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Neurogenic Detrusor Overactivity After Spinal Cord Injury

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Case Vignette

VP is a 60-year-old man who presented to the UPMC Physical Medicine and Rehabilitation (PM&R) clinic with complaints of difficulty with ambulation. History was significant for a prior spinal cord injury from a diving accident 30 years ago with complete return of motor and sensory function within eight months of the initial injury. He reported good overall health until one year ago, when he started to notice mild right-leg weakness and spasms. Work-up by other providers led to the diagnosis of a small nonoperative syrinx in the cervical cord. Initial evaluation revealed that he also was having symptoms of urinary urgency, frequency, and difficulty initiating voiding. He sought management of his weakness and spasticity in the PM&R clinic and also agreed to evaluation for his bladder because his physiatrists were concerned about the possibility of neurogenic bladder as an explanation of his symptoms.

Definition of the Problem

Neurogenic bladder is a term which is frequently used to describe voiding dysfunction due to underlying neurologic disease. Neurogenic bladder is a common problem associated with both traumatic and nontraumatic spinal cord injuries. In North America the incidence of spinal cord injuries is thought to be approximately 12,000 new cases per year, with an estimated prevalence ranging from 300,000 to 1,275,000 persons.[1, 2] Incidence of neurogenic bladder after spinal cord injury is unknown, but is thought to be a relatively common complication.[3]

Neurogenic bladder may be defined as dysfunction of the urinary bladder resulting from an insult to the neurologic system, either by traumatic injury or by nontraumatic disease process. There are different types of neurogenic bladder, which vary by anatomical location and type of voiding dysfunction.[3, 4] Detrusor hyperreflexia, or

neurogenic detrusor overactivity (NDO), is used to describe an overactive bladder that reflexively contracts nonvoluntarily. Often NDO is associated with high bladder pressures (>40cm H_2O). Detrusor sphincter dyssynergia (DSD) describes a combination of problems, in which a bladder with nonvoluntary detrusor overactivity contracts simultaneously with the urethral sphincter to inhibit the urinary stream. Detrusor pressures >40cm H_2O are often seen with DSD.[5] Both NDO and DSD are commonly seen after upper motor neuron (UMN) injuries; thus, persons with NDO or DSD are frequently described as having UMN neurogenic bladders. Injuries affecting lower motor neurons (LMN) often manifest as detrusor underactivity, or acontracile detrusor, and thus may be referred to as a LMN neurogenic bladder.

Pathophysiology and Differential Diagnosis

Coordination of the neural pathways involved with voiding is controlled by the cerebral cortex, the pontine micturition center, and the sacral micturition center.[3] The cerebral cortex controls normal voiding, and at rest the brain inhibits urination.[6] When it is socially acceptable to void, a conscious decision to remove the baseline inhibition of bladder function allows the voiding process to begin. The pontine micturition center is responsible for coordinating the opening of the urethral sphincter with contraction of the detrusor muscle.[6, 7] At the level of the lumbosacral spinal cord, the sacral micturition center controls detrusor and sphincter musculature via input from the parasympathetic, sympathetic, and somatic nervous systems.[8] At rest, the sympathetic nervous system through hypogastric nerves (T10-L2) allows for storage of urine via relaxation of the detrusor muscle and contraction of the internal urethral sphincter.[4, 9] Parasynaptic input via the pelvic nerve (S2-4) allows for detrusor contraction for normal voiding, during which time reduction of sympathetic input allows for relaxation of the internal urethral sphincter. The external urethral sphincter, under voluntary control via the pudendal nerve (S3-5) also is relaxed with the initiation of urination.[6, 8] Under normal conditions, urine is evacuated multiple times a day with low pressures throughout the genitourinary system.[5] Myelinated A-delta fibers and unmyelinated C-fibers carry sensory information from the bladder to the spinal cord via the pelvic and hypogastric nerves.[9] C-fibers do not respond to bladder distension in the normal bladder, but may become active after SCI.

