

Pathophysiological Insights from Urodynamic Studies in Patients with Neurogenic Bladder Due to Upper and Lower Neuron Lesions

Adil Hefdhil Al-Soufi¹, Saif AbdulMohsin Al-Haideri¹, Abbass Awaad Ali¹, Mohammed Bassil Ismail²

¹Department of urology, medical city complex, Baghdad, Iraq

²Department of surgery college of College of Medicine, University of Baghdad

ABSTRACT

Objective: This study aims to delineate the pathophysiological characteristics of neurogenic bladder in patients with upper and lower motor neuron lesions through detailed urodynamic testing, and to correlate these findings with neurological pathology. **Methods:** Conducted at the Urology Department in Baghdad's Medical City Complex, this cross-sectional study involved 43 patients affected by spinal cord injuries, cerebrovascular accidents, and acute disc prolapse, all presenting with urinary incontinence. Urodynamic measures such as maximum bladder capacity, urethral closure pressure, and bladder compliance were meticulously analyzed. **Results:** The median maximum bladder capacity was recorded at 214 ml, median maximum urethral closure pressure at 73 cmH₂O, and median bladder compliance at 19 ml/cmH₂O. Disc prolapse emerged as the most frequent etiological factor, followed by spinal cord trauma. The prevalence of upper motor neuron lesions was marginally lower than that of lower motor neuron lesions. The predominant urodynamic profile was that of an underactive bladder or sphincter, particularly prevalent among patients with upper motor neuron lesions, illustrating the complex dynamics between neurogenic bladder dysfunction and motor neuron status. **Conclusion:** The findings emphasize the necessity of integrating urodynamic data with clinical and pathological evaluations to enhance diagnostic accuracy and therapeutic strategies in patients with neurogenic bladder due to motor neuron lesions. The study highlights the critical role of pathophysiological understanding in the management and prognosis of neurogenic bladder disorders.

Keywords: Urodynamic, Bladder, Prolapse, Incontinence, Urology.

INTRODUCTION

Urinary continence and controlled micturition depend on the highly coordinated function of three major anatomical and functional components: the detrusor muscle of the bladder, the bladder neck, and the urethral sphincter. These components are regulated by intricate neural circuits involving both the central nervous system (CNS) and peripheral nervous system (PNS). Central to this regulation are the Pontine Micturition Center (PMC), periaqueductal gray (PAG), cerebral cortex, spinal cord nuclei, and autonomic and somatic pathways[1, 2]. Any disruption within these circuits—whether from spinal cord injury, cerebral stroke, or peripheral nerve damage—can result in neurogenic bladder, a complex urological condition characterized by abnormal bladder storage and voiding dynamics[3]. Neurogenic bladder is a pathological manifestation of neurological impairment affecting the lower urinary tract. It is commonly observed in patients with upper motor neuron (UMN) lesions, such as those resulting from spinal cord trauma or cerebrovascular accidents, and lower motor neuron (LMN) lesions, as seen in conditions like cauda equina syndrome or advanced disc prolapse[4]. These neurological impairments disrupt the feedback loops and signal pathways that normally regulate the bladder's detrusor function and sphincter control. Consequently, patients may present with diverse symptoms, including urgency, frequency, incontinence [5], retention, and recurrent urinary tract infections. From a pathophysiological standpoint, upper motor neuron lesions often result in detrusor overactivity and detrusor-sphincter dyssynergia (DSD)—a condition in which the bladder contracts against a non-relaxing sphincter, leading to high intravesical pressures and an increased risk of upper

