



Community Paediatric Review

VOL 7 NO 2 JUNE 1998

An initiative of the
Centre for Community
Child Health &
Ambulatory Paediatrics,
Royal Children's
Hospital, Melbourne

EXECUTIVE INDEX

Primary nocturnal enuresis	1
Rickets	2
Immunisation	3

Primary nocturnal enuresis

Definition and prevalence Nocturnal enuresis is defined as intermittent wetting during sleep after the age of five years. It is classified as either primary or secondary enuresis; primary is when the child has never been dry and secondary when the child who has been dry at night for six to twelve months starts to wet again.

Bedwetting is the most common urological complaint among children. Figures indicate that there are possibly 200,000 Australian children older than five years who have primary nocturnal enuresis. [10–15% of five year olds, 7% of seven year olds, 5% of ten year olds, and 1 – 2% of fifteen year olds wet the bed.]

Genetics In primary enuresis there is often a family history of bed wetting. If one parent wet the bed as a child, there is a 40% chance of their child wetting the bed. If both parents were bedwetters there is an 80% chance of bed wetting in their children.

It is thus reassuring for the child and the parents to know that the bedwetting is probably inherited and not the result of being lazy or a behavioural problem. Family, social and economic background, stressful family situations, and changes in parenting as in divorce or separation, also appear to be unrelated to primary enuresis.

Pathophysiology Nocturnal enuresis is usually not associated with organic pathology of the urinary tract. It is more likely caused by a mismatch between nocturnal urine production, functional bladder capacity during sleep, and the ability to wake to a full bladder.

Assessment Before any treatment is commenced it is important the child be examined by a medical practitioner. A full examination of lower back, external genitalia and a peripheral neurological assessment of the lower limbs is essential. Urinalysis is performed to exclude infection although these children do not have a higher incidence of urinary tract infection

than those without primary nocturnal enuresis. No other investigations are indicated.

Management The most effective treatment for primary nocturnal enuresis is conditioning therapy using either a pad and bell alarm or a small personal alarm. Up to 70% of children respond to this therapy and will have a lasting cure.

The child is the active participant in the treatment program, with help from a parent. The first few weeks of treatment can be very trying with disturbed nights, but families should be reassured that this will gradually improve. A smaller wet patch in the bed and more urine to “finish off” in the toilet shows that the child is beginning to respond to treatment.

Spontaneous waking to use the toilet and then sleeping through the night without wetting follows.

Once a program is commenced, there should be regular reviews by the doctor or continence nurse in order to offer support and encouragement and to monitor progress – emphasizing gains however small rather than failures – and help with any problems.

The relapse rate after successful treatment has been shown in some studies to be as high as 50%. In some cases this can be reduced if “overlearning” is introduced towards the end of treatment. This simply means that the child has an extra drink before bed to ensure the bladder becomes full during the night. Provided this is tolerated, bladder control is progressively strengthened.

It is important to prepare the parents and



child for the possibility of relapse. If the child does relapse treatment should recommence as soon as possible to avoid the child developing a sense of failure.

Medication For children who have not responded to conditioning treatment, medication is an option. Desmopressin, which is an antidiuretic hormone, is available on authority in Australia as a nasal spray.

For many years imipramine [Tofranil] has been used in the treatment of nocturnal enuresis. With the availability of desmopressin there is no indication for the use of imipramine. It is less effective than Desmopressin with a much higher incidence of adverse side effects, of which it is the neurological effects are of most concern. In overdose, imipramine may be fatal.

Rickets

Vitamin deficiency rickets is characterized by failure in mineralization of growing bone. It occurs as a result of decreased exposure to sunlight in association with low Vitamin D intake.

The major source of Vitamin D is 7-dehydrocholesterol in the skin, which is activated by the ultraviolet rays in sunlight to cholecalciferol. Cholecalciferol is hydroxylated in the liver and kidney to 1–25 dihydroxycholecalciferol which functions as a hormone. The foods that contain Vitamin D include egg yolk, fatty fish, liver and fish oils. Milk, including human milk, may not be an adequate source as it has variable amounts of Vitamin D depending on maternal sunlight exposure and diet. The normal diet of infants therefore contains negligible amounts of Vitamin D. Infant formula and margarine are fortified with Vitamin D.

The most important function of Vitamin D is to maintain extracellular fluid concentration of calcium and phosphorous in the normal range. It regulates mineral metabolism in the bone, promotes intestinal absorption of calcium and phosphorous and the reabsorption of phosphorous in the kidney.

