LETTERS

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The editors will decide, as before, whether to also publish it in a future paper issue.

Problems with scoring bruises

We write to draw attention to two problems with the recent study on a scoring system for bruising by Dunstan et al.1

Firstly, the authors did not publish confidence intervals for the likelihood ratios (LRs) derived from different score threshold values (table 3), thereby not allowing readers to judge whether the LRs are statistically—let alone clinically—significant.

Secondly, the authors neglect the phenomenon of spectrum bias. This is a well described feature of many tests, whereby sensitivity and specificity (and hence derived LRs) of a test vary with disease severity or prevalence.

Examples of spectrum bias have been described with several tests including exercise electrocardiography, Multivariable analysis. Am J Med 1984;74:64–71.

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 meningococci in two patients; in another two patients, the nasopharyngeal swab was positive while the throat swab was negative. In three 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 poor. 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 rifampicin, is effective in eradicating nasopharyngeal carriage, and in children treated with cefotaxime, additional prophylaxis with rifampicin is not necessary. However, no recommendations for the use of cefotaxime alone can emanate from these findings as the sample size was small and study design did not compare cefotaxime with gold standard treatment (either rifampicin or ceftriaxone). We are keen to coordinate a follow up multicentre study this winter involving paediatric intensive care units across the country to compare the efficacy of ceftriaxone with cefotaxime on eradication of meningococcal carriage. Interested units are kindly requested to contact us.

J Clark, R Lakshman, A Galloway, A Cant
Newcastle General Hospital, UK

Correspondence to: J Clark, Department of Child Health, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE, UK; julia.clark@nuth.northy.nhs.uk

References

Pneumocystis carinii pneumonia in an infant with transient hypogammaglobulinaemia of infancy

Transient hypogammaglobulinaemia of infancy (THI) is characterised by prolongation of the physiological decline in serum immunoglobulin concentrations seen in the first six months of life.1 The incidence reported from an Australian paediatric centre was estimated as 23 per 10⁵ live births.2 It has been reported that THI does not usually predispose to significant infection.

A male infant born at term to non-consanguineous parents presented at 3.5 months of age with cough, tachypnoea (70 breaths/minute), wheeze, crepitations, and hypoxia. A chest radiograph showed consolidation and patchy opacification in the hilar regions and upper lobes. *Pneumocystis carinii* was identified in brochoalveolar lavage by toluidine blue staining. The immunological findings of this child were consistent with those of THI with an IgG level less than the fifth centile3 and absent serum IgA and IgM which resolved with age (IgG at presentation 3.9 g/l (normal: 1.39–8.04); at 5 months 2.23 (1.39–8.04); at 10 months 1.77 (2.02–11.76); at 17 months 7.51 (2.71–13.78); IgA at 5 months <0.07 g/l (normal: 0.14–0.69); at 13 months 0.14 (0.17–1.34)) and evidence of specific antibody production to tetanus, diptheria, and *Haemophilus influenzae* type b following immunisation.1 T cell numbers (total lymphocytes 6.2 × 10⁹, CD3 68%, CD4 56%, CD8 15%) and phytohaemagglutinin induced proliferation were normal. At 3 years the child was well with normal IgG, IgA, and IgM levels.

*Pneumocystis carinii* pneumonia presenting in the first three months of life 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 infants and agammaglobulinaemia.4 This is the first description of *Pneumocystis carinii* pneumonia in a patient with THI.

