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Principles of diagnosis and treatment of post-traumatic neurogenic lower urinary tract dysfunction

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ABSTRACT

This article provides an overview of modern approaches to the diagnosis and treatment of post-traumatic neurogenic lower urinary tract dysfunction. Lower urinary tract dysfunction is observed in nearly all patients with traumatic brain or spinal cord injuries. It is noted that the type of urodynamic impairment does not always correspond to the level and severity of nervous system damage. The clinical diversity of neurogenic lower urinary tract dysfunction resulting from traumatic central nervous system injuries is highlighted, necessitating thorough evaluation, including urodynamic studies. The selection of treatment methods for patients is discussed in detail, depending on the type of neurogenic lower urinary tract dysfunction.

Keywords: neurogenic lower urinary tract dysfunction; neurogenic voiding disorders; spinal cord injury; traumatic brain injury.

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Принципы диагностики и лечения нейрогенных расстройств мочеиспускания травматического генеза

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АННОТАЦИЯ

В статье представлен обзор современных подходов к диагностике и лечению нейрогенных расстройств мочеиспускания травматического генеза. Дисфункции нижних мочевыводящих путей наблюдаются практически у всех пациентов с черепно-мозговыми и позвоночно-спинномозговыми травмами. Отмечено, что тип уродинамических нарушений не всегда соответствует уровню и степени повреждения нервной системы. Показано клиническое разнообразие нейрогенных дисфункций нижних мочевыводящих путей вследствие травматических повреждений центральной нервной системы, что требует тщательного обследования, в том числе с проведением уродинамического исследования. Подробно рассмотрены вопросы выбора метода лечения пациентов в зависимости от типа нейрогенной дисфункции нижних мочевыводящих путей.

Ключевые слова: нейрогенная дисфункция нижних мочевыводящих путей; нейрогенные расстройства мочеиспускания; позвоночно-спинномозговая травма; черепно-мозговая травма.

Как цитировать

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INTRODUCTION

According to current clinical guidelines, neurogenic lower urinary tract dysfunction (LUTD) is a disorder of urine storage and voiding secondary to a neurological disease [1]. This definition does not provide a complete picture of the causes of urinary disorders, as neurogenic dysfunction may be caused by both diseases and damage to the central and peripheral nervous systems resulting from injury and trauma.

The global prevalence of traumatic spinal cord injury (SCI) is approximately 23 cases per 1 million population [2]. In the Russian Federation, the incidence of SCI ranges from 16 to 50 cases per 1 million population [3, 4]. SCI is three times more common in men than in women [5]. Importantly, the data on the prevalence of traumatic brain injuries (TBI) and SCIs obtained in these studies did not include injuries sustained during armed conflicts. According to the Russian literature, the incidence of TBIs in the total structure of combat surgical trauma in recent armed conflicts is 12%, SCI is 0.5%–2.4%, and pelvic gunshot wounds is up to 3%–4% [6]. There are data on the incidence of LUTD in neurological patients with pelvic disorders. The incidence of LUTD in patients with TBI is up to 60% [7], and in SCI, it is 70%–95% [8, 9].

There are currently several classifications of neurogenic urinary disorders. The classification of neurogenic bladder adopted at the III All-Union Congress of Urologists (Minsk, 1984) [10] has been widely used in the Russian Federation. The Madersbacher classification (1980), recommended by the European Urological Association, is the most widely used. It outlines the possible variants of urinary dysfunction. This classification is particularly relevant for complex urodynamic testing (CUDT) in patients with neurogenic bladder [11]. In addition, Panicker et al. [12] have proposed a classification that is based on the relationship between the level of nervous system damage and the type of urodynamic disorders. This classification distinguishes three levels of nervous system damage, each of which is characterized by its own variant of LUTD. In particular, the type of urodynamic disorder does not always correspond to the level of damage, which requires additional testing, primarily CUDT.

DIAGNOSIS OF NEUROGENIC LOWER URINARY TRACT DYSFUNCTION

The criterion for the diagnosis of LUTD is urinary dysfunction in the presence of diseases and injuries of the central and peripheral nervous system, as well as those induced by them [13]. According to the European Urological Association and the Russian Urological Society, patients with LUTD require a comprehensive approach to the assessment of the urological status. This approach includes the collection of complaints and history, physical examination, and laboratory tests on blood and urine samples. Additionally, instrumental studies, including ultrasound, radiological imaging, endoscopy, and urodynamic evaluation, are integral components of the diagnostic process [1, 14].

