Treatment of Voiding Dysfunction in Neurogenic Bladder

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ABSTRACT

Neurogenic lower urinary tract dysfunction (NLUTD) may cause emptying failure, storage failure, or both. The aim of treatment is to keep intravesical pressure low to avoid upper urinary tract damage. Another treatment goal is to maintain continence and consequently improve the patient's quality of life. Urodynamic study is essential for the diagnosis of NLUTD. The selection of treatment is based on patient performance, cost-effectiveness, technical complexity and possible complications. There is no single treatment of NLUTD which fits all patients. We reviewed the treatment options for NLUTD to provide guidelines to achieve treatment goals.

Key words: neurogenic bladder, autonomic dysreflexia, clean intermittent self-catheterization

INTRODUCTION

Neurogenic lower urinary tract dysfunction (NLUTD) is caused by many diseases or events affecting the nervous systems controlling the lower urinary tract (LUT). The true prevalence of NLUTD is still not known because of a lack of large size surveys which follow the 2009 European Association of Urology (EAU) guidelines.

NLUTD may be caused by brain tumors, dementia, mental retardation, cerebral palsy, normal pressure hydrocephalus, basal ganglia neuropathy, cerebrovascular disease, spinal cord lesions, peripheral neuropathy or surgical complications. Abdominoperineal resection of the rectum and radical hysterectomy for cervical cancer may cause NLUTD, implicating the importance of pelvic autonomic nerve preservation [1-9].

Normal LUT function relies on intact peripheral and central nervous systems. Although many different classification systems have been suggested, the description of NLUTD should be individualized since there is no perfect classification for neurologic lesions and LUT dysfunction.

Madersbacher et al [10] presented a simple classification of NLUTD according to clinical practice. It is based on the detrusor pressure during the filling phase and urethral sphincter relaxation during the voiding phase. The classification reflects the concept of NLUTD which can be classified as "failure to store" or "failure to empty". In Madersbacher's classification, a suprapontine lesion may cause detrusor overactivity and normal sphincter function, a spinal lesion may induce both detrusor and sphincter overactivity, a lumbosacral lesion may induce both sphincter and detrusor overactivity or underactivity, and a subsacral lesion may cause both detrusor and sphincter hypoactivy. Overactivity or underactivity of the urethral sphincter without bladder dysfunction may also cause NLUTD (Table 1).

Table 1. Madersbacher's Classification

Location of lesion	Effect on detrusor muscle	Effect on sphincter
Suprapontine	Overactive	Normoactive
Spinal lesion	Overactive	Overactive
Lumbosacral	Overactive	Underactive
	Underactive	Overactive
	Underactive	Normoactive
Subsacral	Underactive	Underactive

During urodynamic evaluation of patients with NLUTD, autonomic dysreflexia is an emergency condition which can be life threatening. Autonomic dysreflexia is caused by massive sympathetic outflow above the splanchnic sympathetic nervous system, caused by bladder irritation or fecal irritation in patients with spinal cord injury above the T5-6 level. Once the systolic blood pressure increases more than 20 to 30 mmHg with concomitant stimuli, it is considered a dysreflexic episode [11]. Other signs include sweating, blushing, and piloerection. If hypertension or bradycardia occur with possible increased intravesical pressure, keeping intravesical pressure low by changing body position or catheterization is imperative; if uncontrolled high blood pressure persists, antihypertensive medication such as a calcium channel blocker or nitrate is needed [12].

Making the diagnosis of NLUTD requires an extensive general history and physical examination including neurological status, and anal sphincter and pelvic floor function. Other tests including urinalysis, blood chemistry, a voiding diary, post-void residual (PVR) and free flowmetry, and incontinence quantification may also be necessary. Urodynamic study is the only method to assess dysfunction of the LUT, and should include filling cystometry, detrusor leak point pressure (DLPP) investigation, pressure flow study and electromyography (EMG) [13,14].

There are three main NLUTD problems, failure to store, failure to empty, and a combination of these two symptoms. Goals of treatment: The aim of treatment of NLUTD is to keep detrusor pressure or intravesical pressure low, although the urodynamic investigation results may vary among patients [15].

According to EAU guidelines, the aims of treatment of NLUTD are as follows in order of priority:

1. To protect upper urinary tract function
2. To improve urinary continence
3. To regain some LUT function
4. To improve the patient's quality of life via freedom from a catheter

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or satisfactory voiding management.

