# Rapid maxillary expansion in the treatment of nocturnal enuresis

**By Donald J. Timms, Consultant Orthodontist**

Normal bladder control should be mastered by age 3 or 4, and nocturnal enuresis (NE) in children over five must be considered abnormal. According to Parks,[1] 15 percent of boys and 10 percent of girls are enuretic at age five; half that number remain enuretic at age 10, and by late adolescence, the numbers fall to one percent to three percent. The causes of nocturnal enuresis have been blurred by the absence, in most cases, of frank urinary pathology. Treatment is often based on the use of antidiuretics as well as such subterfuges as restricting liquid intake prior to bed and electric alarms for arousal. However, these children usually have another distinct feature in common, namely an upper airway obstruction (UAO).[2-8]

An UAO would also account for the prevalence of respiratory tract infections, otitis media and other sleep disorders found in many of these cases. Most of the cited references are limited to single case histories; the most trenchant publication is that by Weider and Hauri.[9] They analyzed 35 enuretics between the ages of 3½ and 11 years who had their tonsils and adenoids removed. It is interesting that the four children who continued bedwetting were those in whom the UAO was incompletely relieved.

This aspect of enuresis has not been extensively studied, but physicians are now taking a greater interest in breathing and sleep disorders with emphasis centered on sleep laboratories. Nocturnal enuresis can now be grouped with certain other obstructive respiratory parasomnias, including snoring, sleep apnea, myclonus, idiopathic polycythemia, cardiopulmonary disorders, etc. In most children the accusing finger points to adenoidal hypertrophy and/or anterior nasal stenosis, in the absence of more sinister pathology. The otolaryngologist[9] has demonstrated relief by adenotonsillectomy but there are surgical difficulties in dealing with anterior nasal stenoses. This is where the orthodontist can become involved with rapid maxillary expan-

**Abstract**

There is growing consensus that upper airway obstruction is a causative factor in nocturnal enuresis. This phenomenon has an unhappy history, although some surgeons in the past have touched on its treatment through the relief of upper airway obstruction. Only recently have sleep laboratory investigations presented a clearer, though still incomplete, picture of the etiology of nocturnal enuresis through disturbed sleep patterns. The obstruction is usually an adenoidal hypertrophy or, less commonly, an anterior nasal stenosis. While the otolaryngologist can readily cope with the former, surgical difficulties make treating the latter problematic. In many cases, the constriction can be reduced by rapid maxillary expansion. In the ten cases examined in this study, nocturnal enuresis ceased within a few months of maxillary expansion.

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**Key Words**

Nocturnal enuresis • Upper airway obstruction • Rapid maxillary expansion

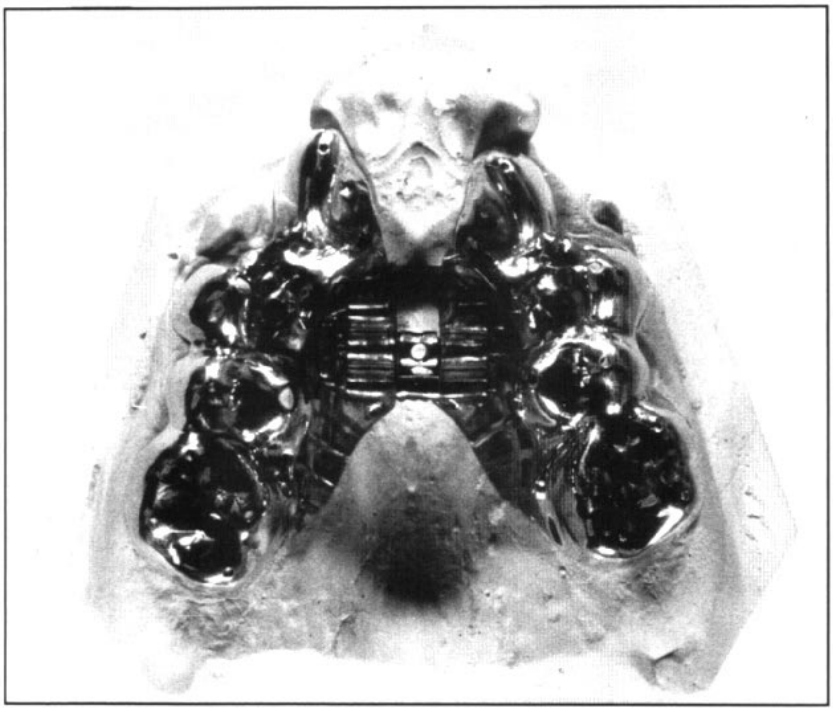

**Figure 1**
**Occlusal view of the expansion appliance used in most of the cases in this study.**

sion (RME). Rhinomanometric investigations[10-14] have found that RME will radically reduce nasal airway resistance (NAR) and these airway obstructive cases illustrate this therapeutic mode.

