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Umbilical disinfection: lessons from history

A baby girl was born by spontaneous vaginal delivery at 38 weeks to a healthy mother in a district general hospital. She was the second child in the family; there was no family history of concern, her mother was well, and the pregnancy had been uneventful. Breast feeding was established, and mother and baby were discharged home the following day with no specific advice as to umbilical care. At 10 days of life there was a yellowish discharge from her umbilicus; this was treated with topical fucidin by the general practitioner. Six days later there were increasing difficulties with breast feeding as the infant became more listless. The following morning, the mother noted swelling of the left knee: on admission a few hours later the infant was hypotonic, sleepy, and afebrile with a discharging umbilicus.

Aspirate from a tense effusion of the left knee, umbilicus, and blood cultures all grew *Staphylococcus aureus*, which was sensitive to flucloxacillin but resistant to fucidin. The infant was treated with intravenous antibiotics and a joint washout by a paediatric orthopaedic surgeon that day. The baby appears to have had a good outcome: six months later she shows normal development and equal limb length. This case shows a systemic complication consequent to umbilical colonisation and sepsis by a staphylococcus resistant to fucidin.

Late in the nineteenth century, staphylococci were among the first bacterial organisms to be clinically identified as the causative agent in epidemics of pemphigus neonatorum, a pustular skin condition associated with maternal mastitis.[1] It was found that newborns are rapidly colonised by staphylococci, streptococci, or *Escherichia coli* on the umbilical stump; infection or toxin production readily led to omphalitis.[2] The application of antiseptic treatments became critical to reducing epidemics of perinatal illness.

Over the last 25 years, the use of umbilical antisepsis has almost disappeared in British hospitals. Concerns about the potential toxicity of chlorhexidine, the unsightly appearance of triple dyes, and the limitations of other methods combined with a low incidence of pathology has resulted in a non-interventionist approach in most maternity units. A Cochrane review has concluded that, in the developed world, simple hygiene is as good as any other treatment.[3] In this same time frame, however, antibiotic resistance has spread, altering the microbial environment of maternity and neonatal units. Rates of neonatal staphylococcal septicaemia—always a changing pattern—may be increasing. Admission times of mothers and infants in district general hospitals have shortened too, facilitating a rapid bacterial exchange between our hospitals and communities among healthy individuals.[4] This combination of events may become a risk factor in the spread of antibiotic resistance in the United Kingdom and a major source of neonatal infection. A parallel pattern has been observed in Japan with neonatal toxic shock syndrome caused by the spread of an methicillin resistant *Staphylococcus aureus* (MRSA) clone.[5]

It might be reasonably argued that there should be a reintroduction of formal umbilical sterilisation, aimed at reducing the bacterial load in our maternity units. Reducing the burden of colonising organisms by the use of topical or systemic antibiotics has been shown to reduce subsequent invasive infection in renal dialysis.[6 7] This approach has been limited by the emergence of antimicrobial resistance to mupirocin, and antisepsis may therefore be a more appropriate long term strategy. Would the production of an appropriate guideline, ensuring umbilical disinfection as well as maternal and staff hand washing in maternity units reduce risks to infants and limit bacterial traffic and potential antibiotic resistance? The debate on the cost effectiveness of this approach should be reopened and the lessons of history reconsidered.

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Effect of laser photocoagulation for retinopathy of prematurity on C reactive protein

Retinopathy of prematurity (ROP) is the major cause of blindness in premature babies, and in the developed world it is a disease of the most immature and unwell infants. Treatment is needed more often in extremely premature babies, taking place in 14% of those born before 26 weeks completed gestation.[1] Treatment of severe ROP was originally by cryotherapy, but is now more commonly undertaken with laser, either as transpupillary or trans-scleral diode photocoagulation.

C reactive protein (CRP) is an acute phase protein first discovered in the 1930s. It is a component of the innate immune system. Increased production is thought to be beneficial because of its ability to bind phosphocoline and thus recognise foreign pathogens.[2] Tissue damage including burns, trauma, and surgery is known to increase CRP.

Babies undergoing treatment for ROP often have regular CRP measurements to help identify the onset of sepsis. There is currently no evidence as to whether cryotherapy or laser photocoagulation cause a rise in CRP irrespective of sepsis.

To ascertain whether CRP is affected by treatment of ROP, a retrospective case note review of infants treated with laser photocoagulation was undertaken. The notes of 16 infants (11 female and five male) requiring treatment for ROP (mean gestational age 24 weeks and 6 days) were reviewed. Fifteen of the babies received laser photocoagulation by transpupillary diode laser, and one received trans-scleral diode treatment.

CRP concentration before surgery and the first measurement after surgery were obtained. CRP was measured using an automated discretionary discrete analyser (Olympus 600; Olympus Optical Equipment). Units are mg/l.

The time interval between the operation and CRP measurement was 12 hours to 12 days; the median time was three days. CRP concentration before surgery ranged from < 5 to 53 (median < 5). After surgery, the range was < 5–51 (median < 5). The data were non-parametric. A Wilcoxon signed rank test was used to determine whether the null hypothesis (lasering does not alter CRP concentration) was correct. This study did not find a significant difference in CRP concentration after surgery. (Sample size means this study has a 90% power to show significance ($p < 0.05$) if there were a difference in CRP concentration of 20.)

Our negative findings give some reassurance that if, after treatment for retinopathy, there is concern about infection, a raised CRP concentration is unlikely to be a result of the procedure and more likely to reflect sepsis. However, a prospective study of CRP concentrations after treatment for retinopathy will be necessary to confirm our findings.

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