determining if one method was biased compared with the other. However, this does not answer the more important question, 'Does the TMS-derived gestational age agree sufficiently well with the LMP-derived age for individual babies?' To answer this question it is necessary to consider the distribution of the within-person differences between the ages derived by each method (see, for example, Wu *et al.*²).

A separate criticism of this paper concerns the division of LBW babies into term and preterm groups. While it may be of interest to see if the results are different for the two groups, the only relevant analysis is that for all LBW babies together. The whole point of the exercise is to see if the TMS method can accurately assess gestational age, and distinguishing between LBW babies of different gestations is an important aspect which Cater's approach cannot fully consider. Even if the statistical approach were valid, a conclusion that the method works for term babies is of limited value if you do not know exactly which babies these are.

I recently made similar remarks³ about a paper by Serfontein and Jaroszewicz⁴ in which two other methods of assessing gestational age (Dubowitz's and Robinson's) were compared. Serfontein and Jaroszewicz⁴ had used correlation—a method which Dr Cater rightly rejects, but for the wrong reason. Their reply to my previous letter dismissed my criticisms as minor. They were however, fundamental, as are my criticisms of Dr Cater's paper. It is not statistical quibbling to raise these points. Each type of analysis tests a different hypothesis; if you choose an incorrect analysis you will test an inappropriate hypothesis.

As in the earlier paper, Dr Cater's conclusion that two different methods are comparable is totally unjustified. In both cases the authors have collected the correct data to enable them to answer their question, but have used incorrect analyses, and in both the data they present suggest that agreement may not be particularly good.

A minimum requirement for the comparison of methods is a scatter diagram relating the methods and indicating the line of equality, together with an analysis of the within-person differences between methods.

References

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Dr Cater comments:

I am grateful to Mr Altman for his interest and comments. The infants studied were a sample from a larger survey of LBW babies and their controls were matched for paternal occupation, ordinal position in the family, maternal height, smoking, and sex of the baby. Those of certain gestation were selected as controls and were therefore not strictly matched.

Mr Altman is right in saying that the paired *t* test will not prove the equivalent of TMS and LMP methods. It is however a necessary if not sufficient condition if the two methods are equivalent for the means to be the same. It is precisely for this reason that the further analysis of subgroups is relevant since the equivalent of means for these subgroups should also be demonstrated.

I cannot agree that the only relevant analysis is to consider all the LBW babies together. Detection of bias in the TMS for particular subgroups may suggest possible recalibration to improve the scoring system.

As clinical methods for corroborating maternal menstrual data in neonatal work become more widespread, it is important to be aware of both the clinical and the statistical limitations of the available scoring methods. Further study of this is proceeding.

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Exchange transfusion in newborn infants

Sir,

We were interested to read the report by Pelet¹ of the effect of exchange transfusion on opsonisation and granulocyte function. While he clearly shows that exchange transfusion increases the capacity of serum to promote phagocytosis of heat-killed bakers' yeast, this may not be the same phenomenon as correction of the isolated defect of yeast opsonisation by plasma as described by Soothill and Harvey.² Newborn infants have a number of immunodeficiencies and one in particular, namely deficiency of factor B of the alternative pathway of complement,³ is likely to be important in the pathogenesis of defective yeast opsonisation. We have shown that children with fulminant hepatic failure (FHF) not only have defective yeast opsonisation, but also profound defects of functional activity of both C3 and factor B (V F Larcher, R J Wyke, A P Mowat, R Williams, in preparation). The addition of subopsonising amounts of normal sera to FHF sera restores yeast opsonisation, as it does in children with primary yeast opsonisation deficiency, but addition of factor B-depleted sera, even in equal volumes, did not provide more than 50% restoration of opsonising activity. On the other hand, the opsonising capacity of sera from patients with primary isolated yeast opsonisation deficiency can be fully restored by the addition of factor B-depleted sera.