SARS is caused by a coronavirus, does this result of a novel coronavirus infection. If diagnostic test becomes available, we do not know and we are impatiently waiting for, because once the test becomes available, our lives can hopefully change back to normal again, and fear of the unknown (that is, can children be healthy carriers of the SARS virus?) will be considerably lessened.

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Childhood SARS in Singapore

Since SARS (severe acute respiratory syndrome) came to Singapore, our lives have changed considerably. Fear has taken over, resulting in irrational handling and panic reactions by some, while others reacted as non-believers. It is the human response to the unknown, and the fear for the future.

In our Department of Paediatrics, as well as other departments at the National University Hospital, extensive measures have been taken (the “no risk” policy), trying to prevent further spread of the disease. Apart from extensive anti-infectious measures (gloves, masks, changing clothes, isolation procedures, etc.) we divided our medical staff into two teams, who are not allowed to have any direct contact with each other, switching every two weeks to take over patient care. This would allow us, if SARS started spreading, to save at least half of our medical staff (as it seems the incubation period of SARS is within two weeks). Furthermore, parents and children avoid coming to the hospital as far as possible, resulting in empty waiting rooms and outpatient clinics. All teaching activities and research projects have been stopped for several weeks.

On the other hand, and in contrast with our colleagues in Hong Kong, we have not been able to identify any case of SARS in a child at the National University Hospital, although extensive and detailed search has become a routine procedure for all of us, especially in those children with fever and/or respiratory symptoms. Does this mean that SARS is mainly an adult and adolescent disease in Singapore? It seems to be, although no validated diagnostic test of SARS has so far become available, and diagnosis is merely based on history and clinical picture (that is, a diffuse pneumonia with fever, progressing rapidly to acute respiratory distress syndrome). Or can it be that SARS in children has a different clinical picture, including a less severe disease or an atypical presentation (that is, acute diarrhoea) or even an asymptomatic carrier state? We do not know and we will only know when a sensitive and specific diagnostic test becomes available.

There is now evidence that SARS could be the result of a novel coronavirus infection. If SARS is caused by a coronavirus, does this mean that children can be the source of SARS? Again, we do not know, but it has been shown that other coronaviruses can be detected in children. However, the question remains as to why SARS affects mainly adults (at least in Singapore). Several explanations are possible. Moreover, other infections are known to cause severe symptoms in adults, such as Varicella, Mycoplasma pneumoniae, or Chlamydia pneumoniae. Could it be that SARS is like a kind of “booster” to these infections, needing previous contacts (and immunological priming), or does it need some co-infection (possibly Chlamydia pneumoniae), or a specific immunological condition of its host? Another reason may be that children have limited contact with the virus, as most SARS patients are infected within the hospitals (85%), and children visit hospitals less than adults, especially since the outbreak. The latter explanation however seems unlikely, as a number of SARS patients, with children of their own, spread the disease to adult members in their family, without affecting their children.

A lot of work still needs to be done on childhood SARS, and research can only start meaningfully once an extensively validated diagnostic test becomes available. This is what we are impatiently waiting for, because once the test becomes available, our lives can hopefully change back to normal again, and fear of the unknown (that is, can children be healthy carriers of the SARS virus?) will be considerably lessened.

Reference

Food allergy in childhood

We were surprised by some of the content of Clark and Ewan’s paper1 and their interpretation of our data. As death certification of allergy deaths is problematic, we collected reports through the prospective BPSU mechanism and other sources as listed in our article. No additional deaths in the study period have been brought to our attention since publication. Our finding of no deaths due to peanut under the age of 13 is supported rather than refuted by the study of Bock,2 cited by Clark and Ewan. In Bock’s study, which looked at all ages, there were 10 childhood deaths (compared with 8 in ours) and they found no deaths due to peanut allergy under the age of 12. The death of a child aged two years was due to brazil nut, not peanut.

