Non-neurogenic female voiding dysfunction

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Purpose of review

The pathophysiological mechanisms of female voiding phase dysfunction are poorly understood, and there are neither standard definitions nor guidelines for diagnosis and treatment. The aim of this review is to present up-to-date data and controversies associated with non-neurogenic female voiding dysfunction.

Recent findings

Conceptually, voiding phase dysfunction may have bladder or urethral causes. Bladder causes include detrusor contraction of inadequate magnitude or duration to effect bladder emptying (detrusor underactivity), or the absence of detrusor contraction (detrusor arreflexia). Urethral causes consist of bladder outlet obstruction as a result of urethral overactivity (functional obstruction), or anatomical (mechanical obstruction) pathologies. The specific prevalence and contribution of each of the above mechanisms is unknown. Furthermore, a correct and timely diagnosis may be difficult, because clinical features are very similar to those of other lower urinary tract symptoms, and diagnostic modalities are often inconclusive or even misleading. A full urodynamic evaluation is essential in making the diagnosis; however, standard urodynamic definitions are still lacking. In the following review, we will present recent findings associated with the prevalence, etiology and diagnosis of each of the different categories of female voiding phase dysfunction, and highlight new advances presented during the past year.

Summary

Further epidemiological and pathophysiological investigations are needed to evaluate the causes and main risk factors of voiding dysfunction in women. A better understanding of the pathophysiological mechanisms associated with this challenging condition may provide the possibility to use appropriate diagnostic and treatment modalities, thus avoiding unnecessary interventions.

Keywords

bladder outlet obstruction, female, urodynamics, voiding dysfunction.

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Abbreviation

LUTS lower urinary tract symptoms

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Introduction

The lower urinary tract has but two functions: the storage and timely expulsion of urine. The bladder fills with urine from the kidneys and when the urge to void is felt, micturition can be postponed until a socially convenient time. During micturition, the sphincter relaxes and the bladder contracts and empties. When the lower urinary tract fails to maintain its functions, lower urinary tract symptoms (LUTS) ensue. LUTS are further categorized according to when they occur in the micturition cycle: the storage or emptying phase. Storage symptoms include urinary frequency, urgency, urge incontinence and nocturia. Emptying symptoms consist of hesitancy, straining to void, intermittent urinary stream, poor stream, a feeling of incomplete bladder emptying and urinary retention. Most research on lower urinary tract function has previously focused on the storage phase of the micturition cycle, or the study of urinary incontinence. However, the availability and increased use of various treatment modalities, as well as new imaging techniques, have recently revived the clinical awareness and interest in female voiding phase dysfunction.

Prevalence and etiology

Data concerning the prevalence of voiding phase dysfunction in women are scarce. Previous studies [1–4] reported 2–25.5% prevalence rates among women referred for the evaluation of LUTS. The most likely reason for this wide variation in reported prevalence rates is the lack of standard definitions for the diagnosis of female voiding dysfunction.

Conceptually, voiding phase dysfunction may have bladder or urethral causes [5]. Bladder causes include detrusor contraction of inadequate magnitude or duration to effect bladder emptying (detrusor underactivity), or the absence of detrusor contraction (detrusor arreflexia). Urethral causes consist of bladder outlet obstruction as a result of urethral overactivity (functional obstruction), or anatomical (mechanical obstruction) pathologies. The term 'detrusor/external sphincter dyssynergia' describes a detrusor contraction concurrent with an involuntary contraction of the urethra. Detrusor/external sphincter dyssynergia occurs in suprasacral neurological lesions. Further discussion regarding neurological voiding dysfunction is beyond the scope of this review.

The specific prevalence and contribution of each of the above mechanisms are unknown. Previous studies [4,6,7]

reported a higher incidence of detrusor underactivity than bladder outlet obstruction among neurologically intact women referred for evaluation of persistent voiding symptoms. However, recent studies [8,9] reported up to a 23% prevalence rate of bladder outlet obstruction among women referred for the evaluation of LUTS. Among patients with bladder outlet obstruction, previous anti-incontinence surgery and severe genital prolapse are the most common etiologies, accounting for half of the cases [9].

