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Review

Pediatric urinary incontinence: Classification, evaluation, and management



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KEYWORDS

Urinary incontinence;
Diurnal enuresis;
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Abstract

Objective: To review the classification, evaluation, and management of pediatric urinary incontinence.

Methods: An examination of texts and peer-reviewed literature was performed to identify subject matter relevant to the stated objectives, with the experience of the senior author used in cases where the literature failed to provide guidance.

Results: On the basis of our review, we identified the International Children's Continence Society's (ICCS) statement standardizing the terminology for lower urinary tract function in children and present a logical classification scheme for incontinence. After an epidemiology review, we discuss the appropriate evaluation of the incontinent child, of which the cornerstones are a detailed history and thorough physical exam. Finally, a concise discussion of the management of daytime incontinence, nocturnal enuresis, and neurogenic and anatomic incontinence is presented, with deference to evidence-based approaches where available. Depending on the type of incontinence, the management strategies can include behavioral, pharmacologic, and/or surgical approaches.

Conclusion: Pediatric urinary incontinence is a common condition which, after appropriate evaluation, can be successfully treated.

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Abbreviations: UI, urinary incontinence; ICCS, International Children's Continence Society; PVR, postvoid residual (urine); MNE, monosymptomatic nocturnal enuresis; NMNE, non-monosymptomatic nocturnal enuresis; FDA, United States Food and Drug Administration; LUTS, lower urinary tract symptoms; VCUG, voiding cystourethrogram; EMG, electromyography; DDAVP, desmopressin; TCA, tricyclic antidepressant; TENS, transcutaneous electrical nerve stimulation; PTNS, posterior tibial nerve stimulation; NGB, neurogenic bladder; BTX-A, onabotulinum toxin – type A; CIC, clean intermittent catheterization; AUS, artificial urethral sphincter.

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Introduction

The purpose of this article is to introduce the up-to-date nomenclature for pediatric urinary incontinence after which a general framework to guide the practitioner in classifying, evaluating, and managing an incontinent child is provided. A detailed discussion of the pathologic processes and long-term management of specific conditions causing incontinence is outside the scope of this review.

Definitions and classification

Urinary incontinence (UI) is any involuntary or uncontrollable leakage of urine [1]. As urinary incontinence can be present in a variety of situations, the International Children's Continence Society (ICCS) has unified the definitions and terminology of pediatric urinary incontinence and lower urinary tract symptoms [1]. *Continuous incontinence* refers to constant leakage of urine and can occur even in infants and young children. *Intermittent incontinence* is defined as any urine leakage in discrete amounts in a child at least 5 years old. When a child has never been dry of urine, they have *primary incontinence*. However, if a child becomes incontinent after a previous period of good urinary control, they have *secondary incontinence*. When intermittent incontinence occurs while the child is awake, it is called *daytime incontinence*. Conversely, when occurring while asleep it is described as *enuresis* or *nocturnal enuresis*, which are synonymous terms. When enuresis occurs in isolation, that is when there is no history of lower urinary tract symptoms (except nocturia), it is defined as monosymptomatic nocturnal enuresis (MNE). Children with enuresis and any other lower urinary tract symptoms (LUTS) are defined as having non-monosymptomatic nocturnal enuresis (NMNE). These LUTS can be daytime incontinence, urgency, frequency, dysuria/stranguria, or others. When bedwetting occurs in a child with concomitant daytime lower urinary tract symptoms causing incontinence, the child has a dual diagnosis of nocturnal enuresis with daytime urinary incontinence. *Urge incontinence* refers to urinary leakage resulting from urgency. *Giggle incontinence* is an unusual phenomenon most often found in girls in which nearly complete voiding occurs during or immediately after laughing. The use of terms 'total incontinence' and 'diurnal enuresis' is discouraged. The former term is replaced by continuous incontinence, whereas children with both daytime and nighttime incontinence, formerly described as 'diurnal enuresis', have a dual diagnosis – daytime incontinence and nocturnal enuresis. Neurologically intact children with urodynamically proven intermittent voluntary contractions of the external urethral sphincter or pelvic floor during voiding are said to have *dysfunctional voiding*.

Children with urinary incontinence will be found to have one of three etiologies for their incontinence (Table 1). Anatomic lesions can lead to continuous incontinence, a vital clue that can be elicited from the history and examination. In females, ectopic ureters with orifices distal to the external urinary sphincter will cause continuous incontinence, as do the sphincteric deficiencies seen in children with epispadias, bladder and cloacal exstrophy. Treatment for other congenital urinary tract anomalies such as posterior urethral valves and ectopic ureteroceles can damage the normal sphincter control mechanisms and secondarily cause urinary incontinence.

Neurologic lesions in the brain, spinal cord, or autonomic and/or somatic peripheral nervous system(s) can result in incontinence. The most common cause of neurogenic bladder dysfunction in

Table 1 General classification of urinary incontinence.

Anatomic	Congenital
	Ectopic ureter Bladder exstrophy-epispadias complex Persistent urogenital sinus with high genitourinary confluence Urethral duplication
	Acquired
	Iatrogenic injury to external urethral sphincter (e.g. after posterior urethral valve ablation or ectopic ureterocele excision)
Neurologic	Congenital
	Myelodysplasia Occult spinal dysraphisms Sacral agenesis
	Acquired
	Central nervous system lesion (e.g. cerebral palsy, tumor, radiation, stroke, multiple sclerosis) Peripheral nervous system injury (e.g. injury to pelvic plexus and bladder efferents/afferents)
Functional	Daytime incontinence
	Nocturnal Enuresis

children is myelodysplasia [2], a term used to describe various abnormalities resulting from failed fusion of the vertebral column with associated malformations of the spinal cord and surrounding structures. More specifically, myelodysplastic patients can have a protrusion of the meninges with intact spinal cord elements (meningocele), protrusion of spinal cord elements with the meninges (myelomeningocele), or evagination of fat, meninges, and spinal cord elements (lipomyelomeningocele). Occult spinal dysraphisms are conditions such as fatty filum terminale, intramedullary lipoma, neurenteric cysts, terminal syringohydromyelia with subtle skin defects and physical findings like midline nevi, hemangioma, lower midline hairy patch, or an asymmetric gluteal cleft that can suggest a tethered spinal cord as a cause for urinary incontinence. Occult spinal dysraphisms are distinct from spina bifida occulta, which is the mildest type of vertebral bony defect in which the sacrum has failed to fuse in the midline but does not involve the spinal cord or dura. Spina bifida occulta is an incidental finding as patients have no symptoms.

While spinal dysraphisms may be the most common cause of neurogenic pediatric urinary incontinence, any acquired condition that interrupts the normal cortical control mechanism, spinal pathways, or bladder efferents and afferents can result in neurogenic urinary incontinence. The conditions that cause this are wide and varied, but some examples include: hypoxic insults like cerebral palsy or pediatric strokes affecting the brainstem or higher urinary control centers, brain or spinal cord tumors that primarily or as a result of their treatment result in incontinence, and radical pelvic surgery for congenital anomalies (e.g. a 'pull-thru' operation for imperforate anus) or cancers (e.g. a radical pelvic exenteration for rhabdomyosarcoma) that damage peripheral autonomic or somatic nerves and sacrifice continence.