urinary tract damage. In contrast, lower motor neuron lesions are typically associated with detrusor areflexia and impaired sphincter tone, contributing to urinary retention and overflow incontinence. These contrasting patterns underscore the necessity of accurate diagnostic differentiation between UMN and LMN lesions to guide appropriate intervention. Urodynamic studies (UDS) serve as a gold standard in the diagnostic evaluation of neurogenic bladder. Through the measurement of parameters such as maximum cystometric capacity, bladder compliance, detrusor pressure, and urethral closure pressure, clinicians and laboratory specialists can quantitatively assess lower urinary tract function[6]. Urodynamic data provide objective insights into detrusor contractility, bladder storage capability, and sphincter coordination—parameters that are vital not only for diagnosis but also for prognostic evaluation and therapeutic decision-making. Despite the recognized clinical utility of urodynamic studies, there is a paucity of region-specific data, particularly in low-resource settings like Iraq, where neuro-urological services are limited. Furthermore, few studies have systematically explored the correlation between urodynamic profiles and neurological lesion types in patients with neurogenic bladder [7]. A deeper understanding of this correlation is essential to improve diagnostic accuracy, optimize management strategies, and prevent long-term complications such as renal impairment, bladder wall fibrosis, and recurrent infections[8]. Therefore, the present study was designed to investigate the urodynamic characteristics of neurogenic bladder in patients with confirmed upper or lower motor neuron lesions[9]. By analyzing urodynamic parameters such as bladder capacity, compliance, and

sphincter function, and correlating these findings with underlying neurological diagnoses, we aim to provide pathophysiological insights that support more targeted and effective diagnostic and therapeutic approaches. This work contributes to bridging the gap between neuroscience, urology, and clinical laboratory medicine, with an emphasis on diagnostic pathology as it relates to functional bladder disorders.

Urodynamic Evaluation

Various components of Urodynamic Studies can be elucidated separately together with other components. It is not uncommon to have occasional discrepancies in urodynamic terms' meanings [10]. In this context, all the terms that are used in this discussion are in accordance with the definitions in the current guidelines stated by the International Continence. In general, UDS are classified into non-invasive and invasive tests. Non-invasive studies are chosen initially for assessment as they are characterized by being readily accessible, inexpensive, and with low related morbidity [11]. Non-Invasive urodynamic tests include: Uroflowmetry and surface electrodes EMG [12]. Invasive Urodynamic Tests include Filling cystometrography (CMG) [13], Pressure flow studies (PFS) [14], Videourodynamics (VUDS) [14], and Electromyography (EMG) [15].

Urodynamic Findings in Upper and lower Motor Neuron Lesions

There was a considerable relationship between the anatomical place of lesion and the anticipated neurologic dysfunction. Insults previously mentioned pontine micturition center (PMC) were typically related with (detrusor hyperreflexia), while detrusor sphincter dyssynergia (DSD) almost always results from lesions below PMC. Blood pressure Monitoring during CMG can aid to define the presence of Autonomic Dysreflexia in such conditions which is expected at lesion above the level of sixth thoracic spine [16, 17]. Patients suffering from supracellular injuries in the spinal cord exhibit hyperreflexia and spasticity. Conversely, infraconal injuries are often linked to poorer motor neuron characteristics including areflexia and loss of muscle tone. In contrast, a thorough neurologic and urodynamic examination is necessary for cauda equina and conus lesions in order to provide an accurate diagnosis and ensure appropriate treatment [17]. Detrusor Sphincter Dyssynergia (DSD): refers to a urodynamic finding describing a state of BOO that results from the involuntary urethral sphincter activation at the detrusor muscle contractions time. Such description is used only in the presence of neuropathology. Dysfunctional voiding, on the other hand, is to be used in the absence of neuropathology [18]. Conditions that are commonly associated with DSD may include SCI, MS, spina bifida. Diagnosis reached typically by incorporating EMG, urethral pressure profiles and VCUG (when available) [19]. Overactivity of detrusors (DO) It is described as involuntary, patient-incapable detrusor contraction throughout the filling phase. When the bladder is full, CMG diagnoses DO, which may result in signs including urgency or frequency, high filling detrusor pressures, and incontinence at low storage volumes [20].

PATIENTS AND METHODS

Design and Settings

Cross-sectional research was undertaken in the Urology department at Hospitals of Nursing Home and Ghazy Al-Hariri for

Surgical Specialties. The included 43 patients selected conveniently from patients attending the urology outpatient clinic or referred from orthopaedic and neurosurgery teams with motor neuron lesion throughout the duration from October 2021 to April 2022.

