

CME FEATURE

Part I: Assessment and Diagnostic Strategies for Neurogenic Bladder

Diagnostics and treatment options for NGB are continually advancing, and clinicians need to remain up-to-date to accurately assess and optimally manage patients

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STATEMENT OF NEED: Urologists and other healthcare professionals caring for patients with bladder dysfunction should be knowledgeable about the symptoms of neurogenic bladder (NGB) and integrate assessment and diagnostic approaches that may facilitate identification of patients for earlier treatment. Providers should also be knowledgeable about the various treatment options for NGB, including options for patients who fail behavioral or oral therapy.

TARGET AUDIENCE: This activity has been designed to meet the educational needs of urologists and other clinicians involved in the treatment of patients with bladder dysfunction.

EDUCATIONAL OBJECTIVES: After completing the activity, the participant should be better able to:

- Identify patients for individualized treatment earlier using diagnostic strategies.
- Describe management options for neurogenic bladder, including evidence-based treatments and novel therapeutic approaches.

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BY DAVID A. GINSBERG, MD

NGB can lead to a higher risk for renal complications.

Pathophysiology and etiology

The two main functions of the bladder are storage and voiding, and a normal cycle depends on coordination between the bladder and the urethral sphincter.⁶ Normal micturition involves autonomic and somatic pathways from the lumbosacral spine to the urethral outlet, bladder neck, and detrusor muscle—and a change in any of these can produce symptoms of NGB.⁷ Furthermore, an alteration in bladder wall innervation, or of the pelvic floor and urethra, can trigger overactivity.

Neurologic disorders often damage and disrupt the central or peripheral pathways involved in the central control of the lower urinary tract.⁸ Damage to areas of the nervous system may result in detrusor overactivity, with or without detrusor-sphincter dyssynergia, detrusor underactivity, detrusor areflexia, and impaired contractility.

The causes of neurologic conditions leading to NGB include supraspinal, spinal, peripheral, or mixed.⁸

Supraspinal disorders involve central nervous system lesions that occur above the pontine micturition center. They

Neurogenic bladder (NGB) affects many adults in the United States and is commonly caused by multiple sclerosis (MS), spinal cord injury (SCI), cerebral vascular accident, and Parkinson's disease. Many of these patients have bothersome urinary symptoms, including urinary incontinence (UI), urinary frequency, and urgency. The primary urodynamic finding for most patients with NGB is an uninhibited bladder contraction, or neurogenic detrusor overactivity (NDO). Bladder disorders have been reported in 40% to 90% of patients with MS and in 37% to 72% of patients with Parkinson's disease, with a higher rate of UI in patients with cerebral vascular accident and Parkinson's disease than in the general population.¹

In addition to their physical burden, urinary symptoms such as incontinence can significantly impair quality of life.² The occurrence of urinary tract infections (UTIs) in the NDO population is common.³ UI also increases the risk for skin irritation and breakdown as well as depression.^{4,5} In some cases,



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include conditions such as Parkinson's disease, Shy-Drager Syndrome, cerebral palsy, frontal lobe lesions, stroke, cerebral trauma,⁸ and MS. Bladder dysfunction at this level usually presents as neurogenic detrusor overactivity combined with synergistic activity between the bladder and sphincter (i.e., when the bladder contracts, the sphincter appropriately relaxes).

