Dysfunctional Voiding - Recognition and the Impact of Multimodality Treatment

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Abstract: The case analysis involves the unusual symptomatology and urodynamic findings of a 12 year old girl who presented to the out-patient department with new-onset daytime incontinence. A visual representation of the stabilization of her bladder dynamics following therapy is presented in the form of serial urodynamic studies. What follows is a précis of the subject which will help readers to understand the concept of "dysfunctional voiding" and ascertain the true nature of the underlying condition by recognizing the pattern of symptoms and ruling out other disorders with appropriate tests. Various treatment modalities are also touched upon briefly.

Keywords: Daytime incontinence, Dysfunctional elimination, Non-neuropathic bladder dysfunction, Urotherapy

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Case Summary

A 12 year old girl was referred from a private hospital to the Pediatric Urology Clinic with the complaints of daytime incontinence for the past one year. She was always wet, with dampness of the underclothes, and also voided a small amount of urine in a staccato manner once or twice a day. A history of being able to micturate only in standing position with straining was indicative of a recent change in voiding habits. There were no episodes of nocturnal enuresis. There was no history of constipation or faecal soiling. The renal function tests and urinalysis were essentially normal.

She had undergone every conceivable imaging test in an effort to pin-point the cause of her symptomatology prior to being referred to our centre. Ultrasonography (USG), Micturating cystourethrogram (MCU), computed tomography (CT) of the abdomen, Magnetic Resonance Imaging (MRI) of the urinary tract and spine were non-contributory. Renal diuretic scintigraphy (RDS) and DMSA scan revealed bilateral normal functioning kidneys without scarring. A DRCG scan ruled out vesicoureteral reflux.

What is most interesting though, is the gradual evolution of the urodynamic picture of this child………

In January 2011, she was started on urotherapy including timed voiding, pelvic exercises and behavioural modification along with pharmacotherapy in the form of Oxybutynin. The initial dose was 5 mg twice a day which was stepped up to 7.5 mg thrice a day over the next 6 months. During follow-up in
July 2011, her symptoms improved and she was able to void while seated on the toilet. The constant dribbling improved although she did report occasional involuntary leakage when straining, playing or coughing. She continued to have small volume voids only twice or thrice a day.

By December 2011 she was voiding every 3 hours and did not have daytime incontinence although she did have occasional dribbling prior to going to bed. The dose of oxybutynin was gradually tapered to 5 mg twice a day and she is currently asymptomatic with complete resolution of symptomatology.

The findings can be summarized as:

**Focus Areas**

- To understand the concept of "dysfunctional voiding"
- To ascertain the true nature of the underlying condition

### Table 1. Serial Urodynamic Studies

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Residual volume (ml)</td>
<td>50</td>
<td>20</td>
<td>Nil</td>
</tr>
<tr>
<td>Expected Cystometric Capacity (ml)</td>
<td>450</td>
<td>450</td>
<td>450</td>
</tr>
<tr>
<td>Measured Cystometric Capacity (ml)</td>
<td>139</td>
<td>255</td>
<td>395</td>
</tr>
<tr>
<td>Volume at Pdet 10 (ml)</td>
<td>132</td>
<td>220</td>
<td>281</td>
</tr>
<tr>
<td>Volume at Pdet 20 (ml)</td>
<td>136</td>
<td>252</td>
<td>387</td>
</tr>
<tr>
<td>Leak point pressure (cmH2O)</td>
<td>33</td>
<td>18</td>
<td>26</td>
</tr>
<tr>
<td>Compliance</td>
<td>poor</td>
<td>poor</td>
<td>good</td>
</tr>
<tr>
<td>Uninhibited Contraction</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Comments</td>
<td>Low capacity, normal pressure, high LLP</td>
<td>Low capacity, low, pressure, high LLP capacity doubled</td>
<td>Near normal, no leak during test, capacity 80% of MMC</td>
</tr>
</tbody>
</table>
by recognizing the pattern of symptoms and ruling out other disorders with appropriate tests

- To know the recognized patterns seen during uroflowmetry (UFM) and urodynamic studies (UDS)
- To be well versed with the pillars of treatment: Urotherapy and pharmacotherapy

**Discussion**

The International Children’s Continence Society has been reviewing available literature constantly and attempting to provide consensus statements regarding the various contentious areas of paediatric urology including dysfunctional voiding. The current ICCS guidelines state that “the child with dysfunctional voiding habitually contracts the urethral sphincter during voiding. The term cannot be applied unless repeat uroflow measurements show curves with a staccato pattern or unless verified by invasive urodynamic investigation. Note that the term describes malfunction during the voiding phase only. It says nothing about the storage phase. The use of this expression to denote any kind of disturbed LUT function leads to confusion and is strongly discouraged. Dysfunctional voiding means dysfunction during voiding. Of course, it is entirely possible for a child to experience dysfunctional voiding as well as storage symptoms such as incontinence”.[1]