Inclusions criteria

Patients were included if they had lower motor neuron disease (hypotonia and hyporeflexia) or upper motor neuron disease (Hypertonia and hyperreflexia) with either of the following:

- Patient with spinal cord injury not operated upon who has incontinence
- Patient with CVA who has incontinence
- Patient with Acute Disc prolapse who has incontinence

Exclusions criteria

- Diabetes mellitus type 1 and 2
- Patients who had operated upon
- Those with CNS diseases (Parkinson disease, MS, peripheral neuropathy)
- Multigravida women

Ethical Consideration

Before data collection, each participant was made to provide written consent after the aim of the study has been explained. Data confidentiality was guaranteed during the study and the patients were made to understand that the data collected are for research purposes only. The approval of Arab Council for Medical Specializations was obtained before conduction of the study. The Health Ethics Committee of the College of Medicine at the University of Baghdad granted ethical permission, which was registered under the number 195.

Patients evaluation and selection

The patients have been assessed through history, physical assessment and sent for investigations.

Data Collection

The subsequent data have been collected: Age, Gender, Causes of motor neuron lesion, Radio-Neurological level of the lesion (upper or lower motor neuron lesions).

Urodynamic Investigation

All tests were carried out with Uromic Samba (Med Konsult Medical Technology/Czech Republic). The operation was conducted using a 9-Fr triple lumen UDS catheter. The patient was positioned in a semi-supine posture, and a rectal catheter has been implanted into the anal canal to assess abdomen pressure. The initial infusion rate of normal saline has been set below 10% of the expected bladder capacity (EBC) per minute, with a min baseline capacity of 350 ml for patients. The administration of saline solution was stopped either once the patient reported a feeling of fullness or when a significant amount of fluid leaked from the urethra. The total bladder capacity is the maximum amount of water that is introduced into the bladder until it is filled. Involuntary Detrusor contraction marked by recording an involuntary uninhibited detrusor pressure rise between 5-10 cm H₂O. The highest urethral closure pressure is determined by subtracting the intravesical pressure from the highest urethral pressure. The bladder compliance is measured with a computerized algorithm using the

formula ($\Delta V/\Delta P_{det}$), with ΔV refers to the average volumetric capacity and ΔP_{det} refers to the detrusor pressure change or rise.

Statistical Analysis

The data analysis was conducted utilizing the Statistical Package for Social Sciences (SPSS) version 25 and Microsoft Excel 2016, utilizing descriptive statistics. The continual variables have been characterized by their mean and standard deviation (SD), and a student t-test has been utilized to see whether there was a substantial connection present.

The binomial parameters were presented using frequency and percentage were evaluated using a Chi-square test. A significance level of $P < 0.05$ has been utilized to determine statistical significance.

Results

The mean age of the patients was 48.37 ± 17.5 years (range= 15-78 years). The most common affected age group was older age 56-78 years accounting for 41.86% of the cases. Males were slightly more common than females (55.81% vs. 44.19%) as listed in table 1.

Table 1: Medical Features of the Patients (n=43)

Characteristics	Value
Age, years	
Mean $\pm$ SD	48.37 $\pm$ 17.5
Range	15-78
Age groups (years)	
15-35	13(30.23%)
36-55	12(27.91%)
56-78	18(41.86%)
Genders	
Men	24(55.81%)
Women	19(44.19%)

Types of Urinary Incontinences

The most common urinary incontinence was stress type which was more common in females than males. In contrast, overflow incontinence and urgency incontinence were more common in males than females (as listed in Table 2).

Urodynamic Parameters

Three urodynamic parameters were investigated in this study, all of which were non-normally distributed. Accordingly, they were expressed as median and range. The median maximum bladder capacity was 214 ml (range= 28-827 ml), while the median maximum urethral closure pressure was 73 cmH₂O (range= 10-321 cmH₂O). Finally, the median bladder compliance was 19 ml/ cmH₂O (range= 1.0-151 ml/ cmH₂O) as shown in table 3.

