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 beg 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 *(Middlesbrough)*
JO SIBERT *(Cardiff)*
NIGEL SPEIGHT *(Durham)*
TONY WATERSTON *(Newcastle)*
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Editors' comment:
We ask reviewers to provide new and interesting 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 far more contentious review of the book in question than that of Sunderland[2] and by correspondence elsewhere about a critical review of another book.[3–5]

1 Sunderland R. Book review of *Physical signs of child abuse. A colour atlas.* Hobbs CJ, Wynne JM, eds. *Arch Dis Child* 1996;**75**:356.
2 Finkel MA. Book review of *Physical signs of child abuse. A colour atlas*. Hobbs CJ, Wynne JM, eds. *N Engl J Med* 1996;**335**:983-4.
3 Wilks D, Farrington M, Rubenstein D. Authors of books hit back. *BMJ* 1996;**313**:1010.
4 Denning DW. Reviewer's reply. *BMJ* 1996;**313**: 1010.
5 Ginsburg J. Editor of book hits back. *BMJ* 1996;**313**:1011.

Differential cytology of bronchoalveolar lavage fluid

EDITOR,—Ratjen *et al* suggest that differential cell counts in bronchoalveolar ravage (BAL) fluid can be of value in the differential diagnosis of pulmonary infiltrates in immunocompromised children.[1] Firstly I was confused by figure 1 whose legend states 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 a mean peripheral blood leucocyte count of 6.3, and neutrophil count of 4.1 $\times$ 10^9/1. It therefore remains possible that most were neither leukopenic 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 leukopenia'. Their results, as presented, suggest that differential counts of BAL fluid may be useful in immunocompromised children without systemic leukopenia/neutropenia, but it would have been interesting to see data from a substantial group of infected children all of whom were demonstrably leukopenic (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.

1 Ratjen F, Costabel U, Havers W. Differential cytology of bronchoalveolar lavage fluid in immunosuppressed children with pulmonary infiltrates. *Arch Dis Child* 1996;**74**:507-11.

Hypocitraturia in patients with urolithiasis

EDITOR,—Akçay *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[1]). 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 *v* 0.63 mol/mol, $p < 0.05$). During childhood, girls tended to have slightly lower mean molar ratios than boys (0.27 *v* 0.33). This relationship changed in adolescence, when girls again had higher mean citrate excretions than boys (0.32 *v* 0.28 mol/mol), as observed in healthy adults.[2]

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.[4] 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/g. The mean (SD) citrate excretion in controls, 0.51(0.2), was significantly higher in patients with urolithiasis, 0.181(0.076).

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

1 Akçay T, Konukoglu D, Celik C. Hypocitraturia in patients with urolithiasis. *Arch Dis Child* 1996;**74**:350-1.
2 Hesse A, Classen A, Knoll M, Timmermann F, Vahlensieck W. Dependence on urine composition on the age and sex of healthy subjects. *Clin Chim Acta* 1986;**160**:79-86.
3 Kok DJ, Papapoulos SE, Bijovet OLM. Excessive crystal agglomeration with low citrate excretion in recurrent stone formers. *Lancet* 1986;**i**:1056-8.
4 Hesse A, Hoppe B, Jahnen A, Bach D. Urinary oxalate, citrate and uric acid excretion in healthy infants and children. *Urol Int* (submitted).

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 polycythaemic (packed cell volume = 0.426). There were no new findings on praecordial echocardiography; no intracardiac thrombus was seen. Thirty six hours later, magnetic resonance imaging showed an acute infarct in the territory of the left middle cerebral artery (MCA). A small, mature lesion was present in the right corona radiata. Magnetic resonance angiography revealed reduced flow in the petrous and cavernous left internal carotid artery, with a high density rim, suggestive of dissection, with reduced flow in the left MCA (see fig 1). She was treated with warfarin; at six weeks she had a significant residual hemiparesis.

Cervicocephalic arterial dissection is a significant cause of stroke in young patients[1] but the diagnosis is considered often only when there is a history of antecedent trauma, although this is not invariable, and dissection may occur spontaneously or after relatively innocuous injuries. Diagnosis is important as anticoagulation is of proved benefit if given before infarction has occurred[2] and the risk of recurrent dissection over 10 years is 12%.[3]

Although an embolic origin for stroke is frequent assumed in children with known cardiac disease, after excision of endocarditis, polycythaemia and right to left shunts, of 25

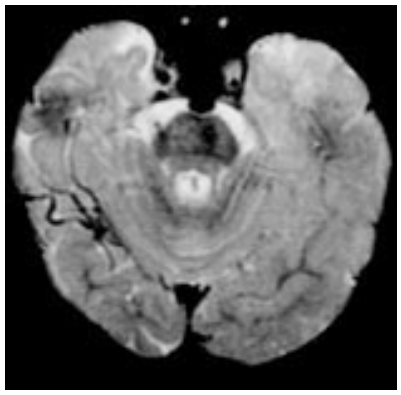

Figure 1 T2 weighted magnetic resonance image (axial section) showing a high intensity rim around the left internal carotid artery which has a smaller lumen than the contralateral vessel. The rim represents the intramural haematoma which has resulted from dissection of the vessel.

such patients we have seen, intracardiac thrombus was demonstrated in only five cases (20%). Fifteen of these children (60%) were found to have structural cerebrovascular abnormalities.

