

## LETTERS TO THE EDITOR

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### Sedation versus general anaesthesia for MRI scanning in children

EDITOR.—We read with interest the article concerning the sedation of children for magnetic resonance imaging (MRI),<sup>1</sup> and would like to support Dr Bray's view that general anaesthesia is a safer and more reliable method of managing children undergoing this procedure.

In our trust we have a large number of children undergoing MRI scanning, the great majority of whom have general anaesthesia. We have three or four planned half day general anaesthesia sessions per week, all covered by a consultant paediatric anaesthetist. We do still occasionally sedate patients when they require a short scan; because of the urgency it is not possible to schedule them into a fixed general anaesthesia session.

Previously, we relied mainly on sedation techniques, but found a large failure rate due to restless patients moving during the scan. In fact, since general anaesthesia has superseded sedation, the quality of scan has markedly improved and scan times have been reduced.

For patients undergoing cardiac MRI scans, periods of breath holding are required during several scan sequences; this would be impossible to achieve unless the patient was paralysed and ventilated.

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1 Lawson GR, Bray RJ. Sedation of children for magnetic resonance imaging. *Arch Dis Child* 2000;82:150-3.

### The debate between sedation and anaesthesia for children undergoing MRI

EDITORS.—Drs Lawson and Bray<sup>1</sup> have presented arguments for and against deep sedation of children by non-anaesthetists. We would like to contribute to the debate by expanding on issues which have influenced and encouraged the development of a nurse led sedation service for magnetic resonance imaging (MRI) at our hospital.<sup>2</sup>

There continues to be a huge demand for MRI and as a result we have had to meet the challenge of providing a sedation and anaesthesia service with limited resources. With safety in mind, in 1996 we sought funding for sufficient staffing to provide an anaesthesia only service for one MR scanner, for four days a week. Funding was refused because of high costs, and because the option of improved sedation by non-anaesthetists had not been fully explored. Fortunately, we have been successful in developing our nurse led sedation service and have needed only a modest increase in anaesthesia sessions from two in 1996 to three currently. We now have two MRI scanners providing a total of eight days a week of clinical service, and we are able to look back and reflect that if we had held the philosophy that only anaesthesia was safe enough this would have severely limited any expansion and flexibility in the totality of the anaesthetic service we provide to the hospital. We believe we have developed a sedation service by non-anaesthetists that is safe and effective.

Everyone seems to agree that conscious sedation, where the patient can be roused by verbal command, is safe for non-anaesthetists but is impractical for imaging in small children because they must be "asleep" to be still enough. We have always accepted the danger of deeply sedated children becoming effectively anaesthetised during imaging. Indeed, one of us (DH) was a member of the working party that developed the guidelines for sedation in adults quoted by Lawson and Bray.<sup>3</sup> We have therefore applied the following definition of sedation for MRI: a technique in which the use of a drug or drugs produces a state of depression of the nervous system such that the patient is not easily roused but which has a safety margin wide enough to render the loss of airway and breathing reflexes unlikely.

We accept that in an ideal world, anaesthetists are the best people to manage deep sedation. However, this statement is too broad and overlooks the fact that sedation is specific to a particular procedure. Gastroscopy for example, requires sedation to a degree which suppresses the gag reflex and consequently airway reflexes are often reduced. Such a "depth" of sedation is unnecessary for non-painful imaging and therefore mortality data about sedation for endoscopy are not helpful in answering the question "is deep sedation by non-anaesthetists of children for MRI safe?"

We believe that our nurse led sedation service is safe because we have developed a protocol that makes any airway or breathing problem extremely unlikely and, if it should occur, our nurses have sufficient resuscitation skills to cope until help arrives. Reducing the risks to acceptable levels depends on the strict adherence to exclusion criteria, the characteristics of the drug regimen, and finally, but most crucially, the judgement, skills, and experience of the nurses. Our nurses are carefully assessed after an

initial training period, and those who are accepted as sedationists receive regular retraining and reassessment. They work to strict protocols devised by a multidisciplinary team consisting of radiologists, anaesthetists, paediatricians, senior nurses, and radiographers. If such a strictly controlled system is not developed, or suitable people cannot be found to implement it, we have no doubt that an anaesthesia service is safer. The references quoted by Dr Bray show that accidents can happen if good practice is not followed.

