Desmopressin for bedwetting

SIR,—The article by Evans and Meadow cast further light on the ongoing controversy about the causes of nocturnal enuresis in children.1 While some stress the importance of small bladder capacity2 and others the importance of nocturnal polyuria 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 non-responders (265 and 2.8 respectively). While these differences were insignificant for the small numbers of children involved (eight and 10 respectively) it seems right to point out that there did 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 younger. A look at the work adenorenal system on nocturnal enuretics who are AVP deficient was done on older children and adults.2,3 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'. Recently we did a short study of nocturnal enuresis in 3 years 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) mOsm/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 polyuria. This type of developmental nocturnal enuresis is probably mainly due to low functional 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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Comparison of BCG vaccination at birth and at third month of life

SIR,—I read with interest the paper by Idirim et al.1 The authors recommend that practice in England be changed 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 concomitant frequency of such children with primary nocturnal enuresis who do not have diurnal symptoms or possible adverse effects in later or social circumstances and who are consistently rather than intermittently wet at night.

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Compromise of both vasopressin secretion and AVP concentrations may be that the authors examined a heterogeneous group of patients. Evans and Meadow included patients with diurnal symptoms, behaviour problems, and adverse family or social factors. They also included children who were only intermittently enuretic. Only 15 of 28 children treated for one month and 17 of 27 treated for three months were dry ≤2 nights per week before treatment.

Evans and Meadow acknowledge the presence of a significant number of children with lower AVP concentrations and volumes of dilute urine in responders. We believe that statistical significance may have been achieved with either a larger group of patients or a more homogeneous patient population. Our experience suggests that desmopressin is most efficacious in children with primary nocturnal enuresis who do not have diurnal symptoms or possible adverse effects. In later or social circumstances and who are consistently rather than intermittently wet at night.

Dr Evans comments:
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. 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 of children secondary onset enuretic children would have 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. Therefore 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 on an enured 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.


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