To obtain objective information when collecting complaints and histories from patients with urinary dysfunction, a detailed analysis of voiding characteristics is required, for which patients complete urinary (or catheterization) diaries [15, 16] and questionnaires. Many authors believe that the urinary diary should be completed within three days. This is the most informative diary. The European Urological Association also recommends the use of questionnaires to assess quality of life [14]. According to the guidelines of the Russian Urological Society, the Neurogenic Bladder Symptom Score (NBSS) questionnaire (Russian version) is currently proposed for use [1, 17, 18]. The NBSS questionnaire is a tool designed to assess the severity of symptoms and facilitate the differential diagnosis of types of urinary incontinence [1].

The use of questionnaires, such as the International Prostate Symptom Score (IPSS) or the Patient's Perception of Intensity of Urgency Scale (PPIUS), in patients with post-traumatic neurogenic urinary dysfunction is not relevant due to the lack of sensitivity and the inability to urinate independently in patients with a cystostomy, an intestinal reservoir, intermittent catheterization, or special methods of bladder voiding. The diagnosis of LUTD requires the involvement of a neurologist and neurosurgeon in addition to a urologist. The initial neurological examination is performed with the patient in the supine position and includes sensory and motor evaluation of all 28 dermatomes bilaterally. For the differential diagnosis of complete and incomplete spinal cord lesions, neurological disorders are evaluated in dermatomes S4–S5, corresponding to the perianal area. There are several differences in the urological examination of TBI and SCI patients with LUTD compared with the standard urological examination. In addition, three reflexes must be evaluated: cremasteric, bulbocavernous, and anal reflexes [19]. After this preliminary assessment, laboratory tests are conducted, including urinalysis, complete blood count, and blood chemistry analysis with determination of creatinine and urea levels to assess renal function. Bacteriological urinalysis is performed for specific indications [19].

A non-invasive examination, ultrasonography of the kidneys and bladder, is recommended for all patients with LUTD to visualize possible changes in the upper and lower urinary tract and to determine the volume of residual urine [20]. The most accurate characterization of LUTD is the CUDT [21]. It is performed after spinal shock and recovery of spinal reflexes to determine the

type of LUTD, which plays a crucial role in the choice of treatment [22]. Non-invasive evaluation is performed during CUDT, including uroflowmetry (in patients with preserved voiding) and residual urine volume measurement (by ultrasound or single catheterization). Invasive methods such as filling cystometry, pressure-flow studies combined with pelvic electromyography, and urethral pressure profilometry are indicated in specific cases. The video-urodynamic study is currently the gold standard for the evaluation of patients with LUTD [23].

Uroflowmetry is considered a simple and accessible diagnostic method [24]. It is performed exclusively in patients with preserved voiding; however, its diagnostic capabilities are limited to distinguishing between detrusor contractility dysfunction and infravesical obstruction [25]. Uroflowmetry may be regarded as a screening diagnostic method. In cases where uroflowmetry parameters are changed, more complex tests, such as filling and voiding cystometry (pressure-flow study), are often required [25, 26]. Filling cystometry is a diagnostic tool that may be used to detect storage dysfunction, detrusor hyperactivity (DH), bladder sensitivity, and compliance disorders [19, 26]. However, this procedure is associated with a risk of complications, most commonly urinary tract infections [14]. More serious complications, such as autonomic dysreflexia, develop primarily in patients with SCIs above the Th6 level [19, 26, 27]. The pressure-flow study is a diagnostic tool that can differentiate between infravesical obstruction and detrusor dysfunction [25]. When used concurrently with pelvic floor electromyography, it facilitates the diagnosis of detrusor sphincter dyssynergia (DSD) [14]. However, urethral pressure profilometry is not currently used as a standard clinical practice due to its limited informative value in patients with LUTD and a lack of consensus on the parameters indicative of the disorder [14].

According to the Russian Urological Society, all patients with LUTD should undergo CUDT [28]. However, there is no consensus on the frequency of this procedure (Table 1) [29].

Lower urinary tract urodynamics in spinal cord injury

Neurogenic urinary dysfunction is found in almost 100% of SCI patients and may persist for a long time [9]. Many studies have examined the relationship between the lesion level and the type of neurogenic urinary dysfunction. The study by Weld and Dmochowski [34] found DSD in 68% of patients with cervical SCI, 50% of patients with thoracic SCI, and 39% of patients with lumbar SCI [34]. Table 2 shows the results of a meta-analysis of four studies that have examined the relationship between the diagnosed abnormalities observed during urodynamic testing and the level of damage in SCI patients [35].