Our latest figures are encouraging. We have sedated almost 3500 children according to our published sedation guidelines and so far no child has required the use of any airway or breathing device. Oxygen saturation has not dropped below 87%. Can anaesthesia, including postoperative recovery by nurses, match these statistics?

It is fair to suggest that a sedation service might be made even safer with anaesthetists present throughout the procedure. Nevertheless in our hospital, we do not believe that such an expense could be justified. Furthermore, if anaesthetists are available they are more cost effective when administering anaesthesia than supervising sedation.

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- 1 Lawson GR, Bray RJ. Sedation of children for magnetic resonance imaging. *Arch Dis Child* 2000;82:150-4.
- 2 Sury MRJ, Hatch DJ, Deeley T, Dicks-Mireaux C, Chong WK. Development of a nurse-led sedation service for paediatric magnetic resonance imaging. *Lancet* 1999;353:1667-71.
- 3 Royal College of Anaesthetists and Royal College of Radiologists. *Sedation and anaesthesia in radiology. Report of a joint working party*. London: Royal College of Anaesthetists and Royal College of Radiologists, 1992.

### Investigation of sudden unexpected deaths in infancy

EDITOR.—The CESDI study report on sudden unexpected deaths in infancy<sup>1</sup> and the paper in this journal by Ward, Platt, *et al*,<sup>2</sup> emphasise the importance of thorough investigation of all sudden infant deaths if the true cause is to be found.

The history of an apparent life threatening event emerges as a significant risk factor for sudden unexpected death and this, together with symptoms of ill health including sweating in the 24 hours before death, suggests that death in these cases may be due to a metabolic cause in a vulnerable infant.

Although inherited metabolic diseases (IMD) are rare because of the reduction in preventable causes following the "back to sleep campaign", they are now likely to form a higher proportion of all sudden unexpected infant deaths, and accurate diagnosis of an

### Guidelines for management of sudden unexpected death in infants under two years old

1. Break the news to parents, explain about the urgency and nature of investigations, and the obligation to inform the coroner, but do not delay taking specimens for metabolic investigations whilst you take a history and examine the baby.
2. Inform the coroner and obtain permission to take specimens.
3. Blood—Perform a heart stab within 30 minutes of death if possible and preferably not over four hours after. Drop some blood onto blood spot cards directly from syringe (for acyl carnitines). Allow to dry at room temperature. Split the remainder into lithium heparin for metabolic tests spin (store plasma at  $-20^{\circ}\text{C}$ ); plain bottle (clotted blood) for toxicology spin (store serum at  $-20^{\circ}\text{C}$ ); blood cultures to incubate at  $37^{\circ}$ ; and consider blood for chromosomes—especially if dysmorphic.
4. Urine—Supra pubic aspirate (SPA) of bladder. Divide urine into three plain bottles. For microbiology store in fridge at  $+4^{\circ}\text{C}$ ; toxicology, spin and freeze supernatant at  $-20^{\circ}\text{C}$ ; biochemistry, for metabolic tests (amino and organic acids), spin and freeze at  $-20^{\circ}\text{C}$ .
5. Nasopharyngeal swab (if less than eight hours after death) for virology into transport medium. Any other body fluids, swabs, etc, store at  $4^{\circ}\text{C}$  for microbiology.
6. Skin biopsy—Send to a metabolic laboratory in culture medium. Store at  $4^{\circ}\text{C}$ .
7. Consider muscle and liver biopsy if there is suspicion of IMD—for example, death of sibling or consanguinity. Contact regional metabolic laboratory for advice.
8. Take a full history, including detailed account of the final 24 hours, position of baby when found, clothing worn, intercurrent illness in family members, and smoking habits.
9. Complete clinical examination—Look for external marks, bruises or injuries and petechiae, look for skull fracture and in fundus for retinal haemorrhages, record rectal temperature and any other signs of illness.
10. Explain to parents about sudden unexpected death in infancy, encourage them to hold the baby and give bereavement support. Give advice about cessation of lactation if necessary.
11. Radiology—skeletal survey.
12. Check child protection register—particularly important if there are young siblings or a twin.
13. Inform general practitioner, health visitor, community child health and hospital records, and cancel all appointments.
14. Document all specimens taken, label, and ensure an unbroken chain of evidence for forensic specimens. Record the site from which specimens were taken—for example, cardiac stab, SPA, urine, etc. Remember to date, time, and sign the records as these may become legal documents.