In general, the anatomic location of a lesion to the nervous system will determine the type of neurogenic bladder dysfunction *(Table 1)*. Any disruption to the cerebral cortex may result in bladder dysfunction. Supraspinal lesions which occur above the pontine micturition center may result from stroke, brain injury, or other lesions of the cortex.[6] Typical voiding patterns at this level present as frequency and incontinence, which is due to neurogenic detrusor overactivity. NDO is characterized by frequent nonvoluntary detrusor contractions.

TABLE 1: Neurogenic Bladder Classification Based on Anatomic Location

Anatomic Location	*Type of Neurogenic Bladder*
Cerebral cortex *(above the pontine micturition center)*	UMN bladder with NDO
Spinal cord *(between the pontine and sacral micturition centers)*	UMN bladder with NDO and DSD
Sacrum *(at or below the sacral micturition center or cauda equina)*	LMN with DU

UMN: upper motor neuron; NDO: neurogenic detrusor overactivity; DSD: detrusor sphincter dyssynergia; LMN: lower motor neuron; DU: detrusor underactivity

Lesions of the spinal cord typically occur between the pontine and sacral micturition centers and are a result of traumatic injury, nontraumatic myelopathy, infection, cancer, inflammation, or multiple sclerosis.[6] These spinal lesions often present with incontinence, frequency, and incomplete emptying, which results in NDO with DSD.[3] Untreated UMN neurogenic bladder either due to NDO alone or NDO with DSD may lead to a high pressure urinary bladder. High pressure systems (as defined by detrusor pressures greater than 40cm H_2O during filling), may lead to secondary complications such as hydronephrosis, vesicouretral reflux, and renal failure.[5]

Sacral lesions occur at the level of the peripheral innervations of the bladder, and are seen in cauda equina syndrome, myelodysplasia, and in peripheral nerve lesions due to other causes, such as diabetes mellitus.[6] Sacral lesions lead to LMN neurogenic bladder, which presents with reduced sensation of bladder fullness with overflow incontinence. Symptoms are due to detrusor underactivity or areflexia, which is defined as complete lack of detrusor function. The sphincter may be competent or flaccid when there is detrusor areflexia.[4] Regardless of the level of spinal cord injury, most bladders are flaccid and areflexic during the initial period of spinal shock.[4, 10] It is important to remember lesion location does not always define the type of bladder dysfunction. For example, lesions at the conus medullaris may result in a mixed picture.[9] Partial innervation, or dysfunction which is due to a combination of pathologic conditions, may result in exceptions to the above classifications.[6] The duration of injury also may contribute to the type of neurogenic bladder.[10] When considering neurogenic bladder as a cause for voiding dysfunction, other causes of pathology must also be considered in the differential diagnosis *(Table 2)*.

TABLE 2: Differential Diagnosis for Causes of Neurogenic Bladder

Supraspinal	✓ Cerebrovascular accident ✓ Parkinson's disease ✓ Brain injury (traumatic or nontraumatic) ✓ Hydrocephalus
Spinal Cord	✓ Traumatic spinal cord injury ✓ Nontraumatic spinal cord injury ✓ Congenital neural tube defects ✓ Vertebral disk protrusion ✓ Fractures of the spine or pelvis
Systemic	✓ Multiple sclerosis ✓ Diabetes mellitus
Infectious	✓ Neurosyphilis ✓ Herpes zoster ✓ Lyme disease
Mechanical	✓ Urethral outlet obstruction ✓ Urethral hypermobility

Evaluation and Work-up

The initial evaluation for neurogenic bladder requires a thorough history and physical examination. A comprehensive history is required to help establish the type of neurogenic bladder dysfunction and to outline symptoms in need of treatment *(Table 3)*. A voiding diary kept by the patient may be useful and includes data on episodes of incontinence, triggers for incontinence, bladder sensation, method of bladder emptying, and post-void residuals.[8] The physical exam should focus on the neurologic and genitourinary systems, with particular attention paid to the motor, sensory, and reflex function at the S4-5 segments.[6] Rectal tone, voluntary rectal contraction, presence of bulbocavernosus reflex, and light touch and pinprick sensory testing at S4-5 should be included. Baseline laboratory studies may include urinalysis, urine cytology, and urine culture to evaluate for urinary tract infection or carcinoma. Blood work to assess renal function is imperative, keeping in mind that serum creatinine will likely be lower than considered normal due to muscle wasting.[11] A 24-hour serum creatinine and calculation of creatinine clearance also may be useful.

