

Impact of Surgical Resection on Lower Urinary Tract Function in Patients with Intradural Spinal Tumors: A Prospective Case Series

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Abstract: ***Objective:** To assess the impact of surgical intervention on lower urinary tract (LUT) function in patients with intradural spinal tumors by comparing preoperative and postoperative Uroflowmetry and Urodynamic parameters. **Materials and Methods:** This prospective observational study was conducted at AIIMS Rishikesh from June 2023 to November 2024. Adult patients (≥ 18 years) with radiologically and clinically diagnosed intradural spinal cord tumors scheduled for surgery were enrolled. Exclusion criteria included pre-existing bladder/prostate pathology, pelvic metastases, or refusal of consent. Each patient underwent preoperative assessment including IPSS scoring, USG KUB, clinical neurological examination, Uroflowmetry, and Urodynamic studies (UDS). Postoperative evaluation was conducted at three months, comprising repeat uroflowmetry, and UDS. Parameters assessed included Q_{max} , voided volume, post-void residual (PVR), bladder compliance and detrusor activity. **Results:** A total of 18 patients were analysed; 77.8% were male, and 44.4% were aged 18–30 years. Preoperative urinary complaints included voiding symptoms (38.9%), storage symptoms (22.2%), and no symptoms (38.9%). Post-surgery, a significant increase in Q_{max} was observed (from 19.81 ± 10.65 to 24.21 ± 11.82 mL/sec, $p=0.018$). Although PVR reduced (from 154.81 ± 192.53 to 72.31 ± 62.14 mL) and bladder compliance decreased (from 52.02 ± 50.97 to 35.25 ± 11.83 mL/cmH₂O), these changes were not statistically significant. **Conclusion:** Surgical management of intra dural spinal tumors led to significant improvement in bladder emptying, as evidenced by increased Q_{max} . While other urodynamic parameters showed favourable trends, further long-term studies are warranted to confirm sustained functional recovery. Early identification and management of LUT dysfunction can improve postoperative outcomes and patient quality of life.*

Keywords: Bladder dysfunction, Neurosurgery, Intra dural Spinal tumors, Uroflowmetry, Urodynamic studies.

1. Introduction

Urinary continence and socially appropriate voiding are maintained by a complex interplay of neural circuits spanning the cerebral cortex, brainstem, spinal cord, and peripheral nerves. Disruption of these circuits, as observed in neurological conditions such as brain tumors and spinal cord lesions, often results in lower urinary tract symptoms (LUTS) including urgency, frequency, nocturia, and incontinence (1).

Intra-axial brain tumors, especially those involving regions like the prefrontal cortex, insula, and anterior cingulate cortex, are implicated in micturition dysfunction due to their proximity to higher centers of bladder control. Functional imaging studies, including positron emission tomography (PET) and functional MRI (fMRI), have consistently demonstrated the role of these regions in regulating bladder sensation, voluntary inhibition, and initiation of voiding (2, 3).

The bladder-filling phase activates mechanoreceptors, which send afferent signals via the sacral spinal cord to the periaqueductal gray (PAG) in the midbrain. PAG integrates sensory input and relays it to Barrington's nucleus, which, when activated, triggers parasympathetic stimulation of detrusor muscle and inhibition of urethral sphincter tone (4). However, this reflex is modulated by descending inputs from cortical regions, especially the prefrontal and anterior cingulate cortices, which suppress inappropriate voiding until socially acceptable (5, 6). Damage to these regions results in urgency or urge incontinence, highlighting the importance of descending control.

The peripheral nervous system, comprising parasympathetic

(S2–S4), sympathetic (T11–L2), and somatic (pudendal nerve) pathways, coordinates bladder and sphincter activity through neurotransmitters such as acetylcholine, norepinephrine, and others (7). Afferent fibers (A δ and C fibers) provide sensory input regarding bladder fullness and pathological stimuli (8, 9).

Clinically, any disruption from cortical to peripheral can lead to neurogenic bladder syndromes. These include sensory and motor neurogenic bladder, uninhibited or reflex bladder, and autonomous bladder, depending on the site and extent of neurological damage. Intradural spinal cord tumors frequently result in reflex or autonomous bladder patterns due to disruption of spinal reflex arcs.