There is no single treatment option which fits all patients; therapy needs to be matched to an individual's needs and abilities while considering the patient's performance, cost-effectiveness, technical complexity and possible complications. Adequate bladder function includes low pressure in the bladder during the storage phase, complete emptying and sterile urine, and these are also the aims of management.

CONSERVATIVE AND MINIMALLY INVASIVE MANAGEMENT TO ENHANCE STORAGE

For neurogenic detrusor overactivity, muscarinic receptor antagonists are used to improve bladder compliance and reduce bladder overactivity [16]. However, the side effect of these drugs may result in discontinuation of therapy [17] oxybutynin [18], trospium chloride [17], tolterodine [19], and propiverine [20] are safe and effective drugs currently available.

Intravesical drug instillation is another choice. The drugs include anticholinergic agents such as atropine [21] and oxybutynin [22]. Intravesical administration may reduce the adverse effect of the drugs because of the metabolic pathway and large amount of drugs needed in the bladder. The other choice is vanilloid agents such as capsaicin and resiniferatoxin, which improve detrusor overactivity by desensitizing the C-fibres. The dosage used is 1 to 2 mmoL/L of capsaicin in 30% ethanol in saline. The reported clinical benefits from a single instillation last 3 to 6 months [23]. Resiniferatoxin is an analogue of capsaicin with more than 1,000 times its potency. The dosage used is 5-10 mmoL resiniferatoxin in 100 mL of 10% alcohol [24]. Both drugs may increase the mean cystometric capacity or decrease incontinence episodes in human models [23,24].

Intradetrusor injections of botulinum neurotoxin type A result in a reversible chemical denervation that lasts for about 9 months [25]. If the first attempt fails, repeated injections may have an effect. Botulinum neurotoxin type A may be effective in the treatment of both idiopathic detrusor overactivity and neurogenic detrusor overactivity [26]. The treatment may cause poor contractility of the detrusor, and there is a risk of urinary retention. Self-catheterization may be required if this occurs [27]. Other adverse effects include generalized weakness [28].

Neuromodulation including sacral neuromodulation and posterior tibial nerve stimulation, which have been approved by United States Food and Drug Administration (FDA), can be performed to treat detrusor overactivity. The mechanism of neuromodulation of bladder function is not clear yet. Benefits have been observed in patients with Parkinsonism and multiple sclerosis. Neuromodulation treatment may play a role for symptoms that are refractory to conservative management [29].

SURGICAL MANAGEMENT TO ENHANCE STORAGE

Some surgical procedures are preserved for refractory bladder storage problem. Percutaneous sacral rhizotomy and sacral rhizotomy for neurogenic detrusor hyperreflexia have shown effectiveness in increasing bladder capacity. There is a recent trend to use these procedures as an adjuvant therapy with anterior root stimulation [30]. In bladder autoaugmentation, the detrusor muscle over the entire dome of the bladder is excised. This procedure does not involve the use of the bowel [31]. It may be performed extraperitoneally or with laparoscopic assistance, which may offer the same advantages. If conservative therapy fails, augmentation enterocystoplasty is well tolerated in selected patients with NLUTD. This procedure may increase the bladder capacity, decrease maximum detrusor pressure, and improve urological satisfaction, such as continence with the use of clean intermittent self-catheterization [32]. The adverse effect of bowel habit changes is not significant. However, some patients may still need several low doses of oxybutynin postoperatively to maintain urinary continence [33].

A pubovaginal sling is an effective treatment for poor proximal urethral sphincter function in women. The success rate is reported to be as high as 92% [34]. Other materials for a sling procedure include a fascial sling, such as a rectus fascial sling [35]. Implantation of an artificial sphincter for both NLUTD and post-prostatectomy patients has resulted in a reported continence rate of 60 percent during a 10-year follow up in males. The side effects include perineal pain and urethral erosion. The outcome of artificial urinary sphincter implantation is better than that for adjustable male sling placement [36].

CONSERVATIVE AND MINIMALLY INVASIVE MANAGEMENT TO ENHANCE EMPTYING

Single use of parasympathomimetics to treat an underactive bladder is not recommended because of side effects such as nausea, gastrointestinal cramps and even cardiovascular depression and related clinical outcomes [37]. However, they are effective in combination with alpha blockers. There is still no conclusion on whether single use of parasympathomimetics enhances bladder emptying long term [38].