Neurological levels

In 21 patients (48.84%) there was an upper motor neuron lesion (sacro - pontine lesion in 19 patients and suprapontine lesion in 2 patients). On the other hand, lower motor neuron lesion (infrapontine lesion) was reported in 22 patients (51.16%).

Causes of Motor Neuron Lesions

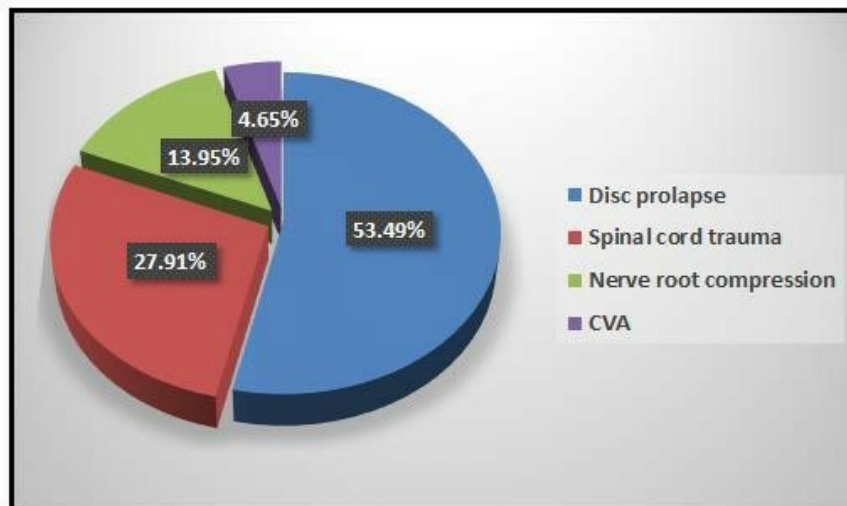
In more than half of the patients (23 patients, 53.49%), the cause of neuron motor lesion was disc prolapse. The second most common cause was spinal cord trauma encountered in 12 patients (27.91%). In six patients (13.95%), the cause of motor neuron lesion was nerve root compression. Finally, in two patients (4.65%), the cause was CVA (Figure 1).

Table 2: Types of Urinary Incontinence Based on Gender

Type of incontinence	Male	Female	Total
Stress	4 (16.6%)	9 (47.4%)	13
Overflow	10 (41.6%)	2 (10.5%)	12
Urgency	8 (33.3%)	2 (10.5%)	10
Mixed	0	6 (31.6%)	6
Urinary Retention	2 (8.3%)	0	2

Table (3): Urodynamic Parameters (n=43)

Characteristics	Value
Max. Bladder Capacity, ml	
Mean $\pm$ SD	276.05 $\pm$ 179.06
Median	214.0
Range	28-827
Max. Urethral Closure Pressure, cmH ₂ O	
Mean $\pm$ SD	93.2 $\pm$ 73.05
Median	73.0
Range	10-321
Bladder Compliance, ml/cmH ₂ O	
Mean $\pm$ SD	30.1 $\pm$ 33.32
Median	19.0
Range	1.0-151

**Figure 1:** Causes of Motor Neuron Lesions

Types of Neurogenic Voiding Disorders

The most frequent neurogenic voiding disorder was underactive bladder/underactive sphincter which was reported in 10 patients (23.26%), followed by overactive bladders/underactive sphincter and underactive bladders/overactive sphincter (each with 6 patients, 13.95%). Each of the normal bladders/underactive sphincter and underactive bladder/normal sphincter was encountered in 4 patients (9.3%). Less common neurogenic voiding disorders were normal bladders/overactive sphincter (3 patients, 6.98%), underactive

bladders/normal sphincter (2 patients, 4.65%), and finally 3 patients (6.98%) had overactive bladders/normal sphincter.