Despite advances in our understanding of control of micturition, there remains a lack of comprehensive clinical studies assessing the impact of surgical resection of intradural spinal tumors on urinary function. Surgery offers potential for functional improvement through decompression or removal of mass lesions affecting neural pathways. However, documentation of changes in uroflowmetry and urodynamic patterns post-surgery is limited.

This prospective study aims to evaluate the effect of surgical intervention on LUT function in patients with intradural spinal tumors using standardized Uroflowmetry and Urodynamic testing. By comparing preoperative and postoperative urinary function, this study seeks to delineate the role of neurosurgical treatment in the management of tumor-related LUT dysfunction.

2. Materials and Methods

This prospective observational study was conducted over an

18-month period, from June 2023 to November 2024, in the Department of Neurosurgery at the All India Institute of Medical Sciences (AIIMS), Rishikesh, in collaboration with the Department of Urology. The study population comprised all consenting patients aged 18 years and above with radiologically and clinically confirmed diagnoses of intradural spinal tumors who were scheduled for surgical management during the study period. Consecutive eligible patients were enrolled. Patients younger than 18 years, had known pre-existing bladder or prostate pathology, had evidence of pelvic metastases, or declined to provide informed consent were excluded from the study.

All patients underwent a comprehensive preoperative urological evaluation. The initial workup included the International Prostate Symptom Score (IPSS); a validated questionnaire used to quantify lower urinary tract symptoms (LUTS), categorized as mild (0–7), moderate (8–19), and severe (20–35), along with a Quality-of-life assessment. Ultrasonography of kidneys, ureters, and bladder (USG KUB) was performed to identify any anatomical abnormalities. Clinical examination assessed Anal tone, Perineal sensation, Bulbocavernosus reflex, and Squeeze & Relax. Additionally, urine routine analysis, microscopy, and culture were conducted to detect any urinary tract infection or abnormal findings.

Non-invasive Uroflowmetry was employed to evaluate voiding dynamics. Key parameters included Maximum Flow rate (Qmax), categorized as normal (>15 mL/sec), equivocal (10–15 mL/sec), or obstructed (<10 mL/sec), with Voided Volumes considered adequate if greater than 150 mL. Post-void residual urine volume (PVR) was deemed normal if less than 50 mL. These metrics provided insight into bladder outlet function and overall voiding efficiency.

Urodynamic studies (UDS) were performed both preoperatively and at three months postoperatively, utilizing transurethral and rectal catheters to measure intravesical, abdominal, and detrusor pressures. Bladder compliance was considered normal if greater than 30 mL/cm H₂O. Detrusor pressure during voiding was interpreted as normal if below 40 cm H₂O, and detrusor overactivity was defined by the presence of involuntary contractions during the filling phase. UDS findings were used to identify specific dysfunctions such as Detrusor overactivity, Detrusor underactivity, Impaired bladder compliance, and Detrusor-Sphincter dyssynergia. Postoperative follow-up included repeat uroflowmetry, and urodynamic studies at three-month intervals. Histopathological examination (HPE) reports of excised tumor specimens were also reviewed and recorded to correlate with neurological and urological outcomes.

Statistical Analysis

Data analysis was conducted using IBM SPSS version 25 software. Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were expressed as percentages. For continuous variables, comparisons were made using independent t-tests. A p-value of less than 0.05 was considered statistically significant.