Detrusor underactivity

Detrusor underactivity or detrusor arreflexia are common, although poorly understood, causes of female voiding dysfunction [4]. Previous studies suggested age-related deterioration in detrusor contractility. Elbadawi *et al.* [10–12] showed histological changes consistent with detrusor degeneration, as well as increased collagen content, with age. Although these degenerative changes are not necessarily associated with voiding dysfunction, clinical urodynamic studies have demonstrated age-related impaired bladder emptying. Similarly, detrusor ability to maintain a sustained contractile pressure was found to be reduced in old versus young animals [13,14]. Most recently, Pagala *et al.* [15••] reported age-related, region-specific changes in the contractile responses of the bladder. Isometric contractions of longitudinal detrusor, circular detrusor and trigone segments of young and old rats were monitored after electrical, potassium and bethanechol stimulation. Study results suggested that during aging there is (1) a decrease in muscarinic receptor-mediated activation of contraction, especially in the longitudinal detrusor; (2) an increase in collagen in the circular axis of the bladder that leads to decreased compliance and increased contractile response in the circular detrusor; and (3) decreased membrane depolarization in the trigone. These findings indicate that the effect of aging is specific to different regions and functional components of the bladder, probably as a result of changes in muscarinic receptors, collagen and depolarization.

Bladder overdistension as a result of impaired detrusor contractility may occur after pelvic surgery, labor and delivery, epidural anesthesia, anticholinergic medications, or, in elderly women, without an obvious cause. Bladder overdistention may further cause ischemic and neuropathic changes within the bladder wall, resulting in irreversible detrusor damage [16–18].

Anatomical bladder outlet obstruction

Previous anti-incontinence surgery and severe urogenital prolapse are the most common anatomical etiologies of bladder outlet obstruction, accounting for half of the cases [9]. A large meta-analysis undertaken by the American Urological Association [19] estimated the probability of temporary urinary retention lasting longer than 4 weeks at 5% after retropubic and transvaginal suspensions and 8% after sling procedures. The risk of permanent retention was estimated at less than 5% [19]. Whereas some investigators recommended using preoperative pressure flow parameters to predict women at risk of the development of postoperative voiding difficulties, others found no such correlation [20]. The later view is supported by a recent study that showed postoperative voiding dysfunction to be associated with the type of surgery, advancing age, previous vaginal bladder neck suspension, first sensation at increased bladder volume, higher postvoid residual urine volume (preoperative), and postoperative cystitis. Preoperative pressure flow studies were not found to have a predictive value for postoperative voiding difficulties [21••].

The specific pathophysiology of postoperative bladder outlet obstruction is controversial. Klutke *et al.* [22] studied pressure–flow parameters after three surgical anti-incontinence procedures: Burch colposuspension, modified Pereyra and anterior repair. Greater obstructive effects were achieved with the Burch procedure, which was also the most successful surgery. The authors concluded that the success of anti-incontinence surgery depends, at least partly, on creating obstructive voiding. In contrast, no obstructive effects were found after the pubovaginal sling procedure when the sling tension was adjusted according to the leak-point pressure [23]. Other recently published studies [24,25,26•] showed a low (less than 2.5%) incidence of prolonged postoperative urinary retention if the sling is not tied with any tension.