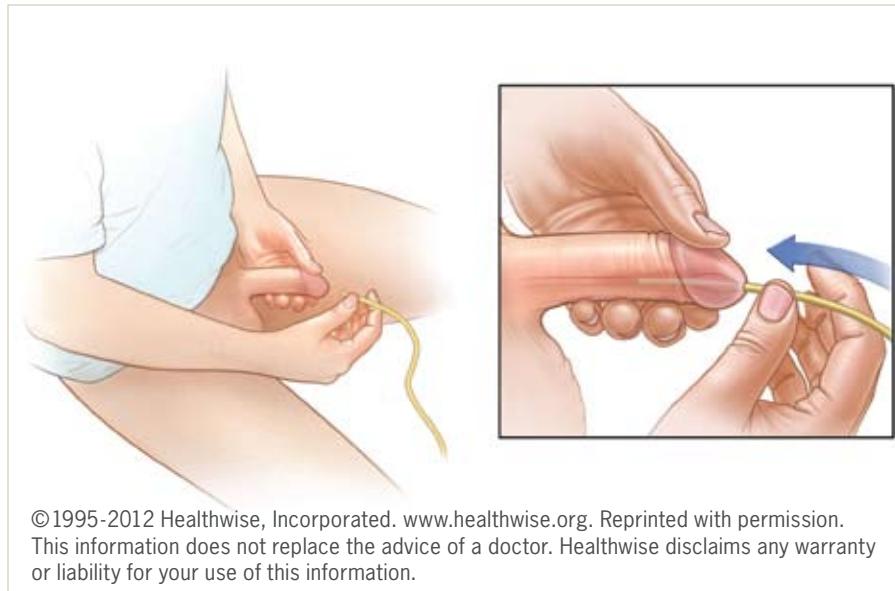
Suprasacral spinal neurologic conditions include SCI, spinal stenosis, spinal cord infarction, MS, transverse myelitis, cervical spondylosis, and intervertebral disk disease.⁸ Patients with complete cord transections above the level of the sacrum generally present with NDO and striated sphincter dyssynergia (i.e., when the bladder contracts, the external sphincter inappropriately contracts at the same time).⁶ Detrusor external sphincter dyssynergia (DESD) is associated with a high risk for other urologic complications such as hydronephrosis, vesicourethral reflux, urosepsis, and urolithiasis.⁸ In addition, patients with lesions at T6 and higher are also at risk for symptoms of autonomic dysreflexia.

Lesions in the sacral spinal cord, including those in patients with spina bifida, diabetes mellitus, herpes zoster, and herniated lumbar disk disease, often lead to an acontractile detrusor.⁸ In addition, poor bladder compliance can be seen, which places the patients at risk for upper urinary tract damage if bladder storage pressures are too high. These patients are at risk for poor sphincteric activity as well. Among patients with peripheral nerve lesions caused by diabetes mellitus, herpes zoster, and herniated lumbar disk disease, bladder dysfunction generally manifests as detrusor areflexia.⁶

It is important to realize that there are a great many exceptions to these general patient categorizations. Neurologic lesions can often be incomplete or span multiple levels; thus, each patient benefits from a complete and thorough neurourologic evaluation.

Diagnosis and assessment of NGB

Appropriate management relies on an accurate diagnosis. In some patients with NGB, the original cause of their symptoms may be misdiagnosed, resulting in failure of initial therapy.⁹ Assessment includes a comprehensive medical and voiding history, a physical examination, laboratory tests, computed tomography urogram or renal



Intermittent catheterization is the preferred method of catheterization for patients with NGB.

ultrasound (as indicated), endoscopic examination (as indicated), and urodynamic studies (Table 1).⁸

Presenting symptoms can be roughly divided into failure to empty or failure to store. Typical symptoms of disrupted bladder emptying include feelings of fullness and difficulty emptying the bladder, such as a slow stream while straining to void. Other patients report hesitancy, an interrupted or diminished stream, a sensation of incomplete emptying, lower abdominal discomfort, nocturia, and recurrent UTIs.⁷ Failure to store usually presents as frequency, urgency, and UI. Depending on the level and severity of the neurologic

lesion, patients may not have any bladder sensation; symptoms therefore may not be a reliable indicator of significant NGB dysfunction in certain patients.

Issues related to NGB include childhood urologic history, prior surgical procedures, and other medical conditions. Current medication use should be evaluated, as certain drugs can impact bladder function. In addition, issues related to bowel and sexual function are commonly seen in patients with NGB and may have to be addressed.