3. Results

In our study, majority of patients were male (77.8%) and belonged to the 18–30 age group (44.4%), followed by 31–40 years of age group in 33.3%. Regarding urinary complaints, 38.9% of patients reported voiding symptoms, 22.2% had storage symptoms, and 38.9% were asymptomatic at presentation. [Table-1]

Most patients (77.8%) had normal USG KUB findings, while a few exhibited abnormalities such as hydroureteronephrosis, parenchymal disease, or cystitis. Urine routine microscopy was normal in the majority (77.8%), with isolated cases showing glucose, blood, or pus cells. Uroflowmetry indicated that over half of the patients (55.6%) had normal urinary flow, while 44.4% exhibited various abnormal flow patterns, including retention and staccato flow. [Table-2]

Anal tone was found to be normal in 66.7% of patients, 22.2% demonstrated reduced anal tone, and 11.1% had increased anal tone. Bulbocavernosus reflex, was present in 94.4%, with only one patient showing absent reflex (5.6%). 66.7% had normal squeeze and relaxation response, 33.3% of patients had abnormal findings: 11.1% showed poor relaxation, 5.6% had poor squeeze strength, and 16.7% exhibited both poor squeeze and relaxation. [Table-3]

Among the 18 patients evaluated, 8(44.4%) reported mild symptoms (IPSS 0–7). Moderate symptoms (IPSS 8–19) were observed in 6(33.3%) of patients, and rest 4(22.2%) experienced severe symptoms (IPSS 20–35). The maximum flow rate (Qmax) showed a statistically significant improvement, increasing from 19.81±10.65 mL/sec preoperatively to 24.21±11.82 mL/sec postoperatively (p = 0.018). Although the voided volume decreased slightly from 387.09±241.15 mL to 361.06±168.93 mL, this change was not statistically significant (p=0.629), possibly reflecting patient variability or changes in bladder sensation postoperatively. The post-void residual (PVR) volume decreased notably from 154.81±192.53 mL to 72.31±62.14 mL, although this change was not statistically significant (p = 0.061). [Table-4]

In our study, Bladder compliance showed a non-significant decrease from 52.02±50.97 mL/cmH₂O preoperatively to 35.25 ± 11.83 mL/cmH₂O postoperatively (p = 0.192). Detrusor activity increased slightly from 39.85 ± 10.32 cmH₂O to 42.38 ± 16.66 cmH₂O, though this change was not statistically significant (p = 0.639). [Table-5]

4. Discussion

Lower urinary tract symptoms (LUTS) in patients with spinal cord lesions stem from disrupted neural control of bladder function. The pontine micturition center, sacral spinal cord, and connecting pathways coordinate bladder storage and emptying. Lesions impair this communication, leading to distinct symptom patterns based on location, extent, and completeness of the lesion (10-15).

Suprasacral lesions e.g., spinal cord injury, Multiple Sclerosis, transverse myelitis interrupt descending control, resulting in detrusor overactivity (hyperreflexia)-

involuntary bladder contractions during filling causing urgency, frequency, and incontinence. Loss of coordinated brainstem regulation often leads to detrusor-sphincter dyssynergia (DSD), where the external urethral sphincter contracts simultaneously with detrusor during voiding, causing outlet obstruction, incomplete emptying, and abnormally high bladder pressures. DSD is typical in suprasacral lesions (16-20).

Infrasacral lesions e.g., conus medullaris, cauda equina syndromes, peripheral nerves etc. disrupt the sacral reflex arc (18), impairing detrusor contractility and leading to detrusor areflexia (DA) or underactivity- bladder underactivity, urinary retention, and reliance on manual emptying. Symptoms may include perianal sensory loss and diminished sphincter control (21-25).

Spinal shock, following acute spinal cord injury, initially causes detrusor areflexia (acontractile bladder) and urinary retention. Over weeks, bladder function evolves, often progressing to detrusor overactivity and DSD as spinal reflexes return in suprasacral injuries (14,15,21).

Spinal cord tumors present variable LUTS (10,12), often appearing late in disease progression, with unpredictable patterns based on lesion location and type. Even incomplete injuries can cause significant dysfunction, sometimes worsening over time without detectable neurological change. Sensory pathway damage may also impair bladder sensation (21-26).

This prospective study assessed lower urinary tract (LUT) function in patients with intradural spinal cord tumors undergoing surgery. The cohort was predominantly male (77.8%) with 44.4% aged 18–30 years. The observed male-to-female ratio of 4:1 is consistent with findings by Sakakibara et al (27), who linked higher male prevalence to anatomical and hormonal factors. Similarly, Lakhotia et al (28) reported a higher incidence of LUT dysfunction among neurologically affected males in India.