J M Smart, A S Kemp
Department of Immunology, Royal Children’s Hospital, Flemington Road, Parkville 3052, Australia; kempa@cryptic.rch.unimelb.edu.au

D S Armstrong
Department of Respiratory Medicine, Royal Children’s Hospital

References

M Williams
Intensive Care Unit, Charing Cross Hospital, London, W6 8RF, UK

B Krishnan
Department of Paediatrics, Guy’s Hospital, London SE1 9RT, UK

Correspondence to: Dr Williams; matt@williams.org

References
Procalcitonin as a prognostic marker in children with meningococcal septic shock

Carrol and coworkers confirmed the findings from Karabocuoglu et al who reported that procalcitonin (PCT) was higher in children with severe meningococcaemia (fever, petechiae or purpura, and hemodynamic instability) than in children with systemic meningococcal infection without shock (291.29 ± 167 vs 19.7 ± 25 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 value at 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 cut-off values: PCT > 130 ng/ml (the best cutoff value of the PCT level was determined by χ² optimisation (Fisher’s test; p = 0.0004)), CRP < 100 mg/l, 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 MSS. These results accord with those of Hatherill et al who observed, in 37 children with MSS, that admission PCT level (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 (AUC 0.73 (0.59–0.88) vs 0.83 (0.71–0.93); statistical comparison not performed).

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The PRISM score is accepted in PICUs worldwide and has been reported to accurately predict outcome of meningococcal disease. It is, however, as a 24-hour observation period, it cannot be used as an inclusion criterion for clinical trials. Admission PCT could represent a good alternative tool if further studies confirm its ability to predict mortality.

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 be 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 allow better reporting and access to mortality data than that of the US. However, the records acquired as described seem to represent the same underreporting issues as those in the US. Is it really unlikely that the BPSU misses a significant number of cases? Based upon 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 food each year.

A paper published in 2001, described methodology in which a National Registry had been established and was well publicised to US allergists. Very few reports were made by allergists and none by other physicians. No cases were initially reported by physicians who conduct research in food allergy. Nearly all the cases were ascertained from the press. These news articles appeared in local newspapers and were not reported in media with a large regional or national circulation. In an earlier effort to account for all cases of food anaphylaxis, only in Colorado, a significantly

**Table 1** Performance characteristics of PCT, CRP, and PRISM score in 35 children with MSS

<table>
<thead>
<tr>
<th>Severity index (%)</th>
<th>PCT</th>
<th>CRP</th>
<th>PRISM value</th>
<th>PRISM probability</th>
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<tr>
<td>Sensitivity</td>
<td>100</td>
<td>64</td>
<td>100</td>
<td>91</td>
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<tr>
<td>Specificity</td>
<td>63</td>
<td>46</td>
<td>63</td>
<td>83</td>
</tr>
<tr>
<td>Positive predictive value</td>
<td>51</td>
<td>35</td>
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<td>83</td>
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<tr>
<td>Negative predictive value</td>
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<td>95</td>
</tr>
<tr>
<td>Well classified</td>
<td>74</td>
<td>51</td>
<td>74</td>
<td>86</td>
</tr>
</tbody>
</table>

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 underreported? The fifth issue concerns the under reported. The fifth issue concerns the under reporting of cases

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 have reactions 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 have food allergy, and never tell their physician. We could argue about the possible progression of these episodes to near fatal or fatal reactions, but the point we wish to make is that they are not being 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 the proportion and said as much, excluding this group from our definition of severity.

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does not lie in assigning a maintenance fluid allotment. Rather, the source of error lies largely with failure to accurately estimate the volume of deficit and the tendency to automatically assume a severe degree of dehydration. From our experience with over 450 consecutive cases of moderate and severe DKA, and our weight gain data, severe DKA (le severe ketoacidemia) does not necessarily mean severe dehydration; the converse is also true. The degree of dehydration ranges from negligible (<1%) to extreme (>20%). Severe ketoacidemia, however, does cause vasoconstriction which may be manifested peripherally by cool, mottled skin, and Kussmaul breathing which leads to extreme evaporation of mucousa. The striking appearance of a parched mouth and the presence of cool, even mottled skin without a critical assessment of vital signs and examination of distal (foot) pulses often results in an erroneous impression of shock and “severe dehydration.”