Loss of genitourinary and gastrointestinal function are important sequelae of SCI.¹⁰ Constipation can affect voiding function, leading to incontinence or

retention. When taking a history, it is important to realize that some patients may have coexisting problems such as stress UI or benign prostatic hyperplasia, in addition to lower urinary tract dysfunction from NGB.

Physical examination should include a focused neurourologic evaluation. In addition to standard evaluation of the abdomen, back, rectum, pelvis, and genitalia, clinicians need to evaluate the sacral dermatomes, including perianal sensation and anal sphincter tone. The neurologic evaluation should be primarily focused at the sacral 2-4 level and assess the bulbocavernosus reflex, voluntary external sphincter control, and reflexes in the sacral dermatome levels. Deep tendon reflexes in the lower extremities, clonus, and plantar responses should be included.^{7,8}

A urinalysis, with or without a urine culture,⁸ should be done to rule out UTI, glucosuria indicative of diabetes, and blood or protein in the urine that might indicate renal disease. If the results suggest an associated condition, the patient may need further diagnostic testing.

Urodynamic assessment can help determine the underlying neurologic issue, categorize the vesicourethral dysfunction, and provide a basis for appropriate therapy. In fact, clinical evaluation is often unable to identify the type of bladder dysfunction; therefore, optimal therapy is dependent on an appropriate urodynamic evaluation, as follows:^{6,11}

- **Uroflow rate**—the volume of urine voided per unit of time. It is primarily used to screen for bladder outlet obstruction.
- **Postvoid residual volume**—considered part of the basic evaluation for UI. A high rate suggests acontractile bladder or bladder outlet obstruction.
- **Cystometrogram** (with or without electromyogram)—a filling cystometrogram evaluates bladder capacity, compliance, and the presence of detrusor overactivity, while a voiding test records voiding detrusor pressure along with the rate of urinary flow.
- **Electromyography**—can help determine the presence of coordinated or uncoordinated voiding, and allows for an accurate diagnosis of detrusor sphincter dyssynergia that is common in suprasacral SCIs.
- **Videourodynamic study** (multichannel with fluoroscopy)—combines

Table 1. Initial Evaluation

History and physical examination <ul style="list-style-type: none"> • Urologic • Neurologic: S2-4; bulbocavernosus reflex • Bowel function • Autonomic dysreflexia • Erectile dysfunction
Neurologic examination
Urinalysis with or without urine culture
Renal function study (serum creatinine)
Computed tomography urogram or renal ultrasound (as indicated)
Endoscopic examination (as indicated)
Urodynamic testing <ul style="list-style-type: none"> • Uroflow • Postvoid residual volume • Cystometrogram with or without electromyogram • Pressure-flow (micturition) study • Videourodynamic study (multichannel with fluoroscopy)

Adapted from: Kennelly MJ, DeVoe WB. *Rev Urol* 2008;10(3):182-191.

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Case Study: Managing Neurogenic Bladder

Mr. J. is a 25-year-old male graduate student. Two years ago, he suffered a complete SCI at T4 as a result of a motor vehicle accident. Although confined to a wheelchair, he has recovered sufficiently to return to graduate school to complete his degree and obtain a job as an educator.

He has good health overall, with strong upper body function, and lives independently. He has continuing UI, managed with anticholinergic medication and CIC, which his schedule, hand function, and mobility permit.

Mr. J has tried 3 anticholinergic drugs and formulations without success. He has a moderate degree of dry mouth and a worsening of baseline constipation. He also has occasional symptoms of autonomic dysreflexia and 2 to 3 UTIs per year, treated easily with no complications. He fears a higher dose will lead to worse adverse effects and asks about alternative options.

His clinician evaluates Mr. J. Urodynamics reveal detrusor overactivity and DESD with a detrusor pressure of 60 cm H₂O at 165 mL. This and the fact that he continues to leak urine between catheterizations suggest that he has failed therapy with CIC and anticholinergics. Since Mr. J prefers to continue CIC, alternatives such as an indwelling catheter or reflex voiding to a condom catheter are not options. To continue with CIC and maintain low intravesical storage pressures urodynamically and continence clinically, his only choices are intradetrusor injection of botulinum toxin A (onaBoNTA) or surgery.

