Dysfunctional voiding: A review of the terminology, presentation, evaluation and management in children and adults

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ABSTRACT

Dysfunctional voiding (DV) is a voiding disorder characterized by dyssynergic striated sphincteric activity in the absence of a proven neurological etiology. It can present at any age with a spectrum of storage and voiding symptoms that may resemble florid neurogenic bladder. There is a striking lack of clarity regarding what this entity represents, the diagnostic methodology and treatment. The limitations of existing guideline documents are analyzed. Specifically, use of the term "habitual", the assumption that bladder changes are secondary to the outlet, the emphasis on "staccato" voiding and the implication of striated urethral sphincter are discussed. Literature shows that DV may also present with continuous slow flow or normal flow. Dyssynergia may be at the level of the striated urethral sphincter, the pelvic floor or both, better termed "striated urethral sphincter-pelvic floor complex" (SUS-PFC).A diagnostic algorithm is provided so that patients are evaluated on merit rather than on the basis of different philosophies of individual centers. High-risk markers such as hydronephrosis, vesicoureteral reflux, renal failure or marked voiding difficulty should prompt a formal urodynamics evaluation and imaging for neurological etiology. Patients with predominantly storage symptoms with incidental staccato voiding can be managed initially, on the basis of non-invasive evaluation. Conservative urotherapy including biofeedback is appropriate initial management for patients without high risk factors. Treatment and evaluation should be escalated based on response. Patients with severe DV will need treatment similar to neurogenic bladder including clean intermittent catheterization and measures to control storage pressures.

Key words: Dysfunctional voiding, guidelines, International Children's Continence Society, International Continence Society, terminology, staccato voiding, urodynamics, uroflow

INTRODUCTION

Dysfunctional voiding (DV) is a voiding disorder due to dyssynergic striated sphincteric activity in an individual without apparent neurological disease. Bladder outlet obstruction results in both voiding difficulty and storage symptoms (presumably) due to bladder changes consequent to obstruction. It may present at any age. In young children it is often associated with urinary incontinence, nocturnal enuresis or recurrent urinary infection while adults classically present with voiding difficulty. The disease has a vast spectrum of presentation and the implications of a diagnosis of DV may vary from the innocuous to grave. There can be a profound impact on the quality of life.[1] Florid disease can present in a form virtually indistinguishable from classical neurogenic bladder and such patients may progress to bilateral hydronephrosis and end-stage renal insufficiency.[2]

The diagnosis of DV is commonly made in both adult and pediatric urology. However, there is a striking lack of clarity and consensus in the literature regarding what condition the term DV represents. Evaluation protocols are markedly variable and are dependent on the philosophy of the treating unit more than patient factors.[3]

This review aims to critically analyze current terminology and its limitations in adults and children. Presentation and treatment of DV is reviewed with an aim to formulate

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Table 1: Terms that have been used to describe DV or conditions very similar to DV in literature

Subclinical neurogenic bladder[72]

Non-neurogenic neurogenic bladder[5]

Hinman syndrome[6]

Hinman Allen Bladder

Dysfunctional bladder[73]

Dysfunctional elimination syndrome[50]

Idiopathic detrusor sphincter dyssynergia

Sphincter overactivity voiding dysfunction

Achalasia[74]

Occult neurogenic bladder[75]

Learned voiding dysfunction[18]

Occult neurological bladder

Occult neuropathic bladder[9]

Occult voiding dysfunction[9]

Ochoa Syndrome (Urofacial syndrome)[17]

Anxious bladder[76]

Fowler's syndrome[23]

strategies for the triage based on patient presentation. The Pubmed database was queried with the terms "dysfunctional voiding", "voiding difficulty", "non-neurogenic neurogenic bladder", "dysfunctional elimination syndrome", "occult neurogenic bladder" and other related terminologies to yield the publications on which this review is based. Data collection was hampered by the lack of quality data, paucity of controlled trials and contamination of literature by patients who might not have been suffering from genuine DV.

TERMINOLOGY AND DEFINITION

Possibly, Beer first described voiding difficulty, recurrent infections and sphincteric incoordination in 1915.[4] Over the subsequent century a bewildering array of terms has been used to describe individuals with external sphincter dyssynergia in the absence of a neurological diagnosis [Table 1]. Hinman and Allen's names became associated with a more florid form of the disease.[5,6] The current term "dysfunctional voiding" was first used by Allen in 1977.[5] This term has subsequently been adopted by several standardization documents.

