Desmopressin for bedwetting

Str,—The article by Evans and Meadow cast further light on the ongoing controversy over the causes of nocturnal enuresis in children.1 While some stress the importance of small bladder capacity2 and others the importance of nocturnal polypuya caused by deficient arginine vasopressin (AVP) secretion,3 many take the compromise of both factors playing their part.4 In this paper, enuretics responsive to desmopressin had higher nocturnal volumes (345 ml) and lower nocturnal urinary AVP concentrations (0.9) compared with nonresponders (265 and 2.8 respectively). While these differences were insignificant for the small numbers of children involved (eight and 10 responders respectively) it is clear that there do appear to be 'a trend towards desmopressin responsive enuretics having lower AVP concentrations and larger volumes of dilute urine at night'. It could well be that significant differences are found with larger numbers of children. It was also interesting that the age of desmopressin responders who appeared to produce more dilute urine was 12 compared with only 9 in the others. This supports our own feeling that the cause of nocturnal enuresis is more likely to be lack of nocturnal AVP secretion in older children and adults and smaller function bladder capacity in those younger. A lot of the work advocates nocturnal enuretics who are AVP deficient was done on older children and adults.2,5 There was therefore no real support for Evans and Meadows suggestion that 'it is possible that the diurnal variations in AVP are less well developed in younger children'.5

Recently we did a short study of nocturnal enuresis in 5 year olds to see if the enuretics produced more dilute urine overnight. At the routine medical on joining school, children were asked to bring along the first urine specimen of the day for measurement of osmolality. (This study was passed by our local ethics committee.) Fifty five children produced samples: 46 who were virtually dry and nine who wet the bed most nights. Mean (SD) osmolality of those who were dry was 801 (180) and of the enuretics 760 (264) mmol/kg (not significant). While it is possible that early morning osmolality may not be fully representative of total overnight urine production if the child has previously wet the bed, we feel this study does indicate that enuresis in young children is unlikely to be due to nocturnal polypuya. This type of developmental nocturnal enuresis is probably mainly due to low function bladder capacity at night. In studies of nocturnal enuresis a firm distinction should be made between such children with developmental enuresis and older children and adults.

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9 Drs Morton and Daniels raise the interesting hypothesis that enuretic children can be divided into two groups: children who suffer from maternal polyuria secondary to vasopressin deficiency and younger children who have smaller function bladder capacities. Certainly our desmopressin responsive patients were older and other authors have made the same observation.7 However such a clear cut distinction is unlikely in clinical practice as the majority of these older children will have been enuretic when younger. It seems unlikely that this group with the onset occurring later will have had the same underlying physiological abnormality.

Dr Robson and Leung find that desmopressin is more effective in children with primary nocturnal enuresis without diurnal symptoms or adverse behavioural or social circumstances. It is likely that most treatments for enuresis would be more effective in such a group of children. The observation that children who have consistently rather than intermittently responded better is interesting and contrasts with our experience that children who wet the bed every night responded extremely poorly to desmopressin. In our experience an unselected group of children attending a local enuresis clinic and in other authors’ experience3 desmopressin results in a minority of children becoming dry while on treatment and even fewer remaining dry when treatment is withdrawn.


Comparison of BCG vaccination at birth and at third month of life

Str,—I read with interest the paper by Idirim et al.1 The authors recommend that practice be changed in this country in which BCG is given to newborn infants, and that the vaccine be given at the end of the third month instead. I would be concerned that the uptake and consequent reduction in the number of bladder infections in children who are likely to be vaccinated at 3 months of age will be considerably lower than when given in the neonatal period when the babies are in effect a ‘captive’ population. In the study reported, at the end of the first year 7% of those immunised in the neonatal period did not have a BCG scar as against 0-6% of those immunised at 3 months. I would suggest that even in this country, if BCG was to be planned to be given at 3 months the number of children not immunised would far exceed 7%, and that as a result although the immunisation itself was more effective, the protection from tuberculosis itself will be less powerful. The risk of serious complications after immunisation (lymphadenopathy with fistulation) was not significantly different between the groups. It would, therefore, seem hasty to change any immunisation programme until further evaluation had taken place.

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Dr Idirim and colleagues comment: We read with interest the letter of Dr Gorham and are pleased to learn that Dr Gorham accepts that immunisation is more effective at the end of the third month. We do not agree that the number of children coming for vaccination will be fewer at the third month. As more than half of deliveries are not vaccination at 3 months of age and many children are vaccinated in the community hospitals in developing countries, to reach the newborn for BCG vaccination will be more difficult in those countries. Instead every developing country has a vaccination program (diphtheria, pertussis, tetanus, and polio) for children, starting in the second