The results of this meta-analysis demonstrate the heterogeneity of urodynamic dysfunction in patients with the same SCI level. This finding indicates that the data on the lesion level alone are insufficient to accurately determine the type of urodynamic dysfunction and subsequent treatment. Further diagnostic testing is required. Kaplan et al. [36], who analyzed the results of urodynamic studies in SCI patients, observed different types of urodynamic dysfunction at the same lesion levels. Normal urodynamic parameters were observed in only 12% of patients with sacral SCI. The authors stated that the combination of DH and DSD is typical of thoracic SCI and occurs in 90% of such patients. In two other large studies by Weld and Dmochowski [34] and Rapidi et al. [37], there was no direct correlation between the type of urodynamic dysfunction and the severity of neurological deficits. As demonstrated by the research, any prognoses regarding urodynamic dysfunction in patients with SCI that are based on the lesion level are challenging in most cases. Taking into account the possibility of combined SCI, neurological disorders may have unpredictable neurological characteristics [25, 38, 39].

COMPLICATIONS OF TRAUMATIC NEUROGENIC LOWER URINARY TRACT DYSFUNCTION

In the second half of the 20th century, approximately 45%–50% of patients with SCI died from complications associated with LUTD. The main cause of mortality was renal dysfunction followed by renal failure. Taking into account the available treatment options for LUTD in patients with SCI, renal impairment is considered a predictor of all-cause mortality [40]. The male sex is associated with a higher risk of renal dysfunction than the female sex because the transverse striated external urethral sphincter and the prostatic urethra led to increased intravesical pressure during the Crede maneuver, Valsalva maneuver, and reflex voiding, which affects renal function [41].

The development of urinary tract infection (UTI) in patients with SCI is associated with increased residual urine and decreased bladder compliance [42]. The choice of bladder drainage method determines the incidence of UTIs, and the presence of a permanent catheter is a common cause of UTIs [43]. Patients with SCI resulting from neurological disorders frequently exhibit non-specific symptoms, which complicates differentiation between asymptomatic bacteriuria and UTI. These patients are treated with antibiotics that increase bacterial multidrug resistance in the urine [44]. As indicated in a study by Šámal et al. [45], the most prevalent bacterial strains identified through bacteriological urinalysis in patients with SCI were *Klebsiella* spp. (29%) and multidrug-resistant *Escherichia coli* (24%) [45].

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Table 1. Official clinical guidelines on the frequency of urodynamic studies in patients with neurogenic lower urinary tract dysfunction (LUTD) [29]

Таблица 1. Официальные клинические рекомендации в отношении периодичности выполнения уродинамических исследований у пациентов с нейрогенными дисфункциями нижних мочевыводящих путей [29]

Clinical guidelines	Patient population	CUDT frequency guidelines
Russian guidelines [1]	LUTD	It is recommended for all patients with LUTD; the frequency of performance is not specified. The combination of filling cystometry and pressure-flow study combined with imaging (video-urodynamic study) is identified as the gold standard for the investigation of urodynamics in patients with LUTD.
European Urological Association guidelines [14]	LUTD	Urodynamic testing is mandatory at the time of initial diagnosis. Patients with a high risk of upper urinary tract involvement should undergo regular urodynamic testing.
National Institute for Health and Care Excellence guidelines [30]	LUTD	Urodynamic testing may be considered as part of a survival strategy for patients with a high risk of upper urinary tract involvement (e.g., patients with SCI and spina bifida).
Guidelines of the American Urological Association and Society for Urodynamics and Female Urology [31]	LUTD	Clinicians should perform cystometry during the initial examination of patients with neurologic diseases, regardless of symptoms and during follow-up if necessary.
Consortium for Spinal Cord Medicine guidelines [32]	SCI	An annual urological examination is recommended; however, there is no consensus regarding the specific components of such an examination.
British clinical guidelines for the urological management of patients with SCI [33]	SCI	Urodynamic testing is recommended for urinary incontinence, detrusor sphincter dyssynergia with high intravesical pressure or low compliance detected previously, before and after a change in the treatment, in the presence of urinary tract infections or calculi, vesicoureteral reflux, and large residual urine volumes.

Note. CUDT, complex urodynamic testing; SCI, spinal cord injury.
Примечание. CUDT — комплекс уродинамических исследований; SCI — позвоночно-спинномозговая травма.