OnaBoNTA, FDA-approved for neurogenic detrusor overactivity for patients with NGB who failed or cannot tolerate therapy with anticholinergics, reduces incontinence episodes and can significantly improve urodynamic parameters. Mr. J decides to pursue this option instead of a more invasive procedure such as bladder augmentation.

the radiographic findings of voiding cystourethrogram with multichannel urodynamics.

Evaluating current treatment options: an overview

Treatment includes nonpharmacologic and pharmacologic interventions, bladder injection, surgery, and urinary diversion using catheters (**Table 2**).

The primary goals of treatment include preserving the upper urinary tract, maintaining adequate bladder capacity, promoting low-pressure micturition, and avoiding bladder overdistention.⁸ Another key objective is to prevent UTIs and minimize the use of indwelling catheters. The patient as a whole needs to be considered, and

therapy should minimize risk while maximizing social, emotional, and vocational acceptability. The ability/willingness of the patient to perform clean intermittent catheterization (CIC) also must be considered.

In patients with elevated bladder storage pressures or vesicoureteral reflux at higher risk for renal complications, the primary goal is to protect kidney function and prevent further complications. Conversely, in patients not at high risk for kidney damage, quality-of-life issues such as UI may be of greatest concern.

Clinicians need to consider the patient's degree of disability, mobility, hand function, cognitive status, general condition, likelihood of progression of the neurologic disease, goals and concerns, and available resources.¹²

Behavioral/nonpharmacologic interventions

Behavioral/nonpharmacologic interventions include lifestyle interventions, the use of external appliances such as pads and/or portable urinals, and the use of CIC, or condom or indwelling catheters for patients with incomplete bladder emptying.¹³⁻¹⁵ These conservative approaches are often used in combination with medications.¹³

Lifestyle intervention includes timed voiding, fluid alterations, pelvic floor muscle exercises, biofeedback, toiletting assistance, and bladder education/retraining.¹⁶ These interventions, primarily applicable to patients with idiopathic overactive bladder (OAB), may not be helpful for all patients with NGB.

Urinary diversion via catheterization is a mainstay of anti-incontinence therapy.

Intermittent catheterization

Intermittent catheterization is the preferred method for patients with NGB who cannot adequately void volitionally. CIC decreases the risk of infection¹⁷ and lowers the risk for long-term complications such as hydronephrosis, bladder and renal calculi, and autonomic dysreflexia.¹⁸ However, for patients with poor hand function or who lack a caregiver, CIC is not an option. It also may be less than optimal in patients with bladder capacity <200 mL, abnormal urethral anatomy such as bladder neck obstruction and strictures, poor cognition, or who cannot adhere to

a catheterization schedule.¹⁸ Using a hydrophilic-coated catheter reduces and delays the onset of symptomatic UTIs and can also decrease the risk of complications related to UTIs.¹⁹

CIC can be more challenging for obese patients and those with higher-level injuries, and for the female confined to a wheelchair because of difficulty accessing the urethra. Women with NGB may more often use a chronic indwelling catheter (or wear a diaper).^{20,21} However, for most patients with adequate upper-extremity function and hand function, CIC works well over the long term without complications.²²

Indwelling catheterization

Indwelling catheters, often considered a last resort, may still be the best option for certain patients. Remaining on anticholinergics may be preferable for bladder health with an indwelling catheter. It may be used in patients with acute central nervous system trauma who require precise monitoring of urinary output, those with no available alternatives, and those unable, or lacking a caregiver, to perform CIC or reflex voiding.^{6,18}

Long-term indwelling catheters cause UTIs and may lead to reduced compliance and a thickened bladder. Over time, patients may develop vesicoureteral reflux that leads to bladder stones, a higher risk for kidney stones, and renal damage. A suprapubic catheter is less traumatic to the urethra and more hygienic.¹⁸

