Optimizing Therapy and Management of Neurogenic Bladder

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Abstract

Clinicians managing patients with neurogenic bladder (NGB) and neurogenic detrusor overactivity (NDO) are faced with a myriad of complex choices when deciding on appropriate medical and/or surgical interventions to relieve bothersome symptoms associated with NGB and NDO, especially urinary incontinence. Therapies must provide maximum benefits while minimizing patients' risk for adverse events. A thorough knowledge and understanding of available and emerging medical and surgical treatment options for NGB/NDO is vital to assist clinicians in choosing appropriate treatment pathways and optimize response to therapy and individual outcomes.

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For author information and disclosures, see end of text.

Neurogenic Bladder: Goals of Therapy

Neurogenic bladder (NGB) with lower urinary tract dysfunction is found in patients with underlying neurologic diseases, including multiple sclerosis (MS), Parkinson's disease, traumatic spinal cord injury (SCI), and spina bifida, among others.[1,2] Once a diagnosis of NGB has been made and neurourologic evaluation has been performed, clinicians are faced with the challenge of finding appropriate therapies and treatment pathways to relieve urinary incontinence (UI) and other symptoms associated with NGB while avoiding adverse events and treatment complications to enhance patient quality of life (QoL).[1] Individualized treatment of NGB will depend on the type of neurologic disease underlying the lower urinary tract dysfunction and its general evolution (prognosis, degree of disability, progression), patient symptoms, urodynamic findings, and also on the general health and condition of the patients and available resources.[1,2] There are several critical goals for management of the patient with NGB dysfunction, especially for patients with poor bladder compliance and/or neurogenic detrusor overactivity (NDO) and detrusor sphincter dyssynergia, which can result in elevated intravesical storage pressures and subsequent upper urinary tract damage. Clinicians should strive to use treatments that (1) achieve and maintain urinary continence to avoid the physical and psychological damage associated with incontinence, (2) preserve renal function by maintaining appropriately low bladder storage pressures, (3) minimize risk of other conditions associated with NGB such as urinary tract infections (UTIs) and bladder stones, and (4) optimize the patient's QoL. Management of NGB may include such interventions as timed voiding, reflex voiding into a condom catheter or diaper, intermittent catheterization, indwelling urinary catheterization, oral and/or intravesical medications, and surgical reconstruction of the lower urinary tract. Patient education is a key component of any management plan for NGB.[1] Clinicians must be aware of conservative and lifestyle-based treatments, along with standard, new, and emerging medications and other technologies to optimize treatment of NGB dysfunction and improve health-related QOL (HRQoL) in patients with NGB and underlying neurologic disease.

Conservative and Lifestyle-Based Therapies

Bladder Retraining/Fluid Schedule/Catheterization

Regular and adequate emptying of the bladder is a critical com-

ponent of optimal healthcare for patients with NGB. Regular bladder emptying (through self-catheterization or other means) reduces intravesical bladder pressure and allows for appropriate cycling of bladder contents to lower patient risk for UTIs.[1,3] Behavioral training is a frequent component of urologic management in patients with neurologic disorders. Behavioral measures are most valuable in patients who have some degree of bladder control and intact bladder sensation. This has been found to be helpful for patients with neurologic lesions involving the brain such as cerebrovascular disease, Parkinson's disease, multiple system atrophy, dementia, and cerebral palsy, as well as for patients with MS, incomplete SCI, transverse myelitis, and diabetes mellitus.[2,4]

Several different bladder management techniques may be utilized to assist in optimizing bladder function in patients with neurologic disorders and NGB/NDO. Timed voiding consists of fixed intervals between episodes of urination/toileting. It is initiated by the patient and/or their caregiver and can depend on both bladder function and the working schedules of the caregivers if they are involved. The primary goal of timed voiding is for the patient with urge incontinence to void before urinary urgency and incontinence occur.[2,4] Depending on the individual patient and the state of his or her bladder and neurologic disorder, bladder retraining may be utilized to avoid incontinence and involuntary bladder contractions by decreasing voiding intervals. The maintenance of a voiding diary by the patient/caregiver can be helpful in both the evaluation of voiding dysfunction and following therapy. Bladder expression is a bladder emptying technique sometimes used for patients with a combination of an areflexic detrusor and a sphincter that is also areflexic or anatomically incompetent, such as following sphincterotomy. The most common techniques used are Valsalva (abdominal straining) and Crédé maneuver (manual compression of the lower abdomen). Although bladder expression techniques can be effective in bladder emptying, they can be complicated by potential urine reflux into the upper urinary tract, genital-rectal prolapse, hemorrhoids, and incomplete emptying. Urine reflux into the prostate and seminal vesicles may cause epididymo-orchitis in male patients.[2] None of these techniques are strongly recommended because of the risk of long-term urinary complications and the inability to empty the bladder adequately.[5]

