These results suggest that normal yeast opsonisation is also, to some extent, dependent on adequate functional levels of factor B. Therefore, unless activity of other complement components is measured in parallel, care is necessary in the interpretation of the yeast opsonisation test.

Finally, the deficiency of yeast opsonisation found in two of Pelet's donors may not have been due to incorrect storage of blood but to primary yeast opsonisation deficiency, which occurs in 5% of the normal population. The use of plasma from such individuals was thought to have been the explanation for the poor opsonic response to plasma infusion in one of our patients with protracted diarrhoea and defective yeast opsonisation.⁴

We believe that these observations stress the need to screen potential donors of blood for exchange transfusion for white cell and opsonic function if this procedure is to be used rationally in the treatment of septicaemia. The opsonising capacity of sera from normal healthy individuals does not seem to alter markedly with time (personal observations) so that there is a case for the prospective estimation of this parameter at least in potential donors.

References

- 1 Pelet B. Exchange transfusion in newborn infants: effects on granulocyte function. *Arch Dis Child* 1979; **54**: 687-90.
- 2 Soothill J F, Harvey B A M. Defective opsonisation. A community immunity deficiency. *Arch Dis Child* 1976; **51**: 91-9.
- 3 Stossel T P, Alper C A, Rossen F S. Opsonic activity in the newborn: role of properdin. *Pediatrics* 1973; **52**: 134-7.
- 4 Candy D C A, Larcher V F, Tripp J H, Harries J T, Harvey B A M, Soothill J F. Yeast opsonisation in children with chronic diarrhoeal states. *Arch Dis Child* 1980; **55**: 189-93.

VICTOR LARCHER AND ALEX P MOWAT
Department of Child Health,
King's College Hospital Medical School,
Denmark Hill,
London SE5 8RX

Dr Pelet comments:

I appreciate Drs Larcher and Mowat's comments concerning a possible defect in the 'functional activity' of both C3 and factor B. There is increasing evidence that *in vitro* factor B and C3 activation is defective in newborn sera.¹⁻³ I found no correlation between the level of factor B or C3 and the grade of opsonisation deficiency as indicated by the opsonisation index. Furthermore, studying synthesis of C3 and analysing the degradation products both of factor B and C3 in normal and septicaemic newborn sera before, during, and after exchange transfusion, I came to the conclusion that *in vivo* as well as *in vitro*, such an activation deficiency should be postulated.³ Larcher and Mowat's observation on FHF seems to be another clinical situation where a similar defect is observed. In cases of hepatic failure there could be an additional and different mechanism, since liver is one of the sites for the synthesis of C3.

I fully agree that potential donors of blood for rational septicaemia treatment should be screened for white cells and sera functions.

References

- 1 Winkelstein J A, Kurlandsky L E, Swift A J. Defective activation of the third component of complement in the sera of newborn infants. *Pediatr Res* 1979; **13**: 1093-6.
- 2 Adamkin D, Stitzel A, Urmson J, Farnett M-L, Post E, Spitzer R. Activity of the alternative pathway of complement in the newborn infant. *J Pediatr* 1978; **93**: 604-8.
- 3 Pelet B. C3, factor B, α -1-antitrypsin in neonatal septicaemia with sclerema; effects of exchange-transfusions and kinetic study of C3 using phenotypes as markers. *Arch Dis Child* 1980; **55**: in press.

BERNARD PELET
Service de Pédiatrie,
Centre Hospitalier Universitaire Vaudois,
1011 Lausanne, Switzerland

A suggested child-health clinic form

Sir,

Professor Illingworth¹ invited comment on intervention programmes for children who speak late and have been properly tested for hearing defect, and of the effectiveness of such programmes. Cooper *et al.*² set out to find and prove an effective way of helping language development in children with early language handicaps. Their 5-year study³ of 119 children in the age range 2 to 4½ years, together with field trials at clinics, showed that most 'programme' children made accelerated progress in all language-related areas of development and that this improved rate of progress was maintained. Sonksen⁴ showed that the accelerated progress occurred in children with all degrees of handicap and there was no evidence that it was related to degree, nor was there a relationship to the paediatric categories of 'causal' or 'developmental'. The Wolfson intervention programmes⁵ introduced last year by our speech therapists at the Newcomen Centre give encouraging results.

Professor Illingworth also questions the need for routine vision tests for children under age 5 years, relying instead on nystagmus, opacity, or persistent squint to reveal treatable visual acuity problems. The Stycar distant vision tests in daily use at the Newcomen Centre give convincing evidence of reliability in picking up visual impairment, and lead to early referral to an ophthalmologist for refraction. Near vision tests are more difficult to interpret. The 6-month-old infant's interest in a 1 mm sweet is taken as an indication of adequate near vision. Ophthalmologists are becoming increasingly concerned by the late discovery of children with squint and amblyopia, which have escaped detection until visual acuity is tested. Ingram,⁶ in a review of all cases referred to hospital and school eye clinics in his district in one calendar year, found that the majority (69%) of amblyopes presented after 5 years of age. Little more than half the children with esotropia had a cosmetically noticeable squint. He pointed out that no improvement can be expected for either straight-eyed amblyopia, or for the