Vitamin D deficiency rickets occurs in breast-fed infants of mothers who are not exposed to sunlight for social, religious and cultural reasons. In addition season, latitude, glass, clothing, pigmentation of skin and sunscreen reduces the sun's photosynthesis of Vitamin D in the skin. Rickets can also be caused by genetic factors (hypophosphatemic and vitamin deficiency rickets), diseases of the kidney, and conditions that reduce the intestinal absorption of Vitamin D such as liver diseases, pancreatic disease,

It is important to remember that drugs alleviate rather than cure the problem, and that there is a high relapse rate once medication is withdrawn.

Summary Nocturnal enuresis is a universal problem and traditionally it has not been managed very well in the community for a variety of reasons. Assessment and management is usually straightforward, and it is a condition that can easily be managed in a community setting.

AUTHOR

ZELIA JOY

CONTINENCE AND UROLOGY SERVICES

ROYAL CHILDREN'S HOSPITAL, MELBOURNE

cystic fibrosis and anticonvulsant therapy.

Rickets, also known as the "English Disease", was described in 17th century England and continued to increase in epidemic proportions at the turn of this

century in Northern Europe, North Eastern America and Australia. Education on the importance of sunlight exposure, along with Vitamin D supplements and environmental measures including the fortification of milk, has led to a marked reduction in the incidence of rickets. However, rickets continues to be a health concern among the Asian and African migrants to developed nations.

In Australia an increase in the incidence of rickets was noted in the seventies among children born to migrants from the Mediterranean countries including Greece, Italy, Turkey, Lebanon and Yugoslavia. More recently it has been detected in children of migrants from Asia (particularly India and Sri Lanka) and Africa, reflecting the changing migration patterns to Australia.

Clinical features Children with rickets present in a number of ways, depending on the age of the child, severity and duration of the illness. They may have lethargy, weakness, growth failure and bone deformities, or present with tetany (spasms of hands and feet, stridor) and convulsions from hypocalcemia. Infants can have increased sweating of the scalp, a bossing deformity of the head and a ping-pong ball sensation on pressing the skull over the occiput or posterior parietal bones (called craniotabes). The chest may have palpable enlarged costochondral junctions called rachitic rosary and Harrison's groove

which is a chest indentation caused by the pull of the diaphragm on the softened ribs. The wrists and ankles may be widened due to epiphyseal enlargement, while bowed legs and exaggerated lumbar lordosis can occur in toddlers. Infants with rickets may also be iron deficient. Adults with vitamin deficiency have vague musculoskeletal symptoms including back and thigh pain, change in gait and difficulty in rising.

Investigations Investigations include serum level of calcium which is low or normal, phosphorous which is usually low and alkaline phosphatase which is markedly elevated. Serum parathormone level is high and a low 25-hydroxy-Vitamin D level confirms the diagnosis. X-ray changes can be seen in areas of active growth – in the wrists, knees, ankles and costochondral junction. Widening, cupping and fraying of the metaphysis (end of the long bone) can be seen while in severe rickets osteopenia (demineralization of bones) and fractures occur.

Treatment The treatment of rickets consists of the daily oral administration of 2000-5000 units of Vitamin D for 6 weeks along with oral calcium. Blood levels of calcium, phosphorous and alkaline phosphatase are monitored

during the treatment. The alkaline phosphatase will begin to normalize within 4 weeks but may take a few months to return to normal. X-ray changes of healing will be seen in 2-4 weeks. Failure to respond to therapy may indicate poor compliance or rickets caused by aetiology other than Vitamin D deficiency.

Prevention Vitamin deficiency is an entirely preventable disease. Community nurses are ideally situated to provide anticipatory advice to high-risk groups about the possible development of rickets and its prevention. They should be advised on the beneficial effects of moderate sunlight exposure and vitamin supplements (0.45 ml of Pentavite drops) to breast fed infants. Breast feeding mothers can also be advised to have calcium and Vitamin D supplements. Children may be monitored for signs of rickets while assessing their growth and development. Investigations and treatment need to be initiated early before severe bone deformities develop.

AUTHOR

DR. MARIANNE LOBO

CENTRE FOR COMMUNITY CHILD HEALTH &
AMBULATORY PAEDIATRICS
ROYAL CHILDREN'S HOSPITAL, MELBOURNE

Immunisation

Immunisation is the best form of prevention we have to protect children from infectious diseases such as diphtheria, tetanus, whooping cough, Hib disease, measles, mumps, rubella and polio. Why then do we as health professionals fail to protect the children in our care?

