Voiding dysfunction – A review

V. Sripathi
Department of Pediatric Surgery and Pediatric Urology, SMF Children’s Hospital, Anna Nagar, Chennai and Apollo Hospitals, Greams Road, Chennai, India

Correspondence: Dr. V. Sripathi,1, Damodara Mudali Street, Chetpet, Chennai–600 031, Tamil Nadu, India. E-mail: sripathi3@vsnl.com

ABSTRACT

In a child who is toilet trained the sudden onset of daytime wetting with frequency or urgency is alarming to the parents. Initially this subject was subdivided into a number of descriptive clinical conditions which led to a lot of confusion in recognition and management. Subsequently, the term elimination dysfunction was coined by Stephen Koff to emphasise the association between recurrent urinary infection, wetting, constipation and bladder overactivity. From a urodynamic point of view, in voiding dysfunction, there is either detrusor overactivity during bladder filling or dysynergic action between the detrusor and the external sphincter during voiding. Identifying a given condition as a ‘filling phase dysfunction’ or ‘voiding phase dysfunction’ helps to provide appropriate therapy. Objective clinical criteria should be used to define voiding dysfunction. These include bladder wall thickening, large capacity bladder and infrequent voiding, bladder trabeculation and spinning top deformity of the urethra and a clinically demonstrated Vincent’s curtsy. The recognition and treatment of constipation is central to the adequate treatment of voiding dysfunction. Transcutaneous electric nerve stimulation for the treatment of detrusor overactivity, biofeedback with uroflow EMG to correct dyssynergic voiding, and behavioral therapy all serve to correct voiding dysfunction in its early stages. In established neurogenic bladder disease the use of Botulinum Toxin A injections into the detrusor or the external sphincter may help in restoring continence especially in those refractory to drug therapy. However in those children in whom the upper tracts are threatened, augmentation of the bladder may still be needed.

KEY WORDS: Dysfunctional voiding in children, Detrusor Sphincter Dyssynergia, Constipation, Dysfunctional Elimination Syndrome

Dysfunctional voiding is a ubiquitous disorder in children. It is estimated that 15% of 6 year olds suffer from this condition.[1] The recognition and management of voiding dysfunction is of utmost importance because if neglected upper tract changes and renal failure will ensue in these children.

PHASES IN THE DEVELOPMENT OF BLADDER CONTROL

Bladder functional development proceeds in fairly well defined stages. The newborn voids about 20 times a day. As he becomes older, the frequency comes down to about 11 times at two years of age. This decrease is accompanied by an increase in the volume of every void. There is a gradual increase in bladder capacity from about 1-2 ounces in the newborn period to an adult capacity at 12 years of age. As a rule of thumb the increase in bladder capacity is one ounce per year of age.

At three years of age the striated urethral sphincter comes under voluntary control. The initiation and termination of voiding now becomes volitional. This is ‘toilet training’ as is conventionally known.

The final phase of micturition control is at four years of age when the sacral micturition reflex arc is brought under cortical control. This means the cerebral cortex is able to control the detrusor by initiating or inhibiting a contraction at any given volume.

DEFINITION OF VOIDING DYSFUNCTION

Any functional disturbance in voiding after completion of toilet training can be called ‘dysfunctional voiding’. A more comprehensive definition is as follows:

Voiding dysfunction means any abnormal holding and disturbed voiding pattern seen in a child without anatomical or neurological disease.

These definitions cover a wide group of diverse conditions such as:

- Nocturnal Enuresis
- Increased Daytime Frequency
- Wetting and recurrent urinary infections (due to}
trusor overactivity)
- Dysfunction elimination syndrome (combined bladder and bowel dysfunction)
- Lazy bladder syndrome (overflow incontinence due to an underactive detrusor)
- Dyssynergic voiding (inability to relax the pelvic floor during voiding)
- Giggle incontinence (detrusor overactivity stimulated by giggling)
- Non-neurogenic neurogenic bladder (NNNB) or the Hinman Allen Syndrome (dysfunctional voiding with upper tract changes)
- Ochoa syndrome (NNNB with facial grimacing during smiling)

TYPES OF VOIDING DYSFUNCTION

In spite of the bewildering variants enumerated above, voiding dysfunction can be simplified into two types:
- Filling phase dysfunction or the ‘purge urge syndrome’
- Voiding phase dysfunction

Filling phase dysfunction
This is the most common type of voiding dysfunction seen in clinical practice characterized by recurrent urinary infections and wetting. Approximately 57% of children between 3 and 14 years of age suffer from this condition at some time [2]. To avoid wetting and social embarrassment children adopt various holding postures. Vincent’s curtsy is done when a female child crosses the legs and bends from the waist downwards with legs crossed. The variations on this include – pinching the glans shut between finger and thumb and sitting on the floor with the heel pressed into the perineum.

