# Bladder: Function and Dysfunction

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INTRODUCTION

The bladder performs several important functions. 1) It must **store adequate volumes** of urine without significant rise in pressure and with sufficient outlet resistance to prevent leakage. 2) Bladder **emptying** requires synchronous activation of all the smooth muscle and an appropriate relaxation of the external sphincter.
The central nervous system function of controlling the lower urinary tract is a complex and involving chapter in neurourology. The micturition process can be visualized as a complex of neural circuits in the brain and spinal cord that coordinate the activity of smooth muscle in the bladder and urethra. These circuits act as **on-off switches** to alternate the lower urinary tract between two modes of operation: **storage and elimination**. Injuries or diseases of the nervous system in adults can disrupt the voluntary control of micturition, causing the re-emergence of reflex micturition and resulting in detrusor overactivity and urge incontinence. Urethral dysfunction can also be an important cause of urinary incontinence in both women and men.

1. Basic Concepts in Bladder Physiology:

The viscoelastic behavior of the bladder and urethra depends on both neuromuscular and mechanical properties. Collagen and elastin content have a profound influence on the viscoelastic properties when these tissues are subjected to stress. Also, with injury, obstruction, or denervation, collagen content increases and when collagen levels increase, **compliance falls**. Bladder compliance (C) is defined as the change in volume (V) relative to the corresponding change in intravesical pressure (P): **C = change V/change P.**

**The concept and understanding of bladder compliance is essential since a poorly compliant bladder can lead to upper tract deterioration.**

The bladder surface undergoes incredible change in size from empty to full. The percentage change is truly unmatched by any other organ in the body. The change is accommodated by both the urothelium and the bladder wall smooth muscle and connective tissue.

The bladder has a multilayer vascular plexus, and this fact has morphologic as well as functional implications. The subepithelial capillary plexus may be associated with maintenance of the barrier function of the urothelium, reducing any exposure of the detrusor smooth muscle to substances diffusing from the urine. It may also play a role in epithelial transport function and be necessary for urothelial metabolism.

2. Voiding Mechanics:

Intravesical pressure reflects the combined factors of abdominal (Pabd) and detrusor (Pdet) pressures. Therefore, Pdet = Pves - Pabd

Micturition relies on a neurally mediated detrusor contraction, causing Pdet to rise without a significant change in Pabd. During micturition, Pdet reflects outlet resistance and decrease in urethral resistance is the first urodynamic parameter detected during normal bladder contraction.

## ABNORMALITIES OF FILLING/STORAGE AND EMPTYING/VOIDING: OVERVIEW OF PATHOPHYSIOLOGY

### Bladder Overactivity

Overactivity of the bladder during filling/storage can be expressed as **phasic involuntary contractions**, as low compliance, or as a combination. Involuntary contractions are most commonly seen in association with neurologic disease or injury; however, they may be associated with increased afferent input related to inflammation or irritation of the bladder or urethral wall, bladder outlet obstruction, stress urinary incontinence, aging and idiopathic. Storage failure may also occur in the absence of overactivity because of increased afferent input from inflammation, irritation, irritative causes such as chemical, psychologic, diopathic and painful bladder syndrome (also known as“interstitial cystitis”).

### Decreased Outlet Resistance

It can result from any process that damages the innervation or structural elements of the smooth or striated sphincter, or both, or support of the bladder outlet in the female (neurologic disease or injury, surgical or other mechanical trauma, or aging). Classically, sphincteric incontinence in the female was categorized into relatively discrete entities: (1) so-called genuine stress incontinence and (2) intrinsic sphincter deficiency (ISD), originally described as type III stress incontinence (nonfunctional or very poorly functional bladder neck and proximal urethra at rest). The implication of classic ISD was that a surgical procedure designed to correct only urethral hypermobility would have a relatively high failure rate, as opposed to one designed to improve urethral coaptation and compression. **Stress- or effort-related urinary incontinence** is a symptom that arises primarily from damage to muscles, nerves, or connective tissue, or a combination, within the pelvic floor. Urethral support is important in the female, the urethra normally being supported by the action of the levator ani muscles through their connection to the endopelvic fascia of the anterior vaginal wall. Bladder neck function is likewise important, and loss of normal bladder neck closure can result in incontinence despite normal urethral support. **Urethral hypermobility** implies weakness of the pelvic floor supporting structures which during increases in intra-abdominal pressure, there is descent of the bladder neck and proximal urethra. **Urethral instability** refers to the rare phenomenon of episodic decreases in outlet pressure unrelated to increases in bladder or abdominal pressure.

The modern view is that the majority of cases of effort-related incontinence in the female involve both deficient support and ISD. It is possible to have outlet-related incontinence that is due only to ISD but not due solely to hypermobility or poor support—**some ISD must exist.**

Emptying/Voiding Failure

Decreased Bladder Contractility

**Failure of bladder contractility may result from temporary or permanent alteration in one of the neuromuscular mechanisms necessary for initiating and maintaining a normal detrusor contraction:** psychogenic, overdistention, drugs, severe infection, or fibrosis.

Outlet Overactivity or Obstruction

Pathologically increased outlet resistance is much more common in men than in women. Although it is most often secondary to **anatomic obstruction**, it may be secondary to a failure of relaxation or active contraction of the striated or smooth sphincter during bladder contraction .

The treatment of emptying failure generally consists of maneuvers to increase intravesical pressure, facilitate the micturition reflex, decrease outlet resistance, or a combination. Intermittent catheterization is an effective way to circumvent emptying failure.