Table 2. Treatment for Neurogenic Bladder

Failure to Empty

- Behavioral modification, i.e., pelvic floor exercises
- Intermittent catheterization
- Botulinum toxin A injection into the sphincter
- Suprapubic catheter
- Indwelling catheter
- Sphincterotomy
- Urethral stent
- Urinary diversion

Failure to Store

- Lifestyle modifications, i.e., diet and fluid intake, timed voiding
- Antimuscarinics
- Botulinum toxin A injections into the detrusor
- Indwelling catheter
- Reconstruction

Pharmacologic treatment options

Anticholinergic/antimuscarinic agents, the standard therapy for patients with neurogenic detrusor overactivity,¹⁵ are often used in conjunction with CIC to allow for optimal storage and emptying. However, most are not registered for this indication, and data are limited. Medications with proven efficacy for NGB include oxybutynin, trospium, tolterodine, and propiverine.²³

Adverse effects, such as dry mouth, often limit patient adherence. Constipation may be an issue, as many of these patients also have concomitant neurogenic bowel and may already be using laxatives, suppositories, and manual defecation.²⁴⁻²⁶

Combination therapy with two or three drugs may lead to significant urodynamic and clinical improvements for patients with NGB.²⁷ A meta-analysis showed that longer-acting formulations were more effective and had a lower rate of adverse effects.²⁸

Imipramine

Tricyclic antidepressants suppress OAB via various mechanisms. Imipramine has improved incontinence in adults with NGB.²⁹

Alpha-blockers/muscle relaxants

Alpha-blockers can treat DESD, lower bladder pressure during voiding, and reduce outlet resistance at the external sphincter.¹⁸ Muscle relaxants such as baclofen, benzodiazepines, and dantrolene have been used to treat outlet obstruction at the level of the striated sphincter due to DESD. Efficacy is not optimal in alleviating skeletal muscle spasticity, however, and adverse effects minimize their overall usefulness.³⁰

Botulinum toxin injection: an emerging therapy

Botulinum toxin A (BoNTA) is an alternative for patients who fail or cannot tolerate the adverse effects of anticholinergic therapy. Detrusor injections of BoNTA are considered second-line treatment for neurogenic detrusor overactivity, and the US Food and Drug Administration (FDA) approved onabotulinumtoxinA (ona-BoNTA; Botox®) for this indication in 2011. Two other BoNTA preparations are available, abobotulinumtoxin A (Dysport®) and incobotulinumtoxin A (Xeomin®), and a botulinum toxin B

(rimabotulinumtoxinB; Myobloc™). None have FDA approval for the treatment of neurogenic detrusor overactivity. These preparations cannot be used interchangeably.

BoNTA is usually administered in the office setting, with a local anesthetic. Patients should be closely monitored for autonomic dysreflexia.

The use of BoNTA to treat NDO was first published in 2000 by Schurch et al with 300 U of onaBoNTA injected into the detrusor muscle.³¹ The toxin inhibits acetylcholine release at the neuromuscular junction and prevents peripheral neurotransmitter release at presynaptic cholinergic nerve terminals. The neuromuscular contraction is then blocked and relaxes muscles that may be overactive or spastic.^{18,32}

Many nonrandomized, single-dose studies have validated the use of 300 U of onaBoNTA in this patient population. Further investigation by Cruz et al was done in a multicenter, randomized controlled trial, including 275 patients with NGB.³³ Patients with NGB secondary to MS or SCI were randomized to placebo and two doses of onaBoNTA (200 and 300 U). Significant clinical and urodynamic improvements were seen in both onaBoNTA arms vs placebo, and the 300 U dose was not superior to 200 U. Baseline UI decreased by 21.8 episodes per week in the 200 U arm from a baseline of 33.5, and median time to request retreatment was 42.1 weeks. This study was one of two trials whose findings ultimately led to the FDA approving this drug for the indication of NDO at the dose of 200 U.