This study is partly retrospective in as much as the decision to publicize the data was made after half the cases had been treated. This had no fundamental bearing on the investigation in view of the simple protocol. A short questionnaire was completed in all cases and the RME technique had been standardized by the author over many years.

## Materials and methods

The sample of ten children (seven boys and three girls) 6½ to 15½ years of age, represent all cases referred to the author's clinics in the past six years with a known history of NE. Except for their NE and some respiratory conditions mentioned in Table A (MP was particularly prone to asthma), the children were otherwise healthy. It was not known if any had the experience of obstructive sleep apneas. Referrals were generally for correction of a malocclusion. Two cases (SA and TF) were referred because of their NE and three cases (RE, VS and MP) came from otolaryngologic departments. All ten had attended otolaryngologic clinics for nasal obstruction, were snorers and mouth breathed to varying degrees. Four cases (TC, JM, MP and TF) had had adenotonsillectomies without improving their condition suggesting that their obstructions were in the anterior nasal passages. All had previously received exhaustive treatments for NE, usually with electric alarms and/or antidiuretic drugs.

In most cases, the diagnoses were initially obtained from the patients' hospital records and then confirmed by the parents. All had life-long histories of bedwetting except for a short period of remission in two cases (SC and TC).

The main points raised were:
Ante RME: (1) How long had the child been enuretic and (2) frequency of episodes?
Post RME: (Questioned each month during the retention phase of RME) (1) Any change in frequency and (2) if and when did it stop?

Parents were also asked if other symptoms associated with UAO, such as snoring, respiratory tract infections, otitis media and general sleep behavior, existed. (It is the authors' practice to check for diseases related to UAO once nasal obstruction has been ascertained.)

For appliance rigidity and enhancement of basal expansion, RME was carried out with cast silver alloy cap splints and a Glenrose Mk VI expansion screw[15] (Figure 1). In the case of RE, an articulated screw with curved guides was used to compensate for tooth tipping and increased basal expansion. Instructions were given for 90 degree turns each morning and evening to produce 0.35 millimeter of expansion daily or 2.5 millimeters weekly. No predetermined linear expansion was pursued; the objective was to dilate the anterior nasal airway and expansion was continued as far as the posterior occlusion allowed, i.e. until the upper palatal cusps articulated with the lower buccal. Thus, the expansion varied between six and 10 millimeters, according to the original mediolateral occlusion.

After expansion, the appliances were kept in place for three to four months before removal and replacement with retainers. Patients were then considered for more conventional mechanotherapy. This also provided the opportunity for long-term enuretic follow-ups.

## Results

The frequency of NE in all cases declined following RME and within two months, on average, the patients were generally dry at night. Over a longer period of time, half the cases reported an occasional micturition. Case RE was interesting because he had seasonal allergic rhinitis; six months after his enuresis had stopped he experienced a two-month relapse due to a high pollen count which caused his mucous membranes to swell thus occluding his nasal airway.

Other features usually associated with UAO, such as snoring and respiratory tract infections recorded in Table A, also showed abatements. Most children affirmed that their nasal respira-

**Table A**
Case histories of each patient in this study with nocturnal enuresis.

| Case | Age at RME Yrs | Age at RME Mths | Sex | Malocclusion Angle Class | Enuretic Histories: Ante RME Period | Enuretic Histories: Ante RME Frequency | Enuretic Histories: Post RME | Other conditions associated with nasal obstruction |
|---|---|---|---|---|---|---|---|---|
| RE | 8 | 5 | M | III, no buccal x-bites | Regular since infancy | Most nights sometimes twice | Dry within 3 mths | Snoring, poor neuromuscular coordination (clumsy), allergic rhinitis |
| SC | 15 | 5 | M | I, edge to edge bite unilateral x-bite, crowding | Intermittent since infancy one dry period | Sporadic 1 per week | Dry within 2 mths | Snoring, recurrent otitis media, conductive hearing loss |
| TC | 13 | 4 | F | I, edge to edge bite, unilateral x-bite, crowding | Intermittent since infancy one dry period | Sporadic 1-2 per week | Dry within 2 mths | Snoring, recurrent otitis media, conductive hearing loss |
| SA | 6 | 7 | M | I, no buccal x-bites | Regular since infancy | 2 per week | Dry within 2 mths | Snoring, recurrent rhinitis, bronchitis |
| VS | 8 | 9 | F | I, bilateral x-bites | Regular since infancy | 1-2 per week | Dry within 2 mths | Snoring, respiratory tract infections |
| JM | 11 | 8 | M | Mild II/I unilateral x-bite | Intermittent since infancy | 1 per week | Dry within 1 mth | Snoring, respiratory tract infection |
| JA | 15 | 2 | M | I, no buccal x-bites | Since infancy but reducing | 1-2 per mth | Dry within 3 mths | Snoring, recurrent otitis media, conductive hearing loss |
| MP | 9 | 8 | M | I, no buccal x-bites | Regular since infancy | Most nights | Dry within 4 mths | Snoring, night terrors, allergic rhinitis, asthma |
| JF | 11 | 2 | F | III, bilateral x-bites | Since infancy but reducing | 1-2 per month | Dry within 3 mths | Snoring, otitis media, bronchitis |
| TF | 7 | 0 | M | I, no buccal x-bites | Regular since infancy | Most nights | Dry within 4 mths | Snoring, otitis media |