The International Children's Continence Society (ICCS) terminology document defines a child with dysfunctional voiding as one who "habitually contracts the urethral sphincter during voiding" and goes on to state that the term should be used only if "repeated uroflow measurements show a staccato pattern or unless verified by urodynamics".[7] A majority of the pediatric studies rely on uroflow for making a diagnosis.

The use of the term "habitually" by the ICCS implies that

a) this is a learned behavior and b) sphincter relaxation in these children is under their voluntary control. While undoubtedly there are children who have learnt a faulty technique for voiding such evidence is lacking in a large number of children who present with DV. Should the ICCS continue to retain a word in the definition that is subjective and difficult to confirm? The ICCS document states that DV leads to bladder changes and reflux. While this may be true in some children, the assumption that all bladder changes are consequent to the sphincter does not have a clear scientific foundation. Most literature regarding DV in children comes from an analysis of children with wetting problems, a storage symptom!

The International Continence Society (ICS) defines DV as "intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated muscle during voiding in neurologically normal individuals".[8] However, there is no provision in the ICS document for describing a non-fluctuating slow flow due to dyssynergia in a neurologically intact individual. Also, while the document specifies that the fluctuating flow should be due to contractions of the periurethral striated muscle, needle electromyography that can show activity of this muscle is not commonly employed in most urodynamics laboratories. Surface electrodes show a summation of activity from the pelvic floor, the external anal and urethral sphincters. Also, there is literature to show that DV may occur due to either striated sphincter dyssynergia, pelvic floor dyssynergia or both. Hence, it might be preferable to replace the term "periurethral striated muscle" with "striated urethral sphincter-pelvic floor complex (SUS-PFC)".

It is suggested that the terms "habitual", "periurethral striated muscle", "intermittent" and "fluctuating" be dropped from these documents. Instead, the definition should reflect two key diagnostic components, dyssynergic SUS-PFC and lack of a clear neurological etiology.

It is also suggested that DV should replace the plethora of terms that are used to describe dyssynergic voiding without an apparent neurological etiology. This is in line with the urological community's move away from labels with etiological or syndromic connotations such as "automatic bladder", "prostatism" or "obstructive voiding". Any dyssynergia irrespective of whether the flow is continuous, intermittent or staccato should be included in this definition. It should be clearly recognized that the diagnosis would be made by exclusion and that some individuals with DV would have a revised diagnosis of detrusor sphincter dyssynergia in case a neurological problem were to surface subsequently. The tendency to label any voiding disturbance in a child as DV should be resisted. Instead, such disturbances are best called "voiding dysfunction", a term that does not specify any particular etiopathology.

PATHOGENESIS

Traditionally, DV has been considered a habitual disorder, presumably because the child learnt the wrong habit of contracting the SUS-PFC rather than relaxing it during voiding. This may develop due to inappropriate toilet training or as a response to urgency or pelvic discomfort. Initiating urgency could be due to detrusor overactivity or urinary infection. Accompanying constipation may aggravate urinary symptoms. DV may result from persistent infantile or fetal voiding patterns,[9] have familial or hereditary origins,[10] get triggered inadvertently by disciplinarian teachers at school[11] and may also be associated with behavioral problems. At least some patients with DV represent occult neurogenic problems that will manifest provided these patients are followed longitudinally.[12]

In all individuals with unexplained severe DV, search for an unidentified neurological lesion must be made. A subtle neurological insult could present as DV. Such lesions may or may not be detectable with current imaging technologies. Routine magnetic resonance imaging (MRI) in children with lower urinary tract problems without overt neurological signs and symptoms has a low yield of 7.5% but this may be improved by targeting children with abnormal cutaneous findings.[13] Tethered cord syndrome may be identified in some patients with subtle neurological signs and symptoms. The spinal cord is stretched in patients with a tethered cord due to a fixed, inelastic anchoring of the conus.

More interesting are recent reports regarding occult or minimal tethered cord. Classical tethered cord has been diagnosed on the basis of a pathologically elongated conus or a conus that lies below the L2 level. However, anecdotal successful outcomes following surgical division of the filum in children with apparently normally located cords suggest that the cord may sometimes be abnormally stretched without being at an abnormal location.[14] Such subtle lesions might also explain the occasional presentation in infancy when the problem has its onset before toilet training has commenced.[15] Surgical division of the filum terminale in such patients is controversial but may yield improvements in bladder dysfunction.[16]

Another possible indirect evidence for an unidentified neurological lesion in these patients is the association of a peculiar facial expression in some children with DV.[17] The association of facial expression with bladder function in this "Urofacial Syndrome" has been explained by the proximity of the cortical centers for the bladder and facial expression in the brain. Presumably, this makes an association of abnormality between the two centers more likely.

