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Underactive bladder in women: is there any evidence?

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Abstract

Purpose of review—Underactive bladder (UAB) is a clinical symptom complex only recently gaining recognition as a clinical diagnosis. Lack of consensus agreement on a definition of UAB has limited its recognition and diagnosis in clinical practice. The purposes of this review are to: present existing definitions of UAB, review recent data regarding clinical and urodynamic diagnosis of the condition, and examine up-to-date hypotheses regarding its pathophysiology, with a focus on women.

Recent findings—The process to develop a consensus definition for UAB as a clinical symptom complex is ongoing. Symptoms associated with UAB, such as weak stream, straining to void, and history of urinary retention are well correlated to detrusor underactivity on urodynamics, which frequently develops in elderly women. In addition to aging, UAB may be the end stage of a variety of contributing pathologic conditions such as diabetes and ischemic disease. In some women, UAB may result from a progression from overactive bladder to UAB.

Summary—Existing evidence supports UAB in women as a symptom complex with a clinical and pathophysiologic profile distinguishable from other lower urinary tract-associated clinical conditions. Consensus definitions of clinical and urodynamic diagnostic parameters will be essential to more widespread recognition of UAB.

Keywords

definition; detrusor underactivity; diagnosis; pathophysiology; underactive bladder; women

INTRODUCTION

Overactive bladder (OAB) is defined by the International Continence Society (ICS) as ‘urinary urgency accompanied by frequency and nocturia, with or without urgency urinary incontinence, in the absence of a urinary tract infection or other obvious pathology [¹].’

OAB is a clinical diagnosis, not requiring urodynamic (UDS) confirmation of detrusor

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overactivity. Though the symptomatology, severity, and pathophysiology of OAB are variable, there is widespread recognition of this condition, which has been estimated to impact as many as one in six adults [2,3]. As a result, OAB has been prioritized with respect to treatment development and consensus guidelines [4,5].

In contrast, underactive bladder (UAB) is a novel concept despite detrusor underactivity being a well recognized – although difficult to define – urodynamic finding [6]. Although detrusor underactivity is defined by the 2010 ICS consensus report on terminology as a ‘detrusor contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span [1],’ no specific parameters are provided, which greatly limits the utility of this definition. Detrusor underactivity is common, identified in 12–45% of older women undergoing UDS for lower urinary tract symptoms (LUTS) [7]. However, many practitioners remain unaware of the diagnosis of detrusor underactivity or UAB. We herein aim to present the evidence related to UAB in women as a condition that is definable, commonly encountered, able to be diagnosed via clinical and invasive parameters, and characterized by pathophysiology distinguishable from other LUTS-associated conditions.

DEFINING ‘UNDERACTIVE BLADDER’

Chapple *et al.* [6] proposed the following working definition for UAB following a 2014 consensus group meeting at the International Consultation on Incontinence-Research society and ICS annual meetings: ‘The underactive bladder is a symptom complex suggestive of detrusor underactivity and is usually characterized by prolonged urination time with or without a sensation of incomplete bladder emptying, usually with hesitancy, reduced sensation on filling, and a slow stream.’ There are several important points of discussion that arise from this proposed definition. First, UAB is defined as a symptom complex, paralleling the ICS consensus definition of OAB. As with urodynamic detrusor overactivity in OAB, UAB is not wed to a diagnosis of detrusor underactivity on UDS, although detrusor underactivity may frequently be present. Second, the definition is broad: a patient with UAB may not report voiding LUTS, although, in practice, characteristic LUTS would frequently be present and bothersome enough to prompt evaluation. Thus, the concept of UAB as proposed would appear to be somewhat of a ‘catch-all’ for LUTS related to impaired bladder emptying not primarily attributable to bladder outlet obstruction (BOO). Lastly, the presence of other conditions is not excluded by this definition. For example, patients with OAB who have detrusor overactivity along with impaired contractility (i.e., detrusor hyperreflexia with impaired contractility, DHIC) would be classified as having UAB [8–10]. Furthermore, the cause and pathophysiology of the condition could be highly variable despite similar symptomatology. Though a finalized consensus definition remains a work in progress, the initial ICS panel working definition suggests UAB can be defined as a clinical diagnosis.

EPIDEMIOLOGY

Detrusor underactivity and BOO may be difficult to distinguish without a pressure-flow study. However, detrusor underactivity among women undergoing UDS may serve as a proxy for prevalence of clinical UAB as BOO has been identified in as little as 2.7% of

women undergoing UDS for LUTS [11]. Several studies to date have reported on the prevalence of detrusor underactivity in women, although primarily in an elderly population.

