If you have a burning desire to respond to a paper published in *Arch Dis Child* or *FEBP*, why not make use of our “rapid response” option? Log on to our website (www.archdischild.com), find the paper that interests you, click on “full text” and send your response by email clicking on “submit a response”.

Providing it isn’t libellous or obscene, it will be posted within seven days. You can retrieve it by clicking on “read eLetters” on our homepage.

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.:

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 UTI 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**


**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 10^6 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 of age with cough, tachypnoea (70 breaths/minute), wheeze, crepitations, and hypoxia. A chest X-ray and radiograph showed hyperinflation, detection of ménigooccal DNA in blood by polymerase chain reaction, and convalescent ménigooccal serology. All children were treated with intravenous cefotaxime for seven days. Nasopharyngeal and throat swabs were repeated 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 ménigoocci 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 ménigoocci 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 finding that 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 ménigooccal 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**


3. Stagno S, Pifer LL, Hughes WT, et al. *Pneumocystis carinii* pneumonia in young immunocompetent infants and agammaglobulinaemia. 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**


www.archdischild.com
Procalcitonin as a prognostic marker in children with meningococcal septic shock

Carrol and coworkers1 confirmed the findings from Karabocuoglu et al2 who reported that procalcitonin (PCT) was higher in children with severe meningococcaemia (fever, petechial purpura, and hemodynamic instability) than in children with systemic meningococcal infection without shock (291.29 ± 167 v 19.7 ± 23 mg/l; 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 of survivors and nonsurvivors. Carrol did not specify the time at which the PCT level was determined by the authors of the paper as a Glasgow Meningococcal Septicaemia Prognostic Score (GMS) for PICUs.9

In our study, PCT on admission was as accurate as the median PCT value and PRISM probability of death calculated within 24 h 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 al16 who, in 37 children with MSS, that admission PCT level (values not indicated) whatever the causative organism, admission PCT functioned worse than the PRISM score (p<0.06; CRP <0.05; PRISM v PRISM probability, p<10−5).

Eleven of 35 children died (31%); predicted mortality with the PRISM score was 15.6 (standardised mortality ratio: 0.71; 95% confidence interval: 0.35–1.26) at 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 88 (63–95) % (p<0.1); CRP 100 (94–100) mg/l (p<0.05); 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 of AUC ± SE of PCT, CRP, and PRISM score are given in the table and the figure.

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 by either 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 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 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 we are trying to make is that there are fatalities under reported. The fifth concern is the safety 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 point should be emphasised. However families reading this commentary may become more fearful, than they currently are, about administering epinephrine. We know that epinephrine is not always life saving even when correctly administered. We specifically studied children 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? It is our opinion that the problem in the diabetic population is very much more complex and therefore may rely their vigilance. If families of younger children become less concerned when their children become adolescents it may be difficult to institute a good public education program. This is directly opposite the goal of education programs in the US (The Food Allergy and Anaphylaxis Network, www.foodallergy.org) and UK (The Anaphylaxis Campaign) aimed at making individuals with food allergy and the general population more aware of the problem and the potential for mortality. It is truly unfortunate that we cannot accurately identify all of the individuals who die during allergic reactions to food and use this information to do a better job of preventing these tragedies. We must continue our campaigns of education of medical professionals and the public, and we must be certain that emergency treatment is available when and where it is needed.

J O’B Hourihane
Wellcome Trust Clinical Research Facility, Southampton University Hospitals NHS Trust, Southampton, UK
D Reading
The Anaphylaxis Campaign, PO Box 275, Farnborough, Hampshire, UK
P Smith
Brisbane, Australia
G Lack
St Mary’s Hospital, London, UK
D Hill
Department of Allergy, Children’s Allergy Centre, Royal Children’s Hospital, Parkville, VIC 3052, Australia
A Muñoz-Furlong
The Food Allergy & Anaphylaxis Network, 10400 Eaton Place, Fairfax, VA 22030, USA
5 A Bock
National Jewish Medical and Research Center, Department of Paediatrics, University of Colorado Health Sciences Center, Denver, CO, USA