It is known that high intravesical pressures and interrupted voiding are common urodynamic features in infants. It may stem from the anatomical finding of a complete ring of striated sphincteric muscle (as opposed to the omega-shaped structure seen later in life) and the immature detrusor-sphincter coordination.[2,3] Micturition control comes about gradually as the neural networks evolve and functional bladder capacity increases. Thus, this entity of “non-neuropathic bladder-sphincter dysfunction” cannot be rigidly defined because more often than not, the various symptoms are a part of a spectrum which also depend on the maturity of continence mechanisms at that point in time. It is quite difficult to standardize and define “normal” in a group which consists of children at each conceivable stage of development. Various groups cite the incidence of dysfunctional voiding from 4.2%[4] to 32%[5] in children presenting with wetting.

Prior to labelling a particular child as having “non-neuropathic dysfunction” the clinician should have exhausted all other diagnostic possibilities. One must then know of the entities that need exclusion:[6]

**A). Derangement of Nervous Control Conditions**

a). **Congenital malformations of the CNS** (e.g., myelomeningocele, spina bifida occulta, caudal regression syndrome, tethered cord syndrome)

b). **Developmental disturbances** (e.g., mental retardation, dysfunctional voiding, urge syndrome)

c). **Acquired conditions** (e.g., cerebral palsy, progressive degenerative diseases of the CNS associated with spasticity, transverse myelitis, multiple sclerosis, vascular malformations, and trauma of the spinal cord)

**B). Disorders of Detrusor and Sphincteric Muscle Function**

a). **Congenital conditions** (e.g., muscular dystrophy, neuronal dysplasia, megacolon-megacystis syndrome)

b). **Acquired conditions** (e.g., chronic bladder distension, fibrosis of detrusor or bladder wall)

**C). Structural Abnormalities**

a). Congenital conditions (e.g., bladder extrophy, epispadias, cloacal anomaly, ureteroceles, posterior urethral valves and other urethral anomalies, prune-belly syndrome)

b). Acquired conditions (e.g., traumatic stricture or damage to the sphincter or urethra)

**D). Other Unclassified Conditions**

a). Giggle incontinence

b). Hinman syndrome

c). Ochoa syndrome (urofacial syndrome)

A detailed history is of utmost importance. Utilizing the aforementioned case as a template, the history of new onset daytime incontinence is a clue to the underlying bladder dysfunction. The lack of other clues which would point to a neurological or congenital cause steers us towards a possible non-neuropathic bladder-sphincter dysfunction. Questions about symptomatology should cover both the storage and voiding phases of the micturition cycle.

**Table 2. Dysfunction specific to the phase of the micturition cycle**

<table>
<thead>
<tr>
<th>During Filling</th>
<th>During Voiding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overactive bladder</td>
<td>Dysfunctional voiding</td>
</tr>
<tr>
<td>Urge syndrome</td>
<td>Staccato voiding</td>
</tr>
<tr>
<td>Functional urinary incontinence</td>
<td>Infrequent voiding</td>
</tr>
<tr>
<td>Giggle incontinence</td>
<td>Hinman syndrome</td>
</tr>
<tr>
<td>Ochoa syndrome</td>
<td>Postvoid dribbling</td>
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</tbody>
</table>

This child demonstrated a distinct problem in initiating voiding and the use of manoeuvres such as standing and straining to void, associated with a sudden cessation of the urinary stream midway, points towards the increased pelvic floor activity which is seen in children with dysfunctional voiding. Those
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with overactive bladders often exhibit holding manoeuvres such as standing on tiptoe and pressing the heel into the perineum during the bladder contraction to postpone micturition. This is reported by caregivers as infrequent voiding. A voiding diary maintained by her parents also helped us keep a track of the evolution of her symptoms.

A history of constipation or evidence of faecal impaction should be actively elicited to rule out the entity of "dysfunctional elimination". This term encompasses the association observed between dysfunctional voiding, recurrent urinary tract infections and bowel dysfunction, including constipation, faecal impaction and encopresis. The cause-effect relation may include a distended rectum causing mechanical compression of the bladder/neck and eliciting significant detrusor instability. It has been seen that the treatment of constipation resulted in 89% resolution of daytime wetting, 63% resolution of nighttime wetting and prevention of urinary tract infection.

Physical examination is as helpful as a detailed history. A palpable bladder, a loaded rectum and any abnormality of the spine such as dimples/agenesis/neurocutaneous stigmata of occult spinal dysraphism should be actively sought. Local examination of the genitalia can reveal anatomic obstructions to flow.

Urinalysis and culture/sensitivity become even more relevant in the setting of frequent UTIs (which is one of the presentations of dysfunctional voiding). Organism directed antibiotic therapy and appropriate drugs for chemoprophylaxis are essential components of chemotherapy.

