

PostScript

LETTERS

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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 in 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.¹ 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 more severe symptoms in adults, such as *Varicella*, *Mycoplasma pneumoniae*, or *Chlamydia pneumoniae*. Could it be that SARS is like a kind of "booster" pneumonia, 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 more 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.

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Reference

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Food allergy in childhood

We were surprised by some of the content of Clark and Ewan's paper¹ and their interpretation of our data.²

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,³ 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,⁴ cited by Clark and Ewan. Alves and Sheik 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 cause coded. 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 our figures, and those of Alves,⁴ 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,³ 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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Running around in circles following ischemic stroke

I read Shaffer *et al*'s paper with great interest, but it gave a strong sense of *deja vu*.¹ 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 how far further on from Freud, Shaffer *et al*'s paper moves us forward.

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Lung function and low 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 about the study of the respiratory outcome of children of very low birthweight reported recently by Anand and colleagues.¹ They quote the results of a similar study of younger children, which we reported some years ago in this journal.² Interestingly, the results were similar, suggesting that the functional outcome for low birth weight children was independent of respiratory support in the neonatal period, and independent of IUGR ("small for dates" in our rather old fashioned terminology). We found a highly significant linear relationship between the FEV 0.75 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 birth weight...". It is gratifying that the two studies come to similar conclusions, but disappointing to be utterly misquoted.

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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.¹ 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.² In addition, for a given procedure, the effective (radiation) dose is larger in a small infant than in an adult—that is, the effective dose increases as age decreases.² It has been alleged that, "the lifetime mortality 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".² 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).³ The CT examination of the lower chest was done in a 6 month old baby using 250mA (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.⁴ 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 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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