It is more difficult to explain the DV that occurs in adults for the first time. Undoubtedly, some of these adults represent children who grew up with an unaddressed voiding problem. But many patients completely deny the existence of voiding symptoms in their childhood.[18] Have these individuals learned this behavior much later in life? Women with lower urinary tract symptoms are more likely to recollect having a similar problem in childhood.[19] Also, abnormal sphincteric behavior is commoner in women who had vesicoureteral reflux in childhood.[20] Hence, at least in some women, urinary patterns in adulthood may be the result of unrecognized childhood dysfunction.

In some patients it is conceivable that pelvic pain could be responsible for DV. Cameron described bladder outlet obstruction in 48% of 231 women with painful bladder syndrome for whom urodynamics data was available. They attributed this to DV. According to the authors, bladder pain triggers a reflex contraction of the pelvic floor during voiding leading to DV. However, the authors did not present electromyographic data and DV was not defined in the article.[21]

Some investigators have tried differentiating between pelvic floor dyssynergia and striated urethral sphincter dyssynergia. In a study of 15 women with DV and retention (mean age 38.2 years) Deindl found inappropriate pelvic floor muscle relaxation in 11 and external urethral sphincter activation during voiding in four.[22] Biofeedback training was effective only in women with pelvic floor (pubococcygeus) activation thus having prognostic implications.

Young women presenting with urinary retention have been described to have increased urethral sphincter tone which might be hormonally triggered. Such women may have polycystic ovarian disease. They are typically between the age of 15 and 30 years and have increased sphincter volume and concentric needle EMG demonstration of abnormal decelerating bursts and complex repetitive discharges. This has been labeled the Fowler Syndrome.[23,24]

A higher than expected association of idiopathic hypercalciuria, ranging from 21-30%, has been noted in children with DV syndromes. However, almost all responded to behavioral therapy, dietary modifications and anticholinergics and treatment specifically directed at hypercalciuria was needed in only two percent.[25] The reason for an association between hypercalciuria and DV remains unclear. The authors postulated that calcium microcrystallization may cause injury to the urothelium and this could trigger a variety of urinary symptoms including DV.

It is probable that the entity DV is not homogenous and that there are several distinct etiologies that can lead to it. The end result is one of a dyssynergic sphincteric activity in the absence of a clearly defined neurological reason.

EPIDEMIOLOGY

The true estimate of DV in the general population is not known. Reported population estimates of DV are based on questionable methodology. A wide variation from 4.2-46.4% has been reported depending on the definition used and the methodology adopted.[7,26] It is probable that these figures represent a gross overestimate of the actual prevalence. The highest figure in this data was derived from a population survey in South Korea of 19,240 children.[26] However, although the authors termed the problem surveyed as DV, it appears that the authors were searching for any urinary symptom in the population rather than the specific problem of DV.

In tertiary care centers, DV constitutes up to 40% of referrals in the Pediatric Urology department.[27] In urology centers treating adults as well, DV is usually noted in 0.5-2% of patients.[18,28] Groutz found an equal prevalence in men and women with a mean age was 44.9 years in men and 51.5 years in women.[18]

PRESENTATION AND CLINICAL EVALUATION

Children with dysfunctional voiding often present with urinary incontinence both during the day as well as at night.[7] They may have urinary frequency, urgency, urge incontinence or nocturnal enuresis.[7] Such storage symptoms may result from associated detrusor overactivity, urinary infection or reduced bladder capacity consequent to a large residual urine and may be aggravated by constipation or behavioral disorders. A distinctive facial expression may be noted in some of these patients. Adults often present with a history of voiding difficulty or unexplained retention. Patients might have difficulty in initiating a void in public places, or might need physical or mental cues to void, such as the sound of running water or the need to 'deliberately' relax themselves.[18] Storage symptoms are common and may be the only presenting symptom.[29] Frequency and urgency was found in 94%, urge incontinence in 43%, voiding difficulty in 54% and urinary retention in 9%.[29] In another study, the mean urinary frequency in both men and women was noted to be 12 while the number of nocturnal voids was 3.5 and 3.0 in men and women respectively.[18] Patients with DV have been considered to be at risk for urinary tract infection (UTI),[12] although a recent study has disputed this premise.[30]

The history is directed towards an assessment of the type of urinary symptom, the severity of bother, the health and integrity of the urinary tract and the careful search for a primary neurological cause. Clinical examination must include an assessment of higher mental functions and their age-appropriateness, basic neurological evaluation including back and spine and a focused neuro-urological examination. Bowel function should be evaluated in detail [Table 2].

