seems unlikely because the clinically missed serious cases in the other two hospitals should have been picked up at necropsy, especially in hospital C where the necropsy rate is virtually 100%. Secondly, the higher incidence of the NEC condition in hospital A may be related to an aggressive management of low-birthweight sick neonates in that hospital. Although the perinatal mortality rate in hospital A was lower than in hospitals B and C—indicating that more neonates, particularly premature ones, survived—the infants with necrotising enterocolitis had higher birthweights and apgar scores and had more full-term neonates among them than has been reported for cases from other centres. Thirdly, the higher incidence may be related to the introduction of procedures that have undefined complications and necrotising enterocolitis may represent a new iatrogenic disease caused by some change in clinical practice, perhaps related to the management of labour or the immediate post-delivery period.

We thank the medical and nursing staffs of the three maternity hospitals for their co-operation.

¹ Book LS, Herbst JJ, Atherton SO, Jung AL. Necrotising enterocolitis in low birth weight infants fed on elemental formula. *J Pediatr Surg* 1975; 87:602-5.

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Ophthalmia neonatorum due to beta-lactamase-producing gonococci

Penicillin is highly effective in treating babies with gonococcal ophthalmia. The prospect of seeing ophthalmia neonatorum caused by β -lactamase-producing gonococci has caused dismay. One case, successfully treated with kanamycin, has been reported from Singapore.¹ We report here on premature identical twins with this condition born in London of Ghanaian parents.

Case reports

Twin 1 was born at 2238 on 27 November 1979 after 28 weeks' gestation and weighed 1250 g. Next morning there was scanty crusted discharge on the lid margins of the left eye with slight conjunctival congestion. The right eye appeared normal. A conjunctival smear from the left eye contained 30 polymorphs/high-power field ($\times 1000$) but no organisms; a smear from the right eye contained only epithelial cells. Cultures were taken. Twenty-six hours later, on 29 November, both eyes were swollen, particularly the left. When the eyelids were separated yellow-green pus escaped under pressure. Smears from both eyes contained 200 polymorphs/high-power field with Gram-negative intracellular diplococci.

Twin 2 was born six minutes after his brother and weighed 1280 g. Because of his brother's conjunctivitis he was also seen. The eyes appeared normal. Conjunctival smears contained only epithelial cells. Cultures were taken. Twenty-six hours later there was mild conjunctivitis; a conjunctival smear from each eye contained 20 polymorphs/high-power field and scanty Gram-negative intracellular diplococci.

The father had attended elsewhere because of urethral discharge. A β -lactamase-producing gonococcus was cultured. Spectinomycin, 2 g intramuscularly, produced a good response. The infection had been acquired in Ghana.

The mother was examined on 30 November. No Gram-negative diplococci were found in smears from the urethra, cervix, or rectum, but a β -lactamase-producing gonococcus was grown from the urethra and cervix. Cultures for *Chlamydia* and micro-immunofluorescence for antichlamydia antibody were negative. She was treated with co-trimoxazole, four tablets twice daily for two days, plus spectinomycin, 2 g intramuscularly daily for two days. Follow-up tests on two occasions were negative.

Treatment of twins—From 29 November the conjunctivae of both babies were flooded with sulphacetamide eye drops (BPO; 30%) every six hours. The babies also received erythromycin suspension, 40 mg/kg/day every six hours, by drip feed. The gonococci isolated from both babies produced β -lactamase. The organisms were sensitive to tetracycline, erythromycin, sulphonamides, co-trimoxazole, streptomycin, kanamycin, gentamicin, spectinomycin, and cefuroxime. Gonococci were isolated from both eyes and

the rectum but not the throat, gastric aspirate, or blood in twin 1, and from only the left eye in twin 2. Twenty-four hours after the start of treatment twin 1 showed considerable improvement but Gram-negative intracellular diplococci were still present in the left conjunctiva. The mild conjunctivitis in twin 2 had largely cleared, but a few Gram-negative intracellular diplococci were still present. Despite the clinical improvement the erythromycin was replaced by cefuroxime 100 mg/kg/day intramuscularly in three divided doses for seven days. Material taken at the same time as the smears gave negative cultures. Improvement continued, and further follow-up smears and cultures gave negative results, as did all cultures for *Chlamydia*.

