Concerning use of conjugate pneumococcal vaccine, the most recent CMO letter sent out in August 2002, updates the recommendations issued by the Department of Health (DoH) in January 2002 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 paper which was subsequently cited in the recent RCPCH guidelines for immunisation of immunocompromised children. 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 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. 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 the results of both reviews were similar: “we found no association between temperature difference and the age of the child.”

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. 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 proceeded 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 feverish infant, how accurately does tympanic thermometry measure core temperature? They took rectal temperature and tympanic temperature and restricted their search to work on children. They found two directly relevant studies and one systematic review. They judged that the results of thorough systematic reviews and the underlying temperature and similarly “we found no association between temperature difference and the age of the child.”

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 answering “the results of thorough systematic reviews and . . . 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.

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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 topic. The first 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 answering “the results of thorough systematic reviews and . . . conscientiously, explicitly and judiciously, use them to
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 to whom 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 only painlessly just before weaning from the ventilator.

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

PostScript

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References

LP and Glasgow coma score

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

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 Cant in the same issue of your journal quote a GCS <8. Renneck et al also use a GCS <8 as their cut off figure in their hospital, as do we.

There is little evidence to my knowledge. A retrospective Manchester study found that children with GCS <8 were more likely to die from coning than other children with meningitis (relative risk 4.6, 95% CI 1.06–15.8).

It would welcome correspondence 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.

References

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 most conservative value (<13), which is 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.

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.

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

Is are hospital 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 ability 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 the 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 on the same 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 I would not see 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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Reference

A self-fulfilling prophecy?
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 one in five children has a fever, with fever, we still need to do 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 misused: 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 originally start antibiotics and request audiometrically, we still need to do lumbar punctures.

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Reference

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. The prevalences of anaemia, iron deficiency, iron-deficiency anaemia, and H pylori infection were 81.9%, 91.9%, 31.3%, 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. 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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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 common 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 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.

As the authors point out, pituitary-hypothalamic axis involvement is caused by direct infiltration of these structures by pathologic 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 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.

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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 severe attacks. 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