LITERATURE REVIEW

Nocturnal Enuresis

by

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Abstract

In Indonesia and in other parts of the world a child with nocturnal enuresis is not an uncommon disease. Studies on nocturnal enuresis is still restricted in Indonesia. It is highly recommended to understand the disease because wetting problems can continue to more serious psychic developmental problems in our young generation. This paper represents a review on some aspects of nocturnal enuresis.

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Introduction

Nocturnal enuresis is one of the most common perplexing problems in children; parents complaining of it in daily practice were ever heard though some of the parents are still ashamed to bring their child to the doctor (Budhiman, 1980; Ling, 1965).

It could be that the parents did not know that wetting problems may continue to more serious psychic developmental problems to the child.

Normal voiding is associated with a complex of physiological events involving the somatic and autonomic nervous system. Thus, detrusor contraction is accompanied by relaxation of the external urinary and rectal sphincters which ordinarily are in a state of tonic contractions, and also accompanied by a slight rise in blood pressure and pulse rate.

Neurogenic disorders, such as those produced by transverse myelopathy, result in changes in timing and intensity of these events (Abraham et al., 1966; Borzyskowski et al., 1978).

Enuresis is defined as involuntary discharge of urine, occurring after the age by which bladder control should usually have been established (Borzyskowski and Chantler, 1978; Formann et al., 1979; Kempe et al., 1976; Rubin and Baliah, 1975; Smith, 1978).

Bladder control

Most children become dry at night between the age of 2 and 4 years. Dryness is a natural development which emerges in the absence of any training. Adverse factors during this period may impair the acquisition and security of subsequent dryness (Ling, 1965).

There are still wide variations of the age of achievement of bladder mastery in children such as:

- Hensie (1960) found that an occasional wetting of the bed may be seen in 10 to 20% of children even as late as 9 to 10 years of age.
- Kanner (1960) said that some authors refer to the mode of micturition during the first two or three years of life as physiologic enuresis.
- Rubin and Baliah (1975) stressed that the bladder control at night is usually gained by 3 years of age, however, many children do not develop control until they are 5, 6 or 7 years of age.
- Borzyskowski and Chantler (1978) pointed out that most of the children are dry during the day by the age of 3, and dry at night by 4 but some 10% of 5 years olds wet the bed, as do 5% of 10 years olds.
- Drummond (1979) found that nocturnal enuresis is present about 10 to 15% of otherwise normal 5 years old children and in about 1% of normal children at 15 years.
- Due to the wide variation in the age of gaining bladder control in children, it is considered now that the children are not generally labeled "enuretic" unless the symptom persists beyond the age of 5 years (Drummond, 1979; Hollerman, 1979).

Sex incidence

Most authors suggest that enuresis occurs in both sexes with about the same frequency. Noyes and Kolb (1959) found that this condition occurs twice as frequently in boys as in girls. Kanner (1960) found that enuresis occurs 62% in boys and 38% in girls.

Wetting incidence

Incidence of wetting in children as obtained by Kanner (1960):

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Boys</th>
<th>Girls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>Occasionally</td>
<td>11%</td>
<td></td>
</tr>
<tr>
<td>Frequently</td>
<td>7%</td>
<td></td>
</tr>
<tr>
<td>Almost daily</td>
<td>5%</td>
<td></td>
</tr>
<tr>
<td>Once a week</td>
<td>4%</td>
<td></td>
</tr>
<tr>
<td>Twice a week</td>
<td>4%</td>
<td></td>
</tr>
<tr>
<td>3 — 4 times a week</td>
<td>3%</td>
<td></td>
</tr>
<tr>
<td>Once a month</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Off and on</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>In spells</td>
<td>2%</td>
<td></td>
</tr>
<tr>
<td>Infrequently</td>
<td>1%</td>
<td></td>
</tr>
</tbody>
</table>

Oei and Setyonegoro (1965) described that the type of chief complaint (which initiated the bringing of the child for psychiatric consultation by the parents) out of 556 patients they studied, was only in 11 (1.9%) enuresis.

Nocturnal enuresis commonly occurs only once during the night but in a small number of instances a child may wet as often as 3 or 4 times in one night. Diurnal enuresis alone is relatively rare.

Types of enuresis

1. According to Noyes and Kolb (1959) and Drummond (1979), betwetting may be divided into 2 types:
   1. Persistent type, in which the child has never been dry at night.
   2. Regressive type, in which a previously continent child begins to wet the bed after a stressful episode.

Persistent nocturnal enuresis is often the result of inadequate or inappropriate toilet training experiences. The regressive type of bedwetting is related to precipitating stressful environmental events, such as moving to a new home, marital conflict, birth of a sibling, death in the family, etc.

Betwetting in these instances is often intermittent and transitory; prognosis is better and management less difficult than in those children who have never been continent.

In both types of betwetting, organic pathology can be found in only a very small number of cases. Organic disorders that may cause nocturnal enuresis include nocturnal epilepsy.

Other organic conditions that may also lead to enuresis are o.a.: urinary tract infection, increased urinary volume in diabetes mellitus, diabetes insipidus, obstructive uropathy, chronic renal failu-
It was found that an increased hyperventilation response occurred mainly among children with primary enuresis (bedwetters from birth).

This was true even if the child had an "uropathy". The results for those with secondary enuresis (later onset) were similar to the controls.

Their findings indicate that disturbed cerebral control of the bladder is an important factor in primary enuresis.

Psychological factors are thought to account for the occurrence of secondary enuresis (Kaada et al., 1981).