Children with acute stroke should be thoroughly investigated on each occasion in order to detect all potential risk factors, some of which may contribute to the already significant risk of recurrent cerebral ischaemic events.

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1 Schievnik WI, Mokri B, Piepgras DG. Spontaneous dissection of the cervicocephalic arteries in childhood and adolescence. *Neurology* 1994;**44**:1607-12.
2 Sturzenegger M. Spontaneous internal carotid artery dissection: early diagnosis and management in 44 patients. *J Neurol* 1995;**242**:231-8.
3 Schievnik WI, Mokri B, O'Fallon WM. Recurrent spontaneous cervical artery dissection. *N Engl J Med* 1994;**330**:393-7.

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,[1] 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 unpreventable.

RICHARD E APPLETON
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1 Barker M, Power C, Roberts I. Injuries and the risk of disability in teenagers and young adults. *Arch Dis Child* 1996;**75**:156-8.

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 dystrophin.[1] 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.[2] 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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1 Lucina. *Arch Dis Child* 1996;**74**:474.
2 Fadic R, Sunada Y, Waclawik AJ, *et al.* Deficiency of dystrophin associated glycoprotein (adhalin) in a patient with muscular dystrophy and cardiomyopathy. *N Engl J Med* 1996;**334**:362-6.

BOOK REVIEWS

The Depressed Child and Adolescent. Edited by Ian M Goodyer. (Pp 354; £45 hardback.) Cambridge University Press, 1995. ISBN 9-521-43326-6.

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. For these rea-

sons I looked to this book for clear narrative, a review of current information, and new ideas.

This is the first in a series of Cambridge Monographs in child and adolescent psychiatry designed to cover major syndromes affecting children's mental health. What is different about this series is its emphasis on the effect of normal and abnormal child development on depressive syndromes. There are contributors from both sides of the Atlantic, clinicians and researchers.

The book opens with an historic review which nicely sets the scene for society's development in appreciating childhood depression. Subsequent chapters then review normal development of emotional regulation and physiological changes with age. There are sections on classification, genetics, social and physiological aspects of morbidity and suicidal behaviour. The reviews on treatment consider the child's developmental stage influencing response to treatment; for example, cognitive behavioural therapy, even in the adolescent, may be limited in those individuals who cannot engage in logical reasoning.

The book is rounded off with a review of the natural history of depressive disorder in the young. The concept of scarring by a first episode is mentioned, hopefully justifying adequate child psychiatry services. If we can help these patients overcome their first episode of depression, further episodes need not be so devastating.

The book has several strengths. Firstly the contributors are concise and, with some exceptions, their reviews are clearly written. Secondly the index and reference lists are comprehensive and the latter are usually cited from more accessible journals. Thirdly there are some really helpful clinical chapters. 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[1] who has also looked at the individual's thinking in these situations with useful practical implications.

Similarly the review of psychological treatments was limited by lack of discussion of psychoanalytic approaches, particularly in the treatment of chronically depressed youth with many social adversities and previous abuse.

The main weakness of the book is that some reviewers tried to do too much in a short space. This was shown in the chapters devoted to neuroendocrine, physiological, and family genetic aspects. Some authors seemed bored by the effort of trying to squeeze complex and often conflicting material into short chapters. This was disappointing especially as some ideas are intriguing, such as neuroendocrine 'scarring' caused by repeated stress, for example, bullying. Part of the problem is lack of research in childhood depression, an area which has really only been recognised in the last 30 years or so.

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

1 Laufer M, ed. *The suicidal adolescent*. Karrac Books, 1995.

Recent Advances in Paediatrics. Edited by T J David. (Pp 229; £30 paperback.) Churchill Livingstone, 1995. ISBN 0-443-05308-1.

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 get help. We can seek specialist advice, or undertake an appropriate literature review. Our biggest mistakes occur when we do not know where our areas of ignorance lie: the black holes of continuing education. To guard against this we must read and study widely and when we do this we must pay attention to all branches of paediatrics.

Recent Advances in Paediatrics provides a major contribution in this field. Twelve review articles on topics of interest to all paediatricians are of sufficient depth to cover the subject but short enough to be digested by the non-specialist. All are up to date and well referenced. The paediatrician who reads them can be confident that he has the subjects covered.

Of the 12 chapters, six are in ambulatory or community paediatrics, four 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 cover the whole of paediatrics, but taken with its predecessors it makes a major contribution to the book shelf of any paediatrician.

CHRISTOPHER CHEETHAM
Consultant paediatrician

Colour Atlas of Congenital Malformation Syndromes. Edited by M Baraitser and R Winter. (Pp 233; £65 hardback.) Mosby-Wolfe, 1996. ISBN 0-7234-2073-4.

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 the current book which, although with a new publisher, is an updated revised edition of *A Colour Atlas of Clinical Genetics.* I think that most readers of *Archives* will find the book an interesting, and more importantly, enjoyable romp through dysmorphic syndromes. Overall the book is to be recommended, though it is slightly spoilt by a few features which could easily be changed in another edition.

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 MRCP. 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 ?

JANE HURST
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