Correspondence

Concern over safety of SAGM blood

Sir.

Some Regional Blood Transfusion Centres are recommending for transfusion a product in which electrolytes are suspended in a saline-adenine-glucose-mannitol medium, hence the name SAGM blood. All the plasma from the donated blood has been removed, we understand, for use in the preparation of other blood products.

A Medline search has not shown any reference to the safety of this product in neonates, and we are concerned because several of our members have been urged to use it. Until the product has been properly evaluated in neonates, and possible side effects of its supernatant have been clarified, we would advise caution in its use even for top-up transfusions. Moreover, it is a wholly inappropriate product for exchange transfusions, where its use would result in total replacement of the patient's plasma by a synthetic product. This especially applies to exchange transfusions carried out to provide babies with antibodies or clotting factors in cases of sepsis or haemorrhage.

We are interested to know if any readers have had experience in using SAGM blood in neonates and whether they have recognised any adverse effects.

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Prolonged flare and periventricular leucomalacia (PVL) in preterm neonates

Sir.

We read the paper by Trounce et al with interest. In this article periventricular leucomalacia (PVL) was divided into three appearances: cystic, precystic, and ‘prolonged flare’. The latter was defined as an appearance of relative increased echodensity in the periventricular region seen both in coronal and parasagittal views and persisting for at least two weeks but not undergoing cystic degeneration.

We wonder whether this term ‘prolonged flare’ comprises only ‘large intraparenchymal echodensities’, as defined by McMenamin et al and ‘globular, blotchy, coarse echoes’, as defined by DiPietro et al, or whether it includes also ‘small intraparenchymal echodensities’ and ‘peritritchional echogenic blush’. According to the necropsy results, infants with the latter two findings may or may not have PVL.

In some cases this ‘blush’ disappears when the scan is obtained through the posterior fontanelle. The best explanation for it could be the interface of numerous parallel fibres that are nearly perpendicular to the longitudinal axis of a sonographic beam passing through the anterior fontanelle. In other cases it does not disappear and, these might be those with PVL. On the other hand, a large and globular, coarse periventricular echodensity, often unilateral, may more likely be an ultrasonic manifestation of focal hypoxic-ischaemic lesion caused by a hypotensive insult, than PVL, which is usually bilateral.

The duration of echodensity indicating PVL is the other problem. Why did Trounce et al prospectively, as they say, choose two weeks for it? Why not four weeks? Was this choice arbitrary or was it based on some clinical follow-up results? If it was based on necropsy findings they do not provide any reliable answers as far as surviving neonates are concerned.

References


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Drs Trounce, Rutter, and Levene comment:

Thank you for your interest in our paper. The question is that of comparison between our definition of ‘prolonged flare’ and alternative definitions by other groups. At the outset of our study (1983) we defined carefully a variety of ultrasound appearances. We were aware of McMenamin’s definition but that of DiPietro et al has only recently been published and was not available to us originally, or before we submitted our paper for publication to the Archives of Disease in Childhood. We have been impressed, on scanning many hundreds of infants, that persistent echodensity in the periventricular white matter is a common finding and is often normal, particularly when the occipital region is affected. We rarely saw this appearance in two planes, however, and if it did appear on coronal and parasagittal section it was usually transient, lasting only for a few days.

Our rigorous definition of prolonged flare was developed so that most of these appearances would not be included. On careful reading of the paper by DiPietro et
it would appear that this ‘blush’ is only diagnosed on parasagittal section. This is therefore not at all comparable with our definition, which must be seen on two planes. In addition, these authors give no information on how long the echos were required before they considered the blush to be abnormal. Indeed, the babies died largely within a few days of age, so clearly this would not fit in with our definition. It is, however, interesting to note that the prevalence of PVL in 68 consecutive liveborn infants dying under two months of age in their unit was 26%. This compares very favourably with the 25% incidence of PVL that we recognised prospectively using ultrasound on our 200 very low birthweight babies.

The point of our study was to set up a prospective database of 200 infants who were scanned sequentially and in whom there was clear understanding of what we meant by our ultrasound appearances. Our definitions were, of course, arbitrary as we did not have the benefit of that time of clinical follow up. We have now completed follow up collection on all the surviving infants and the relevance of our definition of prolonged flare will hopefully soon become apparent.

Reference

Tracheobronchial suction in ventilated neonates

Sir,

We congratulate Dr Prendiville et al on their successful attempt to investigate the efficacy of tracheobronchial suction in ventilated neonates.1 This aspect of the routine care of ventilated babies has important consequences. Knowing when, how often, and how to perform endotracheal tube toilet is difficult, and their study provides some of the answers. Unfortunately, continuous monitoring of airways resistance by flow-volume integration using a double pneumotachograph system seems unlikely to be practical for routine clinical use.

The single breath technique for determining airways resistance relies on passive expiration. Most ventilated babies with the respiratory distress syndrome make use of their abdominal muscles to expire actively (South M, Morley C J. Expiratory muscle activity in premature babies. Presented to Neonatal Society, November 1986). The authors state that spontaneous respiration was stopped by increasing ventilator rate. This is unusual in our experience. We find that at faster ventilator rates most babies continue to breathe spontaneously but synchronously with ventilator inflations.2 When breathing synchronously, they can seem to be apnoeic, with linear flow-volume curves. Expiratory muscle activity often continues during synchronous breathing, and this could shorten expiratory time constants and reduce the calculated resistance. Despite these reservations we are sure that the results of this paper are helpful. We would recommend, however, that an independent method for detecting the presence of spontaneous respiration should be used.3

References

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Infantile colic and feeding

Sir,

Unfortunately the paper by Stähleberg and Savilahti1 adds to the confusion that already exists regarding an association between infantile colic and cow’s milk.

They failed to define colic and, therefore, the criteria for entry into the study. The mean age of the babies at entry was nearly 3 months by which time a large number of babies with ‘colic’ will be improving spontaneously. The duration of feeding on each milk preparation was too short to show any important differences, and there was no washout period between each milk, with the likelihood there will be a carry over effect from the preceding preparation.

Analysis of the data in Table 1 in their paper shows that eight of the ten babies received breast milk first with or without the lactase so that the results do not report adequately on the relative impact of formula followed by breast milk. In addition, if the data are analysed by order of administration of treatment2 the proportion of days with colic declines from 86% for the first treatment, through 81% and 79%, to 74% for the last treatment given. There is, therefore, a clear trend of improvement over time so that treatments given first tended to do badly.

The small size of the study also gives cause for concern. The authors conclude that ‘infantile colic is not a symptom of lactose malabsorption’. It is doubtful whether the study could have provided adequate statistical power to detect any true differences that may exist. Finally, a repeated measures analysis of these data should have been done, rather than the statistically invalid methods used in the paper.

It is only by an appropriately designed trial using an adequate sample size that the question of an association between cow’s milk and infantile colic may be answered.

References

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