SCORING SYSTEMS

The AUA score has been used to assess patients with DV. In one study, half the patients had scores in the severe symptom (20-35) range.[18] Farhat *et al.*, devised a DV symptom score by comparing the scores of 104 children aged 3-10years with 54 age-matched controls across 10 questions related to urinary incontinence, voiding habits, urgency, posturing, bowel habits and stressful life conditions.[27] Nine of these questions are scored between 0 and 3 depending on whether

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Table 2: Clinical evaluation for dysfunctional voiding

History

Urinary system: Urgency, frequency, urinary incontinence, enuresis, voiding difficulty, hesitancy, dribbling, maneuvers to tackle urge, toilet training in children, urinary infections

Gastrointestinal system: Bowel evacuation frequency, constipation, encopresis

Diet: Fluid intake, diet consumed, beverage or alcohol consumption

Neurological system: Higher functions, milestones and age-appropriateness of behavior in children, lower limb neurological symptoms

Obstetric and gynecological history in women: Menstrual cycle irregularities, polycystic ovarian disease, parity, difficult labor, vaginal surgeries

Others: Pelvic or lower abdominal surgery, family history of urinary tract problems, emotional disturbance or trauma, school or workplace performance, behavioral disorders

Physical examination

Genitourinary system: Meatus, labia and vagina in women, scrotum and prostate in men, bladder fullness, kidneys

Gastrointestinal system: Rectal examination, fecal incontinence

Neurological system: Gait, back (meningomyelocele or other spinal abnormality, dimple, lipoma tuft of hair, sacrum), deep tendon reflexes, strength of the lower limbs, focused neuro-urological examination including the bulbocaverosal reflex, anal tone and perineal sensation, facial expression

Investigative evaluation (Individualize)

Micturitional diary, urine examination, blood glucose, USG for the bladder with prevoid and postvoid bladder volume, uroflow with EMG, invasive urodynamics evaluation, MRI of the spine

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the problem is noted almost never (0), less than half the time (1), about half the time (2) or almost every time (3). The last question is addressed to the parents to identify a stress situation in the family. This has a yes-no format, and is scored either '0' for no or '3' for yes. The authors derived cutoff values of 6 and 9 for girls and boys respectively for making a diagnosis of DV. Readers are directed to the original article for obtaining the actual elaborate scoring system. Another scoring system is the "Wetting and functional voiding disorder" scoring system.[31] Afshar *et al.*,devised a 14-item Likert scale questionnaire for children with dysfunctional elimination with a cutoff score of 11.[32]

Scoring systems seem to work well for those who devise them with practice patterns determining the discriminatory ability of the system.[33] They are not meant for making a diagnosis of DV.
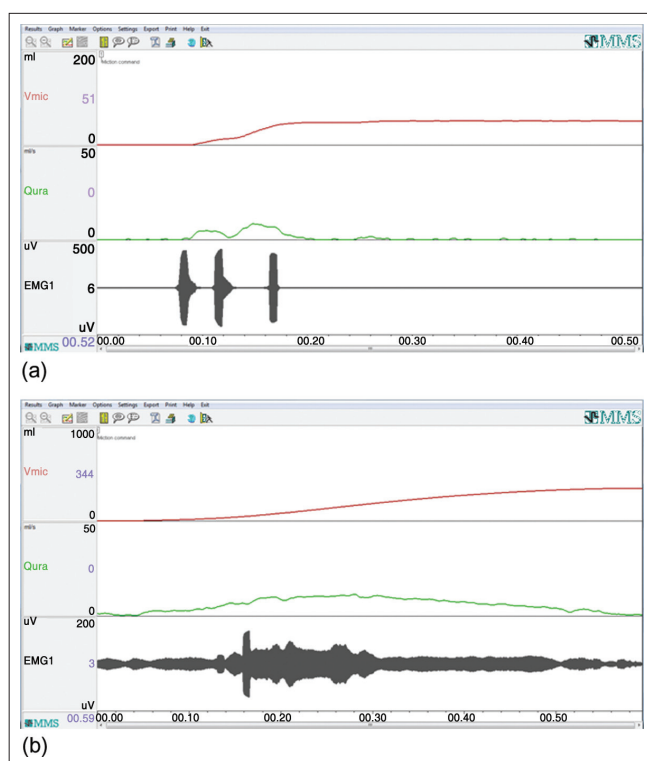

