

PostScript

LETTERS

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Conflicting advice

Concerning use of conjugate pneumococcal vaccine, the most recent CMO letter¹ sent out in August 2002, updates the recommendations issued by the Department of Health (DoH) in January 2002² by making the recommendations for "at risk" under 2 year old children coincide with the manufacturer's recommendations for immunisation of normal healthy children in their Summary of Product Characteristics for their European product licence.

These schedules differ a little bit from those set out in our paper³ which was subsequently cited in the recent RCPCH guidelines for immunisation of immunocompromised children.⁴ In particular, the DoH advice does not draw any distinction between different risk groups, whereas our advice is to give extra doses to children with hyposplenism and various forms of immunocompromise. The DoH does not, at present, advocate use of the conjugate vaccine in any children over the age of two, whereas we do, conscious that many experts feel that there are good theoretical reasons to use the conjugate vaccine in this way (just as conjugate meningococcal C vaccine has replaced polysaccharide vaccine use in older children). Finally, the DoH suggests all recipients in the second year of life should receive two doses of conjugate pneumococcal vaccine, whereas we suggest only one for "at risk" children outwith the very high risk groups mentioned above.

The differences between the two sets of advice have led to some enquiries from colleagues as to how best to proceed and why the two documents differ.

We think it is important to emphasise that both sets of recommendations have been drawn up in the absence of much data as to how best to protect these groups of children. What evidence there is, is summarised in our paper. Further immunogenicity studies in children with HIV and other groups are in progress. In addition, it was reassuring to hear the preliminary results of a large efficacy study in children in South Africa at the International Symposium on Pneumococci and Pneumococcal Diseases in May 2002 which suggested that conjugate pneumococcal vaccine is protective in children with HIV infection, albeit less so than in uninfected

children. However, most pre-licensure studies were done in normal healthy infants, since that was the target group for the license. In the absence of more data, it is not surprising that different expert groups have come up with slightly differing advice. In addition, presumably, as a government agency, the DoH must be constrained to some extent against issuing recommendations which go beyond or which differ from those contained within an official product license—even if that license relates to healthy rather than "at risk" individuals.

Consistently following either set of recommendations seems reasonable under the circumstances—no doubt further modifications to this advice will follow in due course as further evidence emerges.

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<http://www.doh.gov.uk/cmo/cmo0201.htm> (accessed 18th Sept 2002).
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On Archimedes

Using the best available evidence is expected of us in clinical practice. How should clinicians get such evidence? Should we all be formulating questions, searching for the evidence and then appraising it? Or as busy clinicians are we forced to rely on the evidence provided for us in published systematic reviews. Rudolf's recent paper puts one side of the argument.¹ Nine doctors attending at MmedSc Course each spent an average of five hours analysing a clinical problem "in accordance with the principles of evidence based practice". As a result of this work they judged themselves to have improved in structuring clinical questions, searching electronic databases, and in critical appraisal. In addition they succeeded in highlighting the poor evidence upon which we base much of our practice. I have no doubt that their efforts had an educational value, but would they be right to base their clinical practice on the conclusions of five hours work?

In November 2001, as part of the Archimedes series, two middle grade paediatricians attempted to answer the following question: in a febrile infant, how accurately does tympanic thermometry measure core temperature? They took rectal temperature to reflect core temperature and restricted their search to work on children. They found two directly relevant studies and one systematic review. On this they based their analysis.

In August 2002, Craig and her colleagues published a systematic review comparing

tympanic thermometry and rectal thermometry in children.³ They searched eight databases and checked through numerous reference lists. They contacted authors and suppliers of clinical thermometers. They found 44 studies eligible for inclusion, including two unpublished papers and five written in languages other than English. The process of searching for and identifying eligible articles took approximately 80 hours spread over several months (V Craig, personal communication). Given the huge disparity in the number of identified papers, it is surprising that the results of both reviews were similar: in an individual patient, tympanic thermometry may not accurately mirror the rectal temperature. However in the details they differed. Riddell and Eppich tell us that "age and presence of fever significantly affected the rectal tympanic difference".³ Craig *et al* showed that this was not the case, "there was no systematic relation between the temperature difference and the underlying temperature" and similarly "we found no association between temperature difference and the age of the children".³

Riddell and Eppich found 3 papers. Craig *et al* had found 44. What does this tell us?

