isolated finding on a neurodevelopmental examination provide information that is useful to a teacher in planning a teaching programme? It is well known that there is a wide range of normal maturation in this age group—a finding at one examination may be no longer present a few months later. And what of a danger of labelling a child, so that a 5 year old who may be otherwise doing perfectly well is labelled as having a problem?

Finally, there are absolutely no data to suggest that an examination of this type makes any difference to outcome. It would have to be shown that children who underwent such an examination at school entry would have an outcome that was better than children not so examined. Such a study would be extremely difficult to undertake because of the multitude of other variables that effect functioning.2

Given unlimited resources, one could perhaps support these examinations because they may provide reassurance to parents and teachers. In most communities such a situation does not exist, and arguments could be made against them from a cost effectiveness point of view. It seems more appropriate that nurses continue to be involved with children at school entry, but only to perform vision and hearing screening and to review health and developmental problems, perhaps with the aid of detailed health questionnaires completed by parents and consultation with teachers. During the first year or two at school, teachers will certainly identify a further group of children having developmental problems, perhaps with the aid of detailed information by the nurse or teacher, or both, as having problems.

While many would support the continued routine school entry examinations, there is precious little evidence that it is either a cost effective or valid method for reducing school problems. In the face of diminishing resources it is essential to provide harder data to justify their existence.

References


Frank Oberklaid
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Drs Bax and Whitmore comment:

Dr Oberklaid’s letter invites us to write another article! The purpose of our own article was to describe our present clinical method of conducting a school entrant medical examination and not to justify the procedure. We have referred to this in passing in other articles (including one by us to appear in Dev Med Child Neurol soon).3 2

Briefly, to take up some of Dr Oberklaid’s points:

(1) We have good evidence that our routine examinations detected health problems that were not known about in the preschool health service. 94% of children whom we discovered had problems had not seen their general practitioner for those problems within the last 12 months and a third of the children we examined had no available preschool health notes; of those for whom notes were available, two out of five had not been seen since the age of 2.

(2) We do not see why the school health service should aim to employ doctors who are less competent than our aged selves.

(3) In the past teachers have often received driblets and drabs of information from doctors outside school, which are confusing to them. It is indeed our practice to discuss in detail our findings with the teacher and to discuss and explain to them the importance of findings. Our examination aims to report on the present developmental state of the child and we are extremely cautious in talking to teachers about drawing any implications for the future. Nevertheless, our neurodevelopmental examination has proved extremely robust in making predictions, as our own data suggests (see our article to be published in Dev Med Child Neurol) and that of other workers who have used our scheme.3

(4) It is quite true that we are bad at treating many forms of neurodevelopmental disorder from cerebral palsy to learning disorders. There seems to be a new philosophy abroad that you do not diagnose unless you can treat; we believe that this is alien to the whole history of medicine.

(5) We are not businessmen and we are bad at deciding whether things are cost effective. We are dismayed though with the fact that community paediatrics is constantly taking the brunt of the cost effective attack and we wonder if the businessmen involved would devote more time to looking at some of the things that go on in hospital and general practice.

References

3 Michaësson K, Ylinen A, Donner M. Neurodevelopmental screening at five years of children who were at risk neonatally. Dev Med Child Neurol 1981;23:427–33.

Oedema and the aging prima donna

Sir.

It may be a case of the aging prima donna but I was a little disappointed to find that Cartlidge and Rutter in their paper on serum albumin and oedema1 made no reference to our studies in this field.2 4 It is of course disappointing to have published work overlooked. However (and perhaps this is the only justification for this letter), our studies included a wide range of measurements of plasma proteins, albumin, and (directly measured) colloid osmotic pressure that might have made useful data for comparative discussion with the Nottingham data. Most particularly I would have been interested to hear their discussion of our apparent findings of a complex relation between plasma proteins, albumin, and colloid osmotic pressure. For all this I do not have any quarrel with the conclusion of their
paper that the interpretation and treatment of measurements of plasma proteins in the preterm infant is greatly overrated, and I think that Cartlidge and Rutter have done paediatrics a service in drawing attention to the curiously large scatter of plasma proteins concentrations in preterm and newborn infants.