Clark and Ewan were concerned that we did not put sufficient weight on respiratory presentations. Of the 55 non-fatal severe cases, 30 had only upper or lower airway problems. Of the 173 children admitted to hospital not classified by us as severe, 60 had an upper or lower airway presentation for which adrenaline or bronchodilator or both were administered. Even if all these are added to our severe group, the rate of non-fatal severe events only changes from 0.2 to 0.4 per 100 000 children per year.

Allergic deaths indistinguishable from asthma are potentially important but if a child’s symptoms are only asthmatic and no allergen is suspected, then there is no means for attributing such reactions to food or for knowing if a causal link exists. The possibility of an unrecognised link is a key area for further research.

It is misleading to state that the mechanisms of the British Paediatric Surveillance Unit will miss children referred to allergy centres not run by paediatricians. We studied hospital admissions, not outpatient referrals, and all such children should be admitted under the care of paediatricians. The effectiveness of our reporting mechanisms is supported rather than refuted by the study of Alves and Sheikh,3 cited by Clark and Ewan. Alves and Sheikh report all hospital admissions for anaphylaxis in children aged 0–15 years over a four year period in England and Wales. There were 60 for food allergy and a further 240 with no code caused. If, as Clark and Ewan suggest, 94% of the uncoded cases were due to food then there were 288 admissions from 10.6 million children (denominator from Office of National Statistics, personal communication) over four years or 0.68 admissions per 100 000 children per year. This is comparable to our reported 231 admissions over 2 years in a population of 13 million, equivalent to 0.89 admissions per 100 000 children per year.

The inclusion of Clark and Ewan’s own data in their table 1 is inappropriate. Two concerns are that their data are extrapolations and use a completely different definition of severity. But more importantly, their figures are numbers of children with severe allergy per 100 000 child population; whereas ours, and those of Alves,3 are numbers of severe reactions per year per 100 000 child population. Clark and Ewan therefore confuse prevalence of severely allergic children with incidence of severe allergic reactions. Such data cannot be tabulated against each other and their suggestion that our figures are a 62-fold underestimate is therefore without meaning.

We all want to know which children should have an adrenaline auto-injector and what the indications should be for administering it. However in seeking such knowledge, we must be careful not to extrapolate from adult studies. In Clark and Ewan’s own study,4 the high percentage (70%) of children in the study is stated but the median age of a moderate or severe reaction on follow up was 18 years and the three severe cases referred to in which adrenaline was used were actually aged between 27 and 41 years.

We agree with Clark and Ewan’s assertion that problems over the decision to prescribe adrenaline “highlight the need for good data on the clinical features and natural history of nut allergy in children” but would add that studying the epidemiology is also fundamental.

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I read Shaffer’s paper with great interest, as I am concerned in particular with the study of the respiratory outcome of children of very low birthweight reported recently by Anand and colleagues. They quote the results of a similar study of younger children, which we reported some years ago in this journal. Interestingly, the results were similar, suggesting that the functional outcome for low birthweight children was independent of respiratory support in the neonatal period, and independent of UGR (“small for dates” in our rather old fashioned terminology). We found a highly significant linear relationship between the FEV0.75 at the age of 7, and birthweight. Anand and colleagues quote our study as showing “significant differences between those who received ventilatory support and those who did not”. This contradicts even a selected reading of the final line of our abstract, which stated that “the absence of an association between neonatal oxygen score or mechanical ventilation and childhood lung function suggests that the long term effect of neonatal respiratory treatment is small compared with that of low birthweight...”. It is gratifying that the two studies come to similar conclusions, but disappointing to be utterly misquoted.