Answering clinical questions by appraising the available evidence is justifiably the new creed. But done quickly, it risks being done badly. The search for evidence, and its analysis, is best left to those with the necessary time and expertise. The urge to join in is understandable. It should be resisted. Those of us in busy clinical posts should assess the results of thorough systematic reviews and then, in the words of Sackett and his colleagues, conscientiously, explicitly and judiciously, use them to make decisions about the care of our patients.⁴ If we are honest with ourselves, we really haven't time for anything else.

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- 2 **Archimedes.** Towards evidence based medicine for paediatricians. *Arch Dis Child* 2001;**85**:431-4.
- 3 **Craig JV, Lancaster GA, Taylor S, et al.** Infra-red ear thermometry compared with rectal thermometry in children; a systematic review. *Lancet* 2002;**360**:603-9.
- 4 **Sackett DL, Rosenberg WM, Gray JA, et al.** Evidence based medicine: What it is and what it isn't. *BMJ* 1996;**312**:71-72.

Author's reply

As editor of *Archimedes*, and victim of his play,¹ I have the pleasure in responding to the concerns raised by Dr Lopez. I think there are two—a concern with the philosophy of *Archimedes* and a problem with the tympanic topic.

There is a firm and widely held belief that evidence based practice can be achieved only by those "with the necessary time and expertise", and that we should only change our practice after assessing "the results of thorough systematic reviews and . . . conscientiously, explicitly and judiciously, use them to

make decisions about the care of our patients”.

The position of *Archimedes*, and I'd guess many clinicians who believe they practice in an evidence based fashion,² is nearly the opposite. Finding questions, seeking answers, assessing the best of the evidence to answer that question, applying it, and assessing the results is a cycle we all (should) perform in some way or another. I think that's all “evidence based” practice really is. The famous Sackett quote about evidence based medicine being “the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients”³ relies on the current best evidence: not just systematic reviews (which might be categorised as “the best evidence which can be produced”).

Dr Lopez asks “Riddell and Eppich found three papers. Craig *et al* had found 44. What does this tell us?”. Well, possibly that Dr Lopez didn't read the Riddell report⁴ very closely, as their commentary starts with “*The systematic review . . . [had] 44 studies addressing the use of different methods of temperature measurement*” Accordingly, it's not at all surprising that the papers come to the same bottom lines. Any minor differences—reported as comments on subsidiary papers with a much lower level of evidence—wouldn't be that clinically exciting, would they?

But anyway, I think that Dr Lopez and myself would agree on one thing. Compared to starvation in Zimbabwe, an impending Gulf war, and children being raped by their parents, this isn't all that important.

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More lumbar punctures, please!

Applause to Kneen *et al*¹ and Riordan and Cant² for reminding us of the value of lumbar puncture in suspected meningitis. To their arguments I would add that, while the matter may end after seven days' intravenous antibiotic treatment as far as the admitting paediatrician is concerned, it certainly does not for the child or parents of many children who have had meningitis, as recent data shows.³ To be discharged home in ignorance or confusion regarding the diagnosis, as is becoming the norm, does no favours either to the patient or to anyone attempting to manage late complications. In adopting the “treat and do a hearing test” approach we should consider whether we are really motivated by a desire to relieve the child of the risk and discomfort of the procedure or to relieve ourselves of the bother. Undoubted risk and discomfort does not seem to have put us off requesting large numbers of head CT scans (often without contrast—so they do not reliably exclude abscess) in this clinical situation, even though they do not tell us anything useful about raised intracranial pressure.

As both papers point out, the epidemiology and management of bacterial meningitis are changing fast. Has anyone paused to consider how, in the future, we will evaluate either its incidence or the effectiveness of our current management strategies if we can't tell how many cases we have seen and who they were?