Evaluation of the upper tracts of the genitourinary system is often completed with renal scans, renal ultrasound, computer tomography, or intravenous pyelogram.[3,11] Lower tract studies include cystoscopy, cystograms and urodynamics. Evaluation may also be enhanced by referral to a urologist familiar with neurogenic bladder. The decision of which tests should be ordered must be individualized to the patient based on his or her clinical history, as all available tests are not needed in every clinical situation.

Interpretation of Urodynamics

Urodynamic studies (UDS) allow for direct assessment of urinary bladder and urethral sphincter function. It is recommended that all individuals with SCI have evaluation with UDS.[8] Although anatomical location of a neurologic lesion might suggest which type of neurogenic bladder would be expected, UDS often identifies an unexpected type of neurogenic bladder.[12,13] Therefore, UDS is helpful to diagnose neurogenic bladder, establish a baseline for a patient's voiding dysfunction, and to help design treatment plans that might prevent secondary complications.[14]

Accurate interpretation of UDS is important to the evaluation of neurogenic bladder; thus, a consistent format for data collection has been identified.[8,14,15] The range of normal values for UDS testing may vary by setup, technician, patient age, and position.[14] The typical setup for UDS involves the insertion of small catheters into the bladder and rectum (or vagina), and the placement of electromyography (EMG) pads near the external anal sphincter. Each of the catheters is able to record pressure from the surrounding environment. Abdominal pressure (P_{abd}) as measured by the catheter in the rectum is subtracted from vesicular pressure (P_{ves}) as measured by the bladder catheter to determine the true detrusor pressure (P_{det}).[14] Contraction or relaxation of the external anal sphincter as measured by the EMG pads is used to estimate activity of the urethral sphincter.

Routine data collected during UDS should include information related to bladder sensation and capacity, detrusor function and pressure, postvoid residual volume, bladder compliance, and urethral function. Bladder sensation as detected by the patient during filling cystometry is classified as normal, increased, reduced, absent, or nonspecific.[15]

TABLE 3: Important History to Consider in Diagnosing Neurogenic Bladder After Spinal Cord Injury

✓ Etiology and duration of spinal cord injury
✓ Level of spinal cord injury
✓ Pre-injury history of voiding dysfunction
✓ Frequency of urinary incontinence
✓ Frequency of voluntary voiding and quality of urine stream
✓ Sensations of urgency, dysuria, or incomplete emptying
✓ History of secondary complications (such as urinary tract infections)

Early first sensation of filling or early desire to void is classified as increased bladder sensation. Having diminished sensation throughout bladder filling is classified as reduced sensation. First sensation of filling, first desire to void, and strong desire to void (or urgency) are typically documented during filling.[8] In a person who can sense filling, bladder capacity is defined as the bladder volume at which time the filling has stopped and the patient desires to void.[15] The normal bladder capacity is approximately 500 ml. If the patient cannot sense filling, bladder capacity is defined as the point at which filling is terminated by the technician. If there is no sensation of filling after 600–800 ml of fluid is instilled, the technician will stop the study. If involuntary voiding (or leaking) occurs, then bladder capacity is defined as the volume at which the involuntary voiding occurs.

Detrusor function is evaluated by observing detrusor activity during both filling and emptying. The presence of nonvoluntary detrusor contractions during filling suggests neurogenic detrusor overactivity. These uninhibited low-pressure detrusor contractions can be visualized during UDS testing as nonvoluntary contractions of the detrusor muscle,

which may be spontaneous or provoked.[8] Over time, nonvoluntary contractions may generate high pressures (>40cm H_2O) in the bladder.[5, 9] After the bladder has been assessed during filling, it is observed during voluntary voiding. Delayed emptying describes a bladder that empties slower than the normal time span of 15–20 ml urine per second.[16] Post-void residual volume is the volume of urine remaining in the bladder at the end of bladder emptying, and is typically less than 50–100 ml. Incomplete emptying describes failure of the bladder to achieve complete elimination of urine, which results in an above-normal post-void residual that can lead to heightened infection risk.[8] Both delayed emptying and incomplete emptying suggest detrusor underactivity (DU).[8] Failure to empty, which is defined as a lack of contraction during UDS, suggests acontractile detrusor.[8, 15, 17] In addition to observing detrusor function during voiding, pressures are documented. During voiding, detrusor pressures are expected to rise as the muscle contracts. However, pressures greater than 50–80 cm H_2O for men and greater than 40–65 cm H_2O for women would be considered abnormally high.[18]