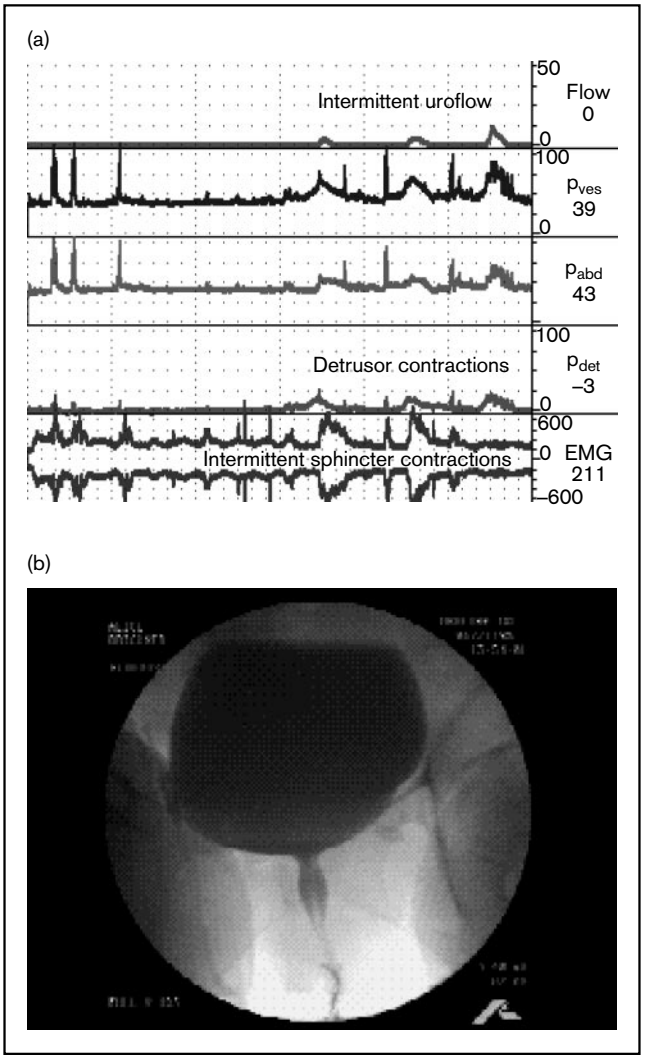
Functional bladder outlet obstruction

Normal voiding is achieved by a sustained detrusor contraction synchronized with urethral sphincter relaxation. Inappropriate sphincter activity during voiding, in the absence of known neurological disease, may result in functional bladder outlet obstruction ('non-neurogenic neurogenic bladder'). It has been suggested that this incoordination of the micturition process is caused by a learned behavioral disturbance, and may be reversed by re-educational therapy [27]. We therefore prefer the term 'acquired voiding dysfunction' to describe this condition.

As no standard definitions for acquired voiding dysfunction have been established, we have recently suggested some clinical and urodynamic diagnostic criteria [28••]. Clinical evidence of the disorder consists of (1) a suggestive clinical history (i.e. LUTS) and difficulty in voiding in public places, or during uroflowmetry/urodynamics, having to concentrate, relax, touch genitalia, listen to running water, etc.; (2) intermittent 'free' uroflow pattern; and (3) the exclusion of neurological disorders, or anatomical causes of bladder outlet

obstruction. A definitive diagnosis is made by the demonstration of typical external urethral sphincter contractions during micturition with needle electromyography or fluoroscopic visualization of the urethra during voiding. The pelvic floor–external sphincter complex can be observed to contract and relax during voiding. The urethra is usually dilated to the level of the external sphincter, whereas the bladder neck is wide open, distinguishing dysfunctional voiding from primary bladder neck obstruction. Typical urodynamic tracing and fluoroscopic visualization of the urethra are presented in Figure 1. Using these strict criteria, 2% of 1015 consecutive adults referred for video-urodynamic evaluation of LUTS were found to have acquired voiding dysfunction. Other patients, with presumed acquired voiding dysfunction, who did not undergo video-urodynamics were not included. The prevalence of acquired voiding dysfunction among adults referred for the evaluation of LUTS is thus likely to be even higher. Contrary to children, in whom the main subjective hallmarks of the syndrome are urinary incontinence and recurrent urinary tract infections, adult patients present mainly with obstructive or irritative symptoms, whereas urinary incontinence is less prominent [28••,29].

Figure 1. Acquired voiding dysfunction



(a) Typical urodynamic tracing. (b) Fluoroscopic visualization of the urethra during voiding. EMG, electromyography; p_{det}, detrusor pressure; p_{ves}, intravesicular pressure

Functional voiding dysfunction may also be associated with transient postoperative urinary retention. FitzGerald and Brubaker [30•] studied 10 women who underwent Burch colposuspension or suburethral sling surgery. Voiding trials were performed 1–2 days after surgery under simultaneous monitoring of the urethral sphincter by electromyogram activity and intravesical pressure. Six patients were unable to void and demonstrated persistent electromyogram activity. Four of these demonstrated no detrusor contraction, whereas two demonstrated detrusor contractions. The authors concluded that failure of relaxation of the striated urethral sphincter contributes to postoperative urinary retention.