Figure 1: (a) Characteristic uroflow in dysfunctional voiding. A 10-year-old boy with daytime urinary incontinence, staccato voiding, elevated residual urine (60 ml) and no hydronephrosis, (b) Dysfunctional voiding in a 58-year-old woman with continuous slow flow and 140 ml residual urine without any underlying neurological etiology. The current guidelines make no provision for categorizing such patients

Table 3: Possible causes for "Staccato voiding"

Dysfunctional voiding

Bladder neck dysfunction

Abdominal straining during the test

Underactive detrusor

Failure to void comfortably in an artificial test environment

Unsupported legs due to inappropriately high toilet seat[48]

INVESTIGATIVE EVALUATION

Uroflow
The diagnosis of DV in children hinges on the repeated demonstration of a staccato pattern on uroflow testing [Figure 1a]. Classically, children show an interrupted pattern (presumably) because their sphincter does not relax completely during voiding. The normal uroflow pattern is a bell-shaped curve with a smooth up-slope and down-slope. An interrupted, staccato pattern usually implies interference by an uncoordinated sphincter. The detrusor being a smooth muscle needs to recruit to produce a contraction. As such it is incapable of sudden contraction and relaxation. Hence sudden changes in flow are unlikely to originate from the detrusor. On the other hand, both contraction and relaxation of the striated external sphincter can occur rapidly and this can cause the staccato voiding that is seen in DV. However, a similar voiding pattern may be observed in a patient with acontractile detrusor who voids by abdominal strain (again, a function of striated muscle).

The staccato pattern of voiding has been considered classical of DV. To label flow as staccato, the fluctuations should be more than the square root of the maximum flow rate.[34] However, staccato voiding is not exclusive to DV [Table 3]. Conversely, patients with DV may show a steady but slow flow [Figure 1b].[3,12] Depending on the severity of disease, the maximum flow rate in both men and women maybe in the low normal range.[12] Women with DV may void with a normal-appearing flow curve.[35]

When combined with EMG or fluoroscopy, increased SUS-PFC activity can be noted. There is sporadic increase-decrease in the EMG activity. Such observations have classically been made on needle EMG and may be more difficult to identify on surface EMG.[18] It may be difficult to demonstrate these findings in young children.

Urodynamics
DV is characterized by bladder outlet obstruction due to dyssynergic external sphincteric activity during voiding in the absence of neurological problems. The definitive test for obstruction, despite its inconveniences, remains urodynamics. Of note, there are no uniform criteria for defining obstruction in women. Groutz identified obstruction in 6.5% of 587 women with voiding difficulty and noted DV in 5% of these women.[36] They termed the finding 'learned voiding dysfunction' although no evidence was presented to justify the term. Kuo differentiated between pelvic floor obstruction and dyssynergic urethral sphincter on videourodynamics and found dyssynergic sphincter in one-fourth and dysyynergic pelvic floor in half of their patients.[29] Both were grouped as DV.

On an average, men with DV are likely to have higher voiding detrusor pressures as compared to women. Groutz

reported Pdet.Qmax and Pdet.max values in men and women to be 43.8 cm H20 and 53.3 cm H20 versus 19.8 cm H20 and 31.5 cm H20 respectively (*P*=0.002 and 0.005). [18] DV has been noted in 14% of young men with voiding difficulty subjected to videourodynamics.[37]

Video may show dilatation of the urethra down to the level of the external sphincter with a wide-open bladder neck.[29] This appearance can easily exclude bladder neck obstruction. However, some individuals can have slow or intermittent flow with lack of relaxation of the entire sphincter mechanism and pelvic floor including the bladder neck without an underlying neurological disorder. What should one label this as? Guideline documents are silent on the video appearance of the urethra for very valid reasons. The data on videourodynamics suffers from marked recruitment bias and remains poorly standardized. There is little data on what degree of dilatation should serve as a cutoff for abnormality. While video can help in making the diagnosis in individual patients, we need to wait for better quality data before it can be included in the diagnostic algorithm.

Urodynamics may prove challenging in children, especially when one attempts to obtain a valid pressure flow trace. Cooperation of the child and the narrow size of the urethra are the major hurdles. A relaxed child-friendly environment and patience on the part of the technician are paramount.