tion was easier but this was only measured in the case of TC because her treatment coincided with a rhinomanometric investigation.[13] Before RME her NAR was 0.55 Pa/cm³/sec (5.5cm$H_2$ O/L/sec) and after RME 0.36 Pa/cm³/sec (3.6 cm$H_2$ O/L/sec) which for her age is close to normal according to Principato and Wolf.[16]

## Discussion

The symptoms in NE are conspicuously evident to the family concerned and consequently the anamnesis would be reliable. Regarding the retrospective cases, some were still receiving orthodontic treatment or entries in their hospital records were sufficient to meet the short questionnaire. From the superficial standpoint, there would be little to link UAO with NE except for the matching in time of the cessation of enuresis with the RME. Given that the children in the sample would eventually stop bedwetting naturally, the chances of coincidence in all ten cases are too remote to contemplate. The absence of a control group is regretted as that would have reinforced this article and the efficacy of the treatment advocated.

At present there are many gaps in our knowledge of NE and very little has been published on it as a UAO related sleep disorder. The scientific study of sleep, at least as far as the laboratories are concerned, is fairly new and other than circumstance, no one has yet come up with an acceptable cause and effect mechanism. During sleep, we lose voluntary control of respiration and in the rapid eye movement stage, the respiratory stimulus is depressed. This can be compounded by airway obstructions leading to apnea and serious physiologic changes in the body based mainly on oxygen desaturation. Many sleepers with UAO are restive, sometimes making jerky movements (myoclonus); this poor neuromuscular coordination may conceivably be reflected in the bladder sphincters. In this respect, one case (RE) was undergoing physical and occupational therapy for 'clumsiness' and dysgraphia. This person improved following RME, prompting his discharge from these therapies.

The mechanism is obviously complex because only a portion of children with UAO suffer the misfortune of bedwetting. Even among individuals with more severe parasomnias, such as obstructive sleep apnea, there are still many who are not enuretic.[17] It is argued that these improvements are due to the restoration of normal sleep patterns through the elimination of respiratory dysfunction caused by UAO. It is advantageous to take the pragmatic approach to treatment by going directly to the answer even if the physiologic bridge is incomplete.

These results not only support other works which have demonstrated a kinship between UAO and NE but stress the relevance of differentiating the obstruction. Weider and Hauri[9] succeeded by adenotonsillectomy where the constriction was in the nasopharynx; the improvements reported here were due to dilation of anterior nasal stenoses (four cases in this study had already had adenotonsillectomy without mitigating their symptoms). The anterior nasal airway includes the liminal or nasal valve, long considered the narrowest section. Little is known of its growth and development. Failure to reach the minimal size necessary for normal conductance of air would compromise nasal respiration and lead to mouth breathing. RME dilates the nasal valve and the inferior turbinate area. The potential of RME to reduce nasal obstruction must be judged by rhinomanometry and all reports using this method, which involves measuring the transnasal pressure difference and airflow, have shown significant improvements. Tests limited to airflow only are equivocal. The ducts within the anterior nasal airway are very narrow, consequently the small increases effected by RME may represent a large percentage increase. Bearing in mind the inherent difficulties in surgical relief of anterior nasal stenosis in the growing child, RME offers a simple therapy which might otherwise be denied to many children. Hopefully, this study will stimulate additional research and bring the orthodontist into this field of health care.

## Author Address

Dr. Donald J. Timms
Oral Surgery Department
Royal Preston Hospital
Sharoe Green Lane
Fulwood
Preston PR2 4HT
United Kingdom

*D.J. Timms is an Honorary Consultant Orthodontist in the Department of Oral Surgery at the Royal Preston Hospital in England.*

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