Finally, functional incontinence can occur in children without any known anatomic or structural neurologic lesions. The etiology of daytime incontinence and nocturnal enuresis is varied. Urinary tract infections can cause inflammation and irritation that lead to transient incontinence. Children can have behavioral issues such as urine

holding that leads to detrusor overactivity and/or overflow incontinence. Detrusor overactivity has also been implicated in nocturnal enuresis, as have lower than normal levels of nocturnal vasopressin secretion and impaired sleep arousal patterns [3–5]. Mood disorders such as attention deficit hyperactivity disorder and anxiety as well as conduct disorders are frequent comorbidities encountered in children with UI [6–8], as is constipation [9,10]. Previously continent children experiencing a stressful life event such as the divorce of parents or loss of a sibling can develop secondary incontinence [11,12].

Epidemiology

Pediatric urinary incontinence is a common disorder affecting children of all ages and cultures. Unfortunately due to their population-based nature, most studies addressing the prevalence of pediatric UI do not classify by the etiology of incontinence, yet they are still helpful in understanding the prevalence of urinary incontinence in general. A sample of 1192 three to twelve year old children from the United States identified daytime incontinence in 10% of children [13]. Kajiwara identified daytime incontinence in 6.3% of seven to twelve year old Japanese primary school children and affected males and females equally [14]. Sureshkumar performed a population based survey and found that 19.2% of school age children had experienced daytime UI at least once in the 6 months prior to survey, with a higher prevalence in females than males [15]. Another Australian study of five to twelve year old school children identified a prevalence of 2% for isolated daytime UI and 4% for daytime incontinence and nocturnal enuresis [16]. Similarly, 6% of school age girls and 3.8% of school age boys had daytime UI in one Swedish study [17].

Enuresis is also highly prevalent. In a study of 10,960 children in the United States, nocturnal enuresis was found in boys at seven and ten years was 9% and 7%, respectively, and in girls at those ages was 6% and 3% [18]. Bloom identified enuresis in 18% of another US sample, though the median age was lower [13]. A large population based study in Great Britain suggested that 20% of children in the first grade occasionally wet the bed [19]. Given the prevalence of pediatric UI and associated symptoms, it is not surprising that it represents 7% and 40% of general pediatric outpatient referrals and pediatric urology visits, respectively [20,21].

As mentioned, no population-based studies investigating the prevalence of pediatric UI do so by their specific etiologies. It is reasonable to conclude from clinical experience and from the relative rarity of anatomic and neurologic causes that the largest proportions of children with urinary incontinence do not have organic causes. Nevertheless, it is always prudent to consider these etiologies in the initial evaluation and subsequent management of the incontinent child.

Evaluation

A detailed history and physical exam are the cornerstones of the evaluation of pediatric urinary incontinence and guide which tests, if any, should be considered. Table 2 shows the evaluation tools available to the clinician faced with an incontinent child.

The history should be broad yet specific and include information from the child as well as the caregiver/parent. Detailed information

regarding when (daytime or nighttime), where (at school/public place and/or at home), how often (once a week or daily), and how much (small or large volume) leakage occurs is important, as is information pertaining to bowel habits, with a determination of the frequency and consistency of bowel movements and presence of any bowel accidents. Frequency-volume charts (i.e. voiding diaries) can be helpful in gathering this information about voiding and elimination habits. The provider should identify if the child has had any previous operations including but not limited to abdominal, genitourinary, and pelvic surgery. Knowing whether there was ever a period of complete urinary control in a child of toilet-trained age can help distinguish between primary and secondary incontinence. Details of the perinatal history are important, as are questions focused on the child's gross and fine motor and behavioral development. Motor impairments could suggest underlying spinal cord pathology also affecting the lower urinary tract. School performance and behavior should be queried. As attention deficit hyperactivity disorder has been associated with urinary incontinence, one should ask whether the teacher has noted the child to be easily distracted or has problems with impulsivity. Conversely, high achieving children who are very focused on school performance can acquire voiding postponement and urge incontinence and should thus be identified. The provider should query whether there is any evidence of stressors at home such as parental conflict or recent losses that could cause developmental regression and secondary incontinence. Importantly, information should be obtained regarding any history of urinary tract infections.

Like a detailed history aimed at identifying clues toward the etiology of a child's incontinence, a focused physical exam can shed light on the etiology as well as direct testing. The gross motor function of a child can simply be observed as he/she walks into the examination suite. An abdominal exam assessing for abdominal masses, a stool filled colon, or distended bladder should be performed. The integrity of the lower abdomen and pubic symphysis should be assessed. In girls, the genital exam should note the presence and relation of the urethra to the vaginal and rectal orifices. A patulous urethra in a girl with a widened pubic diastasis suggests female epispadias. Observation of pooling urine in the vaginal introitus is an important clue that an ectopic ureter might be present. The child's back should be examined, with particular attention paid to recognizing signs of occult spinal dysraphism (hairy tufts, subcutaneous lipomas, skin discoloration, or sacral dimples above the gluteal cleft) and tethered cord (asymmetric gluteal fold). The neurological exam should assess lower extremity reflexes, perineal sensation, anal tone and reflex, and the presence of the bulbocavernosus reflex.

The history and physical exam will provide important clues as to which additional tests will help further identify the cause of urinary incontinence. A urinalysis should be performed in most cases, as infection (bacteriuria, pyuria, hematuria), renal damage with high urine output (proteinuria, low specific gravity), or diabetes (glucosuria) as a cause for osmotic diuresis can be identified. An easy test to add to any physical exam that gives important information regarding bladder emptying is a bladder scan with postvoid residual (PVR) measurement. This will aid the provider in knowing how well the child empties, and can prompt further studies when large PVRs are found on successive tests. Most children should be able to empty to 5 cm³ or less, whereas repeated residual measurements of 20 cm³ or more are concerning for pathology in the bladder and/or urethra [1]. When discovered, high PVRs could require renal and bladder ultrasound, voiding cystourethrogram, and/or urodynamic studies

Table 2 Diagnostic options and their indications in the workup of pediatric urinary incontinence.