References


Lung function and birth weight

At the risk of being accused of raising a trivial issue, I would simply like to ask authors to quote accurately from references within their articles. I am concerned in particular with the study of the respiratory outcome of children of very low birthweight reported recently by Anand and colleagues. They quote the results of a similar study of younger children, which we reported some years ago in this journal. Interestingly, the results were similar, suggesting that the functional outcome for low birthweight children was independent of respiratory support in the neonatal period, and independent of UGR (“small for dates” in our rather old fashioned terminology). We found a highly significant linear relationship between the FEV0.75 at the age of 7, and birthweight. Anand and colleagues quote our study as showing “significant differences between those who received ventilatory support and those who did not”. This contradicts even a selected reading of the final line of our abstract, which stated that “the absence of an association between neonatal oxygen score or mechanical ventilation and childhood lung function suggests that the long term effect of neonatal respiratory treatment is small compared with that of low birthweight...”. It is gratifying that the two studies come to similar conclusions, but disappointing to be utterly misquoted.

CT radiation doses

The hazards of computed tomography (CT) in terms of radiation, are being increasingly recognised. CT scanning, despite representing less than 5% of the total number of x-ray procedures performed, contributes approximately 40% of the total collective radiation dose to the UK population from all medical x-ray examinations. It is well known that children are more radiosensitive than adults—for example, a 1 year old infant is 10–15 times more likely than a 50 year old adult to develop a malignancy from the same dose of radiation. In addition, for a given dose, the effective (radiation) dose is larger in a small infant than in an adult—that is, the effective dose increases as age decreases.

The message is slowing filtering through to the radiology community, but paediatricians should be aware as well. CT is a high dose radiation technique—if a CT study is performed, contributes approximately 40% of the total collective radiation dose to the UK population from all medical x-ray examinations. It is well known that children are more radiosensitive than adults—for example, a 1 year old infant is 10–15 times more likely than a 50 year old adult to develop a malignancy from the same dose of radiation. In addition, for a given dose, the effective (radiation) dose is larger in a small infant than in an adult—that is, the effective dose increases as age decreases. It has been alleged that, “the radiation risk attributable to the radiation exposure from a single abdominal CT in a 1 year old child is in the order of one in a thousand”.

CT scanning, therefore, is a potentially dangerous modality, and when used in children in particular, the dose should be kept as low as reasonably achievable (the so-called ALARA principle).

A recent images in Paediatrics case contained one important piece of CT information in an otherwise excellent CT image of a lipoblastoma (admittedly far more immediately dangerous than a CT study). The CT examination of the lower chest was done in a 6 month old baby using 250 mA (mA = milliamperage, which is the main determinant of CT dose). Unless the authors have an extremely old scanner, this is an adult setting and is much too high for a child. A perfectly good diagnostic study can be done in an infant of this age with an mA of 50, which is one fifth of the dose. In fact, an adequate study might even be achievable with an even lower mA, but the CT scanner manufacturers have been slow to facilitate such low dose techniques, ignoring the needs of children. Using excessive adult CT doses in children is, in my experience, an unfortunately common error in the UK. This has also been noted in the USA. The message is slowly filtering through to the radiology community, but paediatricians should be aware as well. CT is a high dose radiation technique—if a CT study is truly justified in a child then weight (not age) adapted paediatric parameters should be used, which can give adequate diagnostic information with minimised radiation risk.

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References


Running around in circles following ischemic stroke

I read Shaffer et al’s paper with great interest, but it gave a strong sense of déjà vu. The authors and ADC readers may find of interest Sigmund Freud’s comments on this particular topic from his classic Infantile Cerebral Paralysis published in 1897: “The consideration of cranial trauma as another etiological factor defeats all attempts to view hemiplegic cerebral paralysis as a single etiological entity.”

He then cites over 20 cases from the medical literature at that time from Osler, Abercrombie, and Henoch amongst others. He adds: “In a number of cases the role of trauma as an etiological factor of infantile cerebral palsy is indisputable. It would be important for the principal concept of infantile cerebral paralysis, if in definitely traumatic cases there would be specific deviations in the disease from the non-traumatic ones in regard to course or complications. No special study has been undertaken so far in this regard.”

