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## 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.:

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 stress testing and UDI diagnosis.

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.

### References


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### 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 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 meningococcus 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 ceftriaxone, 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.

### References

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### 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. The incidence reported from an Australian paediatric centre was estimated as 23 per 10^5 live births. 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 with cough, tachypnoea (70 breaths/minute), wheeze, crepitations, and hypoxia. A chest radiograph showed hyperinflation and patchy opacification in the hilar regions and upper lobes. *Pneumocystis carinii* was identified in bronchoalveolar 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 centile and absent serum IgA which resolved with age (IgA 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, diphtheria, and *Haemophilus influenza* type b following immunisation. T cell numbers (total lymphocytes 6.2 × 10^9/l, CD3 68%, CD4 56%, CD8 15% and CD4:CD8 ratio 1.84) and phagocytomaglutinin 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. This is the first description of *Pneumocystis carinii* pneumonia in a patient with THI.

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### References


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Procalcitonin as a prognostic marker in children with meningococcal septic shock

Carrol and coworkers 1 reported that procalcitonin (PCT) was higher in children with severe meningococcal meningoencephalitis, fever, peripheral purpura, and hemodynamic instability than in children with systemic meningococcal infection without shock (291.29 ± 167 v 19.7 ± 23 mg/ml; p < 0.001). 2

Unfortunately, information is lacking in the report of Carrol et al, 1 namely: a clear definition of severe MCD (defined in their paper as a Glasgow Meningococcal Septicaemia Prognostic Score ≥8) and median PCT values (defined as ecchymotic or necrotic purpura with shock, needing fluid expansion (median for the first 24 hrs: 90 ml/kg; Q1–Q3: 48–120) and catecholamine infusion) admitted to our PICU between July 1999 and May 2002. We estimated the accuracy in predicting death of PCT, C reactive protein (CRP), procalcitonin levels (PCT functioned worse than the PRISM score whatever the causative organism, admission PCT level (values not indicated) 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) v 0.83 (0.71–0.93); statistical comparison not performed). 3

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) v 0.83 (0.71–0.93); statistical comparison not performed). 3

For each severity index, we calculated the area under the ROC curve (AUC) and the standard error (SE) and determined the significance of comparisons. 4

Eleven of 35 children died (31%); predicted mortality with the PRISM score was 15.6 (standardised mortality ratio: 0.71; 95% confidence interval: 0.35–2.26). The median (Q1–Q3) PCT and CRP levels and PRISM value and probability of death were the following: survivors v nonsurvivors PCT 73 (15–210) v 277 (208–606) mg/ml (p = 0.001); CRP 92 (44–160) v 72 (41–109) mg/l (p = 0.025); PRISM value 17 (8–22) v 33 (26–37) (p < 0.01); PRISM probability 19 (4–42) v 88 (63–95%) (p < 0.10). Performance characteristics and AUC ± SE of PCT, CRP, and PRISM score are given in the table and the figure.

Table 1

<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>
</tr>
<tr>
<td>Specificity</td>
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<td>46</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>

Figure 1 ROC curves (AUC ± SE) for PCT, CRP, and PRISM score in 35 children with MSS (PCT v PRISM value, p = 0.45; PCT v PRISM probability, p = 0.31; PCT v CRP, p = 0.06; CRP v PRISM value, p = 10−1; CRP v PRISM probability, p = 10−1).

In conclusion, PCT on admission was as accurate as the PRISM value and PRISM probability of death in children with meningococcal septic shock and can be used to estimate the probability of death in these children.
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.1 A second concern is the reporting of cases only up to age 15. In the paper mentioned above, of 32 fatalities 10 occurred in young-sters up to age 15.2 An additional 10 occurred in adolescents aged 16 to 19. Why did MacDougall et al not include all adolescents?3 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 emer- gency department and are misclassified on death certiﬁcates as asthma fatalities? Indi- viduals that die at home and are classiﬁed as asthma deaths are unlikely to be further investigated in the UK or the US.4 Fourthly, the authors’ definition of severity seems incomplete. Individuals with severe food reactions who self administer epine- phrine often do not go to hospital, are less likely to have reactions that require hospitaliza- tion 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 are allergic to a speciﬁc food, and never tell their physician. We could argue about the possible progression of these epi- sodes 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 dis- agree 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 individuals who die during allergic reactions without treatment, become convinced that the risk as well as those many parents that think it may to difﬁcult to institute a good 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 rehydrating and administering ﬂuid to children on the best data available. This must include those parents whose children are truly at high risk as well as those many parents that think any guidelines for rehydration and treatment of the pediatric patient with diabetic ketoacidosis (DKA).1 They review some of the key issues that link ﬂuid therapy to complications from brain swelling, and question the appropriateness of rehydrating and administering ﬂuid to children on the best data available. This must include those parents whose children are truly at high risk as well as those many parents that think any guidelines for rehydration and treatment of the pediatric patient with diabetic ketoacidosis (DKA).1
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 episodes of moderate and severe DKA, and our weight gain data, severe DKA (i.e. 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 very dry oral mucosa. The striking appearance of a parched mouth and the presence of cool, even mottled mucosa. The striking appearance of a parched

...and that aimed to provide some evidence base for... hydraulic state, even when mannitol was given... with or without urinary output replacement). In a retrospective portion of our study in 1990 we compared these same therapies and in nearly 40%, no form of... therapy minimised the risk of brain herniation during treatment. Comments regarding the administration of... that the deficit be better defined. Ration... of “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... 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... have received therapy initiated in outlying hospitals, sometimes in keeping with our recommended approach, and sometimes with our recommendations instituted only after initial contact. In this setting, we have... 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... 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, 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 management strategies really had raised intracranial pressure. We believe, however, that the key to our good outcome is the... fluid and electrolyte therapy on which... superimposed is relevant to its success. It is erroneous to assume that a 100% success rate among our mannitol recipients would be reproducible in the setting of a therapy that violates the fundamental principles of rehydrating the hypertonic state of DKA. Drs. Inward and Chambers ask “do we have it right yet?” and convey concern that certain recommendations do not, as of yet, “have it right”. We agree.

Our work regarding the management of the pediatric patient in moderate to severe DKA has spanned 14 years and nearly 500 consecutive prospectively managed episodes. We remain available to participate in any... the paediatric patient in DKA.

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References

The Position Statement on Injection Technique

The Position Statement on Injection Technique (March 2002, Royal College of Paediatrics and Child Health) discusses needle size and length for childhood immunisation. It concludes that there would seem to be insufficient evidence to advise any reparation to change current practice in the use of 10mm 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 the shorter 25 gauge 13mm (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
Problems with scoring bruises

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Arch Dis Child 2002 87: 449
doi: 10.1136/adc.87.5.449

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