Resnick *et al.* [9] reported on 605 patients in a long-term care facility, of whom 245 (41%) were incontinent. The primary goal of the study was to establish UDS findings among incontinent institutionalized patients. Of the 94 patients undergoing UDS, 77 (81%) were women. In this study, detrusor underactivity was defined as 'failure to empty in the absence of an increase in abdominal pressure' and DHIC as 'involuntary detrusor contraction that emptied less than half of volume instilled.' Twenty-nine (38%) women demonstrated impaired detrusor function, with 23 (30%) demonstrating DHIC and six (8%) demonstrating detrusor under-activity. In a subsequent study of incontinent women with mean age of 87.6 years, Resnick and colleagues [12] identified DHIC in 45% of study participants, nearly one-quarter of whom had been misidentified as having stress urinary incontinence. These studies established DHIC as a common diagnosis in the elderly and identified impaired detrusor function as an important pathophysiological contributor to incontinence in elderly women.

Several subsequent studies have evaluated the prevalence of detrusor underactivity in women in the ambulatory setting [10,13,14]. Groutz *et al.* [13] interviewed 206 consecutive women with mean age 62.6 in a urogynecology practice for symptoms of 'voiding difficulties', specifically: incomplete emptying, double voiding, intermittent voiding, weak stream, hesitancy, straining to void, positional voiding, and need for prolapse reduction. Women were categorized by the presence or absence of clinical symptoms and presence or absence of impaired emptying on UDS from either BOO, acontractile detrusor, or underactive detrusor. Impaired emptying was defined by $Q_{\max} < 12$ mL/s with 100 mL voided or postvoid residual > 150 mL on two or more readings. A total of 127 women (62%) reported symptoms of voiding difficulties. Approximately 40 women (19.4%) demonstrated impaired emptying on UDS, only one of whom had demonstrable BOO. Only 27 of these 40 women (68%) were symptomatic, underscoring the correlation but not absolute relationship between the UAB symptom complex and urodynamic detrusor underactivity. Nonetheless, the prevalence of UAB symptoms was high (3 in 5 women), even in this relatively younger, ambulatory population. Failure to demonstrate impaired emptying on UDS in patients with UAB symptoms may be related to challenges in defining and demonstrating urodynamic parameters for detrusor underactivity. It may also support a role for afferent signaling abnormalities in the pathophysiology of UAB.

In a study of postmenopausal women age 55 and older, Valentini *et al.* [14] identified detrusor underactivity, defined as 'impaired detrusor contraction leading to prolonged voiding time and high residual volume,' in 62/449 (14%) consecutive patients undergoing UDS. The motive for UDS was incontinence in two-thirds of patients. Detrusor underactivity was identified in 42 (13%) and 20 (17%) women without and with neurologic disease, respectively, excluding women with spinal cord injury from the analysis.

In a similar study of women older than 70, Abarbanel and Markus [10] identified impaired detrusor contractility, defined as $P_{\det}$ at $Q_{\max}$ of < 30 cmH₂O and $Q_{\max} < 10$ mL/s, in 12/99 (12%) consecutive community-dwelling women who underwent UDS for LUTS. It is

notable that despite variable definitions for detrusor underactivity, prevalence rates were fairly similar across all three studies in the ambulatory setting [10,13,14]. In a recent population-wide cross-sectional study in Metro Detroit, 20% of the 291 women surveyed reported difficulty emptying their bladder, suggesting UAB is also prevalent outside of the clinical setting [15].

CLINICAL DIAGNOSIS

It would be especially helpful if identified clinical risk factors for UAB could facilitate diagnosis so the need for UDS would be obviated in many patients. Older age has been consistently found to be a significant predictor of detrusor underactivity on UDS [9,10,12–14,16,17]. Other identified risk factors in detrusor underactivity prevalence studies include history of urinary retention, presence of a permanent indwelling catheter [10], and possibly the presence of neurologic condition [10,14]; however, subset analyses have generally been limited because of small sample size. Fortunately, two more recent studies have shed light on potential clinical and noninvasive predictors of urodynamic detrusor underactivity [16,17].

Jeong *et al.* [16] reported a rate of detrusor under-activity, defined as $Q_{\max} < 12$ mL/s and $P_{\det}$ at $Q_{\max}$ of > 10 cmH₂O, in 13% of 547 women over age 65 who underwent UDS. The prevalence of diabetes and clinical urinary symptoms did not differ significantly in women with and without detrusor under-activity nor did postvoid residual. Mean $Q_{\max}$ was significantly lower on noninstrumented uroflow in women with detrusor underactivity (7.8 ± 7.3 versus 15.1 ± 12.6 mL/s, $P < 0.001$), although the clinical utility of this finding is limited because $Q_{\max}$ is also decreased in patients with BOO.