A forceful detrusor contraction forces urine into the urethra. This is squeezed back into the bladder by a strong contraction of the urethral sphincter. Organisms colonizing the perineum are also taken into the bladder thus initiating a urinary infection.

Voiding phase dysfunction
The pelvic floor or urethral sphincter contracts during voiding. This dyssynergy between the detrusor and the external sphincter results in high bladder pressures which may cause vesico-ureteric reflux. Also residual urine resulting from incomplete voiding perpetuates urine infections. Persistent high pressure voiding results in bladder wall thickening, bladder neck hypertrophy and a triangular dilatation of the urethra terminating to a point at the external urethral sphincter [Figure 3]
- Clinically demonstrated Vincent’s curtsy (bending from the waist downwards with legs crossed. The variations on this include – pinching the glans shut between finger and thumb and squatting on the floor with the heel pressed into the perineum)
- Infrequent voiding (3 or fewer times daily)

To this can be added:
- Constipation diagnosed either clinically (history and

EVOLUTION OF VOIDING DYSFUNCTION

The purge urge syndrome is believed to be due to immaturity of cortical control over the detrusor.

Voiding phase dysfunction is believed to be acquired due to habitual postponement of voiding and defecation.

DYSFUNCTIONAL ELIMINATION SYNDROME (DES)

This term was coined by Stephen Koff to emphasize the association between severe idiopathic constipation, wetting, bladder instability and recurrent urinary infections [3]. Constipation and urinary infections are closely related because the loaded colon presses on the bladder and:
- reduces functional bladder capacity
- provokes ‘earlier voiding’ sensation
- provokes ‘Unstable contractions’
- provokes chronic pelvic floor spasm
- contributes to post void residual

In addition encopresis selects uropathogenic E.Coli in the perineum leading to severe urinary infections.

RECOGNISING VOIDING DYSFUNCTION

All children with voiding dysfunction present with wetting and / or soiling. Any three of the following six parameters signify presence of voiding dysfunction:[4]
- Thick walled bladder on sonography (3 mm in distended state and 5 mm in the contracted state) [Figure 1]
- Trabeculated bladder on cystography
- Large bladder on cystography (50% larger than expected bladder capacity for age) [Figure 2]
- Spinning top deformity of the urethra on cystography. This appearance denotes contraction of the external sphincter during voiding. This results in a triangular dilatation of the urethra terminating to a point at the external urethral sphincter [Figure 3]
- Clinically demonstrated Vincent’s curtsy (bending from the waist downwards with legs crossed. The variations on this include – pinching the glans shut between finger and thumb and squatting on the floor with the heel pressed into the perineum)
- Infrequent voiding (3 or fewer times daily)
INVESTIGATIONS IN VOIDING DYSFUNCTION

The classic investigations in urinary infections include renal ultrasound, urine culture and sensitivity, micturating cystogram and DMSA. To this would be added cystometry to diagnose bladder overactivity (filling phase dysfunction) and uroflow EMG to diagnose dyssynergic voiding (voiding phase dysfunction).

In uroflow EMG, electrodes are placed in the perineum to pick up activity from the pelvic floor. In a child with voiding phase dysfunction there is intense pelvic floor activity signifying that the urethral sphincter is contracting during voiding. This modality can be used repeatedly to monitor progress of treatment and being non invasive is well accepted by children [Figure 5].

TREATMENT OF VOIDING DYSFUNCTION

This can be divided into the following segments:

- Treatment of urinary infection
- Treatment of constipation
Sripathi V: Voiding dysfunction in children

• Behavioural modification – this includes re-training children like avoiding drinking water or milk prior to bed in those with nocturnal enuresis, timed voiding in infrequent voiders, double voiding in those with postvoid residuals and relaxation during voiding. Bringing attention to voiding and defecation is called ‘cognitive training’. For instance in a child with vaginal voiding altering the pattern of sitting on the toilet will stop vaginal pooling of urine and dramatically cure the wetting [Figure 6]

• Biofeedback – this involves using an objective measure like the uroflow EMG to tell children how far they have progressed in achieving a goal like pelvic floor relaxation during voiding.[5] Complete voiding can be demonstrated by showing low or no residuals after voiding.