Diagnostic option	Indications	Comments	Findings
History	<ul style="list-style-type: none"> All patients 	<ul style="list-style-type: none"> Helps guide practitioner towards neurogenic, anatomic, or functional incontinence 	
Physical exam	<ul style="list-style-type: none"> All patients 	<ul style="list-style-type: none"> Helps guide practitioner towards neurogenic, anatomic, or functional incontinence 	
Urinalysis	<ul style="list-style-type: none"> All patients 		<ul style="list-style-type: none"> Bacteruria/pyuria: infection Proteinuria: kidney damage with possible high urine output Glucosuria: diabetes mellitus with possible osmotic diuresis
Bladder scan with postvoid residual (PVR) measurement	<ul style="list-style-type: none"> All patients 	<ul style="list-style-type: none"> Not necessary if planning to order RBUS 	<ul style="list-style-type: none"> PVR consistently > 20 ml on several measures needs workup to identify if neurogenic, myogenic, or anatomic cause is present
Uroflowmetry	<ul style="list-style-type: none"> Most patients 	<ul style="list-style-type: none"> Can include EMG if full urodynamics not planned 	See Fig. 1
Renal and bladder ultrasound (RBUS)	<ul style="list-style-type: none"> Most patients 	<ul style="list-style-type: none"> Provides functional information (estimated bladder capacity, bladder emptying/postvoid residual, bladder wall thickness) 	<ul style="list-style-type: none"> Identifies gross upper and lower urinary tract pathology Identifies constipation Renal scarring suggests vesicoureteral reflux (VUR) Dilatation of ureter and duplicated upper tract suggestive of ectopic ureter Thickened bladder indicates high voiding pressures from distal obstruction (anatomic – PUV; neurologic – detrusor-sphincter dyssynergia)
Abdominal radiography	<ul style="list-style-type: none"> Most patients 		<ul style="list-style-type: none"> Identifies gross bony anomalies Identifies constipation
Urodynamics	<ul style="list-style-type: none"> Failed medical or behavioral therapy Suspected neurogenic pathology 	<ul style="list-style-type: none"> Storage measurements include: maximum bladder capacity, P_{det} at bladder capacity, detrusor leak point pressure, abdominal leak point pressure, bladder compliance, detrusor overactivity Emptying measurements include: uroflowmetry with EMG, maximal P_{det} during void, voided volume, PVR Applicable in those < 6 months of age 	<ul style="list-style-type: none"> Identifies neurologic etiology Provides evidence to guide treatment
Spinal ultrasound	<ul style="list-style-type: none"> Suspected spinal pathology 	<ul style="list-style-type: none"> Applicable in those < 6 months of age 	<ul style="list-style-type: none"> Identifies spinal dysraphisms, tethered spinal cords, and other occult spinal lesions associated with neurogenic incontinence
Spinal MRI	<ul style="list-style-type: none"> Suspected spinal pathology 	<ul style="list-style-type: none"> Applicable in those > 6 months of age Requires sedation in many children 	<ul style="list-style-type: none"> Identifies spinal dysraphisms, tethered spinal cords, and other occult spinal lesions associated with neurogenic incontinence
VCUG	<ul style="list-style-type: none"> Post-UTI Suspected anatomic abnormality (e.g. ureterocele, ectopic ureter, duplicated urethra) Adolescent males with incontinence 		<ul style="list-style-type: none"> Identifies posterior urethral valves Identifies ectopic ureters with orifices at external urethral sphincter 'Spinning top' urethra indicative of dysfunctional voiding
Magnetic resonance imaging (MRI)/urography (MRU)	<ul style="list-style-type: none"> Suspected anatomic pathology (e.g. ectopic ureter) 	<ul style="list-style-type: none"> Requires sedation 	<ul style="list-style-type: none"> Clarifies anatomy of ectopic ureters and their renal component, Müllerian remnants, and other unusual anatomic abnormalities
Cystoscopy	<ul style="list-style-type: none"> Rarely used in diagnosis of incontinence Appropriate when other diagnostic modalities have not provided etiology of incontinence 		<ul style="list-style-type: none"> Confirms findings of diagnostic imaging studies

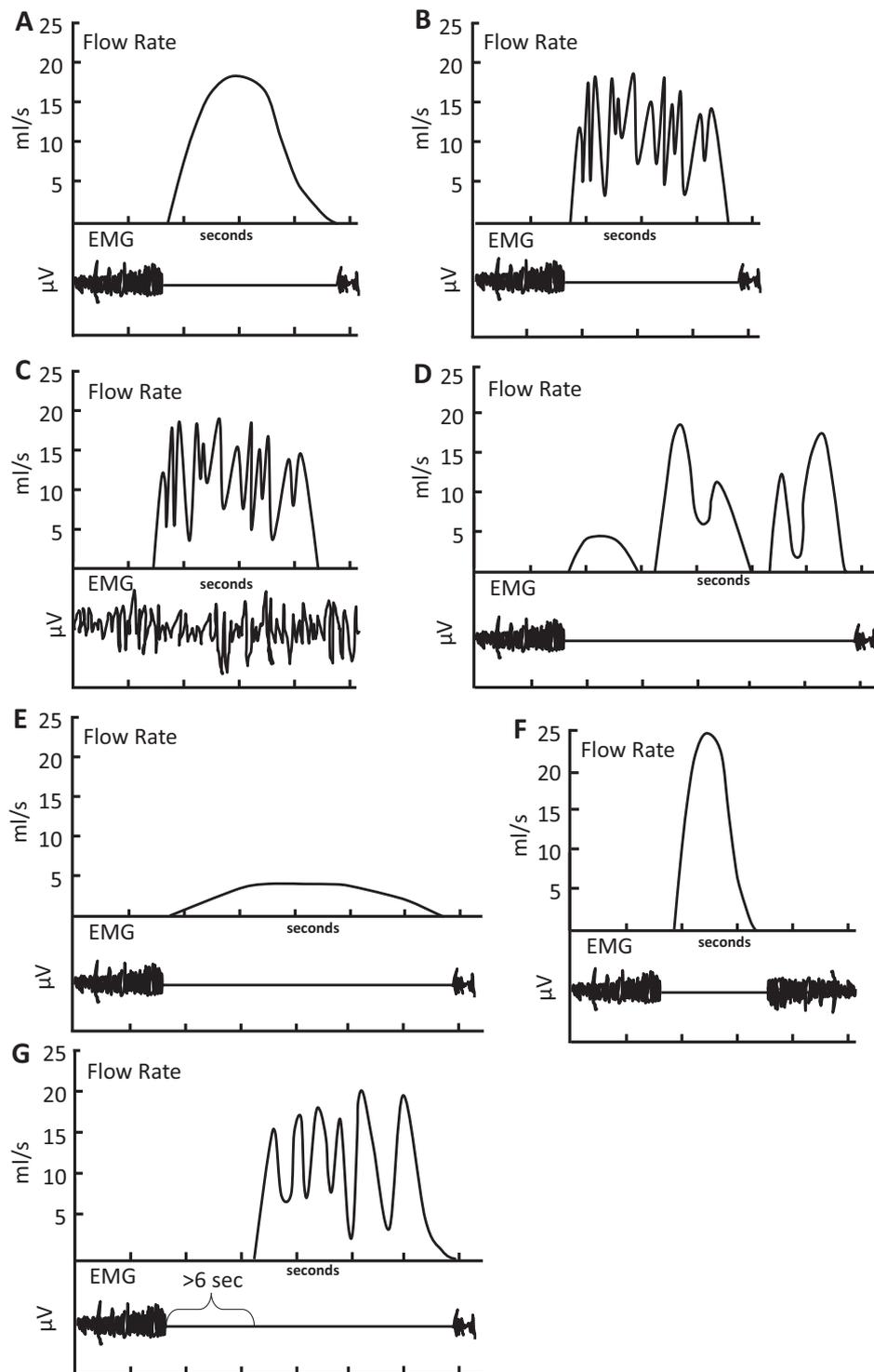


Fig. 1 Examples of uroflowmetry in children. (A) Normal flow showing bell-shaped curve and cessation of external sphincter activity on EMG. (B and C) Staccato shaped flow, which can occur with a weak or unsustained detrusor contraction but quiet external urethral sphincter (as in B) or with periodic bursts of sphincter activity on EMG while voiding with a continuous but varying flow rate (as in C). To qualify as a Staccato pattern, the fluctuations in flow should be larger than the square root of the maximal flow rate. (D) Interrupted or fractionated voiding notable for periods of no urine flow in the absence of EMG activity. Like the Staccato pattern, this can occur with an unsustained detrusor contraction but also result when voiding is achieved via abdominal muscle contractions in the presence of an acontractile bladder. (E) Plateau shaped flow showing low amplitude, prolonged void and cessation of sphincter activity on EMG. This occurs as a result of a fixed anatomic obstruction or a weak detrusor contraction. Plateau shaped flow can also occur with a tonically active external sphincter (not depicted). (F) Tower shaped flow with a high amplitude, short duration of flow caused by detrusor overactivity; may result in urge incontinence. (G) Primary bladder neck dysfunction with urine flow beginning at least 6 s after the cessation of sphincter activity on EMG.