The authors identified BOO in 10% of women, higher than in many reported series [11,13,18], although others have reported rates of BOO of up to one-third [19,20]. Therefore, a significant minority of women may have arrived at detrusor underactivity or clinical UAB following a period of prolonged BOO or may be chronic valsalva voiders in whom BOO (e.g., Fowler's syndrome, cystocele, and iatrogenic) represents the primary disorder for impaired emptying [19,21–23]. This clinical scenario can undoubtedly present a diagnostic and therapeutic dilemma.

Gammie and colleagues [17] addressed the detrusor underactivity/BOO diagnostic challenge in a retrospective study designed to identify clinical predictors of urodynamic detrusor underactivity versus BOO and normal voiding. Detrusor under-activity was present on UDS in 308/1281 (24%) neurologically normal women without urodynamic detrusor overactivity and BOO present in 119 (9%). Detrusor underactivity was defined by the presence of: $P_{\det}$ at $Q_{\max} < 20$ cmH₂O, $Q_{\max} < 15$ mL/s, bladder voiding efficiency $< 90\%$, and absence of clinical obstruction. Women with detrusor underactivity were significantly older than those with BOO or normal UDS (median age 59 years for detrusor underactivity versus 44 for both BOO and normal study). The authors calculated odds ratios for presence of detrusor underactivity versus BOO and detrusor underactivity versus normal study. Table 1 summarizes these findings. No components of the bladder diary were significantly different for those with detrusor underactivity versus BOO.

Although this study by design excludes women with detrusor overactivity (therefore limiting applicability to women with DHIC), a prior study inclusive of only women with detrusor overactivity similarly identified a strong association between voiding LUTS and detrusor underactivity [24]. Both studies support the concept of UAB as a clinical symptom complex often associated with detrusor underactivity that is identifiable and in many cases distinguishable from BOO on history and physical and noninvasive studies. In practice, clinical suspicion is essential, particularly in the subset of patients with UAB may be asymptomatic or minimally symptomatic. For example, women with longstanding and poorly controlled diabetes are at risk for developing diabetic cystopathy, marked by impaired sensation, increased bladder capacity, and failure to empty [25].

URODYNAMIC DIAGNOSIS

When invasive UDS are required, lack of consensus regarding urodynamic definitions of detrusor underactivity presents a challenge in diagnosis. Because the ICS definition of detrusor underactivity does not provide specific parameters for its diagnosis [1], definitions are quite variable from study to study [9, 10, 12–14, 16, 17]. No one measure is perfect, and debate exists regarding whether it is best to focus on the strength, speed, or sustainability of detrusor contractility [7].

A common strategy to measure contractility involves relating P_{det} to urinary flow. Normal median values for P_{det} at Q_{max} and Q_{max} in post-menopausal women without detrusor overactivity over age 55 have been reported to be 24 (18–31) cmH₂O and 18 (16–24) mL/s, respectively [26]. However, consensus standards for clinically relevant abnormal values have not been established.

Another strategy for measuring contractility aims to obtain the isovolumetric P_{det} during a contraction while flow is occluded, thereby not losing the force used to generate flow in the contractility measurement [27, 28]. Projected isovolumetric detrusor calculated as $Q_{max} + P_{det}$ at Q_{max} has been purported to be the most practical measure for clinical practice [29]. Average values for this measure in the elderly female population in which it was developed were 50 cmH₂O with a typical range of 30–75 cmH₂O, but what constitutes a truly ‘normal’ value remains unclear [27]. Furthermore, the measure is probably not applicable to younger women and not applicable to men.

To summarize, no gold standard for measuring detrusor underactivity exists, as existing diagnostic methods and indices remain limited by: complexity (e.g., Watts factor), impracticality or patient discomfort (e.g., occlusion testing), inability to account for presence of both detrusor underactivity and BOO and/or relative lack of applicability to women (e.g., BOO index), or lack of consensus on normal values (e.g., P_{det} at Q_{max} and Q_{max} , voiding efficiency) [7, 21]. However, all of these measures may be more relevant to research than clinical practice, where numbers matter less than overall clinical impression. In many cases where the clinical diagnosis remains unclear, UDS can assist in distinguishing UAB from other LUTS-associated conditions.

PATHOPHYSIOLOGY

Although the end result may appear the same, impaired bladder emptying can arise via multiple mechanisms. The causes implicated in the development of UAB-associated LUTS and detrusor under-activity most broadly include motor and sensory dysfunction, which may arise from damage to: peripheral afferent and/or efferent pathways, the lumbosacral spinal cord, or detrusor muscle (i.e., myogenic failure) [30].

