Identifying Patients with High-Risk Neurogenic Bladder

Beyond Detrusor Leak Point Pressure

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KEYWORDS

• Neurogenic bladder • Spinal cord injury • Urodynamics

KEY POINTS

- The standard workup of neurogenic bladder dysfunction should include patient-based assessments, such as validated questionnaires and 2- to 3-day voiding diaries, a thorough history and physical examination, and baseline urodynamics for patients with spinal cord injury (SCI) and spina bifida. Urodynamic studies (UDS) should be repeated every 1 to 2 years in patients with recent SCI and at regular intervals for children with spina bifida. In adult patients with spina bifida whose linear growth is complete and for patients with chronic SCI whose urinary symptoms have stabilized, UDS should be repeated if urologic symptoms change. In individuals with multiple sclerosis, a history of cerebral vascular accidents, movement disorders, and diabetes, urodynamics may be used more judiciously to help define bladder function if conservative measures fail.
- Nephrolithiasis and bladder stones are more common in patients with SCI and spina bifida than in the general population. Renal and bladder ultrasound every 1 to 2 years should be used both to screen for upper tract changes and evaluate for stone formation.
- Patients with neurogenic bladder should not be treated with antibiotics for bacteriuria alone. Intervention should be prompted by leukocyturia and bacteriuria combined with symptoms of urinary infection, systemic infection, or significant changes in urologic symptoms only.
- Patients managed with indwelling Foley catheters are at increased risk for multiple urologic complications, and alternative bladder management strategies should be pursued.

INTRODUCTION

More than 12,000 spinal cord injuries (SCI) occur annually in the United States, and more than 80% of these individuals will experience urinary tract dysfunction.[1] When combined with the population of patients who have neurogenic bladder as a result of cerebrovascular accidents (CVA), spina bifida, Parkinson disease (PD), multiple sclerosis (MS), and diabetic cystopathy, the number of people with neurologic illness who require urologic care is significant. Despite the clinical demand, there is a paucity of guidelines for the care of these patients. Although most urologists are familiar with dangerous urodynamic parameters and the benefits of clean intermittent catheterization (CIC), these patients face a myriad of risks that may go unrecognized. In this article, the authors briefly discuss the impact of specific neurologic diseases on the urinary tract; examine the risks inherent to

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each; and determine strategies for screening, prevention, and treatment of these complications using the available data.

To understand the presentation of high-risk neurogenic bladder, it is important to understand the cause of neurogenic bladder dysfunction (Fig. 1). Bladder function relies on parasympathetic, sympathetic, and somatic innervation. Conscious control of voiding depends on the complex interplay between the central and peripheral nervous system. In brief, parasympathetic nerves, originating in the S2-S4 region of the spinal cord and synapsing in the pelvic ganglia, have a net excitatory effect on the lower urinary tract.[2] Bladder contraction primarily results from release of acetylcholine and stimulation of the M3 receptors in the detrusor muscle. Although acetylcholine is the primary neurotransmitter involved in parasympathetic stimulation of the bladder, purinergic receptor stimulation via adenosine triphosphate (ATP) may also contribute to detrusor muscle contraction. In the urethra, parasympathetic stimulation results in production of nitric oxide and relaxation of the urethral smooth muscle to allow for physiologic voiding.

Preganglionic sympathetic nerves arise from T11-T12 and course through the paravertebral sympathetic chain to the inferior mesenteric ganglia where they synapse. Postganglionic sympathetic nerves then travel via the hypogastric nerve to their termination on the detrusor and bladder neck/proximal urethra. Via release of norepinephrine, sympathetic innervation has an inhibitory effect on voiding. β3-receptor stimulation in the detrusor muscle prompts relaxation, whereas α-receptor stimulation of the bladder neck and proximal urethra prompts smooth muscle contraction. In the absence of neurologic insult, the external sphincter is innervated by the somatic nervous system and under voluntary control. Motor neurons originating in the ventral anterior horn of the S2-S4 region of the spinal column (Onuf nucleus) synapse in the pelvic plexus and course via the pudendal nerve to the urethra, activating nicotinic receptors and stimulating contraction of the striated or external urethral sphincter. Somatic innervation from this area of the spinal cord is also responsible for pelvic floor muscle innervation, contributing to urinary continence.

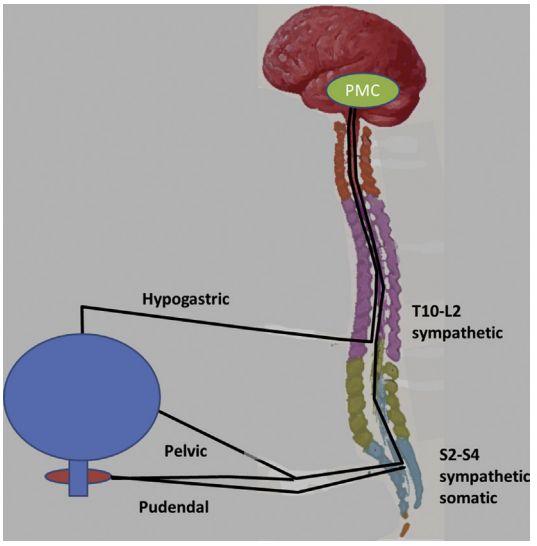

Fig. 1. Neurologic control of micturition. PMC, pontine micturition center.

Afferent nerves within the suburothelial layer of the bladder transmit information regarding bladder distension (myelinated Aδ fibers) and noxious stimuli (unmyelinated C fibers) via the dorsal root ganglia of the thoracic and sacral spine. Although Aδ fibers modulate the sensation of filling in the neurologically intact adult, studies have shown that C-fiber afferent activity may be responsible for triggering reflex detrusor contractions in individuals with suprasacral SCI.[2] In addition to input from afferent nerves, numerous studies have demonstrated the sensory and signaling properties of the non-neural cells of the urothelium.[3] Urothelial cells express a variety of receptors and also release multiple signaling compounds, such as ATP, nitric oxide, and acetylcholine, in response to noxious stimuli and may be capable of triggering detrusor contractions in the absence of neural stimulation.

Central nervous system input to the lower urinary tract is similarly complex, and our understanding of the interplay between mid and hind brain voiding centers and voluntary control of voiding continues to evolve. Normal bladder function can be divided into 2 states: filling and emptying. In the absence of neurologic insult, bladder filling prompts low-level Aδ fiber firing, which prompts bladder relaxation through sympathetic stimulation and urethral smooth and striated muscle contraction through sympathetic and somatic stimulation (collectively known as the guarding reflex). This process may be aided by input from the lateral pons, in an area known as the pontine storage center. When afferent firing reaches a threshold that indicates the need to void, permissive signaling from the periaqueductal gray area (PAG) leads to stimulation of the pontine micturition center (PMC) and activation of the parasympathetic efferent pathways of the sacral cord. This activation results in contraction of the detrusor muscle, relaxation of the urethral smooth muscle, and voiding. Reflex control of voiding would lead to micturition whenever a threshold bladder