Bladder compliance is determined by observing the relationship between bladder volume and detrusor pressure.[15] Compliance by definition is the change in volume over the change in pressure; thus in a bladder with normal compliance, low pressures are maintained as the volume of urine increases during filling. As long as pressures do not increase more than 15 cm H_2O during filling, a bladder would be considered to have normal compliance. Finally, urethral sphincter function is evaluated by observing function during filling and voiding. In the presence of a competent urethral sphincter, EMG activity will increase during UDS filling as the sphincter tightens to prevent leaking. During voiding, urethral relaxation is manifested by absence of EMG activity. In cases of DSD, EMG activity will increase at the same time the detrusor contracts.[15] Persons with an incompetent urethral sphincter will leak urine in the absence of a detrusor contraction.[8]

Treatment Options

Treatment of neurogenic bladder is important to reduce secondary complications such as hydroureter, hydronephrosis, recurrent urinary tract infections, renal or bladder stones, and renal dysfunction. It is thought that prognosis for survival after SCI improves with adequate management of neurogenic bladder.[3] There are multiple components to management of neurogenic bladder. Options for management in persons unable to void on their own include use of indwelling foley, clean intermittent catheterization, Credé maneuver, and reflex voiding. The choice of bladder management is individualized to patient ability and preference.

Pharmacotherapy is the typical choice for initial medical management *(Table 4)*. The pharmacologic treatment of choice is determined by the type of dysfunction identified. Anticholinergic medications

TABLE 4: Common Medications Used to Treat Neurogenic Bladder

Drug Class	*Indication*	*Adverse Effects*
Anticholinergic agents	NDO	Dry mouth, blurred vision, confusion, constipation
a-1-adrenergic receptor blocker	DSD	Dizziness, drowsiness, hypotension, weakness
Botulinum toxin	NDO, DSD	Pelvic pain, hematuria, transient urinary retention
Vanilloid compounds	NDO	Pain, autonomic dysreflexia

NDO: neurogenic detrusor overactivity; DSD: detrusor sphincter dyssynergia

reduce detrusor overactivity and are thought to be the first-line treatment for NDO.[6, 9] Anticholinergic medications work by blocking the action of acetylcholine at muscarinic receptors of detrusor smooth muscle, thereby causing relaxation of the bladder. Alpha-blockers such as tamsulosin help to decrease urethral pressures and facilitate emptying by blocking alpha-1 receptors located throughout the urethra.[19] Alpha-blockers may be helpful to reduce outlet obstruction, to facilitate normal voiding, and to treat symptoms of DSD.[9] In addition to oral medications, local bladder instillations may be considered in treatment programs. Treatment of NDO with bladder instillation of vanilloids such as capsaicin or resiniferatoxin has been shown to be efficacious.[20] Vanilloids are believed to inhibit C-fiber nonvoluntary detrusor contractions that are important in disease but quiescent in normals.[6]

When medical management of neurogenic bladder fails or does not meet the voiding goals of the patient, interventional procedures or surgical options are often utilized. The use of botulinum toxin via cystoscopy is an option to treat both NDO and DSD.[6] Injection of botulinum toxin into the detrusor muscle has been shown to reduce detrusor hyperreflexia by inhibiting acetylcholine action in detrusor smooth muscle.[21] Injection of botulinum toxin into the external urethral sphincter has been shown to reduce symptoms of DSD by inhibiting skeletal muscle contraction via inhibition of acetylcholine release at the neuromuscular junction.[22]