One century after Freud this study has still not been undertaken and thus it is questionable as far as I am concerned with the study of the respiratory outcome of children of very low birthweight reported recently by Anand and colleagues. They quote the results of a similar study of younger children, which we reported some years ago in this journal. Interestingly, the results were similar, suggesting that the functional outcome for low birthweight children was independent of respiratory support in the neonatal period, and independent of UGR (“small for dates” in our rather old fashioned terminology). We found a highly significant linear relationship between the FEV0.75 at the age of 7, and birthweight. Anand and colleagues quote our study as showing “significant differences between those who received ventilatory support and those who did not”. This contradicts even a selected reading of the final line of our abstract, which stated that “the absence of an association between neonatal oxygen score or mechanical ventilation and childhood lung function suggests that the long term effect of neonatal respiratory treatment is small compared with that of low birthweight...”. It is gratifying that the two studies come to similar conclusions, but disappointing to be utterly misquoted.

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References

Oral clodronate as treatment of osteogenesis imperfecta

The benefits of treatment with intravenous pamidronate in osteogenesis imperfecta (OI) have recently been reported.1–4 These include reduced bone resorption, increased bone density, and improved clinical outcomes as judged by apparently lower fracture rates. We would like to report a single case of OI treated by the orally administered bisphosphonate, clodronate, with good effect.

A boy, whose mother was affected with clinically diagnosed type 1 osteogenesis imperfecta, was referred to our unit aged 13 years with a recent onset of severe back pain that had required hospital admission. He appeared of normal stature with blue sclerae and was able to walk independently. He had sustained four previous limb fractures; lateral radiographs of the thoracic and lumbar spine confirmed three vertebral wedge fractures. He was 158.9 cm tall (10th centile) and weighed 49 kg (25th centile). Lumbar spine bone mineral density scanning by dual x ray absorptiometry (DXA, Hologic QDR-1000, Hologic, Bedford, MA) revealed a BMD of 0.396 g/cm² (Z score −5.22, comparing his value to the average young man). Fasting urinary hydroxyproline/creatinine ratio, an index of bone resorption, was 96.6.

His height at that stage lay on the 50th centile (176 cm) and his weight on the 10th (58 kg). The BMD remained considerably below the normal value and clodronate was recommenced at a dose of 800 mg daily.

Eight years after initial referral, his bone mineral density had increased by 60.6% to 0.613 g/cm² (Z score −4.16). To compensate for the expected increase in bone size, the bone mineral adjusted density (BMAD) was computed (BMD(square root of area))/ and showed an improvement of 24.6% in BMAD over the duration of therapy. Clodronate was discontinued when the patient was aged 22. He had reached a height of 177.4 cm and a weight of 60.8 kg, and the spine BMD Z score was −3.92. He had suffered no atraumatic fractures since commencing oral clodronate.

The rationale in using bisphosphonates for osteogenesis imperfecta is the inhibition of osteoclastic bone resorption leading to increased bone density and a potentially lower risk of fracture. This young man exhibited a good response to therapy with oral clodronate, suffering no adverse reactions. The increase in height of 18 cm over eight years, moving him from the 10th to 50th centile, suggests that his growth was not impaired by therapy.5 Many studies have shown that clodronate does not impair mineralisation. We are unable to determine the contribution of remodelling of vertebral fractures to his height gain.

A limitation of this report is that pubertal status was not documented at presentation. However, the increases in lumbar spine density are in excess of the expected average rates in growing children (3–6% per year and 14–16% during puberty).6 The increase was also observed following adjustment for bone growth by BMAD and Z scores relating the measured BMD to age matched controls. The Z score (−5.22 to −3.92) improvement during therapy was not dissimilar to that reported with pamidronate7 in younger children.

We agree that there is increasing evidence of a role for bisphosphonate therapy as part of the multidisciplinary management of osteogenesis imperfecta. Oral clodronate in our patient appeared to elicit a similar response to that of cyclical intravenous pamidronate, suggesting that orally administered bisphosphonates may be of value in the management of this disease.