Initiation of a fluid intake schedule is another important step in managing a patient with NGB requiring intermittent catheterization and may also benefit patients with uninhibited detrusor activity. Fluid schedules permit a predictable degree of bladder filling without the risk of overdistention. Repetitive overdistention of the detrusor can result in permanent muscle damage and lead to a flaccid bladder with overly large capacity known as myogenic bladder. An example of a recommended schedule would be ingestion of 400 cc of fluid with meals, and an additional 200 cc in the morning and afternoon (eg, 10:00 AM, 2:00 PM, and 4:00 PM) with only sips of fluid in the evening to reduce nighttime incontinence. Accounting for fluid loss through respiration and sweating, urine formation would be approximately 1600 cc daily. With intermittent catheterization (IC) performed every 6 hours, the urine volume eliminated with each catheterization would be about 400 cc. IC would be timed to prevent bladder distension and keep bladder volumes at an optimal level of 400 cc to 500 cc. Patients with indwelling catheters will likely benefit from increased fluid intake to help minimize the risks of bacterial colonization, calcium and phosphate crystal formation, and other potential complications related to the use of indwelling catheters. With all the techniques available for bladder management and their complexities, patient education is a crucial component of bladder management. These plans must be individualized for each patient, and both teaching and monitoring of appropriate techniques will assist in achieving optimal bladder management and reduce risks of infection and other adverse events that may negatively affect HRQoL for these patients.[1]

Catheterization

The purpose of catheterization is to empty the bladder of urine.[2] Clean, intermittent catheterization (IC) is the preferred bladder management technique for patients with NGB dysfunction who experience complete or partial urinary retention.[1,3] The purpose of IC is to resume normal bladder storage and regularly complete bladder emptying and urine evacuation.[2] Self-administered IC enhances patient self-care and independence and reduces barriers to activity and sexual functioning compared with use of an indwelling catheter.[1,3] Properly administered IC can make a patient with NGB continent if bladder capacity is adequate; bladder pressure remains low; and a balance is achieved between fluid intake, residual urine volume, and catheterization frequency.[2] The maintenance of appropriately low bladder storage pressures and minimization of detrusor overactivity are often achieved with the simultaneous use of medications such as antimuscarinics, which are discussed later.[6] If the patient has some ability to void voluntarily, voiding can be attempted every 2 to 4 hours or when the urge to void occurs. IC can be used as part of a bladder retraining program to gradually reduce residual urine volumes.[7] Complications may occur with IC, the most frequent complication being UTI.[1] One review of the risks of IC indicated an 11% prevalence of asympto-

matic UTI and a 53% prevalence of symptomatic bacteriuria reported in various studies.[1,8] Catheterization frequency and the avoidance of bladder overfilling are key measures to prevent infection, and asymptomatic bacteriuria (presence of bacterial infection in the urine without patients experiencing symptoms) is often not treated with antibiotics.[2,8] Antibiotic prophylaxis for use in IC overall is controversial; however, it may be indicated if a patient on regular IC continues to get recurrent UTI without another demonstrable cause.[1,8] The best UTI prevention techniques are appropriate education of patients and caregivers involved in IC, good patient compliance, proper materials, and application of optimal catheterization techniques.[8] Closed IC systems are available that do not come into direct contact with the inserter's hands and are designed to reduce the risk of bacterial contamination. Catheter trauma is fairly common but does not usually create lasting damage. Development of urethral strictures and false passages may occur with long-term IC. Rare complications include loss of a catheter within the bladder and bladder perforation.[1]

Indwelling catheters may be used for patients with uncontrollable incontinence or for urinary retention when use of IC is not possible or practical for the patient with NGB. Indwelling catheters are common in the long-term care setting, and antimicrobial prophylaxis is usually not recommended in these patients to avoid development of drug-resistant bacterial colonization of the bladder. Many bacterial species can colonize indwelling catheters as biofilm communities embedded in a polysaccharide matrix that can occlude the catheter lumen and trigger both upper UTI and septicemia.[9] Suprapubic catheters may also be used and have some advantages over standard urethral catheters. These catheters are usually easier to manage hygienically, especially in regard to catheter changes; have less tendency to kink; are easily reversible; and provide less interference with social functioning and sexual intimacy.[1] In addition, avoidance of a long-term urethral catheter removes the risk of urethral erosions or trauma.[1,2] Along with elevated risk of UTIs, less frequent complications include exit site infection, bleeding, bladder spasms, and technical difficulties with catheter usage. These catheters also need to be changed routinely. Long-term bladder health for those patients with an indwelling catheter may be optimized with concomitant use of antimuscarinics.[6]