Surgical procedures may help to restore bladder function after SCI and reduce risk for secondary complications of neurogenic bladder. Sphincterotomy in men treats DSD while maintaining upper tract function and is an option for men who are willing to wear a condom catheter because the procedure often causes constant leakage of urine.[23] It may be performed via cystoscopy as a transurethral sphincterotomy (TURS) procedure. However, complications include stricture, erectile dysfunction, bleeding, and need for revision. Urethral stent placement is an alternative to TURS. Complications to stenting include stent migration, stone encrustation, urethral trauma, and possible need to replace stent.[3] Bladder augmentation via cystoplasty utilizes intestinal segments to surgically increase bladder capacity.[3] Cystoplasty is beneficial in persons with NDO, with a small contracted bladder, and recurrent episodes of incontinence. By increasing bladder compliance, problematic high pressures are reduced. Complications of bladder augmentation include gastrointestinal side effects, persistent urine leakage, bladder perforation, vitamin deficiencies, mucus secretion, and bladder stones.[3] The use of functional electrical stimulation (FES) to allow for voiding involves surgical implantation of electrodes onto the sacral nerves following a dorsal rhizotomy.[3, 24] An external device is triggered when voiding is desired. Bladder FES can be used to help treat NDO, DSD, and DU.[9] Additional surgical treatment options include surgical creation of sacral micturition reflex[25], urethral sphincter prosthesis[26], and urinary diversions.

Case Vignette Outcome

VP presented for scheduled genitourinary testing. Routine blood work showed no abnormalities. Cystography showed a hypertrabeculated bladder

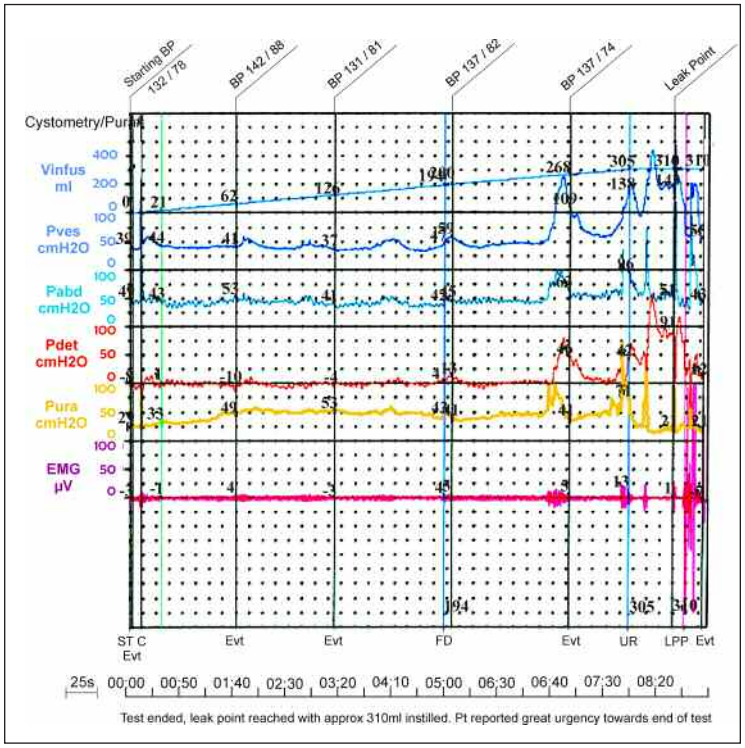

Figure 1

with bilateral diverticula, suggestive of longstanding neurogenic bladder. There was no evidence of vesicoureteral reflux. Renal ultrasound was negative for hydronephrosis or hydroureter. Urodynamics *(Figure 1)* suggested the presence of neurogenic detrusor overactivity and mild detrusor sphincter dyssnergia. He had a sense of urgency with only 300 ml of water instilled, with a leak point noted at 310 ml. He had mild elevation in detrusor pressures with filling, and was noted to have both nonvoluntary bladder contractions and mild sphincter contractions. He was able to void voluntarily with no post-void residual volume noted. After review of studies, he was started on low-dose oxybutynin for neurogenic detrusor overactivity with DSD. He was referred to urology for prostate evaluation. Two months later, VP returned to clinic for follow-up. Evaluation by urology had been negative for underlying prostate hypertrophy or other nonneurogenic pathology. He reported that his previous symptoms of urgency and frequency had resolved with current management. His systemic spasticity and leg weakness also had improved after physical therapy and treatment of spasticity.

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