Association of Neurological Level with Urodynamic Parameters

Although the median max. bladder capacity, max. urethral closure pressure, and bladder compliance was higher in lower motor neuron lesion (289.5 ml, 92 cmH₂O and 22 ml/cmH₂O, respectively) than upper motor neuron lesion (189 ml, 71 cmH₂O and 15 ml/cmH₂O, respectively), the differences were not significant (Table 4).

Table 4: Association of Neurological Level with Urodynamic Parameters

Parameters	Upper Motor Neuron Lesion (N=21)	Lower Motor Neuron Lesion (N=22)	P Value
Max. Bladder Capacity, ml			
Median	189.0	289.5	0.520
Range	40-827	28-770	
Max. Urethral Closure Pressure, cmH ₂ O			
Median	71.0	92.0	0.193
Range	10-276	18-321	
Bladder Compliance, ml/cmH ₂ O			
Median	15.0	22.0	0.193
Range	1.0-70	1.0-151	

Association of Neurological Level with Neurogenic Voiding Disorders

Each of underactive bladder/underactive sphincter, overactive bladder/Overactive sphincter and overactive bladder/Normal sphincter were more frequent in upper neuron motor lesion (28.57%, (23.81% and 4.76%, respectively) than lower neuron motor lesion (18.18%, 4.55% and 0%, respectively) even though the differences were not significant. Normal bladder/underactive sphincter and normal bladder/overactive sphincter were reported in (22.73% and 13.64%, respectively) in patients with lower motor neuron lesion, while none of

patients with upper motor neuron lesion had such neurogenic findings (table 5).

Association of Neurological Level with Bladder and Sphincter Activity

The most notable results in the present investigation were that underactive bladder was more common in upper than lower motor neuron lesion (66.67%vs. 36.36%) with a significant difference. Otherwise, the frequencies of overactive bladder, underactive sphincter and overactive sphincter were almost similar between upper and lower motor neuron lesion with no significant difference (Table 6).

Table 5: Association of Neurological Level with Neurogenic Voiding Disorders

Neurogenic Voiding Disorders	Motor Neuron Lesion	Motor Neuron Lesion	P- Value
	Upper (N=21)	Upper (N=21)	
Underactive Bladder/Underactive Sphincter	6(28.57%)	4(18.18%)	0.221
Overactive Bladder/Overactive Sphincter	5(23.81%)	1(4.55%)	
Underactive Bladder/Overactive Sphincter	3(14.29%)	3(13.64%)	
Normal Bladder/Underactive Sphincter	0(0%)	5(22.73%)	
Overactive Bladder/Underactive Sphincter	2(9.52%)	2(9.09%)	
Underactive Bladder/Normal Sphincter	2(9.52%)	2(9.09%)	
Normal Bladder/Overactive Sphincter	0(0%)	3(13.64%)	
Underactive Bladder/Normal Sphincter	1(4.76%)	1(4.44%)	
Overactive Bladder/Normal Sphincter	2(9.52%)	1(4.76%)	

Table 6: Association of Neurological Level with Bladder and Sphincter Activity

Neurogenic Voiding Disorders	Motor Neuron Lesion		Pvalue
	Upper (N=21)	Lower (N=22)	
Underactive Bladder	14(66.67%)	8(36.36%)	0.047
Overactive Bladder	7(33.33%)	7(31.82%)	0.924
Underactive Sphincter	9(42.86%)	9(40.91%)	0.897
Overactive Sphincter	9(38.1%)	9(36.36%)	0.907

Discussion

Results of the study reveal that the neurogenic voiding disorder that was most frequent was underactive bladder/underactive sphincter and was reported in 10 patients (23.26%), followed by overactive bladder/underactive sphincter and underactive bladder/overactive sphincter (each with 6 patients, 13.95%). Each normal bladder/underactive sphincter and underactive bladder/normal sphincter was encountered in 4 patients (9.3%). Patients with traumatic or nontraumatic spinal cord injuries exhibited primarily four distinct types of neuropathic bladder dysfunction:

1. Overactivity of the detrusor and sphincter muscles
2. Sphincter and detrusor muscle hypofunction
3. The condition involves a decrease in the functioning of the detrusor muscle and an increase in the functioning of the sphincter muscle.
4. Detrusor muscle hyperactivity and sphincter muscle hypoactivity [21].