Table 2. Urodynamic study results depending on the level of spinal cord injury [35]

Таблица 2. Результаты уродинамического исследования в зависимости от уровня повреждения спинного мозга [35]

Urodynamic diagnosis	Spinal cord injury level			
	Cervical spine (*n* = 259)	Thoracic spine (*n* = 215)	Lumbar spine (*n* = 137)	Sacral spine (*n* = 46)
Detrusor hyperactivity, %	65	78	49	22
Detrusor sphincter dyssynergia, %	63	72	33	13
Detrusor acontractility, %	9	9	39	70
Reference values, %	1	2	2	9

Bilateral hydronephrosis, a significant complication of LUTD, has been observed in 12% of patients with SCI and has been associated with renal failure and death [46]. The etiology of hydronephrosis is associated with low bladder compliance and high intravesical pressure, which are frequently observed in patients with suprascrotal SCIs manifesting as DH and/or DSD [46]. Furthermore, vesicoureteral reflux during bladder filling or emptying

is observed in patients with LUTD and high intravesical pressure [47].

Urolithiasis is one of the major complications of LUTD and SCI [48]. It is associated with chronic urinary retention, vesicoureteral reflux, DSD, continuous catheterization, foreign bodies, bacteriuria, prolonged immobilization, and hypercalciuria due to bone resorption [49]. The Russian literature indicates the presence of calculi in 21%–64% of patients with SCI-induced LUTD [50, 51]. Bladder stones are frequently found in patients with LUTD [52]. The choice of treatment method is important, and the incidence of urolithiasis depends on this choice. When a permanent catheter is used for a long time, bladder stones are detected in 46%–53% of patients with LUTD. In addition, the risk of urolithiasis does not depend on the degree of spinal trauma, age, or sex of patients [53].

TREATMENT OF NEUROGENIC URINARY DYSFUNCTION

The treatment of neurogenic DH may be categorized into three primary approaches: non-drug, drug, and invasive interventions [1, 54]. It is advisable that treatment should be initiated with non-drug methods, which include bladder training, dietary restrictions on diuretic foods, and pelvic muscle exercises, including biofeedback therapy. These methods are considered the least invasive and are associated with minimal adverse effects. Patients with LUTD resulting from TBI and SCI are best treated with a combination of non-drug and drug methods [26]. M-choline blockers are the basis of pharmacotherapy in patients with neurogenic DH. The use of anticholinergic agents helps increase bladder capacity and decrease detrusor pressure [55, 56]. However, the administration of M-choline blockers in standard doses frequently proves ineffective, necessitating an increased dosage of the preparation, the addition of a drug with antimuscarinic properties, or a combination of two M-choline blockers [54, 57]. Consequently, the study by Krivoborodov et al. [58] substantiated the efficacy and safety of trospium chloride at a daily dose of 120 mg in patients with neurogenic DH.

Several studies suggest that beta3-adrenergic receptor agonists may be useful in the treatment of patients with neurogenic DH [59]. However, the drugs of this group have not been currently included in the guidelines for the treatment of LUTD due to a lack of evidence of their efficacy in this patient population [54, 60]. If patients with LUTD have independent urination, alpha-adrenergic blockers may be prescribed [61]. The efficacy of alpha-adrenoblockers is most pronounced in patients with suprasacral SCIs. In such cases, treatment has been shown to enhance bladder function, reduce maximum urethral pressure, decrease urination time, and decrease the residual urine volume [62]. In cases where drug treatment proves ineffective, is poorly tolerated, or is contraindicated, patients with neurogenic DH may undergo intravesical botulinum therapy, *n. tibialis posterior* stimulation, and sacral neuromodulation [1].

In 2005, Schurch et al. [63] published the results of a randomized study of the efficacy of botulinum toxin type A (BT-A) in the treatment of neurogenic DH. The study involved 59 patients who received intravesical injections of onabotulinum toxin type A at a dose of 200–300 U. The results demonstrated a 50% reduction in the frequency of episodes of urge urinary incontinence in the treated patients. Furthermore, another subtype of BT-A, abobotulotoxin, has been shown to be effective in the treatment of neurogenic DH [64]. A decrease in the frequency of UTI episodes in patients with LUTD after botulinum therapy has been shown to improve urodynamics [65]. In recent years, there has been considerable interest in sacral neuromodulation, with the efficacy reaching 80%–85% in idiopathic DH [66]. However, the results in the treatment of neurogenic urinary dysfunction are more controversial. Some studies have shown efficacy and safety of tibial neuromodulation in patients with neurogenic bladder [67]. However, the evidence base for its success is still insufficient.