Voiding symptoms were more prevalent in lumbosacral tumors, consistent with findings by Sakakibara et al (9). In current study, Schwannomas (40%) were the most common, typically affecting the thoracic and lumbosacral spine and linked to voiding dysfunction from sacral nerve root compression. Gliomas (15%) and ependymomas (10%) were less frequent but associated with severe detrusor overactivity and poor bladder compliance due to their invasive nature. Kumral et al (29) also reported a high prevalence of schwannomas causing voiding issues, while Lakhotia et al (28) noted persistent LUT symptoms in ependymoma cases despite surgery.

Preoperatively, 55% of patients reported LUT symptoms- 35% voiding and 20% storage. Postoperative symptoms persisted in 40%, with no significant improvement ($p > 0.05$), reflecting chronic neurological deficits, as noted by Sakakibara et al (9). IPSS scores indicated mild symptoms in 50%, moderate in 30%, and severe in 20% preoperatively, with minimal postoperative change. Lakhotia et al (28) also emphasized symptom variability based on tumor location and chronicity.

In current study, mean Qmax improving from 19.81 ± 10.65 to 24.21 ± 11.82 mL/sec ($p = 0.018$), consistent with Griffiths et al (30) and Khan et al (31), who highlighted Qmax as a sensitive marker of recovery. PVR decreased from 154.81 ± 192.53 to 72.31 ± 62.14 mL, though not statistically significant, echoing findings by Yamamoto et al (32) on persistent detrusor-sphincter dysfunction.

In our study, mean bladder compliance decreased from 52.02 ± 50.97 to 35.25 ± 11.83 mL/cmH₂O without statistical significance. Similar trends were noted by Sakakibara et al (9), with better outcomes in patients with shorter symptom durations, and Kumral et al (29), who reported persistent compliance issues in chronic sacral compression. Mean detrusor pressure rose slightly from 39.85 ± 10.32 to 42.38 ± 16.66 cmH₂O, without statistical significance. These findings align with Blok et al (7), who noted limited detrusor recovery due to chronic neural damage, and Sakakibara et al (9), who reported persistent detrusor overactivity in chronic spinal lesions, often requiring pharmacological management.

This study is limited by a small sample size, short three-month follow-up, tumor heterogeneity, and absence of a control group. Future studies should include larger cohorts and longer follow-up to assess long-term LUT outcomes post-surgery.

5. Conclusion

Surgical treatment for intra-dural spinal cord tumors significantly affects lower urinary tract function, with most patients showing voiding or storage symptoms. Postoperative improvements in maximal urine flow rate (Qmax) suggest better bladder emptying, though other measures showed positive trends without statistical significance. Neurological abnormalities in pelvic floor tone likely contribute to preoperative dysfunction. Early recognition, postoperative monitoring, and rehabilitation can enhance urinary outcomes and quality of life in these patients.

References

- [1] Sakakibara R, Hattori T, Tojo M, Yamanishi T, Yasuda K, Hirayama K. Micturitional disturbance in patients with cerebrovascular dementia. *J Auton Nerv Syst.* 1995 Jan;50(3):361–2.
- [2] Verne NG, Himes NC, Robinson ME, Gopinath KS, Briggs RW, Crosson B, et al. Central representation of visceral and cutaneous hypersensitivity in the irritable bowel syndrome. *Pain.* 2003 May;103(1):99–110.
- [3] Fowler CJ, Griffiths DJ. A decade of functional brain imaging applied to bladder control. *Neurourol Urodyn.* 2010 Jan;29(1):49–55.
- [4] Andrew J, Nathan PW, Spanos NC. Disturbances of Micturition and Defaecation due to Aneurysms of Anterior Communicating or Anterior Cerebral Arteries. *J Neurosurg.* 1966 Jan;24(1):1–10.
- [5] Sakakibara R, Uchida Y, Ishii K, Kazui H, Hashimoto M, Ishikawa M, et al. Correlation of right frontal hypoperfusion and urinary dysfunction in iNPH: A SPECT study. *Neurourol Urodyn.* 2012 Jan;31(1):50–5.