The authors gratefully acknowledge the contributions from many colleagues in Birmingham, Walsall, and Wolverhampton to the development of these guidelines.

index case could well prevent death in a subsequent sibling.

Factors suggesting IMD include consanguineous parents and previous infant death in the family. Although a history of hypotonia or developmental delay and organomegaly may occur, these disorders can cause death without significant prodromal symptoms and can be precipitated in a previously healthy infant by a stress such as infection. Investigation may be limited at necropsy if suitable specimens are not obtained as soon as possible, blood ideally within thirty minutes and tissue preferably not more than four hours after death. Many metabolic disorders can be diagnosed on blood or urine, but some require fibroblasts or other tissue for analysis. It should be possible to perform a skin biopsy for fibroblast culture in most district hospitals.

The CESDI study acknowledged that lack of information was a major impediment to determining the true cause of death and makes recommendations for investigations and procedures following sudden deaths in infancy. It is disappointing that they make no reference to the collection of specimens or procedures to be followed by staff in the accident and emergency department, or wherever the death is confirmed.

It is important that paediatric residents are aware of the urgency and have a protocol for investigation and collection of specimens that has been agreed with the local coroner. In the West Midlands, we have written guidelines for managing sudden unexpected death in infancy to ensure that vital evidence of IMD, infection, or non-accidental injury is not lost.

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1 *Sudden Unexpected Deaths in Infancy*. The CESDI SUDI Studies 1993–1996. London: The Stationery Office Books, 2000.

2 Ward Platt M, Blair PS, Fleming PJ, *et al*. A clinical comparison of SIDS and explained sudden infant deaths: how healthy and how normal? CESDI SUDI Research Group. Confidential Inquiry into Stillbirths and Deaths in Infancy study. *Arch Dis Child* 2000;**82**:98–106.

### Lymphopenia in lymphatic malformations

EDITOR,—Hodge *et al*<sup>1</sup> draw our attention to the possible association of hypogammaglobulinaemia and global lymphopenia with Proteus syndrome. They suggest that this may

be secondary to the loss of immunoglobulins and lymphocytes into lymphoedematous tissue. We have seen a similar phenomenon in a child with a massive cystic hygroma in the neck. Immunological investigations showed persistent severe lymphopenia (table 1) with low levels of all lymphocyte subsets. Lymphocyte proliferative responses to PHA were normal as were immunoglobulin levels and antibody responses to protein (diphtheria and tetanus toxoid) and polysaccharide (*haemophilus b*) vaccines. He initially suffered recurrent chest and skin infections and oral candidiasis but this responded well to treatment with prophylactic cotrimoxazole and nystatin mouthwashes. As in their case, we feel we may have been observing peripheral sequestration of circulating lymphocytes and that, as a consequence, the clinical phenotype was milder than one would have expected in a child with similar results but caused by failure of lymphocyte production. We would like to extend their suggestion for immunological investigations into Proteus syndrome to other children with large lymphatic malformations.

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1 Hodge D, Misbah SA, Mueller RF, *et al*. Proteus syndrome and immunodeficiency. *Arch Dis Child* 2000;**82**:234–5.

### Infant air travel, bronchiolitis, and the environment

EDITOR,—Probably like most doctors looking after children, we feel uneasy when asked whether it would “be alright” to take small children and infants on plane journeys for holidays. Controversy continues as to whether flying might be harmful for infants,<sup>1–4</sup> and it is questionable whether infants benefit from weekend breaks or long distance holidays in search of better weather. However, we suspect that the air travelling population will get increasingly younger and we will be asked more frequently. As long as solid data about the safety of plane journeys for infants are lacking, anecdotal experience will be the only basis of advice.

