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.

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


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

References


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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

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.8 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.
child with a squint that is not cosmetically noticeable, unless visual acuity is checked earlier than school entry. Assessment of function in each area of development is surely basic to developmental assessment?

References


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Human growth hormone (UK)

Sir,
A paragraph in the Annotation by Milner1 has been interpreted by some of my professional colleagues as meaning that I have acted in breach of Rule 15, paragraph 3 of the Rules of the BPA and been guilty of unprofessional conduct. The collocation on p.734 of a quotation from my address to the annual meeting of the BPA on 28 March 1979, and the immediately succeeding sentence: ‘The next day ... poor growth youngsters’ coupled with Rule 15, paragraph 3 of the Rules of the BPA clearly implies that I communicated my views to the Glasgow Herald, or was a party to the Glasgow Herald publishing its article.

The facts are that I have for some time been treating patients at the Royal Hospital for Sick Children, Glasgow, for growth hormone deficiency with hGH (UK). Recently I became disturbed about the possibility of contaminants in the hGH (UK) adversely affecting some of these patients. I therefore invited the parents of all my patients to come to the hospital and explained to them that I had become aware of the overall nature of the preparation in use and that from my search of the literature I could not find an irrefragable statement that the contaminants had no deleterious effects on children. I made the point that it was right and proper that parents should be aware of the facts. I understand that a parent of one patient communicated with the press.

At the meeting in York I observed a newspaper reporter, for whose presence I was not responsible. This reporter had approached me some time previously but I declined to make any statement. The articles in the Glasgow Herald of 29 March headed:

‘Scots doctor challenges growth treatment’: HGH ‘Wonder cure’ has turned sour for our ‘poor growth’ youngsters: Why are the doctors staying tight lipped?: Fearful parents who stopped hormone therapy on daughter: Medical treatment for one child that can cost a cool £40,000 were neither written nor authorised by me, nor was any matter on the subject communicated to the Glasgow Herald or any of its staff either by me or on my behalf, at this time.

The facts accordingly do not justify the inference naturally drawn by some of my colleagues from the collocation of the sentences on p. 734 of the Archives.

In these circumstances I must request you to publish this explanation and to make a suitable correction and sufficient apology to me for the imputation against my professional character.

Reference


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Upper urinary tract anomalies in the congenital adrenogenital syndrome

Sir,
Mcmillan et al.1 noted there is often an association between congenital adrenal hyperplasia and anomalies of the upper urinary tract. We studied 13 patients with congenital adrenal hyperplasia diagnosed by conventional methods to see if such an association exists. The ages of the patients ranged from 1 month to 8 years. 12 of them (7 boys and 5 girls) had a deficiency of 21-hydroxylase, 10 being ‘salt losers’. The 13th was diagnosed as having a deficiency of 11-beta-hydroxylase; she was hypertensive. The 17-ketosteroids in 24-hour urine collections were raised in all patients, with a wide range of values from 3-0 to 9-2 mg/24 h (10-4 to 31-9 μmol/24 h), well above those found in normal children. Excretion of pregnanetriol in the urine was raised in 12 patients (in one it could not be determined). In all patients intravenous pyelography was performed; only one, in a male ‘salt loser’ with...