Clearly, it can be ill-advised to perform a lumbar puncture at the outset in seriously sick children—but there is always a time later on when the procedure can be done safely, and often also painlessly just before weaning from the ventilator.

As for the habit of replacing the LP (and other necessary investigations) with indiscriminate initiation of cephalosporin treatment in the mild to moderately ill febrile child, this simply encourages misdiagnosis and promotes development of antibiotic resistance.

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LP and Glasgow coma score

Congratulations to the authors on a balanced article on the need for lumbar puncture.¹

One point of possible confusion is the Glasgow Coma Score (GCS) quoted as a contraindication to LP. Kneen *et al* quote a GCS <13 as a contraindication to LP, which would exclude a very large number of children with meningitis. Riordan and Cant² in the same issue of your journal quote a GCS <8. Rennick *et al*³ also use a GCS <8 as their cut off figure in their hospital, as do we.⁴

There is little evidence to my knowledge. A retrospective Manchester study⁵ found that children with GCS <8 were more likely to die from coning than other children with meningitis (relative risk 4.6, 95% CI 1.06–35.8).

I would welcome comments from the authors and others as to whether they have better evidence for the GCS they quote, and if not, what we should advise in the absence of good evidence.

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Authors' reply

We thank Dr Isaacs for his helpful letter. He rightly points out that the published recom-

mendations as to which Glasgow Coma Scale score serves as a contraindication to a lumbar puncture vary between <8 and <13, though we are not aware of any definitive evidence supporting either value. For the purposes of our overview commentary we chose the most conservative value (<13), which is also that recommended in the Advanced Paediatric Life Support Manual produced by the RCPCH advisory committee. Opinions will vary as to what level of consciousness is a contraindication to lumbar puncture (LP). In our clinical practice we do perform LPs on children with lower coma scores if there are no other contraindications to LP. These issues clearly deserve further consideration, but for this editorial our primary concern related to the observation that even many fully conscious children do not undergo LP for the spurious reasons outlined in our article.

In the editorial we refer to a survey of LP practice in Liverpool, which were unpublished observations at the time; these data have now also been published.¹

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Are interhospital transport teams de-skilling the DGH paediatricians?

As one of the referring hospital consultants to the South Thames combined transport service, I can attest to the successful service described in the paper by Doyle and Orr.¹ However, it is rare for a transport team to be immediately available to collect a sick child. This delay compounded by the inevitable travelling time means that the referring unit needs to be able to stabilise and treat the sick child prior to the team's arrival.

Concerns have been voiced that the availability of such teams de-skill paediatricians and place an increased burden on the “in-house” anaesthetists and intensivists. To examine this concern, data collected over the last 2 years from our paediatric high dependency unit (HDU) were reviewed. 153 children were admitted with 35% originating from the A&E department. The vast majority were medical type patients with 42% suffering respiratory problems, 1% required nasal CPAP and 13% required intubation and ventilation. Of these 63% were intubated by “in-house” anaesthetists. 25% of all admissions required transfer to a paediatric intensive care unit (PICU) by transport team. 71% of admissions to the HDU room were discharged to the in-patient ward. There were no deaths occurring in this HDU facility.

In view of the overall infrequency of intubation by local staff but the successful care of these patients, it would not seem as though transport teams are de-skilling the local teams. Indeed good communication and shared protocols enhance the local team's work provided senior experienced staff are available to supervise care until the arrival of the transport team.

The above experience does highlight the benefit of a local HDU facility. Stabilisation and close monitoring is not only good practice, essential for patient care but should reduce the work of the transport team when it arrives. When funding and patterns of care are reviewed locally, more attention should be given to ensuring that local facilities in the form of HDU beds are available. They are not mini PICUs but they do have a purpose.

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A self-fulfilling prophesy?