In this context we would like to report the case of an 11 week old twin boy, corrected age 6 weeks for prematurity of 35 weeks, who was admitted to the Accident and Emergency Department of our hospital directly from an aeroplane after an emergency landing at Manchester Airport. Shortly after take off from London Gatwick for Florida the infant stopped breathing and went blue. On the plane resuscitation was attempted by the parents, a stewardess, and a paramedically trained fellow passenger. With oxygen and mouth to mouth breathing the baby's colour improved and the plane staged an emergency landing in Manchester. On arrival the

Table 1 Lymphocyte counts and subsets in a child with a large cystic hygroma in the neck

Age at test (years)	Lymphocyte counts ( $\times 10^9/l$ ). Normal ranges in brackets				
	CD3 (0.69 to 2.25)	CD4 (0.41 to 1.41)	CD8 (0.28 to 1.20)	CD19 (0.05 to 0.41)	CD16/56 (0.04 to 0.87)
7.3	0.52	0.11	0.33	0.20	0.2
8.2	0.25	0.1	0.17	0.11	0.12
9.9	0.05	0.03	0.03	0.03	0.04

picture was typical of bronchiolitis and respiratory syncytial virus (RSV) infection was subsequently confirmed. There was a three day history of coryzal symptoms and "snuffiness", for which a family doctor was consulted. The family had understood that the good weather in Florida would "do him good".

Although this infant's RSV infection might have resulted in apnoea, hypoxaemia, and hospitalisation anyway, it seems likely that lower oxygen pressures in the aeroplane will have aggravated the symptoms. For this family the Christmas period was spent in a paediatric ward in Manchester and not in a holiday resort in Florida. Although we have no information from the airline, we assume that for the emergency landing the plane would have to empty its tanks, filled for a transatlantic distance, in order to achieve a safe landing weight. We presume these tanks will have been emptied over the Irish Sea. In addition to the potential harmful episode to the child and the inconvenience for the family, this infant's flight probably also caused significant environmental damage.

We accept the contention of Ward Platt *et al* that any danger from air travel must be very small,<sup>4</sup> but that may not be so for infants who are unwell, and some evidence based guidelines on this subject might be helpful. In the meantime we wonder if we should regard suspicion of bronchiolitis as reason to advise against flying.

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- 1 Parkins KJ, Poets CF, O'Brian LM, Stebbens VA, Southall DP. Effect of exposure to 15% oxygen on breathing patterns and oxygen saturation in infants: interventional study (with commentaries by J Savulescu and V Hughes and authors' reply). *BMJ* 1998; **316**:887-94.
- 2 Savulescu J. Public must be warned of weak evidence for serious harm. *BMJ* 1998; **317**:677.
- 3 James PB. Risks associated with hypoxia during flights need to be investigated. *BMJ* 1998; **317**:677.
- 4 Ward Platt MP, Fleming PJ, Blair PS, *et al*. Danger to babies from air travel must be small. *BMJ* 1998; **317**:676.

### Recommendations for using MMR vaccine in children allergic to egg should be consistent

EDITOR.—Two reviews of measles, mumps, and rubella (MMR) vaccine and egg allergy have recently been published. One appears in the Royal College of Paediatrics and Child Health's own journal (*Archives of Diseases in Childhood*),<sup>1</sup> the other has been endorsed by the Committee on Infection and Immunisation of the Royal College of Paediatrics and Child Health.<sup>2</sup> The two articles differ in their recommendations of which children should be given MMR under supervision in hospital. Which of these expert opinions should paediatricians and general practitioners follow? Were the authors of the two articles aware of each others' conclusions? Could the editorial boards of the two journals (which have members common to both) not have informed the authors?