• TENS – transcutaneous electric nerve stimulation of the perineum using electrodes serves to suppress bladder overactivity.[6]

DIAGNOSIS AND TREATMENT OF CONSTIPATION

Constipation is common, not life threatening and needs to be treated in a sustained manner. To begin with it will be useful to define constipation using objective criteria. The following clinical symptoms alone or in combination denote constipation:[7]

• Fewer than three bowel movements per week.
• Soiling if accompanied by evidence of stool retention on plain abdominal X-ray. [Figure 4]
• Palpable abdominal mass.
• Per rectal examination showing firm stool in the rect-
the first step. The gastrocolic reflex can be taken advantage of and the child can be made to sit on a potty after a meal. The can be done two or three times a day for five to ten minutes at a time.

**Diet**

Children should be given five servings of fruit or vegetables every day. Each serving will contain approximately two grams of fibre thereby fulfilling the American Dietary Association recommendation of 20 – 35 grams of fibre per day.

**Fluid**

Children should be encouraged to drink water. Parents of school going children commonly complain that the water bottle is brought back intact from school. To break this habit, water drinking with meals and after brushing teeth thrice daily should be encouraged.

**Posture**

For young children a proper posture on the potty is essential for successful defecation. A footstool should be provided for support and the aperture of an adult toilet seat should be suitably modified so that the child’s buttocks do not ‘sink in’.

**MEDICAL TREATMENT OF INTRACTABLE CONSTIPATION**

**Stage I**

‘Clean Out’ or evacuate hard retained stool by enemas or suppositories. Enemas with 50% glycerine and water are easy to make at home and can be administered through a large bore feeding tube. Similarly adding 5 gms salt in one liter of water makes up a salt water enema which is also quite effective. In refractory cases Poly Ethylene Glycol (Peglec) can be given orally (1.5 gms/kg/day) till the efflux is clear. Unfortunately the child invariably will not drink peglec unless its taste has been masked with an orange flavored drink.

**Stage II**

‘Stool Softening’ is done with the following agents:[8,9]

- Mineral Oil 2-4 ml/kg/day
- Milk of Magnesia 1-3 ml/kg/day
- Polyethylene glycol 0.5-1.5 g/kg/day
- Sorbitol or lactulose 1-2 ml/kg/day

This stage of stool softening needs to be continued for a period of at least 2-3 months to get a lasting effect. The end point will be regular toileting, absence of fear and the stools will be soft or even runny in consistency.

---

**Figure 7**: MCU of two and half year old male showing gross reflux and trabeculated bladder suggestive of Hinman bladder. DMSA showing gross bilateral scarring.
DYSFUNCTIONAL ELIMINATION SYNDROME (DES) AND VESICO-URETERIC REFLUX

In a study of 143 children with primary VUR, 43 showed evidence of DES. These children had breakthrough infections three times more commonly than the others and unsuccessful outcome following surgery was only in the DES group. This underscores the importance of identifying and treating DES in children with ‘primary VUR’. In a multivariate analysis of 2759 children treated in a referral practice an attempt was made to quantify the relationship between DES and VUR. Girls were found to have a higher incidence of DES and VUR when compared to boys. UTI was particularly more common in those children with DES.

BIOFEEDBACK

1. Transcutaneous Electric Nerve Stimulation (TENS)
   Low intensity current is applied through patch electrodes over the S2 and S3 dermatomes. Following application of current there is an increase in β adrenergic activity, reduced cholinergic activity and increased levels of vaso intestinal peptide and serotonin in the bladder. In addition the current stimulates release of endorphins and encephalins in the cerebro spinal fluid. These various events result in relaxation of the bladder vault.

   Alternating pulses at frequencies of 5-10 Hz over 15 minutes is very effective in reducing detrusor overactivity. An increase in frequency to 25-50 Hz enhances detrusor contractility while frequencies of 35 – 50 Hz increase pelvic skeletal muscle contractility as well. The pulse width is usually set at 0.2-0.5 ms. The only side effects reported are skin irritation or mild sensory deficit. Low cost TENS apparatus are freely available in our country and the physiotherapist is quite comfortable in teaching parents how to apply the electrodes and how to manage the therapy. At the same time they also teach the children how to relax the pelvic floor or in those with poor tone exercises to strengthen the pelvic muscles are also taught. TENS therapy needs to be done for at least three months to six months for a substantial benefit. Recurrence is common once the therapy is discontinued.

2. Uroflow EMG
   The use of uroflow EMG to teach pelvic floor relaxation during voiding and to minimize residual has been elaborated previously.