Fifteen of 111 girls evaluated for incontinence were noted to have DV on urodynamics.[38] In these girls, DV was associated with profound urinary tract changes and the classical finding was urodynamic bladder outlet obstruction due to dyssynergic sphincteric activity. Of note, 90% of children with DV show detrusor overactivity, a fact that explains the high prevalence of storage symptoms. Also, in most studies, the entry criterion is incontinence and this introduces a bias towards selection of children with detrusor overactivity. In a large series of children undergoing videourodynamics, 32% of children had DV.[39]

Urodynamics remains an uncomfortable test for children. Glassberg has made a case for using uroflow with EMG as a middle path between the "impressionist" approach of doing minimal tests and a uroflow and the "purist" approach of doing detailed evaluation including a urodynamics evaluation for all patients.[3]

It is suggested that patients be triaged based on their clinical presentation rather than being evaluated on the basis of treatment philosophies of individual units [Figure 2].
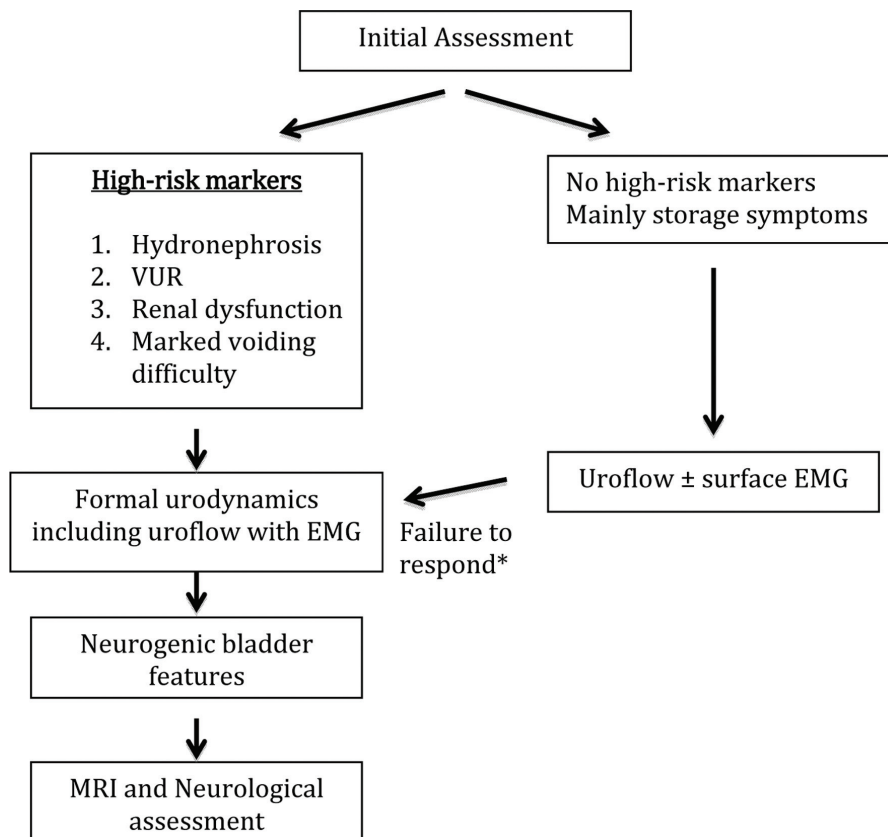

Figure 2: Triage for patients with dysfunctional voiding. *In patients with refractory non-neurogenic DV, two-thirds of patients will show abnormality on formal urodynamics[77]

Ultrasonography

An initial ultrasonography (USG) examination of the bladder including a measure of the residual urine is a basic evaluation step in patients with lower urinary tract symptoms. However, infants and toddlers may not always empty the bladder. In older children, a consistent residual of >20 ml is considered abnormal.[34] Limited data in men suggests that transrectal voiding ultrasound may help in identifying men with DV.[40] In neurologically normal men with voiding difficulty without any obvious cause, 85% of men with bladder neck dilatation of >7 mm on a voiding transrectal ultrasound were noted to have DV on a complete urodynamics.[40] Perineal USG has been used in women with DV to assess sphincter volume.[41] Urethral sphincter volume of more than $1.96\,cm^3$ was predictive of DV in women with recurrent urinary infection. Perineal USG can also be used to evaluate paradoxical pelvic floor movement which may often be seen in children with DV.[42]

Abdominal USG has also been used in lieu of rectal examination to identify chronic constipation in children with DV.[7] Rectal diameter of >3.5 cm signifies constipation.[43]

Cystoscopy

However, in some patients, presumably with longstanding or severe obstruction, trabeculations may be noted.[18] No cystoscopic finding is diagnostic of DV. In patients with suspicion of anatomical abnormalities of the lower urinary tract, a diagnostic cystoscopy may be performed. Routine diagnostic cystoscopy is not recommended.