Afferent signaling is responsible for sensing urine volume during storage and strength of detrusor contraction during voiding [7]. Impaired afferent sensation, a common feature of aging and diabetic cystopathy, can limit or prematurely terminate the micturition reflex [31]. Aging is specifically associated with reduced parasympathetic response to filling both peripherally because of decreased innervation as well as centrally because of diminished response to bladder filling in the brain [32,33].

Diminished efferent signaling is most directly associated with neurogenic impairment from conditions such as multisystem atrophy but is also a common feature of the aging and chronically obstructed bladder [7,32,34]. Injury to the pelvic nerve plexus during surgery is uncommon but is another potential cause for impaired efferent signaling [30]. Reduced efferent input can contribute to or be primarily responsible for impaired contractility and reduced voiding efficiency.

Functional imaging studies suggest signaling from the insula, hypothalamus, periaqueductal grey, and pontine micturition center routed through the lumbosacral spinal cord are essential to micturition [35]. Injury to the lumbosacral cord, which disrupts these signals, is most frequently related to spinal cord injury and herniated intervertebral discs but can also be encountered with spinal stenosis, spinal dysraphism, vascular malformations, and inflammatory and neoplastic conditions [30].

Regardless of whether myogenic failure arises despite intact extrinsic neuronal pathways or because of altered neuronal pathways, decompensation of the detrusor muscle is an important cause of detrusor underactivity and UAB-related LUTS. Although it has been hypothesized that BOO results in bladder wall fibrosis and ultimately myogenic failure, long-term follow-up studies of untreated BOO in men are conflicting with respect to loss of detrusor contractility [36,37]. Furthermore, in women, chronic BOO is uncommon whereas detrusor underactivity is not. Therefore it is more likely that BOO is one potentially contributing factor to long-term bladder dysfunction, unfavorable smooth muscle remodeling, and ultimately myogenic failure [38]. Other factors hypothesized to contribute to this process include aging, denervation, ischemia, and inflammation. Although the end result may be detrusor underactivity, it itself may arise directly from decompensation following a series of insults or through progression from OAB to DHIC to UAB/detrusor underactivity [38]. Taken together, our existing fund of knowledge supports UAB and detrusor underactivity in women as a fairly distinct pathophysiologic state despite in many cases being closely related to LUTS-associated conditions such as OAB.

CONCLUSION

Although yet to be fully defined, UAB appears to be a clinical symptom complex often related to, but unique from, other chronic LUTS-associated conditions such as OAB. Detrusor underactivity is frequently present in older women with symptoms consistent with UAB, particularly weak stream, straining to void, and a history of urinary retention. Urodynamic confirmation of UAB by the presence of detrusor underactivity is hindered by a lack of gold standard measurement and consensus normal values, but can still be useful in cases where the diagnosis remains uncertain. UAB may arise from a combination of disruption in neuronal signaling to or from the bladder as well as myogenic failure. The end-stage result is a unique pathophysiologic state to which aging, ischemia, OAB, and BOO are frequent contributors.

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■ of special interest

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KEY POINTS

- UAB is an evolving clinical concept related to the urodynamic diagnosis of detrusor underactivity.
- It can be defined as a unique clinical condition; however, a consensus definition has not yet been established.
- In women, it is associated with voiding LUTS, particularly straining to void.
- UAB may be the end-stage result of multiple contributing pathophysiologic factors, such as ischemia, neurologic insults, BOO, or OAB.

Table 1

Statistically significant clinical predictors of detrusor underactivity versus bladder outlet obstruction and normal study on urodynamic

Odds of detrusor underactivity versus BOO	Odds ratio	Odds of detrusor underactivity versus normal study	Odds ratio
Signs and symptoms:		Signs and symptoms:	
Stress incontinence	1.91	Decreased urinary stream	10.8
Decreased urinary stream	1.81	Hesitancy	3.71
Enuresis	1.98	Feeling of incomplete bladder emptying	1.62
Absent or decreased sensation	8.56	Impaired mobility	1.98
		Enuresis	1.98
Medical history:		Interrupted urinary stream	10.9
Any history of retention	2.94	Absent or decreased sensation	5.8
Present use of antidepressants	4.34	Palpable bladder	3.34
Patient reported or clinician observed:		Medical history:	
Straining to void	3.13	History of urinary tract infection	1.83
Decreased sensation	5.4	Any history of retention	3.27
		History of spontaneous retention	15.2
		Acute retention after childbirth or procedure	2.08
		Present use of diuretics	1.99
		Present use of antidepressants	149
		Present use of antibiotics	3.13
		History of pelvic surgery with possible denervation	4.28
		Patient reported or clinician observed:	
		Straining to void	13.7
		Decreased sensation	5.28

Adapted with permission from [17■].