Correspondence

This led me to feel that the routine six week developmental physical examination, in its present form, is probably a waste of time.

References

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.

References

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 given4 were used to calculate corrected figures, the results found1 could be interpreted as showing resistant strains from 19% to 25% of the urban population and from 23% to 28% of the rural
population; the technical problem is that testing for antimicrobial susceptibility to obtain minimum inhibitory concentrations was only performed for strains resistant to methicillin by diffusion.\(^1\)

It may therefore prove more reliable to screen for penicillin resistant pneumococci using oxacillin discs that do not yield false sensitive results,\(^3\) so avoiding the risk of primary misclassification of clinical isolates responsible for invasive disease.

**References**


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Drs Klugman and Koornhof comment:

The recommendation for the use of oxacillin in preference to methicillin discs is based on the observation by Swenson *et al*\(^1\) that three of 34 strains resistant to penicillin were falsely identified as susceptible using a 5 µg methicillin disc, compared with 0 of 34 using 1 µg oxacillin discs after incubation on Mueller-Hinton sheep blood agar plates in ambient air. Jacobs *et al*\(^2\) showed that two of 29 compared with one of 29 were falsely identified as susceptible, using the two discs, respectively, after incubation on Mueller-Hinton agar with 5% lysed horse blood in ambient air. Neither of these differences is significant. Though a definitive answer to the advantages of one method over the other rests on an analysis of a larger number of strains, even a combination of the above data fails to show a significant difference between the tests, giving a false susceptibility rate of five of 63 (8%) with the methicillin disc compared with one of 63 (2%) with the oxacillin disc (χ² = 1.57, p = 0.2, with Yates' correction).

The 20% false susceptibility rate quoted by Dr Conde-Glez from another of our papers,\(^3\) is due to his misinterpretation of our presentation of all multiply resistant strains, identified in various day care centres, as multiply resistant penicillin susceptible (MRPS) strains. No inference of the prevalence of false susceptibility can be drawn from that study. A possible estimation of the true prevalence of penicillin resistance in urban and rural children may be calculated, assuming that for every 58 strains identified as resistant using the methicillin disc, a further five are missed. Using this estimation four additional strains to the 43 resistant strains were probably missed out of 206 isolates in the urban study, and three additional strains to the 30 resistant strains out of 96 in the rural study. We believe that the importance of these differences in carrier rates is unimportant.

We do agree, however, that primary misclassification of isolates responsible for clinical disease, especially meningitis, may be important in individual cases. The data of Jacobs *et al*\(^2\) indicate that neither test is 100% sensitive. In areas with high prevalence of endemic resistant pneumococci we recommend that minimum inhibitory concentrations be determined on all pneumococcal isolates from cerebrospinal fluid. Finally the distribution between resistant and intermediately resistant strains cannot reliably be made with either of these disc tests\(^1\) and must rely on MIC data from minimum inhibitory concentrations.

**References**


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**Post-streptococcal glomerulonephritis in Hong Kong**

Sir,

We were interested to read the recent report of Leung *et al* on post-streptococcal glomerulonephritis in Hong Kong.\(^1\) It is now commonly assumed that this condition is rare in developed countries but our recent experience would lead us to think otherwise.

Since July 1985 we have documented a post-streptococcal aetiology in 11 of the 16 patients with acute glomerulonephritis referred to our unit. Nine of the 11 have been diagnosed since December 1986. We have recently reviewed the case notes of all 11 patients.

One obvious difference from the Hong Kong report is that macroscopic haematuria was the commonest presenting symptom in our series. It was present in eight out of 11 patients with only three having oedema as the main complaint. Group A β haemolytic streptococcus was isolated from the throat swabs of eight children, two of whom had received prior antibiotic treatment. In addition nine children had a raised anti-streptolysin O titre, seven a low serum C3 concentration, and seven raised C3 degradation products.

In general the nephritis was mild and no patients required acute dialysis. Four required drug treatment for hypertension. One of our patients presented with a dense right hemiplegia having had eight days of haematuria at home. We noticed that one child in the Hong Kong series presented with a convulsion due to hypertensive encephalopathy and this further illustrates the acute morbidity and occasional mortality associated with this illness.

We also assume that the recent increased prevalence of post-streptococcal glomerulonephritis may be due to an
Carriage of penicillin resistant pneumococci.

C J Conde-Glez

Arch Dis Child 1988 63: 340-341
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http://adc.bmj.com/content/63/3/340.2.citation

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