to elucidate if the high residual is from an anatomic, neurologic, or functional cause.

Similarly, a uroflow with external sphincter electromyography (EMG) is a test that can easily be done in the clinician's office that can provide clues as to the coordination of pelvic floor during micturition. The decision to exclude EMG from the uroflow can be made if a full urodynamic study (which includes external sphincter EMG) is going to be performed. The EMG, whether obtained during uroflowmetry or a urodynamic study, provides critical additional information that can prevent incorrect diagnoses [22]. The child should be instructed to drink fluid in an amount equal to their estimated bladder capacity 60 min prior to the test. Also, two tests (each with a voided volume between 50% and 100% of estimated bladder capacity) should ideally be performed to account for variability between curves generated from the same individuals. As there are no pediatric specific standardized flow rates, the pattern of the flow curve is used to distinguish pathologic from non-pathologic emptying. A normal flow curve should be bell-shaped (Fig. 1). A staccato-like uroflow has peaks and troughs but a continuous urinary stream. This can occur with a weak or unsustained detrusor contraction in the presence of a quiet EMG or with an adequate detrusor contracting against an active external sphincter, as seen in dysfunctional voiding. An interrupted flow curve (fractionated voiding) has periods of zero flow in the absence of EMG activity, and can occur with an unsustained detrusor contraction or result when voiding is achieved via abdominal muscle contractions in the presence of an acontractile bladder. A flat, plateau-like flow curve is suggestive of either a fixed anatomic obstruction, a weak bladder contraction (from either a neurogenic or myogenic cause), or a tonically active external sphincter. A high amplitude, short duration "tower-shaped" flow curve is suggestive of detrusor overactivity potentially resulting in urge incontinence. Glassberg has used uroflow and EMG to detect primary bladder neck dysfunction in those who's time between pelvic floor relaxation and the initiation of urine flow (EMG lag time) was greater than 6 s [23].

The renal and bladder ultrasound should be considered an appropriate non-invasive test in most children with urinary incontinence. The ultrasound should be detailed enough to determine the presence or absence of duplicated renal collecting system, renal morphology and scarring, upper tract dilatation, bladder capacity, and bladder wall thickening or irregularity. Both the upper and lower urinary tracts should be imaged with a full and empty bladder. A plain film of the abdomen should be considered in most cases as well, with particular attention directed towards identifying the amount of stool in the colon, and the presence of any vertebral bony anomalies (possibly indicating a neurogenic cause of urinary incontinence) and/or a widened pubic diastasis (possibly indicating a disorder along the epispadias-exstrophy spectrum).

Urodynamics with external urethral sphincter electromyography are indicated for those with a suspected or proven neurologic lesion (tethered cord on MRI, all patients with spina bifida, patients who have had radical abdominopelvic surgery), incontinent adolescent males with late diagnosis of posterior urethral valves, and in patients who fail behavioral or medical therapy. The specifics of the urodynamics study are described in detail by MacLellen and Bauer [2]. Briefly, the test includes an 11Fr triple lumen urodynamic catheter in the bladder to measure intravesical pressure (P_{ves}), a small balloon catheter in the rectum to measure intrabdominal pressure (P_{abd}), and either a 24-gauge needle electrode placed into the skeletal muscle

of the external urethral sphincter [24] or perineal patch electrodes [25] to measure external urethral sphincter activity. The detrusor pressure (P_{det}) is calculated by subtracting P_{abd} from P_{ves} . These measuring devices are connected to commercially available urodynamic systems to record and display the measurements. Upon inserting the urethral catheter, the bladder pressure should be noted. The initial voiding opportunity allows uroflowmetry with EMG, the detrusor and abdominal pressures during voiding, and the voided and residual urine volume to be measured. The bladder is then filled with warmed 37 degree saline at a rate equal to one-tenth of the child's predicted or known capacity (capacity/10). Key measurements taken during bladder filling and storage include the maximal bladder capacity and its associated detrusor pressure, detrusor leak point pressure, presence of detrusor overactivity, whether leakage occurs with the increased abdominal pressure, and the volume at first leak. The instillation of radiopaque contrast medium instead of saline together with fluoroscopic equipment enables videourodynamics, or real-time visualization of the bladder neck and urethra during the voiding phase, to be performed.

Several other adjunctive tests can play key diagnostic roles when indicated. If the history and/or physical investigations suggest a neurologic etiology, the spinal cord should be imaged to identify anomalies such as syringocele, cord tethering, or malposition, among others. Ultrasound can be used in those younger than 6 months of age, owing to the fact that the vertebrae are not completely ossified. In those older than 6 months of age, MRI is indicated to detect vertebral and spinal cord pathology. A magnetic resonance urography can be performed when there is a suspicion of or to help define the anatomy of an ectopic ureter or other anatomic cause of incontinence such as common cloaca, urogenital sinus, or anorectal anomalies. A voiding cystourethrogram (VCUG) should be considered in incontinent children with a history of febrile UTI and in adolescent males with UI. In the former, unless an obstructed ectopic ureter is found to be entering into the external urethral sphincter in girls, the VCUG is not likely to diagnose the cause of the urinary incontinence (which could be from the infection and inflammation causing temporary irritation to the bladder), but is important to rule out vesicoureteral reflux and other sinister findings. However, a small proportion of males with posterior urethral valves will present in adolescence (owing to less severe disease or lack of prenatal care), in which case the VCUG is diagnostic. Finally, in difficult cases of incontinence, a cystoscopy can identify whether there is urethral (such as a diverticulum, urethral duplication, or large prostatic utricle all which would lead to post-void dribbling), bladder neck, or bladder pathology.