Comment

Strains of gonococci that produce β -lactamase are common in the Far East and probably in parts of West Africa.² Although they are rare in the United Kingdom, they may well become more common. Their advent has created further problems in the treatment of gonorrhoea, particularly in pregnant women and babies, for whom the choice of drugs is limited. All strains of gonococci should be tested for β -lactamase production so that appropriate treatment is given as soon as possible. Prompt tracing of sexual contacts is essential.

The recovery of gonococci from sites other than the eye points to the necessity for systemic antgonococcal treatment in babies with gonococcal ophthalmia.

We are grateful to Dr G J A I Snodgrass for referring the babies, and to Dr S Darougur and Mr J Trehan of the Institute of Ophthalmology, London, for the tests for *Chlamydia*.

¹ Pang R, Teh LB, Rajan VS, Sng EH. Gonococcal ophthalmia neonatorum caused by beta-lactamase-producing *Neisseria gonorrhoeae*. *Br Med J* 1979;i:380.

² Perine PL, Morton RS, Piot P, Siegel MS, Antal M. Epidemiology and treatment of penicillinase-producing *Neisseria gonorrhoeae*. *Sex Transm Dis* 1979;6:152-8.

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Nocturnal enuresis and the buzzer alarm: role of the general practitioner

As a paediatric registrar in Scotland I was struck by the number of children with enuresis attending the paediatric clinic who had failed to be cured by the buzzer alarm. A retrospective study was performed to determine exactly how effective the buzzer had been.

Patients, methods, and results

The notes of patients given a pad and buzzer over a two-year period from January 1976 to December 1977 were studied. The children were attending the paediatric clinic at one of two hospitals: hospital A, a small general hospital; and hospital B, a large teaching hospital. If the paediatrician thought that patients were suitable for treating with the buzzer alarm*, their names were put on the waiting list (several months' wait at each hospital). When the buzzer was collected its use was demonstrated by the ward sister (hospital A) or the ward receptionist (hospital B). After issuing the buzzer, patients were seen on average one to two months later. The notes of 22 patients were studied from hospital A and 40 from hospital B. All the children were over 5 years of age, most being over 7: 50% of those attending hospital A had difficulty using

*The alarms used in hospital A were the Astric alarm from Astric Products Limited, 261 Queen's Park Road, Brighton, Sussex; and the Chiron alarm from Down Brothers and Mayer and Phelps Limited, Church Path, Mitcham, Surrey. Hospital B used the Eastleigh alarm from N H Eastwood and Son Limited, 48 Eversley Park Road, Winchmore Hill, London N2.

the buzzer alarm, and the figure for hospital B was 33 %. By far the commonest difficulty was failure to awake to the alarm. Most of these patients stopped using the buzzer altogether because of the difficulties, most of which occurred in the first week of using the buzzer alarm.

The percentage of patients who could be said to have been cured of their nocturnal enuresis was only 17 % at hospital A and 41 % at hospital B. The figure compares the effectiveness of the buzzer in this study with Meadows's results.¹

Rate at which children became dry using enuresis alarm. Comparison of results at hospitals A and B with Meadows's results.¹

Comment

Many papers have shown how the buzzer alarm should be used.¹⁻⁴ With this knowledge it is possible to determine the reasons for failure of the buzzer to cure enuresis in my study: (1) none of the paediatricians took a special interest in enuretics; (2) the use of the buzzer was not satisfactorily demonstrated before issue; (3) follow-up appointments were not frequent enough; and (4) many patients were seen by a different doctor each time they attended the clinic.

Nocturnal enuresis is a common problem, and effective management should be provided when the family seeks medical advice. Failure to do so may cause the family to lose faith in their doctors and lead to many years of misery for the child. I think that the poor results of my study may be repeated at many hospitals in the United Kingdom. There is no point in children with enuresis attending hospital at three-monthly intervals, year after year, without any improvement. This was the case with an appreciable number of patients in this study. If general practitioners have such patients then they should consider purchasing their own buzzer alarms for the practice. The general practitioner is in an ideal position to manage such children. He has often known the family for years and the child since birth. From this knowledge he is able to judge whether the child is mature enough to cope with a buzzer. He will know the social circumstances of the family and the sleeping arrangements of the children. All these factors are important if the buzzer is to be used successfully. If the practice has an interested health visitor she would be invaluable in helping with home visits.

