

## ANOTHER CONSEQUENCE OF SLEEP DISORDERED BREATHING?

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Reading an article about bedwetting may not be your idea of a good use of time, but if you serve a pediatric patient population, this article may indeed be worth a glance. There is now evidence that obstructive sleep apnea syndrome (OSAS) may be related to bedwetting. And that bedwetting is partially or completely reversible with appropriate treatment of OSAS in some children. In order to understand the possible relationship between bedwetting and OSAS, it will first be necessary to discuss what is currently known about nocturnal bedwetting and its suspected causes and then to review the available literature that seems to indicate that not only is there a relationship between OSAS and bedwetting but that both can be partially or completely eliminated with appropriate treatment. An awareness of this relationship will serve to aid medical professionals to ask more appropriate questions of the parent and the child with a suspected sleep disorder.

Bedwetting, or nocturnal enuresis, is a common sleep problem and is often cited as a consequence of sleep disordered breathing particularly in the pediatric population. Nocturnal enuresis can be a frustrating problem for both the child and their family. It is estimated that in the United States approximately 7 million children have enuresis. Boys with enuresis outnumber girls by more than two-to-one until about 11 years of age when the gender distribution becomes more equal. With maturity, the incidence of enuresis declines though consistent dry nights may still not be present in 1% to 2% of adolescents. Enuresis can take one of two

forms where it can be either primary or secondary. Primary nocturnal enuresis is bedwetting since birth without any dry intervals. Secondary enuresis is bedwetting at night that follows a period of at least three months where the person has achieved a period of sustained nighttime continence. Organic and psychological causes are more common for those individuals with secondary enuresis.

While the precise causes of enuresis are still only partially understood, there are several factors besides OSAS which apparently contribute to this disorder of sleep. Certainly maturation and general child development play key roles in the ability to maintain nocturnal continence. Since functional bladder capacity increases with age, persistent nocturnal enuresis may be a result in a delay in the development of this capacity. By the time a child is 4 years old, functional bladder capacity should reach between 300 to 360 ml. This capacity is felt to be adequate for children to remain dry throughout the night. Some children with enuresis may be slower to develop an adequate functional bladder capacity. Maturation also has an effect on bladder contractions where most children progressively gain more control over uninhibited contractures. Development over the control of bladder contractions as well as the development of diaphragmatic and skeletal muscles generally may contribute to the decreasing incidence of enuresis as the child matures.

Perhaps the most compelling reason for the increase in nighttime continence with maturation is the fundamental differences in sleep states for different age-groups. Young children sleep both quantitatively and qualitatively deeper than adults or even older children and the arousal threshold in response to a full bladder will differ with that sleep-state differentiation. All children have developmental sleep-state differentiations but not all children wet the bed. This notion indicates that there is almost certainly individual variation in sleep-related arousal thresholds. Ultimately, it is necessary for some children to develop the ability to awaken from sleep in response to the feeling of a full bladder. As the child matures, the ability to arouse from sleep in response to the feeling of a full bladder often becomes more likely. This is true if adequate training is used to increase the awareness of these sensations during the night.

While there is evidence that in some cases nocturnal enuresis may be related to partial arousal during the transition from deep sleep to the lighter phases of non-REM sleep, enuresis can occur in any state of sleep. There is no evidence that medically stable enuretic children sleep more soundly or differently than children who remain dry during the night. There is also no evidence that males sleep more soundly than females, though there are still gender differences in the incidence of enuresis.

Other factors that can be related to nocturnal enuresis include genetics, certain medications and organic factors including OSAS. If both parents report that they themselves did not have nocturnal enuresis as children, the chances of their children being enuretic are low; usually about 15%. But if one parent reports enuresis as children, the incidence of enuresis for their offspring increases to 44% and further increases to 75% if

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both parents report enuresis as children. This familial relationship is well known. Diuretic medications may also cause nocturnal enuresis particularly if those medications are used on a long-term basis. Organic factors account for about 10% of enuresis in children. They include urinary tract infections, anatomical abnormalities, severe constipation, conditions that cause polyuria, allergies, neurological disorders, hormonal dysregulation that can increase urine production and OSAS.