Questionnaires developed to assess incontinence

Standardized pediatric incontinence assessment tools nicely complement the physician's history and physical exam. These tools are designed to be filled out by the child or parent/caregiver as proxy and can be used at the initial intake as well as at subsequent visits to help track symptoms over the course of therapy. Some of these tools assess the symptoms and severity of lower urinary tract symptoms (LUTS) and urinary incontinence by incorporating urinary frequency, urgency, the degree of wetness, and coexistent constipation or bowel dysfunction into parent-proxy or child self-reported instruments [26–28]. The Pediatric Incontinence Questionnaire (PIN-Q) was designed to ascertain the effect of incontinence on a child's quality of life [29]. The Bristol Stool Scale can be provided to patients to determine the quality and character of their bowel movements

[30]. There is great overlap in urinary symptoms measured with these instruments, and no one questionnaire has been found to be superior to another, such that the clinician should decide which one (or two) instrument(s) captures the symptoms they desire while also minimizing patient burden. Also, it is important to determine if the selected questionnaire has been validated within a particular cultural, social, and language context. Simple verbatim translation of a questionnaire into another language is discouraged, as the meaning of particular questions can be skewed because the cultural context in which the survey was designed may differ from that of the intended environment.

Management

A thorough history and physical exam with the addition of further diagnostic tests, where indicated, will help determine if the primary etiology is functional, neurologic, or anatomic. The management is then tailored to each type of incontinence, with some overlap in medications and therapies between the former two types of incontinence.

Functional incontinence

a. Daytime incontinence

It is important to emphasize to parents and children that many patients with occasional daytime incontinence or enuresis will fall within the normal developmental spectrum, and that their condition is expected to improve over time. Nevertheless, owing in part to the resultant psychosocial distress and family burden, many patients and families will appropriately desire treatment.

As constipation is highly prevalent in this population [9], and because its treatment has been shown to improve continence [31], bowel management is a major priority in the management of children with daytime incontinence, enuresis, and dysfunctional voiding or dysfunctional elimination syndrome. Although the finding of stool on abdominal film is indicative of constipation, the correlation between the radiographic findings of stool and the degree of constipation is weak. Nevertheless, the radiographic finding of more than normal amounts of stool in the colon should provide strong enough evidence to convince parents who insist that their child has 'normal' bowel movements.

A behavioral approach to bowel management is the first step. After ensuring that the child is ingesting appropriate amounts of fiber and drinking enough fluid, this approach consists of two or more daily attempts at bowel movements. The child should sit on the toilet for at least 5 min, even when evacuation attempts are unsuccessful. If these conservative measures fail to show improvement, the addition of polyethylene glycol 3350 (MiraLax™, GlycoLax) is often successful. The recommended doses for constipation are 0.5–1.5 g/kg daily titrated to effect, with a maximal dose of 17 g/day. However, children can generally tolerate and may require much more than this. Thus, 34–51 g (two to three capfuls) once daily dissolved in 240 mL of water or juice with a sennosides oral laxative once a week can be used for several months. Down titration begins approximately six months later after consistently softer stools, more regular bowel movements, and improvements on abdominal radiograph are noted. Some patients will require a more aggressive bowel cleanout which utilizes a large dose (two of more liters) of polyethylene glycol 3350 with balanced electrolytes (GoLyately®) given over 2 or 3 days,

with or without digital or pharmacologic (bisacodyl, glycerin) rectal stimulation.

The management of the bladder can begin after a bowel management program, when indicated, has resulted in regular soft daily bowel movements. Bladder therapy proceeds in a stepwise fashion, and can be suited to parental/caregiver preferences. Behavioral therapy is a good starting point, the hallmark being the institution of a strict every 2–3 h daytime voiding regimen after educating the child about normal bladder function and sensation. Children are also encouraged to avoid caffeinated, carbonated, and highly acidic fluids. Voiding diaries clarify for both the family and provider the evacuation habits, and need to include information regarding the time and the amount of each void and/or leakage episode, time and consistency of each bowel movement, and amount of fluid intake. Aided by a normal bladder cycling regimen, the goal of behavioral therapy is to re-educate the child about normal bladder sensation and garner central control to suppress bladder urges.

Complementing behavioral therapy is biofeedback therapy, which provides visual and auditory feedback to children about the external urethral sphincter and pelvic floor bioactivity during bladder filling and emptying. Programs and games integrated with pelvic floor rehabilitation are then used to teach children how to respond to normal bladder urges during storage and promote pelvic floor relaxation during bladder and bowel elimination, thus targeting detrusor overactivity and dysfunctional voiding, respectively. Although there are no randomized controlled trials assessing the efficacy of biofeedback therapy, one observational study showed improvement in 89% and 90% of children with daytime incontinence and nocturnal enuresis [32]. Constipation was improved in 100% and resolved completely in 33%. Biofeedback therapy is undertaken over several sessions, with specialists in pediatric urology coaching the patients through the process. This option requires the commitment of families to attend several sessions and an attentive and motivated child.

While behavioral and biofeedback therapy are good non-invasive, non-pharmacologic methods to treat urinary incontinence, pharmacologic therapy plays an important role in pediatric voiding dysfunction, particularly among patients who do not respond to or cannot participate in more conservative approaches.

Oral anticholinergic therapy is used for the treatment of overactive bladder and urge incontinence (Table 3). Although several anticholinergics are used off-label in children with UI, only oxybutynin is approved by the United States Food and Drug Administration (FDA) for use in this population. Both M2 and M3 muscarinic receptors are found in the bladder, but the M3 subtype's stimulation results in direct detrusor contraction and micturition [33]. In blocking these receptors, anticholinergics mitigate the impact of uninhibited bladder contractions and increase bladder capacity, thereby decreasing incontinence episodes and increasing the time between and volume of each voiding episode. The side effects of anticholinergic drugs include dry mouth, blurred vision, facial flushing, headache, tiredness, gastrointestinal discomfort, and constipation. The degree to which these side effects are apparent depends on the specificity of the selected anticholinergic. Oxybutynin is known to decrease sweating, thus causing heat intolerance and overheating during the summer as well as preventing some athletes from tolerating this drug while engaged in sport. Although no short term memory deficits were found in children taking oral anticholinergics [34],

Table 3 Commonly used medications to treat pediatric urinary incontinence.

Medication	Dosage	Indication and notes
Anticholinergics		
Oxybutynin (Ditropan)	0.2 mg/kg bid–0.2 mg/kg qid	<ul style="list-style-type: none"> • Urge incontinence from detrusor overactivity • Transdermal route of administration allows for post-operative use when awaiting return of bowel function and avoids initial first-pass metabolism, potentially reducing side effects • Intravesical administration touted to reduce systemic side effects, but no Level I evidence supports this • Only anticholinergic FDA approved for use in children
Tolterodine (Detrol) ^a	0.01 mg/kg bid–0.04 mg/kg bid	
Hyoscyamine (Levsin) ^a	0.03 mg/kg bid–0.1 mg/kg tid	
Trospium (Sanctura) ^a	10–20 mg/day	
Solifenacin (Vesicare) ^a	5–10 mg/day	<ul style="list-style-type: none"> • Better M3 selectivity purportedly reduces side effects compared to oxybutynin, tolterodine, and trospium • Better M3 selectivity purportedly reduces side effects compared to oxybutynin, tolterodine, and trospium
Darifenacin (Enblex) ^a	7.5–15 mg/day	<ul style="list-style-type: none"> • Low abdominal leak point pressure or urethral pressure profile
Alpha-sympathomimetics		
Ephedrine ^a	0.5 mg/kg bid–1 mg/kg tid	
Pseudoephedrine ^a	0.4 mg/kg bid–0.9 mg/kg tid	
Alpha-adrenergic receptor antagonists		
Tamsulosin (Flomax) ^a	0.4 mg/day	<ul style="list-style-type: none"> • Primary bladder neck dysfunction
Doxazosin (Cardura) ^a	0.5 mg–2 mg/day	
Alfuzosin (Uroxatral) ^a	10 mg/day	
Terazosin (Hytrin) ^a	1–20 mg/day	
Antidiuretics		
Desmopressin	0.2–0.4 mg/night	<ul style="list-style-type: none"> • Enuresis • Controls but does not cure enuresis • Compared to imipramine: no difference in efficacy, more expensive, no anticholinergic side effects • Intranasal formulation not approved for enuresis
Tricyclic antidepressants		
Imipramine	25–50 mg/night	<ul style="list-style-type: none"> • Enuresis • Controls but does not cure enuresis • Compared to desmopressin: no difference in efficacy, cheaper, higher side effect profile
Neurolytics		
Onabotulinum toxin – Type A (Botox) ^a	10–12 IU/kg diluted in 30 ml normal saline, max 300 IU	<ul style="list-style-type: none"> • Neurogenic and non-neurogenic detrusor overactivity in patients not responding to conventional therapy • App. 30 trigone-sparing submucosal injections (1 ml each) under cystoscopic guidance • Repeat injections needed after 3–12 months to sustain effect