A method for estimation of the volume of deficit was described in 1990 and we continue to use this additionally. Successful therapy usually requires not only gradual deficit replacement (evenly over 48 hours) but an accurate estimation of the volume of deficit along with careful attention to the clinical and biochemical response. If the deficit is assumed to be 10−15% but is actually only 3%, that patient will receive excessive water independent of the more gradual timeframe and independent of the deficit. Guidelines that have proposed “safe” limits to fluid volumes administered such as 4 litres/m²/day or 50 ml/kg body weight/4 hours violate the concept of the individualised assessment of the degree of deficit and will invariably overestimate the deficit. The term dehydration should be restricted to the mild to moderately dehydrated child; the problem is compounded when actual body weight is used instead of ideal body weight in fluid allotment. Our approach has been criticised because of the complex disruption of normal metabolism, which leads to metabolic death if left untreated. Shock (decreased peripheral pulses, with or without hypotension), if present, should be corrected rapidly. Insulin should be given preferably by continuous, low dose, intravenous infusion, as soon as possible to begin correction of ketoacidemia/ketoacidosis. Regardless of the serum concentration of glucose, insulin is required to reverse the hepatic, fatty, and ketogenic cycle leading to ketoacid formation.

A delay in insulin administration only serves to enhance and prolong ketoacidemia, thereby extending the period of time within which the patient remains vulnerable to central nervous system and other complications.

Our proposed management strategy may not satisfy the call for simplicity but it is an easily learned approach. It requires an understanding of relevant, known pathophysiology, the monitoring of serial physical examinations and laboratory studies with special attention to correction of acidemia and osmolality, and the anticipatory care that is inherent in the care of the critically ill. Physiologic management was first described between 1988 and 1990, and set forth with additional detail and data in 1994. It is rarely described in its complete form when referenced in texts; mere portions of our recommendations have been cited. In our experience, 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 treatment of DKA, as multicentre studies conducted thus far, all of which compare variations of traditional therapy (empiric volume resuscitation 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 is harmful or undesirable. In our experience, this practice mitigates the development of hyperchloremic acidosis during treatment.

As ours is a referral centre, most of our patients have arrived in hospital of therapy initiated in outlying hospitals, sometimes in keeping with our recommended approach, and sometimes with our recommendations instituted only after initial attempts at correction. 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 typical maintenance allotment (without a deficit replacement component) for the remainder of therapy; some patients required fluid restriction to as little as two thirds the usual maintenance volume.

Our approach has been criticised because of the incidence of mannitol administration in our series. In our mannitol recipients, severe shock, pancreatitis—may require more than 20 ml/kg of fluid resuscitation in the first treatment hour and more than 50 ml/kg in the first four hours. Setting arbitrary fluid volume limits per hour or per day endanger particularly those patients that are the mild and severe ends of the dehydration spectrum. Although the insult would be greater with hypertonic fluid, overhydration occurs readily with isotonic fluids in excess of what their physical examination and laboratory data would dictate. It is rarely described in its complete form when referenced in texts; mere portions of our recommendations have been cited. In our experience, the recommendations simply are not old enough to be reflected in data over the past 20 years. 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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.

L Diggle
Oxford Vaccine Group, Department of Paediatrics, University of Oxford, John Radcliffe Hospital, Oxford OX3 9DU, UK

J Deeks
Centre for Statistics in Medicine, Institute of Health Sciences, University of Oxford, Oxford OX3 7LF, UK

Correspondence to: L Diggle; linda.diggle@paediatrics.ox.ac.uk

Reference


The paper by Parkin et al in the September issue of Archives (Arch Dis Child 2002;87:221–2) was missing acknowledgements. The following paragraph should have been included:

Rita Arseneault, Audrey Bell-Peter, Diana Cohen, Pauline Matthews, Suzanne Stewart, and Olwen Tennis participated in patient enrollment and data collection. Derek Stephens assisted in statistical consultation. Rose Cheung and Carol Collins did the immunofluorescence microscopy and virus isolation tests. Dr Raymond Tellier oversaw virus testing for part of the time while he was on service.

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