Correspondence

References

Diagnosis of neonatal bacteraemia

Sir,

I would like to comment on two letters in your March 1989 issue. Miceli Sopo et al believe that values for positive predictive accuracy of '56–8%,' 59%, or 39–62% are unacceptable while pointing to the 'great potential usefulness of the negative predictive accuracy of the evaluated tests.' However, they neglect to mention the incidence of sepsis in the populations evaluated. As my paper was quoted, I should indicate that the incidence of sepsis in that study was only 8%, so that a positive predictive accuracy of 39% has improved predictiveness by almost five times—hardly 'unacceptable'. Conversely, the chance that sepsis was not present was 92%, so that the negative predictive accuracy of 99% is not quite as impressive as it might at first appear.

In their study there were 17 infected babies (15 with documented sepsis) among 70 preterm infants, with positive predictive accuracy of 59% and negative predictive accuracy of 91–6%. These numbers should be compared with the incidence of infection of 24% and 'no infection' of 76% (that is, the chance of predicting each category without the tests being available).

Dellagrammaticas et al report their experience with C reactive protein, which suggests that the response may vary with the infecting organism, that sensitivity is lower in the first 24 hours after delivery, and that C reactive protein needs to be considered together with haematological tests. Like Mathers and Pohlandt, I have observed that C reactive protein may lag behind the immature/total neutrophil quotient, especially in early onset infection due to group B streptococcus.

These observations emphasise the difficulty in evaluating any diagnostic tests for sepsis, because different organisms may produce different patterns of response. The paper by Kite et al was heavily weighted with cases of coagulase negative staphylococcal bacteraemia (21 of 34). The lack of sensitivity of the immature/total neutrophil quotient with this organism has previously been noted, whereas it is more likely to be deranged with early onset group B streptococcal infection.

It is difficult to escape the conclusion that we need to evaluate more than one test when attempting to diagnose neonatal bacteraemia and that different organisms (as well as different severity of illness) may produce different responses.

References

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Does dexamethasone suppress the ACTH response in preterm babies?

Sir,

We are interested in the need to consider steroid replacement treatment for intercurrent infection or surgery in babies who had been on dexamethasone for chronic lung disease. We used a protocol similar to that of Rennie et al for dexamethasone and stimulation by adrenocorticotropic hormone (ACTH). The cortisol responses to tetracosactrin (Synacthen, Ciba) in our six babies were similar to those reported in their paper. The tetracosactrin (36 μg/kg) was given to six infants one week after the dexamethasone had been stopped and again four weeks later to four infants; the results are shown in the table.

We would agree with Rennie et al that steroid replacement treatment may not be necessary. In our series all of

| Table Cortisol concentrations after treatment with tetracosactrin. Results are median (range) |
|---------------------------------|-----------------|-----------------|
|                                 | One week after stopping dexamethasone | After further four weeks |
|                                 | (n=6) | (n=4) |
| Baseline | 128 (25–400) | 276 (30–375) |
| After tetracosactrin | 802 (345–1420) | 1065 (380–1560) |

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the babies responded to tetracosactrin even if their baseline concentrations of cortisol were low. By contrast three babies out of eight tested by Arnold et al with low baseline concentrations of cortisol did not respond appropriately at initial testing but had normal responses one month later. We would presume that babies with ongoing problems are less likely to respond normally and that in such infants baseline cortisol concentrations should be assessed at times of additional stress, at least for the month or so after cessation of dexamethasone. Babies without ongoing problems appear likely to have normal responses and baseline cortisol concentrations may be expected to be normal.

References

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Immunoreactive trypsin in Shwachman’s syndrome

Sir,

Dr Dossetor and colleagues report finding low serum immunoreactive trypsin in two children with Shwachman’s syndrome. Their observation, however, together with the suggestion that a low serum immunoreactive trypsin may obviate the need for invasive tests of pancreatic exocrine function, hardly seems novel. Low serum immunoreactive trypsin(ogen) concentration has previously been described in Shwachman’s syndrome and low values are known to correlate with low output of trypsin in response to stimulation testing of the pancreas. As cystic fibrosis is the only cause of pancreatic insufficiency more common than Shwachman’s syndrome in young children and is associated with a raised serum immunoreactive trypsin, it is evident that a low serum concentration is likely to point to the diagnosis of Shwachman’s syndrome. Through screening large numbers of children the authors have shown that a low serum immunoreactive trypsin as a test for pancreatic exocrine insufficiency has a high specificity, but apart from this, isn’t this report more a case of reinventing the wheel?

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Drs Dossetor and Heeley comment:
The initial title of our paper was ‘Immunoreactive trypsin in Shwachman’s syndrome in early infancy’, but this was shortened in revision for publication. Unfortunately this has resulted in Dr Puntis missing the point of our paper, which was to show the value of the immunoreactive trypsin test in the investigation of an infant with malabsorption. In the paper of Durie et al, the age of the patients is 2-25 and in that of Moore et al, the mean age of the patients is 5-9 years (although in this paper there may have been one infant with Shwachman’s syndrome, but it is not made clear).

Also a serious flaw in these two publications lies in their control values. In the 1981 paper the mean immunoreactive trypsin in controls under 2 years is 7 µg/l and over 3 years 13 µg/l; but in the 1986 paper, the mean immunoreactive trypsin in controls has risen to 31-4 µg/l with no change in the methodology. In patients with pancreatic steatorrhea, a mean value of 4-9 µg/l is found, so that certainly the authors show low values in pancreatic disease. We, however, showed undetectable concentrations in two infants with Shwachman’s syndrome against a larger number of controls at different ages of infancy, establishing beyond doubt the diagnostic value of the test.

We feel the value of the test is insufficiently known to general paediatricians. With the spread of screening for cystic fibrosis, the immunoreactive trypsin test is now generally available. The diagnosis of significant pancreatic acinar deficiency is as easy as diagnosing iron deficiency with this test. We could, however, agree with Dr Puntis on one point, that in the investigation of an infant with malabsorption, the immunoreactive trypsin test ranks in importance with the discovery of the wheel.

The thermal environment in which 3-4 month old infants sleep at home

Sir,

Dr Wailoo and colleagues have provided valuable data in an area that has hitherto received little attention but may be of considerable clinical relevance. It would be helpful to have more information on two points. First, in calculating the total insulation of clothing and bedding did they make allowance for the proportion of the baby covered by each item? For example, a cardigan and a duvet