^a Not FDA approved for LUT use in children; optimal dose information lacking.

one study noted hyperactivity, insomnia, and agoraphobia in a neurogenic population being administered intravesical oxybutynin [35]. Patients with concomitant constipation should be thoroughly evacuated and maintained on an effective bowel regimen before initiating anticholinergic therapy.

There are few randomized controlled trials assessing the efficacy of oral anticholinergics in children with daytime incontinence. One study compared oxybutynin to biofeedback therapy and placebo, and found no difference in the number of incontinent episodes after nine months of follow-up [36]. Even with the paucity of Level I evidence demonstrating their efficacy, the apparent clinical effectiveness of oral anticholinergics support their use in this population.

Other oral pharmacotherapies can be selected based on specific etiologies of daytime incontinence. Alpha sympathomimetics such as ephedrine or pseudoephedrine can be used to target the bladder neck and external urethral sphincter in patients with low abdominal leak point pressures and intrinsic sphincter deficiency, though this

approach has not been rigorously studied. Alpha-adrenergic receptor antagonists, first described by Austin and colleagues in 1999 [37], can be selected for young males felt to have urinary incontinence that results for primary bladder neck dysfunction. These individuals have impaired relaxation of the bladder outlet which is evidenced by a delay of longer than 6 seconds between external sphincter relaxation and the initial urine flow as seen on uroflowmetry with EMG [23]. As these medications are used off-label for this indication, there is limited data regarding their optimal doses and side effect profiles in children. However, one recent study found no adverse blood pressure effects in children given tamsulosin in adult doses [38]. It is recommended to start patients at the lowest adult dose for a particular agent, with titration as needed. Several observational studies have shown improvements in PVR, uroflowmetry, and/or EMG lag time with alpha blocker treatment [23,38,39]. The one randomized controlled trial of doxazosin versus placebo found no difference in PVR or uroflow measurements, although the study design did not call for dose escalation and the study may have been too small to detect a difference in efficacy [40].

Intravesical onabotulinum toxin – A (BTX-A, Botox) has been used off-label in the treatment of overactive bladder in children with primarily neurogenic and but also non-neurogenic urinary incontinence. A potent but temporary neurotoxin, botulinum blocks the release of acetylcholine from presynaptic nerve terminals, preventing the stimulation of muscarinic receptors in the detrusor muscle and subsequent bladder contraction. With cystoscopic guidance under general anesthesia, injections of a 10 U/ml suspension are placed submucosally throughout the bladder, sparing the trigone. This medication is not FDA-approved for use in children, so dose and efficacy data are sparse. Typically, doses are 10–12 U/kg with a maximal dose of 300 U (equating to 30 injections of 10 U/ml) [41]. The duration of effect ranges from 3 to 12 months, with repeated injections needed to maintain the clinical effect [42–44]. There are no randomized controlled trials comparing this agent to placebo or other oral therapeutics. Hoebeke injected 100 U into 21 neurologically intact children resistant to standard treatment, and demonstrated a 60% complete and 20% partial improvement in urge and daytime incontinence after one injection among the 15 children with at least 6 months of follow-up [44]. Urinary retention lasting for 2 weeks and flank pain from vesicoureteric reflux occurred in 1 female and 1 male, respectively. The advantage of this therapeutic option is the direct action on the target organ, thus avoiding the systemic side effects of oral anticholinergic therapy such as heat intolerance and constipation. The disadvantages are the lack of long-term efficacy and requirement of general anesthesia.

Another non-pharmacologic approach to the treatment of overactive bladder is neuromodulation. Although the therapeutic mechanism is not entirely understood, the stimulation of afferent sacral nerve fibers in the S2–S4 region is believed to inhibit supraspinally mediated signals leading to detrusor overactivity [45]. In children with detrusor overactivity, stimulation of these fibers can occur via transcutaneous or, much less commonly, direct stimulation of the parasacral nerves [46–49]. For parasacral transcutaneous electrical nerve stimulation (TENS), patch electrodes placed in the sacral region are attached to a frequency generator with stimulation applied for 20–120 min. The optimal therapy is difficult to determine as the published studies show significant variation in frequencies applied (2–150 Hz), the duration (20–120 min) and occurrence (twice daily to once per week) of each therapeutic session, as well as the number of months (1–6) TENS was utilized. Nevertheless, 47–62% of subjects report resolution of their symptoms [47,50,51]. By stimulating a peripheral sensory nerve whose roots are located in the L4–S3 region, posterior tibial nerve stimulation (PTNS) is felt to centrally inhibit preganglionic bladder motor neurons in the sacral spinal cord [52]. PTNS uses a pulse generator to stimulate patch electrodes placed superior to the medial malleolus. One group found a 41% and 71% resolution rate in overactive bladder and dysfunctional voiding, which improved significantly with a second PTNS cycle [53]. Barroso compared parasacral TENS to PTNS, and found 70% and 9% complete resolution in the TENS and PTNS groups, respectively [54]. Importantly, the methodology varied significantly between the two groups (i.e. stimulation frequencies, session duration, and number of times per week for each session favored the TENS group), and the authors' conclusion that parasacral TENS is more effective may be overstated. As many of the studies for TENS and PTNS report only results while on therapy, the long-term cure rates for these modalities are not well described. One study suggested that as many as 50% of patients will require chronic transcutaneous therapy [53]. When a stimulator is implanted, cure rates for full response are 40% and partial response are 33% at 2 years of

follow-up [49]. Another study found significant improvements in health related quality of life scores after a median 6-month follow-up after the permanent stimulator was placed [46]. In summary, if families are motivated, willing and able to undergo chronic therapy, neuromodulation has acceptable rates of symptomatic improvement for children with nonneurogenic incontinence.

b. Nocturnal enuresis

Three conditions are felt to contribute to nocturnal enuresis – impaired sleep arousal threshold, nocturnal polyuria, and detrusor overactivity [3–5]. This knowledge helps the practitioner understand the different treatment modalities available for their enuretic patient. Importantly, experts recognize that a significant portion of monosymptomatic enuresis patients likely have underreported or underdiagnosed daytime symptoms, thus explaining the overlapping therapeutic efficacy in many patients. As the therapy for non-monosymptomatic enuresis incorporates strategies used for daytime incontinence and monosymptomatic enuresis, this section will focus primarily on monosymptomatic enuresis.