Diagnosis

The best method of studying the voiding function quantitatively is by analysing detrusor pressure–uroflow parameters. However, factors associated with the technique, setting and interpretation may adversely affect the correct diagnosis. Furthermore, at present, no standard definitions exist for the diagnosis of female voiding dysfunction.

Detrusor pressure–uroflow study

The pressure–flow study is considered to be the best method to assess the voiding phase of the micturition cycle [31]. A non-invasive ('free-flow') uroflowmetry is a composite measure of the interaction between the pressure generated by the detrusor and the resistance offered by the urethra. A low uroflow may thus be caused either by bladder outlet obstruction or impaired detrusor contractility. In order to distinguish between obstruction and impaired detrusor contractility, it is necessary to measure detrusor pressure and uroflow simultaneously. Ideally, the flow pattern in a pressure–flow study should be representative of the equivalent 'free-flow' in the same patient. However, factors associated with the pressure–flow technique and setting may affect the voiding process. In particular, the use of a transurethral catheter may potentially cause urethral irritation or relative bladder outlet obstruction during the study. Data concerning the possible effects of a transurethral catheter on pressure–flow measurements in women are limited and controversial [32–36]. We have recently studied whether a 7 F transurethral catheter affects the urinary flow in women undergoing uro-

dynamic evaluation for LUTS [37]. A urodynamic database of 600 consecutive women referred for the evaluation of voiding symptoms was reviewed. Before the urodynamic evaluation, all patients voided in private using a standard toilet and the free-flow was recorded. The urodynamic study was performed using a 7 F double-lumen transurethral catheter. Only patients who voided similar volumes (varying by less than 20%) in the free-flow and pressure–flow studies were examined. Comparisons were made between free-flow and pressure–flow parameters according to voided volume categories, main urodynamic diagnoses, uroflow patterns and pre-void bladder volumes. One hundred women voided similar volumes in both free-flow and pressure–flow studies. In all but four patients, and in each of the voided volume categories, as well as each of the urodynamic diagnoses, the pressure–flow parameters were significantly different from those of the equivalent free-flow parameters. In particular, maximum flow rate values were significantly lower, whereas flow times were significantly longer, in pressure–flow versus free-flow studies. Intermittent flow pattern was more common in pressure–flow (43%) than in free-flow (9%) measurements. These data imply that a 7-F transurethral catheter adversely affects uroflow parameters in women undergoing pressure–flow studies for LUTS. This may have further clinical implications regarding the interpretation of these parameters, as well as the establishment of an accurate diagnosis.

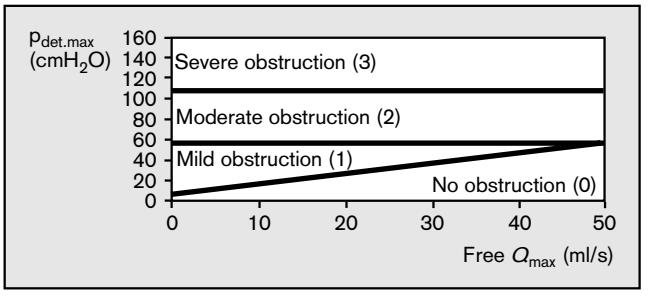
Bladder outlet obstruction

Bladder outlet obstruction nomograms on the basis of pressure–flow data are routinely used in the evaluation of obstructive uropathy in men. Three widely accepted nomograms, the Abrams–Griffiths [38], the linPURR [39] and the International Continence Society [40] nomograms, use the pressure–flow values of Q_{max} and $p_{det}.Q_{max}$ to differentiate between obstructed and unobstructed men. These nomograms are not applicable to women, because normal voiding detrusor pressure is significantly lower in women than in men.