MICTURATING CYSTOURETHROGRAM

Children with DV often show abnormalities on micturating cystourethrogram (MCUG). Spinning top urethra consisting of dilatation of the proximal urethra may be seen in girls while dilatation of the prostatic urethra may be observed in boys.[44] There may be associated mucosal irregularities or reflux.[45] Kutlu *et al.*, found a spinning top urethra in 55% of children with DV evaluated by voiding cystourethrogram.[46] These children had significantly higher voiding pressures and were more prone to recurrent urinary infections and reflux. MCUG is recommended in all patients with symptomatic voiding difficulty. In some situations, a videourodynamics can substitute for a formal MCUG.

Abnormalities on MCUG serve as a marker for potentially more serious disease.

MANAGEMENT

The objective of treatment of DV is to enable patients to learn to relax the bladder outlet during voiding. Since no one method works reliably, several approaches have been tried including suggestion or hypnosis, bladder retraining, bladder drill, biofeedback for the pelvic floor, anticholinergic medication to relax the bladder and alpha adrenergic blockers to relax the bladder neck.[18] Conservative urotherapy including biofeedback is appropriate initial management for patients without high risk factors [Figure 2]. Those with high-risk markers may require early institution of clean intermittent self-catheterization. The management of these patients must proceed along lines similar to that for classical neurogenic bladder including clean intermittent catheterization and measures to control storage pressures and is not discussed further. Patients who progress despite treatment or who present in renal insufficiency will require renal replacement therapy including dialysis and transplantation. Conclusions regarding treatment are hampered by the lack of quality data.[47]

UROTHERAPY

Urotherapy encompasses all non-pharmacological, non-surgical therapies that can help with lower urinary tract function.[7] Urotherapy has been extensively reported in pediatric patients and can reduce constipation, urinary infections and interventions for vesicoureteral reflux.[7] The various components of urotherapy are not standardized and the measures can be combined with pharmacotherapy.[7] Urotherapy needs to be an ongoing process. The first step consists of initial evaluation, education and management. Step two is a series of biofeedback sessions including having the child watch a uroflow curve and teaching how to identify and contract/relax the pelvic floor muscles.[48] Step three consists of ongoing bowel care, preparation of voiding diary, pelvic floor exercises. And the final step consists of identification of persistent behavioral and psychological issues. This system of management when used with escalation of treatment based on response has been noted to give 90-100% success in the children with DV.[7] While interpreting this data one must remember that many children have been diagnosed on the basis of storage symptoms and a staccato uroflow alone.

Impact of bowel care
Several authors have suggested a correlation between bowel symptoms and the urinary tract.[49,50] In a population-based study in South Korea, symptoms of DV were noted in 46.4%, abnormal bowel function in 31.3% and both in 18.4%, a statistically significant association.[51] The commonest explanation has been the common innervation of the two systems (S2 to S4) and their anatomical proximity.[52] It is possible that the abnormal pelvic floor function that is responsible for DV may also lead to constipation independently. However, others have not found a significant association.[53] Aggressive management of constipation may have an independent favorable impact on urinary tract function and one-third of children may become free from recurrent UTIs.[43]

Biofeedback
Behavioral therapy is based on the presumption that the

disorder is a learned one and hence potentially reversible. Biofeedback has been used in children as young as four years of age.[54] Upwards of 80% children will experience improvement marked by a reduction in incontinence and recurrent urinary infection. Multiple sittings and periodic reinforcement is necessary. Improvements in flow rates and residual urine does not always correlate with outcome.[54] Results appear durable at three years and the treatment also seems to help those children in whom urotherapy has failed.[55] The inclusion of biofeedback in urotherapy is more likely to lead to an improvement in residual urine.[56] Two broad approaches to biofeedback are visual feedback of the uroflow curve and teaching perineal muscle identification by EMG electrodes. The former is usually quicker.[7] Cyclical uroflow sessions with audio feedback and charts or animation may be used.[55,57] Biofeedback may also resolve paradoxical pelvic floor movement seen in some children with DV.[42] However, the clinical implication of this finding, seen in 30% of normal children, is not known.[42]

Biofeedback may also be useful in adult women with DV presenting with recurrent UTIs. Biofeedback can be performed using uroflowmetry biofeedback, biofeedback for re-training the pelvic floor or by combinations of these techniques.[58] Biofeedback may be more useful in women with uncoordinated pubococcygeus muscles and less in women with uncoordinated sphincters.[22]

In multiple studies from a few centers, biofeedback has been shown to be useful in suggesting a strong center-specific response factor. Training of instructors, motivation of parents and child, diagnostic algorithms in use at the specific center and the type of patient cohort being treated are all likely to influence the outcome significantly.