The use of absorbent pads or diapers is not recommended as the sole intervention for patients with NGB and significant urinary incontinence, owing to problems such as skin irritation and breakdown and increased risk of UTIs.[1] Condom catheter issues usually center around a poorly fitting appliance. This can usually be minimized with good hygiene practices, frequent changes, and maintenance of lower bladder pressures.[2,10] Some patients may have issues with the fit of the condom catheter. For those with a retractile penis, a malleable penile prosthesis may be helpful.[10]

Pharmacologic Therapies for Neurogenic Bladder

A variety of pharmacologic agents are available for the treatment of NGB as part of a patient's comprehensive bladder management program. The most commonly used class of medications to treat NGB are antimuscarinics. These medications are orally administered in the majority of patients, with intravesical instillation rarely used.[1,5,11] Combination therapy using 2 or 3 different oral agents (antimuscarinics, alpha-blockers, and tricyclic antidepressants) has been found to be effective in patients with NGB and poor bladder compliance.[12] Mirabegron, a beta-agonist, is an oral therapy that has been studied to treat overactive bladder (OAB).[13] Although this does not include an indication for NGB, it is potentially likely this medication will be used either in combination with antimuscarinics or alone in antimuscarinic-refractory patients. Lastly, onabotulinumtoxin A (BoNT/A) was approved by the US Food and Drug Administration (FDA) following phase 3 clinical trials that demonstrated the agent's efficacy in decreasing UI episodes in patients with neurogenic detrusor overactivity.[5,14,15] This treatment is FDA approved for the treatment of NGB (indicated for urinary incontinence due to detrusor overactivity associated with a neurologic condition in adults who have an inadequate response to or are intolerant of an antimuscarinic medication).[16] Clinicians and providers managing patients with NGB must be aware of the characteristics and complexities of different available and emerging therapy options to guide treatment decisions and assist them in optimizing individualized management of patients with NGB.

Antimuscarinics

Antimuscarinic drugs comprise the foundation of first-line treatments for patients with NGB/NDO.[5] The physiologic basis for treating bladder overactivity with these agents is that detrusor contractions are primarily mediated by muscarinic receptors.[17] Acetylcholine release from cholinergic nerves stimulates muscarinic receptors on the smooth muscle within the detrusor.[18] Antimuscarinics competitively inhibit acetylcholine at the muscarinic receptors, leading to relaxation of the detrusor and improved bladder storage.[5] By blocking the action of acetylcholine, these agents stabilize the detrusor, rendering it refractory to parasympathetic nerve impulses. This results in increased bladder capacity and a delay in the urge to void.[18]

