If you have a burning desire to respond to a paper published in ADC or FEP, 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 “submit your response” and send your response by email by 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.

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 the absence of much data as to how best to protect these groups of children, the DoH does not, at present, advocate use of the conjugate vaccine, whereas we suggest only one for “at risk” children outwith the two documents differ.

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 hypoplasen 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 way (just as conjugate meningococcal C vaccine has replaced polysaccharide vaccine)

DoH must be constrained to some extent against issuing recommendations which go beyond or conflict with the principles of evidence based practice. As a result of this work they judged the results of thorough systematic reviews 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. If we are honest with ourselves, we really haven’t time for anything else.

T Lopez

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References


Author’s reply

As editor of Archimedes, and victim of his play, 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 practice.

This 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

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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, is nearly the opposite. Finding questions, seeking answers, assessing the evidence of the evidence to attune that question, applying it, and assessing the results is a cycle we all (should) perform in some way or another. I think that 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..." is not just a throwaway line. 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 found 44. What does this tell us?". Well, possibly that Dr Lopez didn't read the Riddell report very closely, as their commentary starts with "The systematic review of existing 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 difference—reported as conclusions 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.

B Phillips

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References

More lumbar punctures, please!

Applause to Kneen et al. and Riordan and Cant' 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 recent data may end after seven days' intravenous antibiotic treatment. In adopting the 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. In our 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."

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1 Kneen R, Solomon T, Appleton R. The role of lumbar puncture in children with suspected central nervous system infection. Pediatrics 2002; 2: 8. This article is available free online from: http://www.biomedcentral.com

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. 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-skills 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 PICU in a similar condition. 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.

Authors' reply

We thank Dr Isaacs for his helpful letter. He rightly points out that the published recommendations 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 more conservative value (<13), which is less than that recommended in the Advanced Paediatric Life Support Manual prepared 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.

B Phillips

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References
1 Kneen R, Solomon T, Appleton R. The role of lumbar puncture in children with suspected central nervous system infection. Pediatrics 2002; 2: 8. This article is available free online from: http://www.biomedcentral.com
iron deficiency: seroprevalence
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Reference
Carroll and Brookfield
A self-fulfilling prophesy?
Carroll and Brookfield 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 tiny percentage of children with febrile convulsions have meningesitis. By definition though, that percentage is 0.
1 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 meningial 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.
2 Even viral meningitis is very good at misleading to suggest that the patient develops meningitis.
3 The above experience does highlight the need to retrain patients who develop ataxia, cerebellar biopsy usually reveals only gliosis and demyelination,
H pylori positive rates in
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Reference
A self-fulfilling prophesy?
The prevalence of anaemia, iron deficiency, iron-deficiency anaemia, and H pylori infection were 8.1%, 21.3%, 9.1%, 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 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 who already have a low iron iron. When children at puberty are found to have iron deficiency, serum ferritin and transferrin saturation and serum ferritin were obtained according to the presence or absence of H pylori infection.

H pylori infection

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. 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.

References

References

The relationship between Helicobacter pylori infection and iron deficiency: seroprevalence study in 937 pubescent children
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The prevalence of anaemia, iron deficiency, iron-deficiency anaemia, and H pylori infection were 8.1%, 21.3%, 9.1%, 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 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 who already have a low iron level. When children at puberty are found to have iron deficiency, serum ferritin and transferrin saturation, and serum ferritin were obtained according to the presence or absence of H pylori infection.

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.

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 Histio- cyte Society, which include recommendations for the management of CNS complications.44 More details of the Histioctye Society’s activities and their contact address can be found on the Society’s website: www.histio.org-society.

References

Acute ataxia complicating Langerhans cell histiocytosis
Some of the statements in the interesting short report by A Polizzi et al 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 also a reason to suspect that 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.1

As the authors point out, pituitary-hypothalamic axis involvement is crucial 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 direct cerebellar infiltration and not by LCH. 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 (Grois NG, personal communication). It is unlikely, however, that they are also involved in the primary events because the meningitis occurs after the lesion has already been formed. 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-neuronal” antibodies have not been detected in two studies (Grois 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.

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References
Hypocalcaemia and calcitonin precursors in critically ill patients

We read with interest the paper by Baines and colleagues 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, we have similarly found no correlation between plasma concentrations of calcitonin precursors (CTpr) on admission and both the admission and lowest (within 72 hours of admission) adjusted total serum calcium concentrations (unpublished data). The concentrations of CTpr were significantly higher 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 colleagues 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 pancreatitis or in experimental models of the disease. Whilst CTpr concentrations were reported to rise significantly in critically ill patients, they correlated rather weakly with a concomitant fall in serum ionised calcium. A rise in CTpr concentrations did not correlate with the fall in serum calcium concentrations in patients with acute malaria. This suggests that factors other than calcitonin and CTpr are involved in the homeostasis of calcium in the critically ill.

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References

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.