After excluding underlying medical conditions and undertaking an appropriate evaluation to exclude relevant comorbid conditions, the ICCS recommends a stepwise approach to treatment of monosymptomatic enuresis [55]. Prior to instituting any urotherapy, the practitioner should assess and treat constipation. The family should assess nocturnal urine production by weighing diapers and measuring voided volumes during normal feeding and drinking. When present, nocturnal polyuria, defined as urine volumes greater than 130% of expected bladder capacity for age [55], can help direct the practitioner towards desmopressin therapy.

When first presented with an enuretic patient, education and simple behavior maneuvers should be employed. Patients and families should be educated about normal bladder function and be instructed to void regularly throughout the day, immediately before bedtime, and on awakening. The majority of fluid intake should occur throughout the morning and afternoon with minimal drinking in the evening hours. Other simple measures such as reward systems for dry nights can be instituted. Compared to controls, children who underwent the above behavioral therapies were found to have fewer wet nights and lower relapse rates [56]. However, both the response and relapse rate are inferior compared to enuresis alarms and drug therapy.

Alarm therapy is the mainstay of treatment for enuresis in those who do not respond to education and simple behavior maneuvers. Placed under the bed linens or applied to the undergarments, the alarm senses wetness and arouses the patient with an audible or vibratory alarm. An adequate understanding of the technology and features of the device by motivated caregivers is required during the 3 to 6 month therapy period. A systematic review of 3,257 children from 56 randomized trials found that two-thirds of patients become dry while using the alarm, thus making it an excellent first line therapeutic option [57]. Of those who become dry on therapy, nearly half will relapse. Several options exist for those that relapse, including overlearning and repeating another course of treatment. Overlearning is the process whereby newly dry patients are given extra fluids prior to bedtime while still using the device, and has been shown to lower relapse rates. Some advocate that adding desmopressin to alarm therapy improves response in those that failed, although the data is conflicting [58,59].

Desmopressin (DDAVP) is a vasopressin analogue with an antidiuretic effect and represents another good first line therapy option. The ICCS suggest that the best candidates for this therapy are those with nocturnal urine production > 130% of expected bladder capacity for age (nocturnal polyuria) who are able to void at least 70% of their expected bladder capacity [55]. While actively taking the medication, 30 and 40% of children are estimated to be full and partial responders, respectively, with about 1.3 fewer wet nights per week expected [55,60]. Formerly indicated for primary nocturnal enuresis, intranasal DDAVP is no longer recommended given the risk of hyponatremia-related seizure events. Tablets are available in 0.2 and 0.4 mg doses and should be given at least 1 h before bedtime. There are no clear dose-related effects [60]. The medication is well tolerated with the only notable but significant risk being water intoxication causing hyponatremia and seizures in those who ingest excessive fluid prior to bed. Therefore, fluids should be restricted to 200 ml (6 oz) or less during the evening. As there was no difference in wet nights after cessation of therapy comparing treatment to placebo [60], desmopressin does not cure enuresis. However, given the favorable side effect profile and cost notwithstanding, the medication can be continued until short periods of abstinence show the child to be dry.

Of the tricyclic antidepressants (TCA), imipramine is historically the most commonly used in enuresis treatment. In addition to its peripheral anticholinergic effects, it acts centrally to increase vasopressin release and modify the sleep arousal pattern [61,62]. For enuresis, the doses (given at bedtime) are 25 mg for patients younger and 50 mg for patients older than 9 years. Side effects of imipramine include typical anticholinergic effects such as dry mouth and constipation, and mood changes and insomnia have been reported in children taking it for enuresis. At high doses, imipramine is cardiotoxic, so its use in those with long QT syndrome or a family history of sudden cardiac death should be avoided. Therapy should be interrupted every three months to avoid tachyphylaxis. One-fifth of children become dry while receiving TCA therapy for enuresis, though like desmopressin all patients relapse after cessation [63]. No evidence favors desmopressin compared to TCAs [60]. Although the side effect profile favors desmopressin, the lower cost of imipramine may make it more attractive to some families.

The ICCS also recommends a stepwise strategy to treat NMNE: address constipation, if present; diagnose and treat daytime LUTS; identify if comorbid behavioral disorders are present and refer for treatment; apply standard therapy for monosymptomatic nocturnal enuresis [64]. This strategy recognizes that with successful treatment of preceding steps, improvement or cure of nighttime symptoms is possible. The diagnostic and treatment strategies outlined for constipation, daytime incontinence, and monosymptomatic nocturnal enuresis should be employed for patients suffering from nonmonosymptomatic nocturnal enuresis.

Neurogenic incontinence

Patients with neurogenic incontinence will have leakage as a result of detrusor overactivity causing elevations in intravesical pressure above the urethral outlet resistance (a so-called upper motor neuron lesion) and/or from an incompetent external urethral sphincter (a so-called lower motor neuron lesion). Importantly, some children will be found to have both causes for incontinence. This determination is made through a full urodynamic study with cystometry. Other important parameters measured on UDS are the bladder capacity,

which may not provide an adequate reservoir for urine storage, and the intravesical storage pressures, which if high can lead to renal deterioration. The principles of incontinence treatment for neurogenic bladder are the same irrespective of the etiology of the lesion. That is, upper motor neuron lesions causing detrusor overactivity from traumatic spinal cord injury or myelodysplasia are treated in the same manner. As such, this section focuses on general strategies to treat incontinence. It does not discuss specific etiologies and recognizes that lesions evolve and new symptoms in a formerly stable patient should prompt repeat urodynamic studies. Finally, while the maintenance of low intravesical storage pressures is of utmost importance for renal preservation, it will not be discussed here.

Clean intermittent catheterization (CIC) enables efficient and complete bladder emptying in patients with neurogenic bladder who are unable to spontaneously void yet suffer from spontaneous uninhibited bladder contractions causing incontinence. The frequency of catheterization can be adjusted to maintain dryness in these patients. Following CIC, anticholinergics are the mainstay of pharmacologic treatment for neurogenic urinary incontinence caused by detrusor overactivity (Table 1). Establishing a good bowel regimen prior to instituting anticholinergic therapy is of utmost importance in this population that is particularly susceptible to constipation. The anticholinergic doses used to treat neurogenic incontinence may be higher than those needed in non-neurogenic incontinence. If patients do not respond to a particular medication, its dose can be increased or an alternative anticholinergic tried.