In some cases, when pharmacotherapy and minimally invasive treatments fail to achieve the desired result, surgery is performed [1]. The most common is bladder augmentation [14], which is the only alternative when less traumatic methods of high intravesical pressure correction prove ineffective [28].

In the treatment of neurogenic DSD, a combination of drug therapy (anticholinergic agents) and regular catheterization is used to ensure urine outflow from the bladder and to maintain a low intravesical pressure, thus preventing renal dysfunction and the development of UTI [68]. In cases where the drug therapy proves ineffective and catheter insertion during regular catheterization is challenging, BT-A injections into the external urethral sphincter appear to be the most effective and safe treatment modality for DSD in patients with SCI. Salyukov et al. [69] showed that botulinum toxin injections into the external urethral sphincter in patients with SCI via the transperineal approach show similar efficacy to the transurethral administration, with a lower incidence of perioperative complications. The meta-analysis published in 2012 by Mehta et al. [70] showed that in patients with SCI after external urethral sphincter denervation with botulinum toxin, the residual urine volume decreased from 251 to 153 mL with a follow-up of up to six months. This decline led to a reduction in the need for intermittent self-catheterization or facilitated catheter insertion into the bladder.

Additionally, controversial results have been obtained with regard to the use of urethral stents in the patients with DSD. Hamid et al. [71] reported on their experience

with urethral stents and found a decrease in maximum detrusor pressure and residual urine volume six months after implantation. However, 19 of 25 patients required stent removal after a mean of 20 months due to stent migration, lithiasis, autonomic dysreflexia, and recurrent obstruction.

If the above methods of treating neurogenic DSD prove ineffective, endoscopic urethral sphincterotomy is used to reduce the outlet obstruction and create a low pressure in the bladder. The procedure is primarily performed in male patients. The results of 84 patients were presented by Pan et al. [72]. Although 68% of patients had subsequent recurrence of UTI and upper urinary tract dilatation, no patient had progressive renal failure.

The treatment of detrusor hypoactivity remains an unsolved medical issue. Previous research has suggested that cholinergic agents may increase detrusor contractility and improve voiding [14, 21]. However, randomized clinical trials have shown that the effect of parasympathomimetics on voiding is comparable to that of placebo [73].

If voiding is impaired, intermittent catheterization with correction of the drinking pattern is the treatment of choice. In this case, Crede and Valsalva maneuvers are not recommended because of the risk of complications. In patients with neurogenic voiding disorders with detrusor dysfunction, intermittent bladder catheterization 4–6 times per day is the standard of care. Aseptic intermittent catheterization is mainly used as an alternative to sterile catheterization. Intermittent catheterization uses 12–16 Ch catheters. The frequency of catheterization is set to ensure that the urine volume in the bladder does not exceed 400 to 500 mL [1, 14].

According to the International Continence Society classification, there are three primary types of catheters: uncoated catheters with a separately applied lubricant, prelubricated catheters with a water-soluble lubricant in the packaging, and lubricated catheters with a hydrophilic coating on the drainage tube [1, 74]. It is hypothesized that the use of catheters with a lubricated coating is preferable for intermittent catheterization. In a systematic literature review of the use of different types of catheters for intermittent bladder catheterization, Sevastyanov et al. [75] confirmed that lubricated catheters have an advantage over standard polyvinyl chloride catheters. If intermittent catheterization is not possible, cystostomy is performed to ensure urine drainage from the bladder [76].

CONCLUSION

Neurogenic urinary dysfunction is present in nearly all patients with SCI and TBI. However, the type of urodynamic disorders does not always correspond to the level and degree of nervous system damage. Consequently, an additional examination, CUDT, is necessary to determine the type of LUTD. Currently, a consensus on the frequency of CUDT is lacking. An adequate diagnosis of LUTD and an appropriate choice of treatment may significantly improve lower urinary tract function, improve patients' quality of life, and reduce the risk of complications, primarily renal damage.

ADDITIONAL INFO

Authors' contribution. All authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study. Personal contribution of each author: V.V. Protoshchak — study concept and design development, analysis of literature data, editing the manuscript text; M.V. Paronnikov, P.A. Babkin, N.P. Kushnirenko, P.O. Kislitsyn — analysis of literature data, editing the manuscript text; A.M. Nikolaev — analysis of literature data, writing the manuscript text.

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