Correspondence to Dr Bock; Bockdoc@aol.com

References

Authors’ reply
We thank Bock et al for their interest in our article. We respect their views on the interpretation of the data but it is of course for each reader to come to their own opinion on these. We would like to respond to their comments on the accuracy and validity of our data. Did our paper under ascertain deaths? Bock et al base their concerns on our methods of case ascertainment and on comparison with another study. We cannot be certain about this but as the text indicated we used many sources and spoke to many experts in the field. We agree we did not search local newspapers but this would have been almost impossible as few were on CD-ROM in the 1990s. As mentioned, we did search national newspapers and all cases we came across were already known through one of our other sources. Finally, since publication, no-one has told us of a case we appear to have missed. We specifically studied children up to 15 years because this is the group we were interested in. Many recommendations on risks to children are based on inferences from data covering all ages and we wanted to bring a proper paediatric perspective. Indeed the interpretation Bock et al give to the paper they cite is grossly misleading. They suggest extrapolation to a US population would lead to 200 deaths from food each year yet the paper, in which there is only one death (occurring during exercise), covers all ages and reactions to all allergens, not just food.

The issue of whether asthma deaths may have been precipitated by food allergy is a most important question which we addressed “If a child’s symptoms are only asthmatic and no allergens 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 must 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 very 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.

A Colver
Northumbria Health Care Trust and University of Newcastle upon Tyne, Donald Caudwell House, 2 Walker Terrace, Gateshead NE8 1EB, UK
C Macdougall
Newcastle General Hospital, Westgate Road, Newcastle upon Tyne NE4 6BE, UK
A Cant
Paediatric Immunology and Infectious Diseases Unit, Newcastle General Hospital, UK

Correspondence to Dr Colver; allan.colver@ncl.ac.uk

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).1 They review some of the key issues that link fluid therapy to complications from brain swelling, and question the appropriateness of using volumes 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 DKA is a risk factor for cellulitis complications, and that despite published studies 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


Reference
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 (ie severe ketoacidemia) does not necessarily mean severe dehydration; the converse is also true. 13,14 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 vere oral and oral mucosa. 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 23 and we continue to use this approach. Successful therapy usually requires not only gradual deficit replacement (evenly over 48 hours) but an accurate estimation of the volume of deficit along with correction of the clinical and biochemical response. If the deficit is assumed to be 10–15% but is actually only 3%, that patient will receive excess water independent of the more gradual timeframe and independent of the rehydration therapy actually given. 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. Such fluid allotment would oversaturate and moderately overload the mildly to moderately dehydrated child; the problem is compounded when actual body weight is used instead of ideal body weight in fluid allotment. On the other hand, other patients, particularly those with complicating illness—for example, septic 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 at the mild and severe ends of the dehydration spectrum. Although the deficit would be greater with hypotonic fluid, overhydration occurs readily with isotonic fluid as well when water requirements are overestimated.

DKA represents the effects of a 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 counteract the hepatic fatty acid/ carnitine cycle leading to ketoacid formation. 1 A delay in insulin administration only serves to enhance and prolong ketoacidemia, thereby extending the period of time that patients may remain 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. 1 12 Physiologic management was first described between 1988 21 and 1990, 22 and set forth with additional detail and data in 1994. 1 It is rarely described in its complete form when referenced in texts; mere portions of our recommendations are published in what we have called 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 with multicentre studies conducted thus far, all of which compare variations of traditional therapy (empiric volume resuscitation whether or not shock is present, assumption of a large volume of deficit, planned rehydration in less than 48 hours with either 0.45% or 0.9% NaCl, with or without urinary output replacement). In a retrospective portion of our study in 1990 22 we compared these same therapies and found no form of traditional therapy minimised the risk of brain herniation during treatment.

Comments regarding the administration of mannitol base should be better defined. Rapid administration 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 of the dehydrated patient 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 been brought to our hospital during or after their initial treatment by us in oulying hospitals, sometimes in keeping with our recommended approach, and sometimes with our recommendations instituted only after intensive contact. 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, 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. 1 It is possible that not all of our mannitol recipients actually had raised intracranial pressure. In our series, we have not encountered a case of brain herniation during treatment.


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 recommendation to change current practice in the use of insulin 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. 1

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 12mm (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.

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