Correspondence

Selective medical examinations on starting school

Sir,

We read with interest the paper by O’Callaghan and Colver.1 We agree that school health services need evaluation. We disagree, however, with the inference that the sole function of the medical at school entry is to identify treatable physical problems.

The total child population must be examined at some stage. The Newcastle team are choosing to do this three times preschool. One is hardly surprised, therefore, at the low number of new problems identified at school entry. In Peterborough, full medical examination and development assessment preschool is largely reserved for those children screened out by the health visitor.

Seeing the total population preschool is notoriously incomplete, particularly in urban areas. We positively retain the school entry medical for this and the following reasons:

1. School ‘fitness’—medical, educational, and social—is seen in the school setting.
2. Parents and children are introduced personally to the school doctor and nurse; this facilitates self referral if problems arise later.
3. It is perceived as important by both teaching staff (National Association of Head Teachers. The NAHT view of the future requirement for school health service provision.) and by parents (99-6% consent to medical, 88% accompany their child, personal observation).
4. In this authority children enter school at the age of 4 years (an age often chosen for preschool medicals).

During the school year 1986–87, 2877 school entrants were medically examined in Peterborough. Altogether 1770 important problems were identified (that is, required action or follow up); 742 of these were discovered on screening vision and hearing—an activity few would dispute. Furthermore, 17-6% of the children had important educational problems that were discussed with teaching staff. A proportion of the problems were known to parents and family doctors before the medical but appreciable numbers were not (table).

There have been tremendous changes in the nature of preventive child health care over the past 10 years. We are now far more involved with behaviour and adjustment problems and health education. We heartily endorse the comments on the importance of the school entry medical contained in the report Investing in the Future.2 We recommend it as an economical use of doctor time.

References


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Sir,

The excellent paper by O’Callaghan and Colver on selective preschool medication examinations voices the misgivings many of us have about developmental screening or surveillance, in general.1 Quite clearly the concept is too wide and unfocused to allow any useful information to be gathered, other than for epidemiological reasons.

My experiences with the six week developmental check are rather similar; over a two year period I examined 218 consecutive 6–8 week old babies in a community paediatric clinic. (This was part of a separate study, which was looking at acute infantile colic and its possible relationship to maternal psychiatric illnesses as measured by the General Health Questionnaire.)2 From the purely medical point of view, however, developmental surveillance was a very disappointing business. I picked up nothing that I could refer to our local paediatrician as a new abnormality. Even worse, every abnormality I did discover was already known to the general practitioner or hospital clinic. A couple of mothers wanted to know ‘if I was going to shake the rattle’ or in some way test the development of their babies. A pass was expected and, even worse, was equated with being intelligent.

Had I not been looking specifically at the possible association between crying babies and maternal psychiatric problems, however, I would not have picked up a small number of medical reasons for these babies to cry (two urine infections, one case of otitis media) and a far larger number of mothers with psychiatric problems (46 women).

Table  Proportion of problems unknown to mothers and general practitioners (total number of children=2877)

<table>
<thead>
<tr>
<th>Problem</th>
<th>No (%) of children with problem</th>
<th>No (%) of mothers unaware of problem</th>
<th>No (%) of general practitioners unaware of problem</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical speech</td>
<td>546 (19)</td>
<td>142 (26)</td>
<td>231 (42)</td>
</tr>
<tr>
<td>Neurodevelopmental</td>
<td>202 (7)</td>
<td>26 (13)</td>
<td>119 (59)</td>
</tr>
<tr>
<td>Behaviour</td>
<td>117 (4)</td>
<td>42 (36)</td>
<td>89 (76)</td>
</tr>
<tr>
<td>Behaviour</td>
<td>161 (6)</td>
<td>15 (9)</td>
<td>123 (76)</td>
</tr>
</tbody>
</table>

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Trends in birth prevalence of cerebral palsy

We would like to add our comments to the correspondence arising from the recent publication of Professor Pharoah and colleagues on trends in birth prevalence of cerebral palsy.1

There has been an interesting swing in thinking on the origins of cerebral palsy over the past few years. This has arisen mainly from the observations derived from registers which have been compiled of children with cerebral palsy within geographically defined populations. It has become increasingly clear when surveying the problem from this community perspective that although low birthweight infants are at increased risk of cerebral palsy, many infants who later manifest signs of cerebral palsy had neither a low birth weight nor a recognisable perinatal insult.

On the other hand there has been an increasing understanding of the associations between evolving neuropathological changes in the brain of the low birthweight infants, as detected by imaging techniques, and eventual clinical outcome.2 This has led to the sort of claim made by Dr Barson that ‘most infants with cerebral palsy have been admitted to a maternity hospital with a morphologically normal central nervous system whilst in utero and subsequently discharged to the community with pathological cavities in their brains’.3 This may be the perspective of those involved with immediate clinical care of low birthweight infants in a special care nursery but ignores the question of the aetiology of cerebral palsy in other babies.

Preliminary figures from the Oxford Region Child Development Project suggest a different view. This study has been establishing a register of infants with serious impairment, including cerebral palsy, born from 1984 onwards to mothers resident in the Oxford region at the time of delivery. Although the oldest infants on the register are now only 3½ years old, 58 infants from a total 1984 birth population of 31 811 have been diagnosed as having cerebral palsy. This gives a birth cohort prevalence rate of 1.82/1000 live births. Of the 570 infants with a birth weight of less than 2000 g, 17 have cerebral palsy. They represent only 29% of the 58 children with cerebral palsy.

We do not wish to minimise the risk of later motor impairment in the low birthweight population and the need for continued monitoring of the outcome of the increasing numbers of extremely low birthweight survivors. At the same time we would support the view that the aetiological origins of most cases of cerebral palsy must be sought outside the immediate perinatal period.

Carriage of penicillin resistant pneumococci

Sir,

While studying the pharyngeal colonisation by Streptococcus pneumoniae and the absence of penicillin resistance among pneumococci isolated from healthy Mexican children, we noticed the paper by Klugman et al that reported the relative penicillin resistance of S pneumoniae in 303 urban and 156 rural black children; this resistance was seen in 14% of urban carriers and 19% of rural carriers. The authors obtained those figures after screening isolated strains with methicillin discs and subjecting those organisms with halos of less than 25 mm in diameter to a quantitative antimicrobial test to confirm the resistance.2

The prevalence of such strains in South Africa may be even higher. Other investigators have shown that the methicillin discs can miss relatively resistant strains of S pneumoniae in 9% of cases, incorrectly identifying them as sensitive (that is with halos of greater than 25 mm in diameter).3 In addition, another paper by Klugman et al reported that when they were screening for resistance with methicillin discs they found 20% of falsely susceptible S pneumoniae strains, but they offered no explanation for this.

Consequently, if the limits of error given were used to calculate corrected figures, the results found could be interpreted as showing resistant strains from 19% to 25% of the urban population and from 23% to 28% of the rural