Oxybutynin has been in widespread clinical use for this purpose since 1975, with tolterodine tartrate introduced in 1996 as an alternative to oxybutynin.[18] Oxybutynin is available in both immediate- and sustained-release oral formulations as well as transdermal and topical gels.[1] In addition, intravesical administration of oxybutynin has been described in the past.[11] Tolterodine tartrate and trospium chloride are also available in immediate- and sustained-release oral formulations. It appears that most extended-release preparations of antimuscarinics may produce less cognitive side effects and dry mouth than immediate-release oxybutynin.[1] It is thought that trospium, owing to its size and charge as a quaternary amine, does not cross the blood-brain barrier (BBB) and thus produces fewer cognitive side effects.[1,19] BBB permeability can be an important factor when treating NGB because BBB integrity can be disrupted in patients with stroke, MS, Parkinson's disease, trauma, and older age.[5] Multiple antimuscarinic agents are available in different dosages and may be administered orally, or in some cases, through intravesical or topical administration (**Table**).[18] Overall, antimuscarinic therapy can provide substantial benefits to patients with NGB and NDO.[1] A recent meta-analysis that included a total of 960 patients with NDO in 16 randomized clinical trials (RCTs) found that antimuscarinics were associated with statistically significantly better patient-reported cure or improvement, higher maximum cystometric capacity, higher volume at first detrusor contraction, and lower maximum detrusor pressure when compared with placebo. A multitude of agents were studied, and no one drug was found to demonstrate superiority over another. While the drugs were all found to be highly effective, a greater incidence of adverse events, including dry mouth, constipation, nausea, and visual disturbances, was noted in the treatment cohorts versus those receiving placebo in these RCTs. Although the authors urged caution, as the data referred to limited follow-up, they concluded that the findings overall supported the use of antimuscarinic drugs in patients with NDO to reduce the risk of long-term deterioration of renal function associated with NDO.[20] Combining antimuscarinic agents is sometimes considered for patients with NGB; one study found that whereas 63% of patients diagnosed with NGB were using 1 antimuscarinic agent, another 9% of patients were taking more than 1.[1,21] Combining antimuscarinics may be an effective strategy in patients in whom monotherapy has failed, including double-dose antimuscarinic therapy. One study demonstrated that when combined high-dosage antimuscarinics were used, 85% of the patients who previously experienced failed treatment with dosage-escalated monotherapy were treated successfully, including improvements in bladder capacity, detrusor compliance, and reflex volume without a significant increase in adverse events.[22] In addition, Bennett and colleagues found that patients with NGB were more likely to request higher doses of oxybutynin-XL compared with patients with overactive bladder.[23] Therapy combining an antimuscarinic with the tricyclic antidepressant imipramine and/or an alpha-blocker has been found to be effective in patients with NGB and poor bladder compliance. Combination therapy using either 2 or 3 drugs improved bladder compliance, decreased pressures at capacity, and improved clinical outcomes. Combination therapy is a potential pathway to consider in patients in whom antimuscarinic monotherapy proves inadequate.[12] The use of intravesical oxybutynin has been used as well, more so in the past. The theory is that with intravesical administration the efficacy can be maintained with minimization of side effects such as dry mouth and constipation. Oxybutynin has been shown to be both safe and effective when administered in this manner, and other drugs have been tested for this type of use.[1,11] However, the drug can still be absorbed through the detrusor, and side effects related to the blocking of muscarinic receptors such as dry mouth do not appear to eliminated.[24,25] Lastly, the administration of oxybutynin intravesically, multiple times daily, can be challenging for patients and their families and is a primary reason why patients discontinue the treatment.[26]

Although antimuscarinics have obvious benefits, some of these agents have been associated with poor patient compliance, and patients with NGB can be intolerant of some adverse events associated with treatment, such as bowel dysfunction.[5] One epidemiologic study found that during a 1-year period, 38% of patients with NGB taking antimuscarinics discontinued use of these agents and did not restart, more than 80% of patients interrupted antimuscarinic therapy, and approximately 34% stopped and restarted therapy.[1,21] Patient compliance and persistence with therapy can be suboptimal and may vary depending on the drug prescribed.[5] Compliance/adherence/persistence surrounding therapy is an area that clinicians must pay special attention to initially and throughout follow-up when using antimuscarinics for management of NGB/NDO.

OnabotulinumtoxinA

Botulinum toxin is one of the most potent naturally occurring neurotoxins in existence. It is derived from the gram-positive coccus bacterium *Clostridium botulinum* and leads to a flaccid paralysis of striated muscle through a mechanism that involves blocking acetylcholine release at the presynaptic level. Botulinum toxin was first approved by the FDA in 1989 for treatment of strabisumus, blepharospasm, and hemifacial spasm, and it has further been developed and approved for a variety of other applications.[27] There are 7

Table. Antimuscarinic Agents

Medication	Brand Name	Dosage
Oxybutynin	Ditropan	2.5-5 mg/2-4 times per day
Oxybutynin extended release	Ditropan XL	5-30 mg once daily
Oxybutynin extended-release transdermal patch	Oxytrol	3.9 mg per day; system applied twice weekly (every 3-4 days)
Tolterodine	Detrol	1-2 mg twice daily
Tolterodine extended release	Detrol LA	2-4 mg once daily
Darifenacin	Enablex	7.5-15 mg once daily
Solifenacin	VESIcare	5-10 mg once daily
Trospium chloride	Sanctura	20 mg twice daily
Trospium chloride extended release	Sanctura XR	60 mg once daily