BoNTA appears to last 6 to 9 months in most patients. Reinjections do not appear to diminish efficacy³⁴ or decrease bladder compliance and can improve quality of life.³⁵ The most common adverse events are urinary retention and UTI.³³ The risk for UTI may be decreased with BoNTA injection.³⁵

More invasive procedures

An intraurethral stent may be an option for the long-term management of detrusor-sphincter dyssynergia in patients with SCIs.³⁶ Potentially reversible, it can circumvent urethral obstruction while avoiding surgery.¹⁸

A transurethral resection of the external urinary sphincter converts the bladder into an open draining conduit.¹⁸

Considered irreversible, it reduces the intravesical voiding pressure mediated by bladder contractions against a contracted external urethral sphincter from DESD.

Bladder augmentation uses an intestinal segment (usually ileum) to enlarge the bladder and increase bladder capacity. The aim is to restore continence and preserve upper urinary tracts by lowering intravesical storage pressures and minimizing the risk of reflux and hydronephrosis.¹⁸

Ileovesicostomy uses a section of ileum to create a channel leading from the urinary bladder upward to the abdominal wall. It provides for a low-pressure conduit for urinary drainage and decreases detrusor leak point pressures.³⁷

Conclusion

NGB is common in patients with neurologic disorders and requires thorough assessment to select appropriate management. Diagnostics and treatment options are continually advancing, and clinicians need to remain up-to-date to accurately assess and optimally manage patients with this condition. ■

Part II of our series, coming in the July issue, will focus on evaluating treatment options for neurogenic bladder dysfunction.

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CME Post-test

Expiration Date: May 2012

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- The primary urodynamic finding for most patients with neurogenic bladder (NGB) is:
 - Abnormal micturition
 - Neurogenic detrusor overactivity
 - Vesicoureteral reflux
 - Autonomic dysreflexia
- Supraspinal disorders leading to NGB include conditions such as:
 - Parkinson's disease and Shy-Drager Syndrome
 - Cerebral palsy and frontal lobe lesions
 - Stroke, cerebral trauma, and multiple sclerosis
 - All of the above
- Appropriate urodynamic evaluation:
 - Can help determine the underlying neurologic issue, categorize the vesicourethral dysfunction, and provide a basis for therapy
 - Always includes uroflow rate, postvoid residual volume, and electromyogram
 - Should be done to rule out urinary tract infection, glucosuria indicative of diabetes, and blood or protein in the urine
 - Is not needed to determine optimal therapy
- In patients with elevated bladder storage pressures or vesicoureteral reflux, the primary goal of treatment is:
 - To improve quality of life
 - To preserve the upper urinary tract, maintaining adequate bladder capacity
 - To protect kidney function and prevent further complications
 - To prevent urinary tract infections and minimize the use of indwelling catheters
- The preferred method for patients with NGB who cannot adequately void voluntarily is:
 - Indwelling catheterization
 - Intermittent catheterization
 - Condom catheterization
 - Suprapubic catheterization
- Which of the following statements is true?
 - Anticholinergic/antimuscarinic agents are often used in conjunction with CIC to allow for optimal storage and emptying
 - Alpha-blockers diminish total bladder capacity
 - Muscle relaxants are the standard therapy for patients with neurogenic detrusor overactivity
 - Imipramine has improved urinary urgency in adults with NGB
- Second-line treatment for neurogenic detrusor overactivity that is an alternative for patients who fail or cannot tolerate the adverse effects of anticholinergic therapy is:
 - Bladder augmentation
 - Reflex voiding to a condom catheter
 - Intradetrusor injection of botulinum toxin A
 - Ileovesicostomy
- A potentially reversible option for the long-term management of detrusor-sphincter dyssynergia in patients with SCIs that circumvents urethral obstruction while avoiding surgery is:
 - A transurethral resection of the external urinary sphincter
 - An indwelling catheter
 - Endoscopic sphincterotomy
 - An intraurethral stent

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