Carroll and Brookfield¹ quote a widely used definition of febrile convulsion in their second paragraph: “an epileptic seizure occurring in a child aged from 6 months to 5 years, precipitated by fever arising from infection outside the nervous system in a child who is otherwise neurologically normal”. The authors then go on to say that only a tiny percentage of children with febrile convulsions have meningitis. By definition though, that percentage is 0%.

I dispute the assertion that more experienced staff are less likely to recommend lumbar punctures. Over the years, most people miss the occasional case of meningitis and become doubly wary of “absence of meningeal signs” thereafter. Meningeal signs are often misunderstood too; many Senior House Officers believe Kernig sign to have something to do with pain in the back (rather than just a feeling of tightening in the hamstrings). With neck stiffness, they sometimes expect the neck to be rigid rather than just slightly stiff on extreme flexion.

Even viral meningitis is very good at causing sensorineural hearing loss. Unless we routinely start antibiotics and request audiology on all children who have had a convulsion with fever, we still need to do lumbar punctures.

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The relationship between *Helicobacter pylori* infection and iron deficiency: seroprevalence study in 937 pubescent children

Helicobacter pylori infection has been reported to be associated with various unexpected manifestations in childhood. One of them is iron deficiency anaemia at puberty. In 1999, we conducted a double blind, placebo controlled trial in pubescent children with iron deficiency anaemia and coexisting *H pylori* infection.¹ We found that *H pylori* eradication led to resolution of iron deficiency. We have carried out a study of seroprevalence to examine the epidemiological relationship between *H pylori* infection and iron deficiency anaemia at puberty. Haemoglobin, serum

iron, total iron-binding capacity, serum ferritin, and serum IgG Antibodies to *H pylori* were measured in 937 Korean children (475 boys and 462 girls). Their ages ranged from 10 to 18 years. The prevalence of *H pylori* infection was compared between groups, based on the presence or absence of anaemia, hypoferritinemia, iron deficiency, and iron deficiency anaemia. The levels of hemoglobin, serum iron, total iron binding capacity, transferrin saturation, and serum ferritin were obtained according to the presence or absence of *H pylori* infection.

The prevalences of anaemia, iron deficiency, iron-deficiency anaemia, and *H pylori* infection were 8.1%, 9.1%, 3.1%, and 20.8%, respectively. The *H pylori* positive rates in anaemia, hypoferritinemia, and iron deficiency group were 34.2%, 29.5%, and 35.3%, respectively, compared to 19.6% in the non-anaemia group ($p=0.003$), 19.2% in the non-hypoferritinemia group ($p=0.005$), and 19.4% in the non-iron deficiency group ($p=0.001$). The *H pylori* positive rate in the iron deficiency anaemia group was 44.8% in comparison with 20.0% in the non-iron deficiency anaemia group ($p=0.001$). Haemoglobin and iron levels did not show any significant differences between the *H pylori* positive and negative groups. The serum ferritin level was significantly lower in the *H pylori* infected group ($p=0.0002$).

The associations between iron status and *H pylori* were largely restricted to girls rather than boys. We speculate that this is because female adolescents are more vulnerable to iron deficiency. *H pylori* may affect iron absorption metabolism in the stomach and exacerbate the iron deficit in adolescents, especially girls, whose iron is supplied marginally, with anaemia ensuing promptly.²

We believe that this is the only large scale study in children showing an association between *H pylori* infection and iron deficiency. When children at puberty are found to have iron deficiency that is refractory to iron supplementation, *H pylori* infection can be considered to be a possible cause of iron deficiency.