**Etiology of enuresis**

Etiologically there are several theories to explain enuresis (Hollerman, 1979):

1. **Small bladder.**
   At age 2-4 years the bladder capacity is 200-340 ml of urine. It is believed by some that nocturnal enuresis is the result of a decreased bladder capacity; however, not all children with small bladder are enuretic.

2. **Allergy.**
   The bladder wall is believed to be the target organ with resultant edema and subsequent decreased functional capacity. No positive relationships has been identified.

3. **Deep sleep.**
   No difference in depth of sleep between enuretic and non enuretic children has been documented.

4. **Behaviour problems.**
   There appears to be a relationship between behavioral problems and diurnal enuresis and/or encopresis but not for children with nocturnal enuresis. There is one exception to the latter statement; nocturnal enuresis in teenage delinquent boys appears to be related to earlier maternal separation or deprivation (Coucheils et al., 1981).

5. **Intelligence and/or Electroencephalographic patterns.**
   No correlation has been found so far, though Kaada and Retveld (1981), found an increased hyperventilation response in EEG occurring mainly among children with primary enuresis.

6. **Spina bifida occulta.**
   It is commonly diagnosed in enuretic children though no positive correlation exists.

7. **Bacteriuria.**
   A positive correlation is found in girls between bacteriuria and enuresis, but at the maximum only 10% of enuretic girls will have bacteriuria (Heale, 1981).

8. **Familial pattern.**
   If one parent is enuretic 25% of the children will be enuretic; if both parents are enuretic, 50% of the children may become enuretic (Burke and Stickler, 1980).

9. **Developmental delay.**
   This may be associated in familial enuresis or may be the etiological factor in the majority of children with enuresis. By age 10-12 years virtually all children have achieved full bladder control; however, enuresis may persist into adolescence (Hollerman, 1979).

**Differential diagnosis**

Enuresis should be differentiated with urinary incontinence. Enuresis is voiding of urine inappropriately in the absence of neurological or urinary tract disease, at an age when most children have bladder control.

Incontinence is the voiding of urine inappropriately as a result of neurological or urinary tract disease (Borzyskowski, 1978; Ling, 1965).

Thomas et al. (1980) found that about 90% of the incontinent boys aged 5-14 years had nocturnal enuresis; of the incontinent girls in this age range only 60% were bedwetters, the remainder complaining mainly of urge incontinence.

The main causes of incontinence in childhood are anatomical abnormality, mental subnormality and neuropathic bladders (Borzyskowski and Chantler, 1978a Chusid and Mc Donald, 1960).

**Treatment of enuresis**

In enuresis due to organic causes, such as urinary tract infection and diabetes mellitus, treatment is directed at the disease process.

For the psychologically handicapped child professional psychological/psychi-
If improvement occurs the therapy is continued for two to three months, then gradually tapered off over 3 to 4 months. Imipramide changes the sleep pattern and decrease the bladder tones.

Imipramide is rapidly absorbed and in toxic levels can affect the central nervous system (depressed respiration) and has cardiovascular effects (atriyultiasis, hypertension). (Hollerman, 1979; Rutter and Herlov, 1976).

There is no specific antidote for this drug, and dialysis is not effective to remove the drug.

4. The buzzer (waking agent) is apparently the most efficient waking device. If used appropriately 80% of children with nocturnal enuresis will be cured in the period of 1 week to 6 months.

5. Dietotherapy (elimination of dairy products) has been presumed useful in patients diagnosed as having enuresis secondary to food allergy.

The management of the child with enuresis will continue to be a time-consuming venture on the part of any clinician.

Except in those patients who have an underlying organic disease, enuresis is not associated with a profound morbidity or mortality.

The inclination is either to reassure the parents that the condition will be "outgrown" or to use drugs for control. These children are deserving a careful, complete history and physical examination and a program of therapy based on delineation of the underlying etiology.

References


SELECTED ABSTRACTS


Twenty-five children with clinical egg hypersensitivity, confirmed by double blind challenge, were followed for between 2 and 2½ years. Clinical egg hypersensitivity was found to have resolved in 11 children but was persistent in 14. Skin prick test reactions to egg were initially of equivalent size in the resolved and persisting groups, but became negative or diminished in size with resolution of clinical egg hypersensitivity, while remaining positive in the group with persisting symptoms. Symptoms after egg ingestion were categorized as cutaneous, gastrointestinal, respiratory, and angioedema. The adverse reactions of the resolved group were either cutaneous or gastrointestinal symptoms. The persisting group had multi-system involvement and most of them developed angioedema and respiratory symptoms. These differences may be useful as prognostic indicators in clinical egg hypersensitivity.


In order to find the optimal dosage schedule of phenobarbitone for neonatal convulsions, four groups of patients were studied. Twelve infants (group 1) received a mean phenobarbitone dose of 9.5 mg/kg a day given intramuscularly for 3 days followed by 5.8 mg/kg a day given intramuscularly and then orally. Six infants (group 2) received a mean intravenous loading dose of 9.5 mg/kg followed by 6.8 mg/kg a day given intramuscularly or orally. Nine infants (group 3) received a mean loading dose of 14.9 mg/kg intravenously followed by a maintenance dose of 5.9 mg/kg a day. Thirteen patients (group 4) received a mean intramuscular loading dose of 15.2 mg/kg followed by 5.9 mg/kg a day. Blood samples were taken regularly and phenobarbitone levels were determined by gas liquid chromatography.

A mean intravenous or intramuscular loading dose of 15 mg/kg of phenobarbitone safely achieved therapeutic levels within 2 hours of injection and high therapeutic levels were maintained with a dose of 6 mg/kg a day.