In this investigation, patients were primarily allocated to the division of detrusor and sphincter muscle overactivity. On the other hand, only a few reported a contractile detrusor with sphincter muscle overactivity. In a similar investigation, Pesce et al. [22] conducted a study on individuals who had a whole spinal cord injury or damage at the thoracolumbar vertebral junction. In total, 50% of the patients with detrusor areflexia were from the urodynamic diagnosis. Fifty percent of the patients had detrusor hyperreflexia, and, in addition, 16 out of 46 patients in the group had detrusor/sphincter dyssynergia. A total of 22 patients had vertebral lesions located above the L1 vertebra, which is where the spinal cord ends. Out of these patients, 8 individuals (36 percent) displayed detrusor areflexia, while 14 individuals demonstrated detrusor hyperreflexia. Among the latter group, 9 patients additionally experienced detrusor/sphincter dyssynergia. Out of the fifteen individuals who had a lesion affecting the L1 vertebra or a lower vertebral body, three of them (20 percent) exhibited both DO and DSD. The current investigation found no statistically substantial variations in urodynamic variables between lower and upper motor neuron injuries. According to Samson et al. [23], a bladder that is very compliant is linked to a hyporeflexive or reflexive bladder, which is frequently observed in lower motor neuron injuries. Weld and Dmochowski [24] showed that there was a greater number of individuals with inadequate bladder compliance in the group with sacral injuries. Arnold et al. [25] documented two instances of upper motor neuron lesions accompanied by detrusor areflexia. According to Wyndaele's [21] research including 92 individuals who had spinal cord damage, it was determined that relying just on clinical examination is not enough to diagnose neurological bladder conditions. Instead, urodynamic tests are necessary to thoroughly assess the functioning of various components of the lower urinary tract. It is crucial to note that the results of urodynamic investigations

might be affected by many causes such as urinary tract infections (UTIs), stones, and so on [26].

It was discovered that an underactive bladder was more prevalent in upper motor neuron lesions than the lower ones (66.67% vs. 36.36%), and this was the most crucial finding of the present study. It is known that the sacral micturition center remains intact or unaffected (parasympathetic and somatic) when the lesion includes the spinal cord directly above the sacral segments. The sacral micturition center is no longer inhibited by the pontine micturition center, and bladder overactivity results (detrusor hyperreflexia). Utmost patients, the synergistic process that enables the pelvic floor to have complete relaxation throughout a bladder reduction is also lost, which results in a state of detrusor-sphincter dyssynergia. Conversely, once the lesion affects the sacral micturition center, the normal urodynamic repetition will be of a contractile bladder (detrusor areflexia) [12]. However, in clinical practice, these statements may not be always applicable. To elucidate this variation, various theories have been suggested including:

- Another underlying lesion is present at a specific neurological level, but it is not yet showing clinical symptoms.
- The spinal segments farther away from the site of injury are affected by vascular damage caused by trauma.
- There is variation in the relationship between the spinal cord and the spinal column.
- Damage to the detrusor muscle of the bladder is caused by excessive stretching of the bladder [12].

This Investigation Confirmed the Occurrence of an 'Unexpected' Vesicosphincteric Behavior in Approximately 36.36% of the Patients.

The major limitation of this investigation is the small size of specimen. Another source of limitation is the lack of follow-up and the fate of those trauma on long term.

Conclusions

This study provides critical insights into the pathophysiological distinctions between upper and lower motor neuron lesions in patients with neurogenic bladder, as revealed by comprehensive urodynamic evaluation. The findings underscore the diagnostic value of key urodynamic parameters—including bladder compliance, urethral closure pressure, and maximum cystometric capacity—in differentiating between the functional patterns associated with each type of neurological lesion.

