References


5. Does cefotaxime eradicate nasopharyngeal carriage of *N meningitidis*. We enrolled 43 children admitted with an unequivocal clinical diagnosis of meningococcal sepsis into a study to determine whether cefotaxime eradicated nasopharyngeal carriage of *Neisseria meningitidis*. In 28 cases (70%) the diagnosis was confirmed by positive culture from blood, nose, throat, or skin scraping, detection of meningococcal DNA in blood by polymerase chain reaction, or convalescent meningococcal serology. All children were treated with intravenous cefotaxime for seven days. Nasopharyngeal and throat swabs were obtained on the day of admission in 42 of these children, and all children had swabs repeated every day until there were at least two negative swabs.

On admission, the throat and nasopharyngeal swabs were both positive for meningococcus in two patients; in another two patients, the nasopharyngeal swab was positive while the throat swab was negative. In the first two patients, the swabs became negative after 24 hours of treatment, and in one child it became negative after 48 hours. In these children and others in whom the swabs were negative from the day of admission, subsequent swabs remained negative.

Compared to a previous study that reported a nasopharyngeal carriage rate of 50% on admission and showed that the yield of meningococci in throat swabs was unaffected by prior administration of penicillin, the yield from throat and nose swabs in this study (9.5%) was lower. This may reflect the fact that in practice many of these swabs were taken after the child had been given the first dose of cefotaxime.

The study population had a prevalence of physical abuse of 40%, much higher than the general paediatric population. Since test performance—that is, LR—is not independent of the pre-test probability, the LRs generated by a study done on this population cannot necessarily be used in a population with a much lower prevalence of abuse, as the authors have done in table 4. Since spectrum bias tends to reduce test performance as the pre-test probability falls, the LR for any given score threshold would be smaller than that quoted when applied to a population with a lower prevalence of physical abuse.

As most settings would expect to have a lower prevalence of physical abuse than the study, this reduces the value of the proposed scoring system as a clinical tool.

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5. Does cefotaxime eradicate nasopharyngeal carriage of *N meningitidis*. We enrolled 43 children admitted with an unequivocal clinical diagnosis of meningococcal sepsis into a study to determine whether cefotaxime eradicated nasopharyngeal carriage of *Neisseria meningitidis*. In 28 cases (70%) the diagnosis was confirmed by positive culture from blood, nose, throat, or skin scraping, detection of meningococcal DNA in blood by polymerase chain reaction, or convalescent meningococcal serology. All children were treated with intravenous cefotaxime for seven days. Nasopharyngeal and throat swabs were obtained on the day of admission in 42 of these children, and all children had swabs repeated every day until there were at least two negative swabs.

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Compared to a previous study that reported a nasopharyngeal carriage rate of 50% on admission and showed that the yield of meningococci in throat swabs was unaffected by prior administration of penicillin, the yield from throat and nose swabs in this study (9.5%) was lower. This may reflect the fact that in practice many of these swabs were taken after the child had been given the first dose of cefotaxime. The study suggests that cefotaxime, like a mixture of rifampicin and ceftriaxone,2 can eradicate meningococcal carriage. This is the first description of *Pneumocystis carinii* pneumonia presenting in the first three months of life and is an infection typically seen in patients with significant T cell immunodeficiencies and X linked hyper IgM. These were excluded by normal T cell numbers and function and by normal CD40 ligand expression and mutation analysis. There are reports of *Pneumocystis carinii* pneumonia in immunocompetent infants5 and agammaglobulinaemia.2 This is the first description of *Pneumocystis carinii* pneumonia in a patient with THI.

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Procalcitonin as a prognostic marker in children with meningococcal septic shock

Carrol and coworkers confirm the findings from Karabocoglu et al who reported that procalcitonin (PCT) was higher in children with severe meningococcaemia (fever, petechiae and purpuric rash, and hypodynamic instability) than in children with systemic meningococcal infection without shock (291.29 ± 167 μg/l ± 19.7 ± 23 ng/ml; p<0.001). Unfortunately, information is lacking in the report of Carrol et al., namely: a clear definition of severe MCD (defined in their paper as a Glasgow Meningococcal Septicaemia Prognostic Score ≥8) and median PCT values (median for the first 24 hrs: 90 ml/kg; Q1–Q3: 48–120) and cefotaximolamine infusion) admitted to our PICU between July 1999 and May 2002. We estimated the accuracy in predicting death of PCT, C reactive protein (CRP), naphelophony on admission, and the Pediatric Risk of Mortality (PRISM) score within 24 hrs of admission or at the time of death. Sensitivity, specificity, positive and negative predictive values, and percentage of well classified children were calculated at the following cutoff values: PCT >130 ng/ml (the best cutoff value) and CRP >100 mg/l (the best cutoff value); PRISM value >20 and PRISM probability of death >50 %.