immunologically distinct types of botulinum toxin. Types A and B are available commercially, and onabotulinumtoxinA (BoNT/A) is the FDA-approved form for use in urologic indications.[28] The toxin blocks neuromuscular junction presynaptic vesicle fusion, preventing acetylcholine release and blocking signal transmission across the neuromuscular junction.[1,29] This effect clearly impacts the efferent muscle contraction, and there is evidence to support possible afferent effects on bladder function (and for other applications) as well.[1,28] Injection of BoNT/A into the detrusor muscle leads to dose-dependent muscle relaxation due to reduced neural signal transmission.[1] Interestingly, BoNT/A's duration of effect in the bladder, which is composed of smooth muscle, is significantly longer than that seen in skeletal muscle. Long-lasting improvement in NDO, UI, and QoL in individuals with SCI and MS, the patients in whom it has been most extensively studied, has been noted in multiple studies evaluating its use for NGB.[1,14,15,28] At present, this agent is approved for use for treatment of UI due to detrusor overactivity associated with neurologic conditions (eg, SCI, MS) in adult patients who have an inadequate response to or are intolerant of antimuscarinic medications.[16]

The 2 primary trials that ultimately led to the approval of BoNT/A for the treatment of NGB studied 691 patients with either SCI or MS. The studies enrolled 275 and 416 patients, respectively, with UI due to NDO (≥14 episodes per week) who received 30 intradetrusor injections of BoNT/A 200 U, 300 U, or placebo. The primary end point was the change in baseline in UI episodes per week at week 6. Secondary end points included urodynamic end points (maximum cystometric capacity, maximum detrusor pressure during first involuntary detrusor contraction) and QoL using the Incontinence Quality of Life questionnaire (I-QoL) score. The studies found that both BoNT/A doses significantly reduced UI and improved urodynamic outcomes and QoL in the patients studied. Both doses of BoNT/A were well tolerated with no clinically relevant differences in effect or duration between the 2 doses tested. The most common adverse events were urinary tract infections and urinary retention.[14,15] Increased risk of urinary retention and elevated postvoid residual (PVR) were seen in patients who did not perform IC at baseline. Although many initial studies evaluated the higher dosage of 300 U of BoNT/A, these clinical trials trial demonstrated that the 200-U and 300-U doses had similar efficacy, with patients on the 300-U dose experiencing a greater risk of certain adverse events. Thus, the 200-U dose was approved by the FDA due to similar efficacy and an improved risk-benefit ratio compared with the 300-U dose.[14-16] The average duration of effect of BoNT/A in patients studied was approximately 9 months.[15] Another retrospective trial of 200 patients with NGB receiving BoNT/A found a greater than 50% increase in mean bladder volume to first reflex detrusor contraction and maximum cystometric capacity accompanied by a proportional decrease in maximum detrusor pressure. The benefits of BoNT/A administration persisted over more than 6 months and could potentially prove most cost-effective over this duration of therapy (ie, continuous use of BoNT/A for 5.1 months of therapy or more).[1,30] Studies assessing HRQoL following BoNT/A administration in patients with NDO have also been performed, and results in general have demonstrated improvements in QOL as assessed by I-QOL scores or Urinary Dress Inventory (UDI-6) assessment.[31,32]

Other Pharmacologic Interventions for Neurogenic Bladder and Neurogenic Detrusor Overactivity

Other pharmacologic interventions that have been assessed or are currently undergoing investigation as poten-

tial therapies for use in NGB/NDO include several different classes of therapy[1]:

- Tricyclic antidepressants (imipramine, amitriptyline)
- Cholinergic agonists (urecholine)
- Alpha-2 adrenergic agonists (clonidine, tizanidine)
- Alpha-1 adrenergic agonists (dibenzyline, terazosin, tamsulosin, alfuzosin, doxazosin)
- Benzodiazepines (diazepam)
- GABA-B agonists (baclofen)
- Opioids (nociceptin)
- Vanilloids (capsaicin, resiniferatoxin)
- Nerve growth factor
- Nitrous oxide agents

Each class of agents exhibits unique mechanisms of action. Many of these agents are experimental, and not all are strongly recommended as potential treatments for patients with NGB/NDO.[1] In addition to these drugs, beta-adrenergic agonists have found to be potent relaxant agents of the human detrusor muscle in vitro.[33] This class of agents has been studied and found to be effective adjuncts in the treatment of OAB and may have a potential role in the treatment of detrusor overactivity in patients with NGB.[34]

Surgical Interventions for Neurogenic Bladder and Neurogenic Detrusor Overactivity

Surgical interventions are usually reserved for cases in which nonpharmacologic and pharmacologic interventions fail to control NDO in patients with neurologic disorders. Options include neuromodulation and other surgical interventions.[1]