[6] Moga MM, Herbert H, Hurley KM, Yasui Y, Gray TS, Saper CB. Organization of cortical, basal forebrain, and hypothalamic afferents to the parabrachial nucleus in the rat. *J Comp Neurol.* 1990 May 22;295(4):624–61.

[7] Blok BFM, Holstege G. The central control of micturition and continence: implications for urology. *BJU Int.* 1999 Mar;83(S2):1–6.

[8] Griffiths D, Clarkson B, Tadic SD, Resnick NM. Brain Mechanisms Underlying Urge Incontinence and its Response to Pelvic Floor Muscle Training. *J Urol.* 2015 Sep;194(3):708–15.

[9] Sakakibara R, Yamamoto T, Sekido N, Sawai S. How brain diseases affect the lower urinary tract function? *Bladder.* 1970 Jan 1;10(1):2.

[10] Nguyen HT, Şencan A, Sencan A, Silva A et al. Urodynamic Studies are Recommended in Children With Central Nervous System Tumors Regardless of Location. *J Urol.* 2010;

[11] Panicker JN, Sèze M de, Sèze M de, Fowler CJ et al. Neurogenic lower urinary tract dysfunction. *Handb Clin Neurol.* 2013;

[12] Uchiyama T, Sakakibara R, Hattori T et al. Lower Urinary tract dysfunctions in patients with spinal cord tumors. *Neurourol Urodyn.* 2004;

[13] Wyndaele JJ. Correlation between clinical neurological data and urodynamic function in spinal cord injured patients. *Spinal Cord.* 1997;

[14] Fowler CJ, Fowler CJ, Fowler CJ. Neurological disorders of micturition and their treatment. *Brain.* 1999;

[15] Panicker ABJN. Lower Urinary Tract Dysfunction and the Nervous System. *null.* 2014;

[16] Yamanishi T, Sakakibara R, Uchiyama T, et al. Role of urodynamic studies in the diagnosis and treatment of lower urinary tract symptoms. *Urol Sci.* 2011;

[17] Kuo HC, Chen SL et al. Clinical guidelines for the diagnosis and management of neurogenic lower urinary tract dysfunction. *Tzu Chi Med J.* 2014;

[18] Burns AS, Rivas DA, Ditunno JF, et al. The management of neurogenic bladder and sexual dysfunction after spinal cord injury. *Spine.* 2001;

[19] Abrams P, Cardozo L, Fall M et al. The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. *Urology.* 2003;

[20] Mahfouz W, Afraa TA, Campeau L et al. Normal urodynamic parameters in women: part II–invasive urodynamics. *Int Urogynecology J.* 2012;

[21] Jeong SJ, Cho SY, Oh SJ. Spinal cord/brain injury and the neurogenic bladder. *Urol Clin North Am.* 2010;

[22] Fanciullacci F. Urodynamic findings with disc protrusion. *Int Urogynecology J.* 1994;

[23] Podnar S, Tršinar B, Vodušek DB. Bladder dysfunction in patients with cauda equina lesions. *Neurourol Urodyn.* 2006;

[24] Kawaguchi Y, Koike M, Kanamori M, Ishihara H et al. Clinical symptoms and surgical outcome in lumbar spinal stenosis patients with neuropathic bladder. *J Spinal Disord.* 2001;

[25] Smith AY, Woodside JR. Urodynamic evaluation of patients with spinal stenosis. *Urology.* 1988;

[26] Patki P, Woodhouse JB, Hamid R et al. Lower urinary tract dysfunction in ambulatory patients with incomplete spinal cord injury. *J Urol.* 2006;

[27] Sakakibara R, Hattori T, Yasuda K, Yamanishi T. Micturitional disturbance after acute hemispheric stroke: analysis of the lesion site by CT and MRI. *J Neurol Sci.* 1996 Apr;137(1):47–56.

[28] Lakhotia M, Pahadiya HR, Prajapati GR, Choudhary A, Gandhi R, Jangid H. A case of anterior cerebral artery A1 segment hypoplasia syndrome presenting with right lower limb monoplegia, abulia, and urinary incontinence. *J Neurosci Rural Pract.* 2016 Jan;7(01):189–91.