Imaging studies should start with simple and easily available baseline investigations such as a radiograph of the spine and ultrasonography. In the context of dysfunctional voiding, USG has been used to study the pelvic floor musculature during the phases of voiding. Yeung et al have correlated the "bladder volume and wall thickness index" (BVWI) with urodynamic parameters to stratify patients and steer further investigations. A micturating cystourethrogram and a retrograde urethrogram may be required to rule out anatomic obstructions and associated vesicoureteral reflux (VUR).

Urodynamic studies are essential to diagnose this entity, although there is now a trend towards utilizing only the uroflowmetry and electromyography (EMG) aspects. To confirm dysfunctional voiding, a flow/residual urine measurement should be repeated up to 3 times in the same setting in a well hydrated child to ensure that a reasonable volume of urine (100 ml) is expelled with each void. The typical uroflowmetry pattern seen in these patients is a staccato or intermittent flow with reduced maximal flow rate and prolonged flow time. This may be associated with continuous or intermittent perineal muscle activity on EMG. The dynamic obstruction as a consequence of abnormal sphincter activity (internal or external sphincter) interrupts the continuous flow pattern and causes the normal bell shaped curve to disappear. But, as Kanematsu et al have pointed out that without standardizing the evaluation method, a discussion about the role of UFM in diagnosing paediatric overactive bladder or dysfunctional voiding is pointless. Also the comparison of UFM data with parameters such as bladder wall thickness or external sphincter electromyography is not possible. They also warned that the subjective patterning of curves was liable to personal bias.

**Table 3. Indications for a full urodynamic study**

| 1. | Thickened bladder wall on ultrasound with depressed flow patterns suggesting obstruction |
| 2. | Dilated lower ureters suggesting reflux or increased storage pressures |
| 3. | Suspected bladder neck dysfunction |
| 4. | Failure of empirical therapy |
| 5. | Positive neurological history (e.g. Cerebral palsy) |
| 6. | Anatomic problems |

As regards the collation of clinical evidence and its objective assessment prior to categorizing a child, various scoring systems have been used through the years. The dysfunctional voiding symptom score and the wetting and functional voiding disorder score are two such scores although validation is required from studies with larger patient groups.

Urotherapy is a nonpharmacologic and nonsurgical combination of cognitive, behavioural and physical therapy with an aim to normalize micturition pattern and prevent further functional disturbances of the lower urinary tract. A 74% success rate in 48 children with daytime wetting has been reported using behavioural therapy and biofeedback. Behavioral therapy included timed voiding, modification of fluid intake, and pelvic floor exercises. With the inclusion of drugs, the armamentarium to tackle the often recalcitrant problem of dysfunctional voiding has expanded greatly.

The role of multimodal therapy is exemplified by a study conducted at the Children's Hospital of Philadelphia in which a program was instituted in children with dysfunctional voiding involving antibiotic prophylaxis in 59% of 280 patients, anticholinergics in 49%, biofeedback in 25%, and counselling in 15%. At a minimum follow-up time of 6 months in 222 children with daytime wetting, 100 (45%) were cured (i.e., off all medications with no wetting), 82 (37%) exhibited daytime wetting improvement (i.e., on medication, 50% reduction in symptoms), and vesicoureteral reflux resolved in 16 of 30 (53%) children undergoing repeat VCUG. The antimuscarinic agent of choice has traditionally been oxybutynin. Approximately 10% of patients require discontinuation of...
therapy due the onset of side-effects.\textsuperscript{[16]} Common adverse effects include reduced salivation, constipation and personality changes in some. This has led to the emergence of a more bladder specific drug, tolteridaine tartrate. We used oxybutynin in this child and were rewarded with a gradual resolution of symptoms and a marked improvement in the urodynamic parameters over time, the most striking being the increase in measured cystometric capacity which approached the norm for her age.

It is difficult to prove the absolute superiority of any one treatment method over a placebo. This is due to various reasons including the fact that the "placebo" treatment often includes bladder diaries and timed voiding, which themselves are known to be efficacious,\textsuperscript{[17]} as is illustrated by this case where the parents and the children were very compliant with urotherapy. Also, most studies draw their study groups retrospectively and are beset by the problems of poor randomization and small sample sizes. A baseline annual resolution rate of about 15\% exists for paediatric incontinence which may also confound results in the long-term.\textsuperscript{[18]}

Other modalities such as sacral neuromodulation, Botulin toxin injection into the detrusor and the use of alpha-blockers remain at best investigational in children and are more established for the treatment of voiding dysfunction in adults. Whatever the approach, the bedrock of successful management of dysfunctional voiding is correct diagnosis and the elimination of all other neurologic and anatomic abnormalities before embarking on therapy.

\section*{References}