ALPHA BLOCKERS

Strictly speaking, one would not expect an alpha-blocker to improve voiding in patients with DV, a condition that affects the striated sphincter rather than the bladder neck. In small series, alpha blockers have been shown to give symptomatic improvement.[29,59] Matching objective improvement may not be noted and hence in patients with severe DV, urodynamic confirmation of response is warranted. The treatment is well tolerated in both adults and children.[60]

BOTULINUM TOXIN

Small case series show benefit with botulinum toxin injection into the sphincter. Injection of Dysport (Botulinum toxin A) 500 units into the external urethral sphincter of nine girls with refractory DV resulted in a significant increase in voided volume and reduction in post-void residual but no difference in the uroflow rates.[61] Seven girls had resolution of voiding difficulty. Of the five girls with incontinence prior to the injection, incontinence resolved in four at a follow up of six months. In another study, seven of eight adult patients with DV voided after botulinum toxin.[62] Various authors have used between 50-100 units diluted into 1-8 ml usually into four sites. In women, the injection is usually performed periurethrally while in men the injection is given cystoscopically into the sphincter.[62] The effect of botulinum toxin may take one to two weeks. Patients may be offered an indwelling catheter or intermittent self-catheterization in the interim.

NEUROMODULATION

Sacral neuromodulation has been used for intractable lower urinary tract dysfunction.[63] The feasibility and efficacy of neuromodulation has been well demonstrated.[64] Storage symptoms resolve in about three-fourths of children.[64] In two series which included children with DV the response of DV to neuromodulation with the Interstim neuromodulation device was modest. Retention was relieved in one of four children in one study while in the other, two of six children on clean intermittent catheterization could stop catheterizing.[64,65] In this latter study, about 60% of children with voiding difficulty had some benefit.[65] While both studies performed pre-treatment urodynamics, neither reported urodynamic outcomes. Neuromodulation offers the additional incentive of a potential improvement in constipation and irritable bowel symptoms.[64,66]

Percutaneous tibial nerve stimulation performed for 30 min on a weekly schedule for 12 weeks has been shown to benefit patients with DV. Patients with DV were significantly more likely to benefit as compared to those with overactive bladder. However, storage symptoms were more likely to demonstrate an objective benefit as compared to residual urine and flow rates.[67]

In women with the Fowler's syndrome, a form of DV, long-term outcomes at 10 years are now available and show that 78% of women continue to void spontaneously. Neuromodulation needs careful supervision and is not without complication. Failure of the procedure may occur in 25% of patients and 30-50% may need revision.[24]

OTHER THERAPIES

Women with DV have often been subjected to urethral dilatation in the past although literature in this regard is sparse. In a series of women undergoing urethral dilatation for a voiding dysfunction, there was a small increase in flow rates.[68] The benefits of dilatation were sustained at six months in only 19% and there was new onset of stress incontinence in 13%. This series of women, however, did not stick to the strictly defined DV that we have used in this article.

Bladder neck incision was offered to two men out of six in another series and intriguingly both of them improved.[18] This is counter-intuitive and might represent misdiagnosis

or a flaw in our understanding of the condition.

VESICOURETERAL REFLUX AND DYSFUNCTIONAL VOIDING

Patients with DV often have elevated voiding and storage pressures and this can contribute to the development or persistence of vesicoureteral (VUR) reflux. It is important to carefully assess all children with reflux for subtle signs of DV. The treatment of DV in such children can improve the chances of spontaneous resolution of the reflux and may also reduce recurrent urinary infection.[69] Fifteen percent of children with DV show reflux.[39] Children with untreated DV undergoing ureteral re-implantation may be at a higher risk for developing recurrent reflux or a new bladder diverticulum. Three percent of 492 patients undergoing ureteric reimplantation developed a bladder diverticulum. Sixty percent of these had severe dysfunctional voiding on the preoperative urodynamics evaluation.[70] Conservative therapy may help in the resolution of vesicoureteral reflux in those with DV who have it. A significant decrease in the DV symptom score may predict VUR resolution.[71]

TRANSPLANTATION IN PATIENTS WITH DYSFUNCTIONAL VOIDING

There are several reported series of children with dysfunctional lower urinary tracts undergoing transplantation. In most of these reports the term DV has been used rather loosely to include any patient with an abnormal bladder. In general, the graft survival outcomes are 5% lesser at one year than age-matched controls and the incidence of urological complications is higher.[2] All patients with suspected bladder dysfunction undergoing transplantation must undergo a formal urodynamics evaluation.[77]

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