The extra work would be compensated for by the satisfaction of successfully managing what can be a difficult problem, and surely this role for the general practitioner would be in keeping with the spirit of the Court Report⁵—which coined the term general practitioner paediatrician.

¹ Meadow SR. How to use buzzer alarms to cure bed-wetting. *Br Med J* 1977;ii:1073-5.

² Dische S. In: Kolvin, MacKeith, Meadow, eds. *Bladder control and enuresis*. London: Spastics International Medical Publications, 1973.

³ Forsythe WI, Redmond A. Enuresis and the electric alarm: study of 200 cases. *Br Med J* 1970;i:211-3.

⁴ Fraser MS. Difficulties encountered in buzzer training. Communication at the Newcastle Colloquium, 1971.

⁵ Committee on Child Health Services. *Fit for the future*. London: HMSO, 1976.

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Expired air carbon monoxide: a simple breath test of tobacco smoke intake

The amount of carbon monoxide in end-expired alveolar air provides a rapid and accurate non-invasive indirect measure of carboxyhaemoglobin,¹ since carbon monoxide in alveolar air after breath-holding is in equilibrium with the concentration of carboxyhaemoglobin in the blood. The carbon monoxide analyser (Ecolyzer Series 2000, Analysis Automation Ltd) is portable and inexpensive (£930). The method has a potentially wide application as an objective test of the amount smokers inhale and of their claims to have stopped. Although it has been available for several years, more cumbersome and costly methods that are no more accurate, and in some cases require blood samples, continue to be advocated. This paper shows the accuracy of the method and that error due to impairment of lung function among smokers is negligible for most purposes.

Subjects, methods, and results

Parallel venous blood and expired air samples were obtained from two groups of subjects: (1) "normal smokers" from the smokers' clinic at the Addiction Research Unit who gave no history of chronic lung disease, though this was not specifically excluded; (2) patients with emphysema from King's College Hospital chest unit who had been diagnosed on radiological criteria.² Expired alveolar air samples were collected by the method of Jones *et al.*³

Scattergram of expired air carbon monoxide (CO) against carboxyhaemoglobin (COHb). Linear regression lines: (A) for normal smokers; (B) for emphysema patients. (C) indicates line derived from the multiple regression analysis for smokers of average age for sample (39.6 years). Corresponding regression equations: (A) $\text{COHb} = -0.28 + 0.175 \text{ ECO}$; (B) $\text{COHb} = -0.12 + 0.211 \text{ ECO}$; (C) $\text{COHb} = -1.629 + 0.222 \text{ ECO} - 0.0006 \text{ ECO}^2 + 0.018 \text{ age}$. Note ECO refers to linear and ECO^2 to quadratic expired air components.

The subjects were instructed to exhale completely, then to take a deep breath, hold it for 20 seconds, and breathe out rapidly through a three-way connector into, firstly, a 500-ml anaesthetic bag (to allow for the dead space) and, secondly, an impermeable plastic bag⁴ to provide the sample for immediate measurement by the Ecolyzer. The time between sample collection and the last cigarette was never under four minutes. The instrument was calibrated daily with a mixture of 80 parts per million carbon monoxide in air. The blood samples were analysed by an IL 282 CO-Oximeter. The accuracy of this method was checked by comparison with a gas chromatographic technique,⁵ yielding a correlation of 0.99 for 43 specimens over the range 0.6%-12.3% carboxyhaemoglobin. Repeated measurements by CO-Oximeter at four concentrations of carboxyhaemoglobin saturation gave the following values (mean $\pm$ SE, $n=5$): $0.52 \pm 0.06\%$, $4.12 \pm 0.02\%$, $8.12 \pm 0.02\%$, and $14.72 \pm 0.06\%$.

The correlation between the expired air carbon monoxide reading (ECO)