These recommendations also differ from Department of Health advice,<sup>3</sup> which also differs from that given by the Health Education Authority.<sup>4</sup> This debate might be settled if a consensus can be agreed and published in the next edition of *Immunisation against infectious disease*.<sup>3</sup>

In the mean time a pragmatic approach is needed. That is to offer MMR under supervision in hospital to children who have had a severe allergic reaction to egg and to children whose general practitioners, practice nurses, or parents are unhappy for them to be given MMR elsewhere.

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- 1 Lakshman R, Finn A. MMR vaccine and allergy. *Arch Dis Child* 2000; **82**:93-5.
- 2 Khakoo GA, Lack G. Recommendations for using MMR vaccine in children allergic to eggs. *BMJ* 2000; **320**:929-32.
- 3 Salisbury DM, Begg NT, eds. *Immunisation against infectious disease*. London: HMSO, 1996; **38**:141.
- 4 Health Education Authority. *MMR immunisation factsheet*. London: Department of Health, 1997:5-6.

*Dr Lakshman and Dr Finn comment:*

We note Riordan's response to our editorial on the issue of MMR vaccine and allergy<sup>1</sup> and the recommendations put forward by Khakoo and Lack<sup>2</sup> on this topic. While we agree that conflicting advice creates confusion, we cannot agree with his proposed "pragmatic approach". This amounts to a pointless waste of time and resources—greater than that proposed by anyone else to date—which will simply stoke up unfounded concerns about this vaccine, while diverting people from the important necessity to prepare themselves to tackle cases of severe anaphylaxis which, on the rare occasions that they occur, will continue to do so in community clinic settings.

- 1 Lakshman R, Finn A. MMR vaccine and allergy. *Arch Dis Child* 2000; **82**:93-5.
- 2 Khakoo GA, Lack G. Recommendations for using MMR vaccine in children allergic to eggs. *BMJ* 2000; **320**:929-32.

*Dr Marcovitch, Editor in Chief of Archives of Disease in Childhood, comments:*

Dr Riordan asks which expert opinion to follow. The answer surely lies in reading the papers carefully, seeking out any key references quoted, and deciding for oneself who has provided the best evidence. This should be the case for all guidelines, but we know that they are often absorbed undigested, which is one reason why *ADC* erects fairly firm barriers to their publication. Lakshman and Finn's paper was commissioned by the editors as a leading article because, as practising paediatricians, we recognised that all of us have problems responding logically to requests to immunise children in hospital.

When we commissioned this paper we did not know that a college committee was embarking on an enquiry; we learned this only after our leading article had been peer reviewed and was set up for publication. Editors of *ADC* have long been saddened that many of our readers, including members and fellows of the RCPCH, prefer first to

submit their papers elsewhere; we realise, of course, that the artificial constraints of the research assessment exercise result in some authors needing to collect Brownie points by publishing in journals with a higher impact factor, even if their research thereby reaches an inappropriate readership. In this sense, the *BMJ* is our competitor, not our partner, which is why editors do not tell each other what they have in the pipeline.

I realise that this cannot have been the case in this instance as the *BMJ* copied Khakoo and Lack's paper from the specialist journal in which it originally appeared (which probably has a lower score than *ADC* and is read by far fewer paediatricians).

Dr Riordan suggests seeking a consensus. Far better would be to undertake a full literature search of RCTs and subject it to a systematic review. The days of guidelines by GOBSAT (grand old boys sitting at table) are over. At this year's annual scientific meeting of the RCPCH, the journal and the college's quality of practice committee have forged a working relationship that should leave our readers less confused in future.

### Sputum induction for the diagnosis of pulmonary tuberculosis

EDITOR.—We read with interest the study of Zar *et al* on the usefulness of sputum induction in infants and young children for the diagnosis of pulmonary tuberculosis.<sup>1</sup> Bacteriological confirmation of pulmonary tuberculosis in infants and young children remains a problem because it is difficult to obtain sputum. Therefore, in young children, gastric lavage is the recommended method for the collection of respiratory secretions.<sup>2</sup> Since the number of tubercle bacilli and the frequency of positive cultures in specimens recovered by gastric lavage are usually small, gastric washings are ideally performed on three consecutive mornings to maximise the yield.<sup>3</sup>

In this prospective study, children with acute pneumonia with a high risk of pulmonary tuberculosis were included. On 142 children both gastric lavage and sputum induction was performed. The yield of *M tuberculosis* in sputum and gastric lavage was compared, as was the amount of positive cultures in sputum and gastric lavage. The influence of HIV status on the yield was also determined.