One can hardly research childhood OSAS literature without finding nocturnal enuresis listed as a co-morbid symptom of OSAS. In fact, enuresis has been shown to actually be predictive of OSAS in children. Evidence is also available that shows secondary enuresis is a symptom even in adults with OSAS and that these symptoms can be alleviated with proper treatment. While there is evidence that the effect of OSAS on the pediatric population is different from that of adults with OSAS in that sleep is less fragmented and subsequently less likely to yield excessive daytime sleepiness, children with OSAS can exhibit other symptoms of sleepiness. In children with disturbed sleep, symptoms such as behavioral problems that resemble ADHD, learning and cognitive deficits and decreased memory, productivity and school performance are more likely. While it is clear that enuresis is closely related to the presence of sleep disordered breathing, the exact mechanisms for this relationship are still unknown. As some have suggested, OSAS may be related to a hormonal dysregulation that can increase urine production and also to increased levels of catecholamines that can affect metabolic and musculature function. Others have indicated that OSAS in children can further suppress the arousal response and effect bladder pressure or urinary hormone secretion. In this study, they tested the hypothesis that the presence of nocturnal enuresis is related to the severity of sleep apnea. They found that children with a respiratory disturbance index (RDI) of less than or equal to one had a significantly lower

prevalence of enuresis (17%) than did children with an RDI greater than one (47%). In this group of children referred to a sleep laboratory for sleep disordered breathing, 14% of children with an RDI less than or equal to one had frequent enuresis but the incidence increased to 32% for children with an RDI greater than one. Perhaps paradoxically as the RDI of the children in the study increased in severity, there was no such corresponding increase in the prevalence of enuresis. It seems that simply the presence of disturbed breathing not the severity was sufficient to increase the frequency of nighttime incontinence.

Additional support for the notion that enuresis is a co-morbid symptom of OSAS is the recent evidence showing resolution of enuresis following treatment of OSAS. In the pediatric population, treatment of OSAS usually begins with adenotonsillectomy (T&A) if this lymphoid tissue is present and it is felt the child will benefit from this procedure. In one study, the sleep questionnaires of children that underwent polysomnographic evaluations were reviewed retrospectively. It was found that about 30% of children with an Apnea/Hypopnea Index (AHI) greater than one were reported as enuretic. Of those children who were treated, 74.1% had three or more wet nights per week prior to treatment compared to 37% one month after treatment. In 41% of those children who were treated with a T&A, enuresis totally disappeared throughout the remaining time of follow up. In another study using a retrospective chart review of children that underwent polysomnography, almost 33% of the children reviewed had a positive history of enuresis. Of the participants, 41.1% were females and 58.9% were males. Following treatment with a T&A, 61.4% of these children were free of enuresis, 22.8% had a decrease in enuresis and 15.8% had no change in enuresis. These studies show a marked improvement in frequency of enuresis following treatment for OSAS in many children and that this organic basis for enuresis requires the attention of medical professional to assess enuresis as a co-morbid symptom of OSAS.

In assessing the child with suspected disturbed sleep, sleep professionals should invariably ask about the frequency and nature of nighttime enuresis. Since some children who have sleep-disordered breathing have been shown to also be enuretic at night, enuresis and snoring may be in and of itself a reason for a polysomnographic evaluation. Not only can the detrimental effects of OSAS on the cognitive development of children and subsequently their educational outcomes be averted with appropriate treatment, but in some children, treatment of OSAS can result in the decrease or elimination of nocturnal enuresis. Appropriately serving this population improves the quality and function of the child's life as well as improving the quality of life of the child's entire family. Any history and examination of a child with suspected sleep disordered breathing should include questions about the presence or absence of nocturnal enuresis.



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