No standard definitions exist for the diagnosis of bladder outlet obstruction in women. Recently, Lemack and Zimmern [41] analysed pressure–flow parameters of 87 'clinically obstructed' and 124 stress-incontinent women. According to their analysis, Q_{max} of 11 ml/s or less and $p_{det}.Q_{max}$ greater than 21 cmH_2O are reasonable pressure–flow parameters to identify women with bladder outlet obstruction. However, relying on a history of only obstructive symptoms for inclusion is too restrictive. Many patients with bladder outlet obstruction present with various LUTS, and correlation between obstructive symptoms and objective urodynamic findings is poor [4,9,31,42]. Moreover, strict urodynamic cut-off values will fail to diagnose patients who are unable to void with a urethral catheter in place, or those with 'normal' uroflows despite the existence of a relative obstruction. These patients may be diagnosed by using simultaneous fluoroscopic imaging of the bladder outlet during a pressure–flow study. Nitti *et al.* [8] proposed video urodynamic criteria for diagnosing bladder outlet obstruction in women. Obstruction was defined as radiographic evidence of obstruction in the presence of a sustained detrusor contraction of any magnitude. Strict pressure–flow criteria were not used. Twenty-three per cent of their patients met the radiographic criteria for bladder outlet obstruction. Obstructed cases had significantly higher voiding pressures, lower flow rates and higher post-void residual volumes than the unobstructed cases. However, in 11.8% of the patients, Q_{max} was greater than 15 ml/s and in 10.5% of the patients $p_{det}.Q_{max}$ was less than 20 cmH_2O. The authors concluded that pressure–flow studies alone may fail to diagnose obstruction, whereas the use of video urodynamic criteria facilitates the diagnosis of obstruction, even when it is not clinically suspected.

More recently, we proposed a bladder outlet obstruction nomogram for women with LUTS [43]. Two parameters were chosen to construct the nomogram: free Q_{max} and $p_{det\cdot max}$. The free Q_{max} (free-flow study) was preferred over the Q_{max} (pressure–flow study), traditionally used in male nomograms, because of the adverse effect of the transurethral catheter in women undergoing pressure–flow studies [37]. The $p_{det\cdot max}$ was preferred over the $p_{det}.Q_{max}$, used in male nomograms, because separate analysis of these parameters failed to reveal significant differences. Moreover, $p_{det}.Q_{max}$ cannot be plotted in cases of urinary retention because there is no measurable flow, whereas $p_{det\cdot max}$ during an attempt to void also enables analysis of these obstructed patients. The suggested female bladder outlet obstruction nomogram consists of four zones: no obstruction, mild, moderate and severe obstruction (Figure 2). Further analysis confirmed a positive correlation between the subjective

Figure 2. Female bladder outlet obstruction nomogram



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severity of the symptoms and the four nomogram zones. The nomogram may also serve as an instrument to assess treatment outcomes, either after potentially obstructive procedures (such as anti-incontinence surgery), or after corrective surgical interventions (such as urethrolysis). However, one should bear in mind that a nomogram should not be used to dictate treatment; rather, it should be considered as a tool to facilitate diagnosis. Specific treatment plans should be based on overall judgement, taking into consideration the clinical status and all objective findings.

Conclusion

Voiding phase dysfunction may have bladder or urethral causes. Bladder causes include detrusor underactivity or arreflexia. Urethral causes consist of functional or mechanical obstruction. The specific prevalence and contribution of each of the above mechanisms is unknown. Recent studies reported up to a 23% prevalence rate of bladder outlet obstruction among women referred for evaluation of LUTS.

The correct and timely diagnosis of voiding phase dysfunction in women may be difficult, because clinical features are very similar to those of other voiding disorders, and diagnostic modalities are often inconclusive or even misleading. A full urodynamic evaluation is essential in making the diagnosis; however, standard urodynamic definitions are still lacking. Further epidemiological and pathophysiological investigations are needed to evaluate the causes and the main risk factors of voiding dysfunction in women. A better understanding of the pathophysiological mechanisms associated with this challenging condition may provide the possibility to use appropriate diagnostic and treatment modalities, thus avoiding unnecessary interventions.

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