In our study, PCT on admission was as accurate as the PRISM value and PRISM probability of death calculated within 24 hrs of admission or at the time of death, and more accurate than the CRP level in classifying survivors and nonsurvivors of MCD. These results accord with those of Hatherill et al who observed, in 37 children with MCD, that admission PCT levels (values not indicated) was higher in nonsurvivors (11%) than in survivors (p=0.04) and related to the severity of organ failure (p=0.02); however, in the whole group of children with septic shock whatever the causative organism, admission PCT functioned worse than the PRISM score.

The PRISM score functioned worse than the PRISM score because of underreporting of cases. Based on a well characterised population in Olmstead county Minnesota and extrapolating the data to a US population of 280 million, it may be estimated that there are 200 deaths from anaphylaxis reactions to foods each year.

Incidence of severe and fatal reactions to foods

Although the article by Macdougall et al regarding the incidence of severe and fatal reactions to food would seem to be reassuring, we would like to express some concerns and raise some questions about the data presented. The first question is whether the ascertainment of cases is really as complete as the authors suggest. We acknowledge that the UK medical system may miss a significant number of cases? Based on a well characterised group of children with severe infectious purpura: a comparison with severe infectious purpura in children. Clin Immunol 1998;101:552–3.

### Table 1

| Performance characteristics of PCT, CRP, and PRISM score in 35 children with MSS |
|-----------------|-----------------|-----------------|-----------------|
| Severity index (%) | PCT | CRP | PRISM value | PRISM probability |
| Sensitivity | 100 | 64 | 100 | 91 |
| Specificity | 63 | 46 | 63 | 83 |
| Positive predictive value | 100 | 46 | 100 | 83 |
| Negative predictive value | 100 | 46 | 100 | 83 |
| Well classified | 74 | 51 | 74 | 86 |

References

higher number of cases were reported from rural regions as compared to metropolitan areas strongly suggesting either misdiagnosis or inaccurate recording of cases in the emergency department log of busy hospitals. A second concern is the reporting of cases only up to age 15. In the paper mentioned above, of 32 fatalities 10 occurred in youngsters up to age 15. An additional 10 occurred in adolescents aged 16 to 19. Why did MacDougall et al not include all adolescents? A third question must always be raised when fatal food anaphylaxis is studied. Is it not possible that cases of fatal asthma were actually initiated by unidentified allergic reactions to food? All authors in this field are likely to agree that the ultimate cause of death may be irreversible airway obstruction, and all would agree that poorly controlled asthma increases the risk of fatal anaphylactic reactions to food, but we would suggest that the trigger responsible for individual asthma fatalities is not always determined. What about fatalities that never reach the emergency department and are misclassified on death certificates as asthma fatalities? Individuals that die at home and are classified as asthma deaths are unlikely to be further investigated or whether the US or the UK. Fourthly, the authors’ definition of severity seems incomplete. Individuals with severe food reactions who self administer epinephrine often do not go to hospital, are less likely to participate in surveys that require hospitalisation or cause death, and often they do not report these reactions to their physicians unless specifically queried. Some survive the reaction without treatment, become convinced that they avoided a specific food, and never tell their physician. We could argue about the possible progression of these episodes to near fatal or fatal reactions, but the point to be made is that they are frequently under reported. The fifth issue concerns the safe administration of epinephrine. We disagree about the risk to children of the administration of a single dose of epinephrine as opposed to withholding that dose. We have no disagreement about aggressive treatment of asthma concurrently, and in fact we think that administration of epinephrine is not always life saving even when and where it is needed. The issue of whether asthma deaths may have been precipitated by food allergy is an important question which we addressed “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”. Furthermore, such deaths will never have been reported in surveys of food allergy in other countries or in other age groups. No group has been able to address this question satisfactorily and it is a key area for further research.

We are not sure we agree that children, who have self administered epinephrine, often do not go to hospital. However we do not know the proportion and said as much, excluding this group from our definition of severity.

Finally we agree that education of professionals and the public should continue based on the best data available. This include those parents whose children are truly at high risk as well as those many parents that think any immediate hypersensitivity reaction to food means their child is at high risk of an allergic death; when in reality the risk, in the absence of asthma, seems so small, different parents will come to different views about how to proceed faced by a severe but very small risk, just as we all do in many aspects of our lives.