Neuromodulation

Sacral neuromodulation (SNM) involves unilateral or bilateral sacral nerve root stimulation. SNM has been used to treat OAB and has been approved by the FDA for treatment of urge incontinence, urinary urgency and frequency, and nonobstructive urinary retention. SNM has been investigated for the treatment of NGB; however, it is not FDA approved for this indication. The exact mechanism of action of SNM is not fully understood; however, SNM uses electrical stimulation to modulate signaling from somatic and afferent nerves involved in the micturition reflex pathway. Original trials excluded patients with NGB because it was initially believed that intact spinal pathways were required for neuromodulation to occur. However, since FDA approval for use in OAB, clinical trials have been under way studying the use of SNM in neurogenic voiding dysfunction. This therapy is considered (though not yet approved by the FDA) in patients with NGB/NDO who have used conservative therapies including lifestyle changes and bladder training plus antimuscarinic therapy but have not achieved adequate relief from symptoms. A 2-stage procedure is performed. Patients initially undergo a 1- to 2-week trial of SNM to assess likelihood of clinical improvement in lower urinary tract symptoms. In this trial, the nerve located at S3 is stimulated by a lead positioned in the sacral foramen connected to an external stimulator that applies the appropriate electric current. Patients are instructed to keep a voiding diary to record both bladder function and urinary symptoms. If the patient reports more than a 50% improvement in symptoms, the patient moves on to stage 2, in which an implantable pulse generator is placed internally via an incision in the upper lateral buttock and development of a subcutaneuous space to house the pulse generator.[1,35] A recent meta-analysis found a pooled success rate of 68% for the test phase and 92% for permanent SNM (after a successful test stimulation) in NGB with a 0% complication rate for the test phase and a 24% complication rate for permanent SNM.[1,36] Disagreements about the actual applicability of SNM exist. One study evaluating patients with NGB dysfunction due to a variety of neurologic diseases found SNM became ineffective for all patients studied except 1 after a period of 54 months, suggesting a lack of long-term efficacy in this patient population.[37] Another more recent trial evaluating use of neuromodulation in patients with NGB dysfunction, both with and without underlying neurologic conditions, demonstrated that greater than 50% of patients in both groups reported moderate or marked improvement in bladder symptoms with SNM over the course of the 2-year assessment period.[38] Further study is ongoing in this particular therapeutic area.

Other Surgical Interventions

The most effective surgical method to optimize bladder capacity is enterocystoplasty. This involves the harvesting of a piece of bowel that is configured into a patch, which is placed on a bivalved bladder. Patients undergoing bladder augmentation would be expected to have an appropriate increase in bladder capacity and decrease in bladder storage pressures. Risks of these procedures include bladder stone, UTI, bladder perforation, change in bowel habits, and cancer. For patients who are interested in lower urinary tract reconstruction but are unable or unwilling to perform IC per their native urethra, a continent urinary stoma can be constructed to the skin.[1,39]

Bladder sphincterotomy may be performed to improve bladder emptying and is primarily performed in patients with detrusor external sphincter dyssynergia who reflex-void to a condom catheter. Indications may include elevated PVR, elevated detrusor pressures, autonomic dysreflexia, and recurrent UTI. Other options for this problem include the use of urethral stents and "pharmacologic sphincterotomy" with the use of BoNT/A.[1]

In addition, there are surgical options for patients with UI due to an incompetent urinary sphincter. Implantation of an artificial urinary sphincter device is considered the gold standard for treating UI due to sphincteric incontinence in men.[1] Female patients with stress UI as a result of a bad outlet would benefit from sling placement. The majority of non-neurogenic women undergoing sling placement for stress UI will have a minimally invasive Prolene mesh sling placed. For neurogenic stress UI, the goal is often obstruction (and regular IC); therefore, a synthetic sling would not be recommended and autologous tissue, either from the rectus fascia or fascia lata, should be used.[1,40]

Future developments for managing NGB under investigation include lumbar to sacral nerve rerouting, and spinal cord regeneration using genetically modified fibroblasts or neuronal and glial precursors into the spinal cord.[1]

Conclusion

NGB and its associated lower urinary tract dysfunction present difficult symptoms for patients with neurologic disorders and present clinicians with multiple options and challenges in providing appropriate individualized therapy to improve urinary function and HRQoL. Clinicians managing these patients must choose individualized and optimal treatment pathways among an array of standard, new, and emerging treatments. Better understanding of classes of medical therapies and surgical interventions, both available and emerging, will help clinicians managing patients with neurologic disorders and NGB/NDO to enhance both outcomes and QoL in these patients.

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