professor David's personal literature review for 1994. This consists of one line summaries of a wide variety of articles of general paediatric interest collected under topic headings. It serves as an excellent reminder of the ground which has been covered during the year as well as being a starting point for a possible literature search.

The series Recent Advances has an established reputation for quality and this edition has maintained that reputation. It can never be updated and departments of paediatrics should strongly consider this book as an alternative.

The authors recommend the book for paediatricians and geneticists as a guide to the visible recognition of congenital malformations. In fact, the book deals with babies and children with malformations as part of a syndrome rather than addressing isolated malformations. An improvement on the previous edition is the expanded text and references by each condition which is described. Many of the photographs have been updated. There is an increase in the number of dysmorphic syndromes described at the expense of descriptions of the more common birth defects and single gene disorders. I'm not sure this is an unqualified success because it is now difficult to know whether the book is aimed towards paediatricians or geneticists. In fact it falls somewhere in the middle. For paediatricians there are too many rare syndromes which are not distinct enough for easy recognition. Unfortunately, compared with the earlier edition, a decision has been made to omit much of the general introductory text.

I would recommend some sharper editing before the next edition. The grouping of syndromes, for example Rubinstein-Taybi, under syndromes diagnosed by visible abnormalities, is unhelpful. I also found it annoying that the title at the top of the page may not correspond to any of the illustrations on the page.

Most paediatricians will use the book as a teaching or learning aid rather than a diagnostic aid and I'm sure that it will give invaluable help to those preparing for the MRCGP. It is more up to date than Smith's Recognizable Patterns of Human Malformation and departments of paediatrics should strongly consider this book as an alternative.

Finally the cover shows the full body x-ray of a neonate. I asked several consultant geneticists for a diagnosis. There was no consensus. Could the authors please give us the answer in the next edition?

CHRISTOPHER CHEETHAM
Consultant paediatrician

JANE HURST
Consultant clinical geneticist


It is a difficult task to review a book written by two clinical geneticists who taught me any skills I have in the diagnosis of children with malformations. The enthusiasm of the authors to share their skills is recognised by the number of trainees who would like to work with them and by the many teaching aids they have published over the past 15 years. This enthusiasm has resulted in this current book which is considered among the best by TJD avid. (Pp 229; £30 paperback.)
Book review of *Physical Signs of Child Abuse*

SIMON COURT, J R JAMES, CAMILLE LAZARO, PETER MORRELL, JO SIBERT, NIGEL SPEIGHT and TONY WATERSTON

*Arch Dis Child* 1997 76: 174
doi: 10.1136/adc.76.2.174

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