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Physiologic management of DKA

Inward and Chambers provide a provocative description and discussion of the continuing confusion regarding the issues surrounding rehydration and treatment of the pediatric patient with diabetic ketoacidosis (DKA). They review some of the key issues that link fluid therapy to complications from brain swelling, and question the appropriateness of using a volume of fluid calculated by “maintenance plus deficit”, calling for a second revolution in the management of DKA. In the accompanying commentary, Edge makes several statements concerning fluid therapy in DKA, including that “DKA is associated with severe fluid losses”, that “any guidelines for fluid and electrolyte management must be simple to calculate”, that administration of base is a risk factor for diarrhoeal complications, and that despite published guidelines and “changes in protocols”, there is no evidence that the “incidence of cerebral oedema has changed over the past 20 years”. It is our opinion that the problem in the rehydration of the pediatric patient with DKA...
physiologic management was first described since 1988 and 1990, and set forth with additional detail and data in 1994. It is rarely described in its complete form when reference to texts; mere portions of our recommendations are the what we have considered physiologic management. Not only is it unlikely that large numbers of patients outside our own institution have been managed using our guidelines in their entirety, but the recommendations simply are not old enough to be reflected in data over the past 20 years. We suspect that physiologic management is significantly underrepresented in the literature. We are aware of studies conducted thus far, all of which compare variations of standard therapy (empiric volume resuscitation) with or without serum bicarbonate. We note in our prospective study our retrospective portion of our study in 1990 we compared these therapies and found no form of traditional therapy minimised the risk of brain herniation during treatment.

Comments regarding the administration of insulin should be better defined. Rapid infusion or “pushes” of hypertonic sodium bicarbonate should not be given. On the other hand, there is no evidence that administration of physiologic concentrations of base in the rehydration fluids will cause either harmful or desirable. In our experience, this practice mitigates the development of hyperchloremic acidosis during treatment.

As ours is a referral centre, most of our patients have 70% of therapy initiated in outlying hospitals, sometimes in keeping with our recommended approach, and sometimes with our recommendations instituted only after involvement by our centre. In this setting, we have managed certain patients with severe DKA who received resuscitation fluids in excess of what their physical examination and laboratory data would dictate. It is not unusual for such patients to require as little as a third or the usual maintenance volume. Our approach has been criticised because of the incidence of mannitol administration in our series. In our mannitol recipients, several of whom did not receive their initial management by us, there was no central nervous system morbidity or mortality. In another large series of patients there was a 50% failure rate of mannitol to reverse a deteriorating neurologic status, even when mannitol was given before respiratory arrest, with a near 100% failure rate when mannitol was given after respiratory arrest.

It is possible that not all of our recommendations have been applied properly. As the authors of a research study that aimed to provide some evidence base for immunisation practice we would like to respond to this.

The Position Statement on Injection Technique

The Position Statement on Injection Technique (March 2002, Royal College of Paediatric and Child Health) discusses needle size and length for childhood immunisation. It concludes that there would seem to be insufficient evidence to advise any recommendation to change current practice in the use of hypodermic needles. As the authors of a research study that aimed to provide some evidence base for immunisation practice we would like to respond to this. Our study of 119 babies aged 4 months receiving their third dose of DTP/Hib vaccine found that significantly less redness and swelling occurred when infants were immunised using the longer 23 gauge 25mm (blue hub) needle rather than when the shorter 25 gauge (orange hub) needle was used. The magnitude of the reductions was substantial. The position statement is correct to note that in our study the difference in tenderness did not reach statistical significance. However we believe our study still
justifies a recommendation for the use of the longer needle for immunisation in 4 month old infants.

We believe the non-significant difference in tenderness with the different needles must be interpreted with caution, and should not be taken as a rationale for ignoring the significant benefits in terms of reduced redness and swelling. Tenderness was in fact reduced by the same relative amount as redness, but as tenderness occurred less frequently, the results were not formally statistically significant. We have used Bayesian analyses (using an “uninformative” prior distribution) to formally compute the chance that there is a clinically significant reduction (of at least 25% as specified in the protocol) in tenderness between the long and short needles. At six hours the probability of a clinically significant decrease in tenderness with the longer needle is 73%, whereas the chance of a clinically significant increase is only 2%. The evidence is therefore clearly in the direction of the longer needle causing less harm.

We recognise the need for further evidence on which to base immunisation practice at each of the infant immunisation ages. To this end, we are now conducting a randomised controlled trial involving over 600 infants aimed at providing a definitive answer. In the meantime, we reiterate our recommendation to practitioners to use the longer needle for immunising 4 month old infants.

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Reference
Physiologic management of DKA

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