Onabotulinum toxin – type A (BTX-A, Botox) has been used in patients with neurogenic bladder who are either non-compliant with or resistant to anticholinergic therapy. Although there are no randomized controlled trials comparing BTX-A to placebo or other therapeutics, a review of six observational studies using BTX-A in mostly myelodysplastic patients found 65–87% of patients becoming completely dry after injections, with significant improvements in detrusor storage pressures and bladder compliance [41]. Marte's more recent study presented similar results, with 81% of children becoming completely dry between catheterizations [65]. Although no deaths or significant systemic adverse effects have been noted when using BTX-A for pediatric urology indications, patients can experience transient hematuria, UTIs, urinary retention, or persistent flank pain following injections [41,44,65]. BTX-A is a temporary neurolytic: patients who initially respond to therapy will experience a loss of therapeutic efficacy and require repeat injections after 3 to 12 months [42–44,65]. Though not FDA approved in the pediatric population, BTX-A has been shown to improve bladder storage characteristics and improve continence in patients with neurogenic etiologies.

Neurostimulation has been used with limited success in patients with neurogenic incontinence. Capitanucci reported improvements in LUTS in only 1/7 (14%) patients who completed the 12 week posterior tibial nerve stimulation cycle. No patients were cured, and many dropped out of the study due to lack of subjective improvement. Guys randomized children with NGB to receive either sacral neuromodulation or standard therapy with anticholinergic and bladder neck bulking agents [66]. Sacral neuromodulation was performed in the same fashion as in adults with continuous stimulation of the S3 nerve root via an implantable nerve stimulator. At 12 months compliance, bladder filling pressures, and post-void residuals were the same between the two groups, though the conventional therapy group had greater improvements in bladder capacity and the

neuromodulation group had greater improvements in detrusor leak point pressures. Xiao described an invasive neurosurgical procedure in which a lumbar ventral nerve is re-routed to the sacral nerves to provide a new skin to central nervous system to bladder pathway that facilitates bladder emptying [67]. Twelve of 14 (86%) of patients with areflexic NGB had clinically significant improvements in bladder capacity and maximal detrusor pressure at 12 months of follow-up. Improvements from hostile bladder dynamics to near normal urodynamic profiles were seen in 5 of 6 (83%) patients with preoperative detrusor hyperreflexia and/or detrusor-sphincter dyssynergia. The results of clinical trials investigating the Xiao Procedure in the United States are forthcoming, and this procedure should be deemed experimental until these and other studies confirm the findings of Xiao.

Although CIC and anticholinergic medications are the mainstay of treatment for bladder overactivity in the neurogenic population, some patients on maximal anticholinergic doses will continue to have high-pressure, small capacity, overactive bladders with resulting upper urinary tract changes and persistent incontinence. For such patients, bladder augmentation with colon, small intestine, or, infrequently, gastric segments can create a safe, low pressure urinary storage reservoir. Results from several single institution series with limited numbers of patients show significant improvements in bladder capacity, detrusor leak point pressure, and continence rates [68,69]. One study investigated the health related quality of life in myelodysplastic patients, and found no improvements in patients' quality of life after surgery compared to before surgery [70]. Finally, it is important to recognize that the short- and long-term complications of augmentation cystoplasty (ileus, bowel obstruction, bladder stone formation, vitamin deficiency, metabolic acidosis leading to bone demineralization and decreased linear bone growth, and bladder perforation, among others) are significant and can be life-threatening.

Some children with neurogenic incontinence will have an incompetent bladder outlet. In such patients, artificial urinary sphincters (AUS), fascial slings, or bladder neck reconstructions can be used to increase outlet resistance. Each option has its own unique attributes. An AUS can maintain spontaneous voiding in certain children and provide sufficient outlet resistance to improve incontinence. They infrequently become infected or suffer erosions and are durable but can require small revision surgeries owing to their mechanical nature [71]. Autologous rectus fascia or small intestinal submucosa bladder neck slings and wraps have been successfully used in patients with neurogenic sphincteric incontinence [72,73]. In patients with bladder augmentations, slings provide the advantage of preserving the urethra as a pop-off mechanism and negate the complexity and low but present infectious risk of the AUS. However, children with fascial slings may not be able to spontaneously void. Several variations of the bladder neck reconstruction exist, and common to all are urethral elongation and reliance on either increased muscular backing or the Mitrofanoff principle to improve continence. These operations are seldom used in this population for several reasons: their creation uses precious bladder capacity, catheterization can be difficult, and they often do not provide adequate increases in outlet resistance sufficient for continence.

Anatomic incontinence

Treating anatomically related incontinence with surgery can be one of the most satisfying and challenging operations for a pediatric

urologist. Although a thorough description of technique is outside the scope of this article, we briefly discuss the surgical strategies used in some of these conditions.

When an ectopic ureter is suspected in a female with continuous incontinence and verified by radiologic workup, surgical therapy can provide a cure. If a renal scan confirms a poorly functioning upper pole segment drained by the ectopic ureter, a partial nephrectomy with excision of the ureter as far distally as possible will provide immediate dryness. Alternatively, if the functioning segment contributes significantly to renal function, it should be preserved. The presence or absence of lower pole vesicoureteral reflux will determine the surgical approach. If reflux is present, a common sheath ureteral reimplantation is a viable option. If reflux is absent, an upper to lower pole ureteroureterostomy or pyeloureterostomy redirects upper pole urine drainage to the non-refluxing lower pole ureter, thus curing continuous incontinence.

Children with bladder exstrophy or epispadias will require surgery to reconstruct the urethra and bladder neck. Several different techniques are described, though all have the common goals of preserving upper tract function, providing cosmetically pleasing and functional external genitalia, and achieving urinary continence. As the initial bladder closure often fails to provide adequate outlet resistance, a bladder neck reconstruction is used to create outlet resistance sufficient for continence but still allow spontaneous voiding.

There are other rare etiologies of anatomic urinary incontinence such as cloacal anomalies and urethral duplications which require individualized surgical approaches. A good preoperative workup including diagnostic imaging and cystoscopy will help define the congenital anomaly and aid the surgeon in reconstructing normal anatomy.

Conclusion

Pediatric urinary incontinence is a common condition. A thorough history and systematic physical exam will direct the provider towards adjunctive tests, if required. Identification of the parents' motivation and goals for their child's treatment will help the clinician form a management plan and set reasonable expectations. The identification and treatment of constipation is of utmost importance prior to beginning incontinence therapy. If classified as having daytime incontinence, a stepwise approach beginning with behavioral and biofeedback therapy prior to instituting anticholinergic medication should be followed. An evidence-based approach to enuresis suggests that alarm therapy in a household with motivated caregivers will provide durable cures in a majority of children. Although the relapse rates are high for enuretic children on pharmacotherapy, desmopressin and amitriptyline decrease wet nights when the child takes them routinely, and provide a good option for special events like vacations or sleep-overs. Identification of bladder overactivity and/or sphincteric deficiency helps direct the management plan for neurogenic incontinence. The former is often successfully treated with CIC and anticholinergic therapy; augmentation cystoplasty is reserved for patients with high bladder storage pressures and relatively small bladder capacities who continue to leak despite maximal dose anticholinergics. Surgery to increase bladder outlet resistance is used to treat the latter patients with sphincteric deficiency. Surgery is also the mainstay of treatment for those children with a well-defined anatomic etiology for their incontinence.

Conflict of interest

Authors have no conflict of interest to declare.

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