Table 2. Summary of Treatment Options

Conservative and minimally invasive management to enhance storage	Muscarinic receptor antagonists
	Intravesical drug instillation (atropine, oxybutynin, vanilloid agents)
	Intradetrusor injections of botulinum neurotoxin type A
	Neuromodulation (sacral neuromodulation, posterior tibial nerve stimulation)
Surgical management to enhance storage	Percutaneous sacral rhizotomy or sacral rhizotomy
	Bladder autoaugmentation
	Augmentation enterocystoplasty
	Pubovaginal sling
	Implantation of an artificial sphincter
Conservative and minimally invasive management to enhance emptying	A combination of parasympathomimetics and alpha blockers or alpha blockers alone
	Neuromuscular electrical stimulation (intravesical stimulation or sacral root stimulation)
	Intermittent catheterization
Surgical management to enhance emptying	Sphincterotomy
	Botulinum toxin sphincter injection

Drugs such as alpha blockers are effective in decreasing bladder outlet resistance and are appropriate for long-term use. Alpha blocker administration alone may improve bladder storage and emptying, and also improve symptoms of autonomic dysreflexia [39]. Neuromuscular electrical stimulation is divided into intravesical and sacral root stimulation. Intravesical stimulation of the bladder has still not proved beneficial [40-42].

Stimulation of sacral anterior nerve roots in association with a posterior rhizotomy has resulted in "urination on demand" for patients with a low PVR, a reduced rate of urinary tract infections (UTI) and improved continence [43]. The Vocare Bladder System, which has been approved by the FDA, is used in patients with complete spinal cord lesions who are neurologically stable and have good bladder contractility.

According to the EAU guidelines, intermittent catheterization (IC) is the gold standard for NLUTD. Patient education, good patient compliance, and a good catheterization technique are important to avoid complications. Complications of IC include urinary tract infection, prostatitis, traumatic catheterization, false passage and urethral strictures. The frequency of IC may be a risk factor for UTI [44]. It is suggested that the mean volume of each catheterization be less than 400 mL [45]. The average frequency of catheterization should be 4-6 times per day, according to EAU guidelines.

No catheter type, technique, or strategy is better than another for indwelling catheters via the transurethral or supra-pubic route [46]. The reasons for shifting from IC to an indwelling catheter are urethral trauma or damage, worsening of the original disease, personal reasons and loss of personnel.

SURGICAL MANAGEMENT TO ENHANCE EMPTYING

Reducing bladder neck resistance may be effective in decreasing intra-vesical pressure. The procedures include bladder neck or sphincter incision and chemical sphincterotomy. Botulinum toxin sphincter injection is effective in reducing intra-vesical pressure and reducing PVR, and is effective for around 3-9 months [47-49]. Sphincterotomy should be performed stage by stage and can improve LUT parameters. However, with a twelve o'clock sphincterotomy, the reoperation rate for maintenance of urodynamic results is high and may also result in bladder neck contracture [50]. According to EAU guidelines, the sphincterotomy is the standard operation for detrusor sphincter dyssynergia. Balloon dilation of the urethra [51] has not been reported since 1994 and current long term results are unknown. The placement of a urethral stent may provide the same benefit as a sphincterotomy, however complications such as stent migration or recurrent stenosis may limit its use [52,53].

URINARY DIVERSION

The aim of urinary diversion is to protect the upper urinary tract if no other therapy has been successful. Continent urinary diversion such as an appendicovesicostomy, Mitrofanoff procedure, or detubularized sigmoidovesicostomy may be the first choice for patients with an indwelling catheter. The continent stoma can be created by various methods, and is often applied in children and adolescents because of long life expectancy [54,55]. The umbilicus is often used for the stoma site for cosmetic reasons and convenience in self-care [56].

An incontinent diversion such as an ileal conduit, and incontinent ileovesicostomy, is seldom used nowadays because of other alternatives. This can be offered for a devastated LUT or a bed-ridden patient with intractable incontinence. However, in long term follow-up, there is still a risk of upper tract deterioration in patients with an ileal conduit diversion [57].

CONCLUSIONS

There is no single treatment for NLUTD which fits all patients. The treatment choice is complex. However the aims of management, which are to protect the upper urinary tract and obtain better quality of life, do not change. Appropriate individualized therapy may avoid NLUTD-induced complications and improve patients' quality of life.

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