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References

Serum prolactin in coeliac schoolchildren

Literature published suggests that in children with coeliac disease (CD) serum prolactin concentrations are increased, and correlate with the grade of mucosal atrophy. It has been proposed that prolactin is a possible marker of disease activity.1 Other studies, however, have failed to show this correlation in children with CD.2

We studied prolactin levels in children with CD, and the correlation with the severity of intestinal mucosal atrophy.

We used samples from a serum bank obtained during a mass screening for CD in Sardinian schoolchildren, using both antinemysial antibodies and antigliadin antibodies as screening tests, as previously described.3 The sample included 20 children with CD on a gluten containing diet (16 girls, 4 boys, mean age 12.9 years, range 11.5–14.4 years) and 40 sex and age matched normal children (32 girls, 8 boys, mean age 13.0 years, range 11.2–14.8 years). All subjects were euthyroid. Prolactin was assayed in duplicate using a commercial immunoradiometric method; results were analysed by analysis of covariance.

Data are expressed as mean (SE). Prolactin levels were 4.62 (2.1) ng/ml in patients with CD and 5.90 (2.6) ng/ml in controls (no statistically significant difference). No correlation was found between prolactin concentrations and the degree of intestinal damage (Marsh criteria).

Our study did not confirm the increased prolactin concentrations in children with CD reported by Reifen and colleagues.4 Our population differed somewhat in that there was a higher mean age (12.9 v 11.3 years), a narrower age range (11.5–14.4 v 15–18 years), and a different girl:boy ratio (4:1 v 1:1). Furthermore, our study included three potential coeliac subjects (subjects with antienzymosium antibodies positivity but normal intestinal biopsy5) and 11 asymptomatic coeliac children. The hypothesis that the normal prolactin values observed in our study may be due at least in part to the different clinical characteristics of the population studied is plausible, but its validation requires a specifically designed study.
Eating Problems in Children: Information for Parents.

Edited by C Fox, C Joughin. London: Gaskell, 2002; £10.00, pp 621. ISBN 1901242862

How commonly do we encounter the following scenario?

A desperately anxious mother at last convinces her GP that she needs to see a paediatrician because her normally growing toddler is eating nothing. The paediatrician wonders why his time is being wasted, and “reassures” the mother that there is nothing to worry about. Needless to say the anxiety persists with, no doubt, damaging consequences. As a professional, we have to handle these cases poorly. With 30% of preschool children suffering from mild to moderate eating problems, we need a better way to address these issues.

The Royal College of Psychiatrists has produced this small book for parents that should prove helpful, not only to parents but also to paediatricians and other health professionals. It provides information about the epidemiology of eating problems, and gives a useful classification, categorising eating difficulties into nine types, including persistence of eating inappropriate texture of food for age, food refusal, restrictive eating and selective eating. This allows the parent or professional to come to a more specific “diagnosis”, and also a sense of the anticipated course these difficulties are likely to take. In particular, it provides clear warning for those conditions that are indicative of significant emotional or psychiatric conditions.

Giving clear indications to the parent as to when to worry is helpful, as it is likely to encourage a sense of reassurance. The anxiety accompanying the more common eating difficulties the book goes on to provide specific and sensible advice about the practical management for each of the different types of eating difficulty.

At the end of the day, one is left with the finding that for most parents, not surprisingly, reassurance is what is required. I felt, however, that this book could help us proffer the advice in a more substantive form than we do at present, and can give us an approach that is likely to help diffuse the anxiety which contributes to the perpetuation of stressful mealtimes. I suspect the book will prove to be of most value to health visitors, but selected paediatricians who think they can read this book from start to finish and become initiated into the mysteries of metabolic disease are likely to be overwhelmed.

If you can peruse this paperback at your local medical bookshop, do so: you may find it an answer to prayer, and anyway, at just under £30 it won’t bankrupt anyone.