The authors found more positive cultures in the induced sputa compared to the gastric lavages. Therefore they conclude that sputum induction was a more sensitive method than gastric lavage for culture of *M tuberculosis*. However, in order to compare the sensitivity of two diagnostic tests, one should perform both tests in all patients. In this study, 39 patients underwent only one gastric lavage, 77 patients had lavages on two consecutive mornings, and only 26 patients underwent all three gastric lavages.

We therefore disagree with the authors on one of the conclusions, that induced sputum is better than gastric lavage for the isolation of *M tuberculosis* in infants and children. In our opinion, in order to answer the question whether sputum induction is as good as or better than gastric lavage, only the results

from the patients who underwent gastric lavages on three consecutive days should be used.

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- Zar HJ, Tannebaum E, Apolles P, Roux P, Hanslo D, Hussey G. Sputum induction for the diagnosis of pulmonary tuberculosis in infants and young children in an urban setting in South Africa. *Arch Dis Child* 2000;82:305-8.
- Abadco DL, Steiner P. Gastric lavage is better than bronchoalveolar lavage for isolation of Mycobacterium tuberculosis in childhood pulmonary tuberculosis. *Pediatr Infect Dis J* 1992;11:735-8.
- Chernick V, Boat TF. Kendig's disorders of the respiratory tract in children. Philadelphia: WB Saunders Company, 1998;893-4.

*Drs Zar, Tannenbaum, Apolles, Hanslo, and Hussey comment:*

Dr Wiersma and colleagues suggest that the yield from a single sputum induction should be compared only with the results of those children who had three consecutive gastric lavages. Only 26 of our patients had three gastric lavages; among this subset however, four children were culture positive on sputum while only three were positive on gastric lavage.

Although the yield from gastric lavage is improved with increasing number of specimens, it is frequently not feasible to perform this procedure on three consecutive days, particularly in developing countries with limited resources. Moreover, performing three repeated gastric lavages may be very unpleasant, both to the child and the health worker. In practice, even in tertiary institutions such as those in which our study was performed, obtaining three sequential gastric lavages is rarely feasible.

The yield from sputum induction may also be increased with increasing number of specimens.<sup>1</sup> Therefore we would submit that the yield from consecutive gastric lavages should be compared with that of repeated induced sputa. Data from studies of adult patients using paired specimens of induced sputum and gastric aspirates have reported a higher yield from sputa specimens.<sup>2-4</sup> In our study, the findings that a single induced sputum specimen yielded *M tuberculosis* more frequently than repeated gastric lavages (in the majority of children) further strengthens our conclusion.

- Finch D, Beatty CD. The utility of a single sputum specimen in the diagnosis of tuberculosis—comparison between HIV-infected and non-HIV-infected patients. *Chest* 1997;111:1174-9.
- Elliott RC, Reichel J. The efficacy of sputum specimens obtained by nebulization versus gastric aspirates in the bacteriological diagnosis of pulmonary tuberculosis. *Am Rev Resp Dis* 1963;88:223-7.
- Jones FL. The relative efficacy of spontaneous sputa, aerosol-induced sputa and gastric aspirates in the bacteriological diagnosis of pulmonary tuberculosis. *Dis Chest* 1966;50:403-8.
- Lillehei JP. Sputum induction with heated aerosol inhalations for the diagnosis of tuberculosis. *Am Rev Resp Dis* 1961;84:276-8.