## CLASSIFICATION SYSTEMS

The purpose of any classification system should be to facilitate understanding and management. A good classification should serve as intellectual shorthand and should describe the essence of a clinical situation. An ideal system for all types of voiding dysfunction would include or imply a number of factors: (1) the conclusions reached from urodynamic testing, (2) expected clinical symptoms, and (3) approximate site and type of a neurologic lesion or lack of one. **Most systems of classification for voiding dysfunction were formulated primarily to describe dysfunction secondary to neurologic disease or injury and no system is perfect.** There are several classification systems, but the detailed individual description goes beyond the purpose of this chapter.

## HISTORY AND PHYSICAL EXAMINATION

General questioning about the desire to void, frequency, urgency, hesitancy, dribbling, incontinence, nocturia, control, quality of stream and associated constipation is important. The past medical history may reveal prior urological or neurosurgical procedure, as well as neurologic disease.

The abdomen (bladder distension), lower back (spina bifida oculta), perineum, genitalia (masses) and extremities should be carefully inspected and examined. Also, the bulbocavernous reflex (S2-4) which is a contraction of the anal sphincter upon squeezing the glans penis or the clitoris, is very important.

## URODYNAMIC EVALUATION

The purpose of Urodynamics is to study the storage and emptying phases of the urinary bladder. The urodynamic armamentarium is extensive, including such simple tests as uroflowmetry and cystometry as well as more sophisticated studies such as pressure-flow studies, electrophysiologic studies, urethral pressure studies, and videourodynamic studies. The indications are mentioned on table 1.

Table-1 -- Indications and Selection of Patients for Conduction of Urodynamics

| |
|---|
| Patients in whom potential therapy may be hazardous where one would want to be sure of the correct diagnosis before instituting therapy |
| Patients with recurrent incontinence in whom surgery is planned |
| Patients with incontinence and a confusing mix of stress and urge symptoms and those with associated voiding problems |
| Patients with neurologic disorders and those with a mismatch between symptoms and clinical findings |
| Patients with symptoms suggestive of bladder outlet obstruction |
| Patients with persistent symptoms despite presumed appropriate therapy |
| Patients with symptoms who have both obstructive and marked instability symptoms |
| Patients with obstructive symptoms and neurologic disease |
| Young men with suggestive symptoms |
| All neurologically impaired patients who have neurogenic bladder dysfunction |
| Children with daytime urgency and urge incontinence |
| Children with persistent diurnal enuresis |
| Children with spinal dysraphism |

## NEUROLOGIC DISEASES CAUSING VOIDING DYSFUNCTION

Neurologic lesions can affect the filling/storage and emptying/voiding phases of lower urinary tract function in a relatively consistent fashion. The consequences are dependent upon (1) the area(s) of the nervous system affected; (2) the physiologic function(s) and the contents and location of the area(s) affected; and (3) whether the lesion or process is destructive or

irritative. Table 2 describes some of the key points of voiding dysfunction depending on level of injury, etc.

Table 2

| **Type of Injury/Neurologic Anomaly** | **Description of Findings** |
|---|---|
| Lesions above the Brain Stem | Involuntary bladder contractions with coordinated sphincter function. Sensation and voluntary striated sphincter function are preserved but sensation may be deficient. Detrusor areflexia may be present. Urinary incontinence may occur due to the detrusor overactivity. |
| Complete lesions between T6 and S2 | Involuntary bladder contractions without sensation, smooth sphincter synergy, but striated sphincter dyssynergia. Incontinence may occur due to detrusor overactivity, but the outlet obstruction can also cause urinary retention. |
| Lesions below S2 | Detrusor areflexia is the rule after spinal shock. An open smooth sphincter area may result Various types of striated sphincter dysfunction may occur, but a residual resting sphincter tone (not under voluntary control), exists. |

Other Important Points:

1. Those with lesions above spinal cord level T6 may experience, in addition, smooth sphincter dyssynergia and **autonomic hyperreflexia,** which is a syndrome of exaggerated sympathetic activity in response to stimuli (distension of bladder or rectum) **below** the level of the lesion. The symptoms are pounding **headache, hypertension, and flushing of the face and body above the level of the lesion with sweating**. Bradycardia is a usual accompaniment, although tachycardia or arrhythmia may be present. The symptoms resolve quickly if the stimulus is withdrawn.

2. The differential diagnosis in patients presenting with decreased bladder sensation should include: Diabetes, Pernicious Anemia and Tabes Dorsalis.

3. Disk prolapse is in a posterolateral direction, which does not affect the majority of the cauda equina**.** However, in 1% to 15% of the cases , central disk prolapse occurs and compression of the cauda equina may result.

4. Non-neurogenic neurogenic bladder (Hinman syndrome), occult voiding dysfunction, occult neuropathic bladder, learned voiding dysfunction: presents the unusual circumstance of what appears urodynamically to be involuntary obstruction at the striated sphincter level existing in the **absence of demonstrable neurologic disease.**

5. Postoperative urinary retention occurs more frequently after lower urinary tract, perineal, gynecologic, and anorectal surgery. Contributing factors are described on Table 3

Table3.

1. Traumatic instrumentation
2. Bladder overdistention
3. Diminished awareness of bladder sensation
4. Decreased bladder contractility
5. Increased outlet resistance
6. Decreased micturition reflex activity
7. Nociceptive inhibitory reflex
8. Preexistent outlet pathology (e.g., BPH)

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