3. Rectal probes
   The use of a rectal probe to monitor the levator ani and deep external sphincter is more accurate in demonstrating dyssynergic voiding. Biofeedback training with these probes is particularly effective.

4. Intravesical transurethral bladder stimulation
   This is done in children with established neurogenic bladder in order to achieve spontaneous voiding, gain sensation of bladder filling and knowledge of when to catheterize & to increase bladder compliance. The bladder is filled with saline upto one-third capacity and stimulation is done with electrodes for 60 minutes. On an average 45 sessions are required to achieve the stated goals. In one study 18% of children were found to void with conscious control and a further 25% were found to have a reduction in bladder capacity.
NEWER ADVANCES IN DRUG THERAPY

Oxybutinin has been the standard antimuscarinic drug in the treatment of the overactive bladder. The clinical efficacy of the drug depends on receptor affinity, pharmacokinetics and specificity for the bladder. The effectiveness of the drug is measured by receptor specificity and efficacy to side effect ratio. Oxybutinin is non selective but tolterodine is more bladder specific (less effect on salivary glands) and therefore has a better efficacy to side effect ratio. Furthermore since oxybutinin penetrates the blood brain barrier, it is believed to induce central side effects and impair cognitive function.

Solifenacin, the latest molecule, is a selective M3 muscarinic receptor antagonist. This drug has less incidence of dry mouth when compared to tolterodine because of its better bladder selectivity.\(^{13}\)

Oxybutinin when administered intravesically is found to have less systemic side effects. In addition to its antimuscarinic effects it also exhibits local anesthetic properties. This mode of administration is particularly effective for those children who have intolerable side effects on oral administration of the drug.

Botulinum A toxin is a presynaptic neuromuscular blocking agent which induces selective and reversible muscle weakness upto several months following intramuscular injection of small quantities. The use of Botox A has been evaluated in children with detrusor overactivity not responsive to orally administered agents, and in whom high bladder pressures threaten the upper tracts. Another indication for Botox A is intrasphincteric injection in those with severe Detrusor Sphincter Dyssynergia (DSD).\(^{14}\)

Injection into the bladder muscle is done either with a metal needle or flexible needle at doses of 10-12 units/kg upto a maximum of 200 units. The injection is done at 20-30 sites with 0.5 ml being deposited in each site. The trigone is spared. Excellent results have been reported in the following areas:

- Increase in volume of urine held at 20 cms bladder pressure (pressure specific volume)
- Becoming dry between catheters
- Being able to spontaneously void or to achieve continence
- Reducing the dosage of anticholinergic drugs or stopping it altogether
- Reducing pressures during voiding and abolishing post void residuals

The results last for six to nine months.
In an excellent review article on the use of botulinum toxin; Smith and Chancellor have added valuable insights in the use of this agent. They have emphasized the following:

- There is no contraindication to injection in the trigone. The belief that injection into the trigone will induce vesico-ureteric reflux is unfounded. The authors instead recommend avoidance of the posterior wall and dome of the bladder to prevent inadvertent bowel perforation.
- Concomitant usage of aminoglycosides will enhance the toxicity of botox A.
- Large volumes of injection or frequent injections may cause distal complications like upper limb or facial weakness.
- Injection frequencies less than 3 months may induce antibody formation.
- For injection into the external sphincter 100 units of botox A is diluted in 2 ml and 0.5 ml is injected in four sites at 3,6,9 and 12 o’clock positions. For detrusor injections 200 units is diluted in 20 ml saline and 0.5 cc is deposited in various sites.

The hope is that Botox A may reduce or abolish need for augmentation. However long term results, complications and refractoriness to repeated use of this novel therapy are not known at present (especially in children). In our country, the high cost of Botox A may prevent its widespread use.

NON-NEUROGENIC NEUROGENIC BLADDER OR THE HINMAN ALLEN SYNDROME

This represents the end stage of uncorrected dysfunctional voiding. In this condition the changes in the bladder and kidneys are indistinguishable from neurogenic bladder disease. However, in these children, MRI does not show any spinal cord abnormality. Management is identical to neurogenic bladder disease due to spinal anomalies [Figure 7-10].

CONCLUSIONS

40% of children with urinary infections have voiding disturbances and half of them suffer from vesico-ureteric reflux. There is a close relationship between constipation and bladder dysfunction. The elimination of constipation, prompt treatment of urinary infection and behavioral modification will serve to prevent upper tract changes in the long term in these children. Botulinum toxin A is promising to be an effective therapy for those with neurogenic bladder disease refractory to conventional medication.

REFERENCES