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Acute ataxia complicating Langerhans cell histiocytosis

Some of the statements in the interesting short report by A Polizzi *et al*¹ can be

challenged. It is incorrect to suggest that cerebellar ataxia has been reported “only occasionally” in children and that it is commoner in adults with Langerhans cell histiocytosis (LCH). Diabetes insipidus is the only CNS complication that is more common than cerebellar disease and though the precise relative incidence of cerebellar ataxia in children and in adults is unknown, because all published series are institution based, there is no reason to suspect that proportionately more adults suffer this complication. It is also misleading to suggest that the patient described by Polizzi *et al* represents a “unique” occurrence. Cerebellar ataxia may be present at diagnosis or appear during follow up and may be progressive or static. More details of the clinical and pathological spectrum of CNS involvement by LCH can be found in a recent review.²

As the authors point out, pituitary-hypothalamic axis involvement is caused by direct infiltration of these structures by pathological Langerhans cells (“LCH cells”) and accompanying inflammatory cells. In patients who develop ataxia, cerebellar biopsy usually reveals only gliosis and demyelination, but CD1a-positive cells have been demonstrated in a few instances. Therefore, it is likely that the cerebellar lesions are caused by “LCH cell” infiltration followed by cytokine and chemokine mediated neural damage. The same sequence, with fibrosis as the end point, occurs in the liver and lungs of other LCH patients. Immune mechanisms may also be involved, as suggested by Polizzi *et al*, because CD8-positive T cells are also found in the cerebellar biopsies (Gros NG, personal communication). It is unlikely, however, that they represent the primary pathogenetic process. In other words, it is improbable that cerebellar involvement represents a “paraneoplastic syndrome” (ie an autoimmune disorder), as suggested by Polizzi and colleagues, a view supported by the fact that cerebrospinal fluid (CSF) “anti-neural” antibodies have not been detected in two studies (Gros N *et al* and Donadieu J *et al*, unpublished observations).

The combination of reticulo-nodular pulmonary shadowing and ataxia, as in this case, makes the diagnosis of LCH a real possibility. Given the onset of fatal pneumothorax soon after the child’s discharge from hospital, a lung CT scan would almost certainly have shown cystic change and led to tissue diagnosis via lung biopsy or bronchial lavage. With chemotherapy, the prognosis for pulmonary LCH is usually favourable, even if complicated by pneumothorax.³

It is generally recommended that patients with suspected LCH are referred to paediatric oncologists and/or managed on the international evidence-based trials of the Histiocytosis Society, which include recommendations for the management of CNS complications.^{2–4} More details of the Histiocytosis Society’s activities and their contact address can be found on the Society’s website: www.histio.org.society.

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Hypocalcaemia and calcitonin precursors in critically ill patients

We read with interest the paper by Baines and colleagues¹ in which the authors reported a strong inverse relationship between total serum calcium concentrations and disease severity in 70 critically ill children with meningococcal disease. Calcitonin concentrations were measured in a subgroup of 23 children on admission, and significantly correlated with disease severity. In particular, however, the authors found no relation between calcitonin concentrations and total or ionised calcium concentrations. In a study of 69 adult patients with acute pancreatitis,² we have similarly found no correlation between plasma concentrations of calcitonin precursors (CTpr) on admission² and both the admission and lowest (within 72 hours of admission) adjusted total serum calcium concentrations (unpublished data). The concentrations of CTpr were significantly higher² and

of the lowest calcium were significantly lower (median (IQR): 2.16 (2.0–2.18) mmol/l v 2.23 (2.15–2.30) mmol/l, $p=0.017$) in patients with severe attacks ($n=14$, Atlanta criteria) compared with mild attacks. Our data and that of Baines and colleagues¹ support the contention that calcitonin and its precursors have a minor effect on calcium metabolism. Indeed, previous investigators found no correlation between the serum concentrations of serum calcitonin and hypocalcaemia in patients with acute pancreatitis³ or in experimental models of the disease.⁴ Whilst CTpr concentrations were reported to rise significantly in critically ill patients, they correlated rather weakly with a concomitant fall in serum ionised calcium.⁶ A rise in CTpr concentrations did not correlate with the fall in serum calcium concentrations in patients with acute malaria.⁷ This suggests that factors other than calcitonin and CTpr are involved in the homeostasis of calcium in the critically ill.

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CORRECTION

The Archivist piece "Heart disease in Williams syndrome" (*Arch Dis Child* 2002;**87**:420) should have given the reference to the original article in the Journal of Medical Genetics as *J Med Genet* 2002;**39**:554–8.