Patients with upper motor neuron lesions demonstrated a higher prevalence of detrusor overactivity and reduced bladder compliance, consistent with the classical pattern of detrusor-sphincter dyssynergia. In contrast, those with lower motor neuron lesions exhibited features of detrusor areflexia and underactive bladder, characterized by diminished contractility and larger residual volumes. These distinct

urodynamic profiles reinforce the notion that the nature and location of the neurological insult significantly influence lower urinary tract function and clinical presentation. Importantly, the study illustrates the indispensable role of urodynamic testing as a diagnostic tool in the context of neurogenic bladder. When integrated with neurological assessment and clinical history, urodynamic studies provide a precise, objective framework for establishing a differential diagnosis, planning individualized treatment strategies, and preventing long-term urological complications such as recurrent urinary tract infections, renal dysfunction, and bladder wall damage.

In clinical pathology and laboratory medicine, the value of functional diagnostics such as urodynamics is increasingly recognized as complementary to biochemical and histological assessments. This study adds to the growing body of evidence advocating for a multidisciplinary, laboratory-integrated approach to managing

neuro-urological disorders. Further research involving biochemical biomarkers and neuroimaging could enhance the understanding of disease progression and offer novel prognostic indicators.

Ultimately, the integration of clinical neurology, urology, and laboratory diagnostics is vital to improving patient outcomes in neurogenic bladder disorders. The findings from this study advocate for the routine inclusion of urodynamic analysis in the diagnostic algorithm for patients with suspected upper or lower motor neuron bladder dysfunction, especially in resource-limited settings where early, accurate, and cost-effective diagnosis is crucial.

Ethical Approval

The Health Ethics Committee of the College of Medicine at the University of Baghdad granted clearance, with a registration number of 195.