## BOOK REVIEWS

**The new children's hospital handbook 1999.** Edited by Kilham H, Isaacs D. (Pp 409, paperback; \$44.95) Sydney: New Children's Hospital, 1999. ISBN 0 9587 167 1 4

Any publication dropping onto the doormat of a paediatrician in Manchester at present that has a front cover showing a brand new children's hospital under a crystal clear blue sky had better be good if it hopes to receive an even handed review. Having resisted the initial desire to emigrate, I settled down to investigate whether the contents of the text matched the glossy cover. My first impulse for purchasing a book is based on the initial impression gained from a quick flick through the pages. So far so good. This handbook has a concise list of contents, well structured chapters covering the usual general paediatric topics, as well as eye catching sections on infant feeding and nutrition, pain management, the adolescent patient, and psychiatry. However, the ultimate test for any book that claims to be "a useful and practical guide for the management of sick children" is whether it proves to be just that. Many authors have claimed the above, but have produced texts that are too brief to be clinically useful, or in too much depth to provide clear and direct advice in times of trouble.

I can, without reservation, say that this publication definitely delivers. The details on clinical features, investigation, and subsequent treatment are pitched at just the right level to make it eminently useful. It enables you to confidently handle the vomiting diabetic child, develop a logical approach to the prescription of antibiotics in the pyrexial child, as well as manage less common problems such as febrile neutropenia and acute adrenal insufficiency. The chapters on fluid management, endocrinology, and infection are worth particular praise. There are colour coded pages for the most important information, and drug doses are only included when essential or relevant. Any criticisms I have are minor, but would include a rather too brief chapter on cardiology and the inclusion of a section on neonatology that might have been better left to a more specialist text.

I would definitely buy this book for myself, as well as recommending it to colleagues, both junior and more senior. It has the potential to become a valued member of any acute department and I suspect it will secure a well deserved corner in the handbook market.

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**Cerebral palsies: epidemiology and causal pathways.** Stanley F, Blair E, Alberman E. (Pp 251, hardback; £37.50) Cambridge: Cambridge University Press, 2000. ISBN 1 898 68320 4

Descartes, the father of modern philosophy, in his pivotal work *Discourse on method*, published in 1637, set himself the task of doubting everything. From that starting point, he then tried to find a solid base of certainty. Over the years, epidemiologists have attempted to follow the Cartesian approach, not least those epidemiologists who are interested in that heterogeneous group of conditions called the "cerebral palsies". To doubt everything requires imagination and courage, a setting aside of traditional wisdom; to establish certainties requires scientific rigour, clear thinking and sheer hard work.

For years, the conventional wisdom has been that most children with cerebral palsy have suffered brain damage at birth, which has led to a movement disorder of varying severity with or without additional intellectual and sensory deficit. This view has been challenged from time to time, but in the last 15 years, the dissenting voices have been loud and persistent. There is now a wide acceptance that not more than 10% of cerebral palsy in term babies is due to adverse intrapartum events. This recognition has opened the way to thinking about the alternative and infinitely more complex pathways that lead to this clinical entity. The problem now is to define these, to unravel the sequences of events, their temporal order, their interdependence, and their relative importance.

The authors of *Cerebral palsies: epidemiology and causal pathways* have taken up this challenge. In a systematic, lucid, way, they give the current data on cerebral palsy frequency, the current thinking on risk factors, and present for us a series of hypothetical causal pathways, most of which have an appealing biological plausibility. The authors are to be congratulated for their imagination and clear thinking. It is an elegantly written book, a landmark in the ongoing saga of the epidemiology of the cerebral palsies. In turn, they have thrown down a challenge for us—to test these possible pathways using sound methodological approaches, some of which, I suspect, have yet to be developed. The research agenda in this field appears to have been set for a number of years to come.

But perhaps the most exciting prospect is that this painstaking epidemiological work will be much enhanced by the advances in two rapidly developing fields. First, neuroimaging techniques now provide a powerful tool for assessing the timing and the structural and metabolic changes in brain injury. Secondly, there is an increased understanding of the complex biochemical changes that make up the maternal, fetal, and neonatal response to infection or hypoxia, and the genetic factors which modify this response. This opens up exciting new preventive and treatment possibilities. The next edition of this book will almost certainly need to draw on a wide multidisciplinary expertise in order to encompass these major advances in our understanding of the pathophysiology of the cerebral palsies.

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