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Drs Cartlidge and Rutter comment:
We were sorry to discover that Professor Baum has carried out so much work in the field of neonatal oedema to which we made no reference. This was no intended slight (we were unaware of his work), but his disappointment is understandable. When we set out to perform the study we carried out a search of the published works through *Index Medicus*, using the preterm infant, oedema, and albumin as key words, and choosing papers that seemed relevant by their titles. We knew of the study from 1971 on colloid osmotic pressure in the fetus and newborn infant, but as it contains no data on albumin concentrations or oedema in preterm infants we did not refer to it. The study on colloid osmotic pressure in erythroblastosis fetalis does contain data on albumin concentrations in preterm infants but the title gives no clue to this. The most important reference, the monograph *Oedema in the newborn*, certainly contains a lot of information on oedema and albumin concentrations in the preterm infant, some of which we have duplicated. It does not appear in *Index Medicus*, however, and is not referred to in any article that we have come across. We regret that we were unaware of it, but the exponential rise in the volume of medical publications (to which these letters will contribute) makes such rediscovery of the wheel more likely.

We have since read and enjoyed 'Oedema in the newborn.' The chapter on oedema in the preterm infant provides data on cord albumin concentrations at different gestational ages from 33 weeks onwards. Our values are somewhat lower, perhaps because they were all obtained after birth, but show a similarly large scatter—our findings on infants below 33 weeks' gestation further emphasise the importance of gestation on serum albumin. Baum suggests that oedema in preterm infants is not looked for now compared with the earlier days of neonatology, except in very immature infants. Our data strongly supports this. We do not know why very immature infants develop oedema—it does not seem to relate to illness or serum albumin. Clearly, we have both shown that subcutaneous oedema is not simply due to a low colloid osmotic pressure produced by a low serum albumin concentration. Baum has found that levels of colloid osmotic pressure in preterm infants without oedema are often below 20 cm of water, levels that would produce generalised oedema in a child with the nephrotic syndrome. Perhaps the low mean arterial pressure of the preterm infant results in a low hydrostatic pressure and therefore protects against severe oedema. Whatever the reasons are for oedema, the message seems to be that hypoalbuminaemia of prematurity is a usual finding, not a disorder that needs treatment.

Correspondence

Cystic fibrosis and diabetes mellitus

Sir,

Cystic fibrosis is associated not only with chronic suppurrative lung disease and exocrine pancreatic insufficiency but also with endocrine dysfunction of the pancreas, leading to impaired glucose tolerance, decreased insulin production, and diabetes mellitus in some children. Measurement of glycosylated haemoglobin (HbA1c) has been shown to be useful for assessment of children with impaired glucose tolerance.

We measured stable HbA1c concentrations by a modification of the Corning electrophoresis method in 21 children with cystic fibrosis, aged 1–16 years, regularly attending our clinic, and in 50 normal children, aged 1–16 years. Three of the children with cystic fibrosis were receiving insulin for clinical diabetes mellitus. The mean (SD) HbA1c concentration in the remaining 18 children was 8.1 (1.5)% (range 6.0–11.3%) compared with 6.0 (0.8)% (range 4.3–7.7%) in the normal group (p<0.001). All of the children with cystic fibrosis had HbA1c concentrations equal to or greater than the mean of the normal group and in 55% the HbA1c concentrations were more than two standard deviations greater than the normal group mean.

The degree of glucose intolerance may be relevant to morbidity and problems of growth in children with cystic fibrosis and requires further study. Treatment with prednisone has been shown to improve pulmonary function in such children, and although that study detected no change in HbA1c concentrations, careful monitoring of glucose tolerance would seem to be indicated if this treatment is begun.

We suggest that HbA1c concentrations will be of value for monitoring children with cystic fibrosis and if repeated at six to 12 month intervals would indicate any progressive impairment of glucose tolerance. This would allow earlier detection and treatment of associated diabetes mellitus.

References


3. Auerbach HS, Williams M, Kirkpatrick JA, Colten HR.
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J D Baum

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Updated information and services can be found at:
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