Is there a place for “drive thru” management of neonatal fever? Not yet!

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Perspective on the paper by Marom *et al* (see page F15)

The non-specific symptoms and signs of severe bacterial infection (SBI) in the neonate means that for reasons of safety many children with fever are hospitalised and treated with antibiotics for non-existent bacterial infections. Psychologists, microbiologists, parents and hospital managers have taught us that admission is something to be avoided, although for some families the perceived safety of a children's ward can be very reassuring. Thus, a study that helps to discriminate the neonate with infection from one without infection is to be welcomed.

What have Marom *et al*¹ shown? There was an extremely low rate of SBI (1/166; 0.6%) among infants fulfilling low-risk criteria, as follows: previously well, good appearance, no focal signs, a low erythrocyte sedimentation rate, a white cell count of 5000–15 000/mm³ and normal urinalysis. The relatively high risk for SBI in the other group (107/220; 48.6%) was accounted for by a large number of children with urinary tract infections (n = 54) and with otitis media (n = 36); otitis media is regarded as relatively unusual in the neonate. In this study all infants were treated, but it was suggested that further research be performed with low-risk babies observed but left untreated with antibiotics.

Several factors need to be considered in this discussion. Neonatal infection is of overwhelming importance; >20% of the 133 million children born in developing countries each year develop neonatal infection.² For many of these children, hospital assessment or admission is not an option, and preventative strategies such as immunisation and breast feeding are of added importance. However, in this population, the World Health Organization Young Infant Study³ identified 14 physical signs of value. When any one of these was positive there was a sensitivity of 87% and a specificity of 54% for SBI.

Different patterns of neonatal infection exist, with considerable regional variation

in the proportion of early infections, such as those caused by group B streptococci and Gram-negative bacteria. In the UK, for instance, there were 376 reported neonatal group B streptococcal infections in 2001, giving an incidence of 0.5 in 1000 live births, which is likely to be an underestimate.⁴ It is possible that early-onset neonatal infections produce more subtle signs, making the proposed assessment less reliable. There do not seem to be any early-onset infections in Marom *et al*'s series; indeed, he refers to the publication of Chiu and Lin,⁵ who described an infant with *Escherichia coli* bacteraemia and meningitis, originally allocated to the low-risk group.

The work of Dagan *et al*^{6,7} provides the foundation for Marom *et al*'s study: infants aged <3 months were considered to be at low risk for SBI if they had been previously well, had no signs of ear, soft-tissue or skeletal infection, had a white cell count of 5000–15 000/mm³, <1500 band cells/mm³ and normal urinalysis. This approach in a population with a median age of 37 days provided a sensitive indicator for SBI.⁶ The authors went on to apply the criteria for low or high risk to infants aged ≤2 months, with almost half discharged home after the initial assessment.⁷ Of the 148 infants classified as being at low risk, six switched to the high-risk group. The authors conclude that management of fever in selected young infants is feasible if meticulous follow-up is provided. This seems to have been the view of a panel of paediatricians and infectious disease specialists who believed that:

... under most circumstances all febrile infants less than 28 days of age including those in the low risk group should have a sepsis evaluation and be hospitalised for parenteral antimicrobial therapy pending culture results ... an alternative management strategy for low risk infants less than 28 days of age is a complete sepsis evaluation, hospitalisation and careful observation

without parenteral antimicrobial therapy pending culture results⁸

The same author, Baraff, 7 years later,⁹ modified this view only slightly: “treatment of infants younger than 4 weeks of age as outpatients with either strategy should be done only when the parents are reliable and close follow up is assured”.

So if we were to adopt a screening system, what tests would be included? A difficult question because of the wide variation in tests available: for instance, the counting of immature neutrophils or band cells is labour intensive and is rarely practised in the UK; the erythrocyte sedimentation rate is favoured in the USA and the C reactive protein level in Europe. And ideally, each group of tests needs to be tested on the population to which it is applied. Recent work has suggested that measurement of procalcitonin is more sensitive and specific than CRP in predicting SBI, especially in young infants.^{10,11} Although many paediatricians in hospitals are using a combination of tests on young infants with fever, office-based paediatricians seem to be successful in “using individualised clinical judgements” alone.¹²

I believe that the real issue for the neonate with fever is recognition by parents and medical attendants that this requires urgent assessment; death and disability from neonatal infection are all too common in affluent societies because the importance of fever is ignored.

So how should we deal with fever in the neonate—a condition that can have devastating results? By education of parents and their medical carers. Certainly in the UK, there is no substitute for observation and treatment in a paediatric ward where the child will have the benefit of an experienced medical assessment and ongoing observation. Screening tests undoubtedly have an important role. We hope to have further guidance from the National Institute of Clinical Excellence on management of fever in the young child in early 2007. Meanwhile, I recommend the screening of all neonates with fever using procedures described above, combined with observation in a hospital setting for up to 48 h if antibiotics have been withheld.

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Superior sternal cleft, cutaneous, and airway haemangiomas

A term baby, born to a 19 year old healthy mother by emergency caesarean section because of failure to progress, was noted to have a superior V shaped sternal cleft, measuring 4 cm across with lower ribs meeting in the midline (fig 1). The mother had no significant medical history.

Apart from a visible pulsatile heart, cardiological examination was normal. She did not have a midline abdominal raphe. At three weeks, she gradually developed superficial capillary haemangiomas over the lower sternum, lower lip, neck, and right side of the face (fig 1).

A computed tomography scan of the thorax at 2 months of age revealed a membrane in the place of the sternum, with normal lungs, major vessels, and vertebrae.

At 3 months of age, she presented with stridor. Excision of the sub-glottic haemangioma was performed, with partial excision of the vocal cord. She is fed by gastrostomy because of aspiration. She is developing well while awaiting repair of the sternal defect.

Superior sternal cleft is almost always an isolated abnormality but can occasionally be associated with cervicofacial haemangiomas (vascular dysplasia)¹ and PHACES (posterior fossa brain malformations, facial haemangiomas, arterial anomalies with coarctation of aorta, cardiac defects, eye abnormalities, sternal cleft, and supraumbilical raphe) syndrome.² The aetiology of this condition (OMIM 140850) is not known. Facial haemangiomas are common in female infants.³ Most cases are sporadic with few reported recurrences in siblings.⁴ Studies on ventral body development in mice indicate impairment in Hoxb gene expression as a possible cause of sternal defects.⁴

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Figure 1 Superior sternal cleft with superficial capillary haemangiomas over the lower sternum, lower lip, neck, and right side of the face. The patient's parents have given permission for publication of this figure.

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