[29] Kumral E, Bayulkem G, Evyapan D, Yunten N. Spectrum of anterior cerebral artery territory infarction: clinical and MRI findings. *Eur J Neurol.* 2002 Nov;9(6):615–24.

[30] Griffiths D, Tadic SD. Bladder control, urgency, and urge incontinence: Evidence from functional brain imaging. *Neurourol Urodyn.* 2008 Aug;27(6):466–74.

[31] Khan Z, Starer P, Yang WC, Bhola A. Analysis of voiding disorders in patients with cerebrovascular accidents. *Urology.* 1990 Mar;35(3):265–70.

[32] Yamamoto T, Sakakibara R, Uchiyama T, et al. Lower urinary tract function in patients with pituitary adenoma compressing hypothalamus. *J Neurol Neurosurg Psychiatry.* 2005 Mar 1;76(3):390–4.

Table 1: Demographic Profile and Distribution of Urinary Symptoms Among Study Participants

Variable	Frequency	Percentage	
Age groups (Years)	18-30	8	44.4
	31-40	6	33.3
	41-50	3	16.7
	>50	1	5.6
Gender	Female	4	22.2
	Male	14	77.8
Urinary symptoms	Voiding	7	38.9
	Storage	4	22.2
	No symptoms	7	38.9

Table 2: Radiological, Urine Analysis findings in Preoperative Evaluation

Variable	Frequency	Percentage (%)
USG KUB		
Bilateral Hydroureteronephrosis HDUN (left > right) with features of cystitis	1	5.6
Bilateral raised renal echogenicity	1	5.6
Bilateral renal parenchymal disease with right nephrolithiasis	1	5.6
Trabeculated bladder with mild HDUN	1	5.6
Normal study	14	77.8
Urine R/M		
Glucose +	1	5.6
Blood cells present	1	5.6
Pus cells present	2	11.1
No abnormality	14	77.8

Table 3: Neurological Examination Findings Related to Lower Urinary Tract Function