References

- [1] C. L. Lee *et al.*, "Sophisticated regulation of micturition: review of basic neurourology," *Journal of exercise rehabilitation*, vol. 17, no. 5, p. 295, 2021.
- [2] W. C. de Groat, D. Griffiths, and N. Yoshimura, "Neural control of the lower urinary tract," *Comprehensive Physiology*, vol. 5, no. 1, p. 327, 2015.
- [3] C. J. Fowler, D. Griffiths, and W. C. De Groat, "The neural control of micturition," *Nature Reviews Neuroscience*, vol. 9, no. 6, pp. 453-466, 2008.
- [4] D. Griffiths and A. Apostolidis, "Neurological control of the bladder in health and disease," *Pelvic Organ Dysfunction in Neurological Disease: Clinical Management and Rehabilitation*, pp. 1-39, 2010.
- [5] M. Drake, C. Fowler, D. Griffiths, E. Mayer, J. Paton, and L. Birder, "Neural control of the lower urinary and gastrointestinal tracts: supraspinal CNS mechanisms," *Neurourology and Urodynamics: Official Journal of the International Continence Society*, vol. 29, no. 1, pp. 119-127, 2010.
- [6] A. T. Prabhakar *et al.*, "Neural correlates of urinary retention in lateral medullary infarction," *International Neurourology Journal*, vol. 23, no. 3, p. 205, 2019.
- [7] D. Griffiths and C. Fowler, "The micturition switch and its forebrain influences," *Acta physiologica*, vol. 207, no. 1, pp. 93-109, 2013.
- [8] J. W. Kim, S. J. Kim, J. M. Park, Y. G. Na, and K. H. Kim, "Past, present, and future in the study of neural control of the lower urinary tract," *International Neurourology Journal*, vol. 24, no. 3, p. 191, 2020.
- [9] C. Karmonik, T. Boone, and R. Khavari, "Data-driven machine-learning quantifies differences in the voiding initiation network in neurogenic voiding dysfunction in women with multiple sclerosis," *International neurourology journal*, vol. 23, no. 3, p. 195, 2019.
- [10] S. D. Tadic, C. Tannenbaum, N. M. Resnick, and D. Griffiths, "Brain responses to bladder filling in older women without urgency incontinence," *Neurourology and urodynamics*, vol. 32, no. 5, pp. 435-440, 2013.
- [11] A. W. Partin, R. R. Dmochowski, L. R. Kavoussi, C. A. Peters, and A. J. Wein, *Campbell Walsh Wein Urology, E-Book: 3-Volume Set*. Elsevier Health Sciences, 2020.
- [12] S.-F. Chen, C.-L. Lee, and H.-C. Kuo, "Change of detrusor contractility in patients with and without bladder outlet obstruction at ten or more years of follow-up," *Scientific Reports*, vol. 9, no. 1, p. 18887, 2019.
- [13] C. A. Raptidi, N. Karandreas, C. Katsifotis, M. Benroubi, K. Petropoulou, and C. Theodorou, "A combined urodynamic and electrophysiological study of diabetic cystopathy," *Neurourology and Urodynamics: Official Journal of the International Continence Society*, vol. 25, no. 1, pp. 32-38, 2006.
- [14] B. Stuart, J. Shefner, M. D. Kelly, and M. M. Darbey, "The effects of delayed diagnosis and treatment in patients with an occult spinal dysraphism," *The Journal of urology*, vol. 154, no. 2, pp. 754-758, 1995.
- [15] J. Groen *et al.*, "Summary of European Association of Urology (EAU) guidelines on neuro-urology," *European urology*, vol. 69, no. 2, pp. 324-333, 2016.
- [16] P. Abrams *et al.*, "The standardization of terminology of lower urinary tract function: report from the standardization sub-committee of International Continence Society," in *Textbook of female urology and urogynecology*: CRC Press, 2010, pp. 1098-1108.
- [17] B. Schurch, D. M. Schmid, G. Karsenty, and A. Reitz, "Can neurologic examination predict type of detrusor sphincter-dyssynergia in patients with spinal cord injury?," *Urology*, vol. 65, no. 2, pp. 243-246, 2005.
- [18] K. V. CARLSON, S. ROME, and V. W. NITTI, "Dysfunctional voiding in women," *The Journal of urology*, vol. 165, no. 1, pp. 143-148, 2001.
- [19] I. Perikash, "Autonomic dysreflexia and detrusor-sphincter dyssynergia in spinal cord injury patients," *The Journal of Spinal Cord Medicine*, vol. 20, no. 3, pp. 365-370, 1997.
- [20] I. Perikash, "Urodynamic patterns after traumatic spinal cord injury," vol. 38, ed: Taylor & Francis, 2015, pp. 134-134.
- [21] J. Wyndaele, H. Madersbacher, and A. Kovindha, "Conservative treatment of the neuropathic bladder in spinal cord injured patients," *Spinal cord*, vol. 39, no. 6, pp. 294-300, 2001.
- [22] F. Pesce, V. Castellano, E. F. Agrò, A. Giannantonio, F. Tamburro, and G. Vespasiani, "Voiding dysfunction in patients with spinal cord lesions at the thoraco-lumbar vertebral junction," *Spinal cord*, vol. 35, no. 1, pp. 37-39, 1997.
- [23] G. Samson and D. D. Cardenas, "Neurogenic bladder in spinal cord injury," *Physical medicine and rehabilitation clinics of North America*, vol. 18, no. 2, pp. 255-274, 2007.
- [24] K. J. Weld and R. R. Dmochowski, "Association of level of injury and bladder behavior in patients with post-traumatic spinal cord injury," *Urology*, vol. 55, no. 4, pp. 490-494, 2000.
- [25] E. Arnold, A. Anthony, J. Fukui, and W. Utey, "Bladder function following spinal cord injury: a urodynamic analysis of the outcome," *British Journal of Urology*, vol. 56, no. 2, pp. 172-177, 1984.
- [26] M. Gablishvili, T. Rukhadze, V. Kuchava, G. Nemsadze, and M. Mchedlishvili, "Prognostic value of uterine leiomyosarcoma's histological grade: Clinico-pathological analysis of 22 cases with literature review," 2023.



This is an open-access article distributed under the terms of the Creative Commons Attribution License.