R A F Bell

Children’s Headache


Headaches in children are a common problem—70% of school children have headaches at least once a year, with 25% suffering from recurrent headaches. This book is part of the Clinics in Developmental Medicine series, and provides a comprehensive overview of the subject. The book is divided into clear chapters, which makes it easy to dip into. It includes interesting sections on pain perception in children and neonates, as well as a good epidemiology section. Throughout the book there are summary tables of recently published studies. In the later chapters there are case histories, including parental descriptions, which break up the occasionally slightly long winded text. There is an extensive list of references at the end of each chapter.

I found the chapters on migraine enlightening, especially the theories on pathophysiology of migraine. The diagnostic criteria for migraine are easy to read and clear. There is an excellent overview on the psychological treatment of headaches, regardless of diagnostic type. Again, the evidence is summarised in clear tables. There is a practical section on managing abdominal migraine. Causes of headaches are divided into separate chapters for specific and rare causes, which was helpful when I used the text when on call.

The final chapter talks about setting up a headache clinic, including a discussion on diagnostic tests. There is a headache questionnaire for parents, which I would find very helpful. There is also advice on the role of the multidisciplinary team in management.

This book would be a valuable addition to a general paediatric department, both in outpatients and for reference when on call.

A Marjaria

CORRECTIONS

In the article by Nixon et al (Arch Dis Child 2002;87:306–11), Dr Claire Wainwright should have been included as an author. Dr Wainwright’s contribution was the establishment of the methodology and early patient recruitment and testing. Dr Wainwright moved from The Royal Children’s Hospital at the end of 1997, and was funded by The Royal

BOOK REVIEWS

A Clinical Guide to Inherited Metabolic Diseases, 2nd edn

Edited by JTR Clarke UK: Cambridge University Press, 2002, £29.95, pp 306. ISBN 0521890764

Dr Clarke’s enthusiasm and erudition are evident on every page of this book, which is handsily sized, and, wonder of wonders, costs only £30. Most of the chapters are written with a clinical approach, and the chapters on basic principles in understanding inherited metabolic disease, neonatal screening, hypoglycaemia, metabolic acidosis, storage diseases, and dysmorphism will be read with a sensation of increasing revelation by just about any paediatrician, and those with a secure background in biochemistry and metabolic disease will pick up many nuggets of wisdom.

Why then, do I simply not recommend every paediatrician who sometimes deals with metabolic problems—and there must be few of us who do—not to rush out and buy a copy before such a gem either goes out of print or rises in price? My caveat is that this book’s clinical approach coupled with its encyclopaedic coverage of some topics means that several chapters leave the non-expert mentally breathless, and this effect is made worse by the absence of the structural formulae of the molecules named; this may be of little moment to those with these formulae at their fingertips, but that excludes rather a lot of us. By contrast the metabolic sections of “Nelson” and “Forfar and Arniel” clearly benefit from their inclusion.

So, for those on a unit dealing with many patients with inborn errors of metabolism, this book is invaluable, but general and trainee paediatricians who think they can read this book from start to finish and become initiated into the mysteries of metabolic disease are likely to be overwhelmed.

If you can peruse this paperback at your local medical bookshop, do so; you may find it an answer to prayer, and anyway, at just under £30 it won’t bankrupt anyone.

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www.archdischild.com

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References

Children's Hospital Foundation, Brisbane and the Cystic Fibrosis Research Inc, Queensland. The authors apologise for the omission.

The authors of the letter “Childhood SARS in Singapore” in the August issue (Arch Dis Child 2003;88:742) were written incorrectly. The authors' names should be P Van Bever, C P P Hia, S C Quek.

In the acknowledgements for the leading article by Duke et al (Arch Dis Child 2003;88:563–5), Dr Diana Silimperi should have been acknowledged as part of the Paediatric Quality Care Group. The authors apologise for the error.

An error occurred in the paper by Riordan M, Rylance G, Berry K in the November issue (Poisoning in children 1: General management. Arch Dis Child 2002;87:393–6). In Table 2, pupillary constriction associated with signs of increased sympathetic nervous system activity should read as mild pupillary dilation. Anticholinergic agents are likely to produce a more marked dilation. The authors apologise for the error.