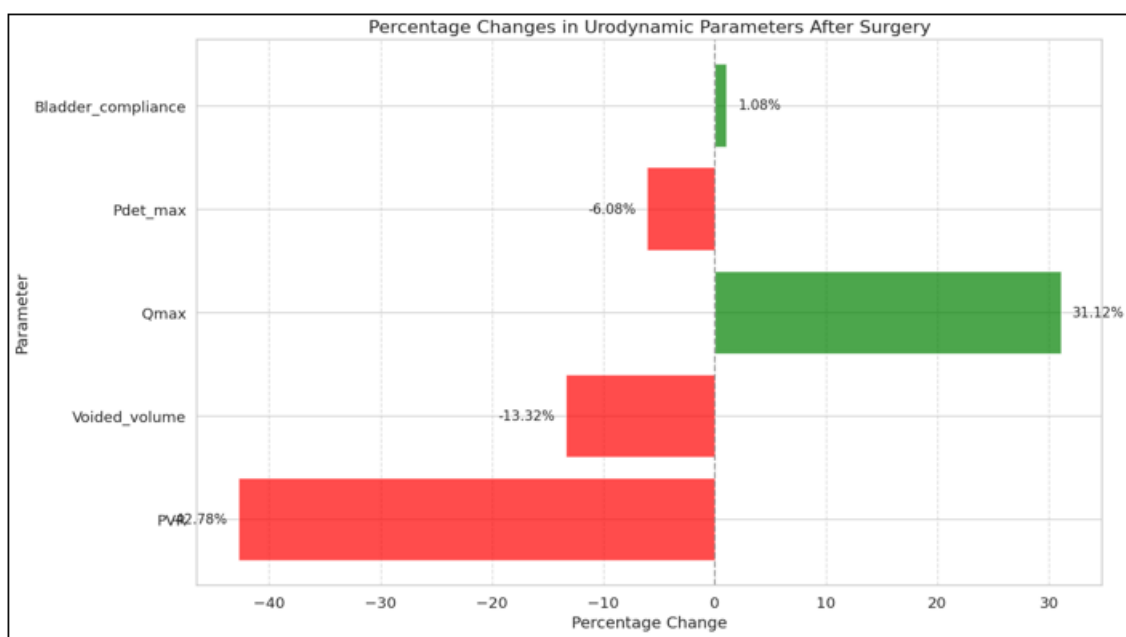
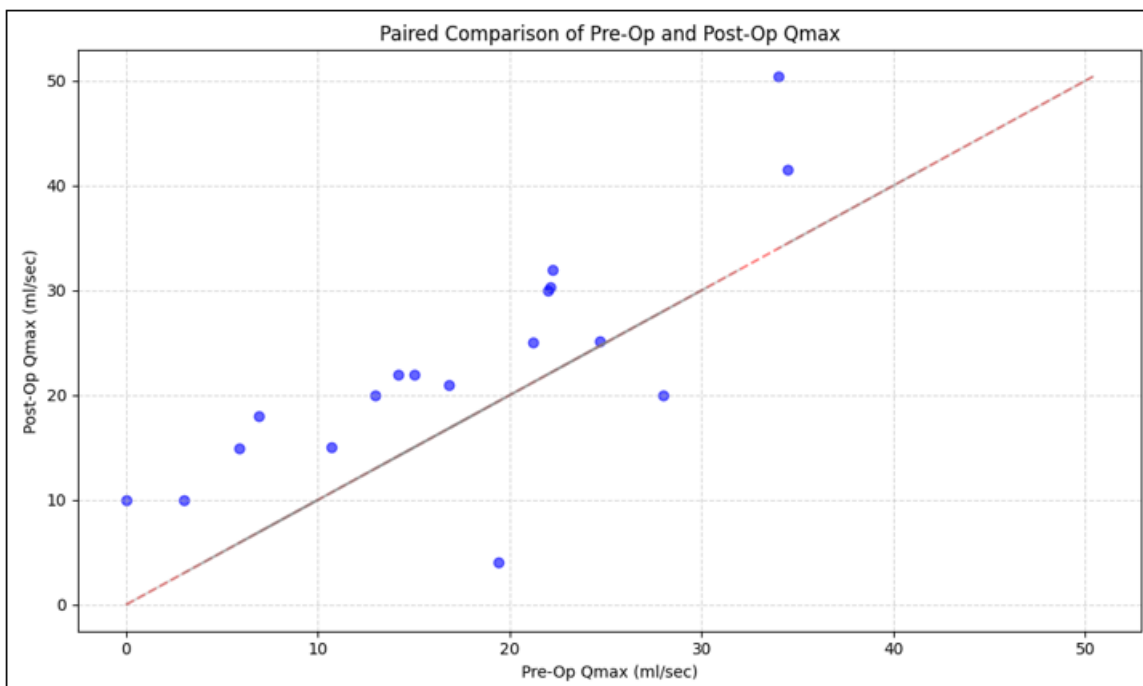
Variable		Number (n)	Percentage (%)
Anal tone	Increased	2	11.1
	Reduced	4	22.2
	Normal	12	66.7
Bulbocavernosus reflex	Absent	1	5.6
	Present	17	94.4
Squeeze and reflex	Poor relaxation	2	11.1
	Poor squeeze	1	5.6
	Poor squeeze and relaxation	3	16.7
	Normal	12	66.7

Table 4: Change in uroflowmetry parameters from pre-operative to post-operative status

Uroflowmetry	Pre-operative	Post-operative	P value
Qmax (ml/sec)	19.81±10.65	24.21±11.82	0.018
Voided volume (ml)	387.09±241.15	361.06±168.93	0.629
Post void residual volume (ml)	154.81±192.53	72.31±62.14	0.061

Table 5: Change in variable from pre-operative to post-operative

Variable	Pre-operative	Post-operative	p value
Bladder compliance (ml/cm H ₂ O)	52.02±50.97	35.25±11.83	0.192
Detrusor activity (cm H ₂ O)	39.85±10.32	42.38±16.66	0.639



Comparison of Pre-operative and Post-operative Urodynamic Parameters