For all of us involved in paediatric care, the protection of children through immunisation should be high on our agenda.

There are a multitude of reasons why children fail to be vaccinated appropriately – apathy, limited access to services, parental beliefs, lack of parental initiative, economics – the list goes on.

One area in which health professionals add to the list is with missed opportunities, because we fail to keep our knowledge base up to date. Studies undertaken reveal that both public and private sector physicians as well as nurses have major deficits in their knowledge regarding the contraindications to vaccination.

Contraindications to vaccines are rare, but we tend to cling to historical myths and as a result we fuel the myth and cloud reality with a smoky haze. Contraindications can be broadly divided into three main areas – absolute, relative and false.

Absolute contraindications are rare. Pertussis vaccine has remained an area of concern for most medical and nursing professionals as well as parents. The absolute contraindications for pertussis vaccination are as follows :

- anaphylaxis to a previous pertussis dose,
- encephalopathy within 7 days of a previous pertussis dose,
- defer pertussis vaccine if the child has an evolving neurological illness.

However, it is important to be aware of relative contraindications when deciding on further vaccinations and the settings in which they will occur.

Relative contraindications to pertussis vaccination are :

- Convulsions with or without fever within 3 days
- Persistent inconsolable crying for more than 3 hours
- Unusual high pitched crying within 48 hours
- Hypertonic/hyporesponsive episode within 48 hours
- Temperature greater than 40.5°C within 48 hours that is unexplained by any other cause
- Severe local reaction involving circumferential swelling and redness.

Despite what might appear a severe reaction, a relative contraindication is NOT an absolute contraindication and vaccination may continue.

Health professionals now have access to both DTPw and DTPa. It is preferred that DTPa be used to continue the primary course in children who have experienced relative contraindications as opposed to omitting pertussis vaccination.

Aside from pertussis vaccination, it is important when performing a prevaccination assessment to be aware of areas that will impact on whether a child will be vaccinated or not. The prevaccination assessment can be divided into 3 areas.

The first is **condition**. All vaccines should be deferred if the child has a significant acute illness or temperature more than 38.5°C. If immunocompromised or pregnant, all live vaccines should be deferred. OPV should be deferred if there is an intestinal upset.

The second is **allergies**. Advice should be sought if there are allergies to streptomycin if IPV is to be given, neomycin for live vaccines as well as IPV. Advice should be sought regarding MMR vaccination if the child has an egg anaphylaxis.

The third comes under the broad heading of **drugs**. If the child is on immunosuppressants then defer live vaccines. If previous live vaccines has been given in the last month, defer other live vaccines. If the child has received a blood transfusion or immunoglobulin within the last 3 months live vaccines must also be deferred.

If you find you need to defer vaccination for one of these conditions, make an appointment for review prior to the end of the consultation with the intent of vaccination at the earliest possible time.

False contraindications are the largest group of reasons why parents and unfortunately health professionals defer vaccination.

The following are NOT reasons to defer vaccination :

- A family history of convulsions
- A family history of adverse events following vaccination

- A stable neurological condition such as epilepsy or cerebral palsy
- A previous condition such as pertussis, measles, mumps or rubella
- Contact with an infectious disease
- The child is on antibiotics
- The child has asthma, eczema or hayfever
- The child is on low dose steroids (inhaled or topical)
- The child is breastfed
- The child's mother is pregnant
- The child has a history of neonatal jaundice
- The child is underweight
- The child is older than the recommended age in the schedule
- Recent or imminent surgery
- The child is on replacement corticosteroids
- The child has a history of allergies.

As mentioned earlier, this last point is not an absolute contraindication but may be a relative contraindication and should be discussed further prior to vaccination.

Whatever the reason for delay – absolute, relative or false – every child has a right to be protected against preventable diseases such as whooping cough, measles, etc. We as health professionals have a duty to ensure that all children are given this right. Promoting and facilitating opportunistic immunisation is one way of ensuring our children's health. Let's stamp out the fires caused by vaccination myths.

FOR FURTHER INFORMATION the Royal Children's Hospital, Melbourne has an Immunisation Telephone Enquiry Service, Mon.–Fri., 9.50am–5pm. (05) 9345 6399.

AUTHOR
CATHY GOUCHER
IMMUNISATION COORDINATOR
ROYAL CHILDREN'S HOSPITAL
MELBOURNE