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How Do Urodynamics Findings Influence the Treatment of the Typical Patient With Overactive Bladder?

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Abstract Overactive bladder (OAB) is a clinical symptom complex whose hallmark is the symptom of urinary urgency, usually accompanied by frequency and nocturia, with or without urgency incontinence. Historically, urodynamics (UDS) evaluation has not been recommended in the initial evaluation of OAB, since it is defined primarily by clinical symptoms. As the pathophysiology of the OAB complex has become more clearly elucidated from recent studies, the role of UDS has again become a topic of discussion as a tool that can provide objective data to reflect these new findings. The utility of UDS in the diagnosis and treatment of OAB is still evolving, but in certain clinical scenarios, especially when empiric treatment has failed, it can provide definitive information that can identify associated pathologies and/or alter the treatment course. Herein, we will discuss the current literature regarding use of UDS in OAB patients and offer our own opinions as to its use.

Keywords Urinary bladder · Overactive bladder · Urodynamics · Hypersensitivity · Bladder outlet obstruction · Detrusor overactivity · Urgency · Lower urinary tract symptoms · Sensory · Motor · Nocturia · Classification · Lower urinary tract symptoms

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Introduction

The International Continence Society defines overactive bladder (OAB) syndrome as "urgency, with or without urge incontinence, usually with frequency and nocturia" [1]. However, a central tenet of this article is that we consider OAB a symptom complex (not a syndrome) with a differential diagnosis that can be classified by urodynamics (UDS) studies. Furthermore, although there are no clear-cut treatment guidelines that emanate from this viewpoint, we believe that the distinctions discussed herein are important vis-à-vis furthering our understanding of OAB and developing new treatments on the basis of recognizing the underlying pathophysiology detected by a combination of UDS and clinical evaluation.

OAB is a prevalent condition with 9 %–17 % of both men and women reporting symptoms, and its incidence increases with age [2]. OAB is primarily defined by symptoms, and historically, the UDS finding of involuntary detrusor contractions has served as the presumed pathophysiologic mechanism behind OAB symptoms. Early studies primarily focused on the prevailing theory that detrusor overactivity was the underlying cause of symptoms, and thus, for many years, anticholinergic pharmacotherapy was the principal modality of treatment [3]. The rationale for anticholinergic therapy relies on the prevention of binding of acetylcholine to the muscarinic postganglionic parasympathetic receptors, thereby inhibiting involuntary detrusor contractions. But such a simplistic view has been gradually challenged. Numerous mechanisms underlying OAB symptoms and pathophysiology have been proposed highlighting unique and independent pathways in both sensory (afferent) and motor (efferent) etiologies of OAB. In other words, altered function of the detrusor muscle, peripheral motor and sensory nerves, and central neural elements all contribute to the disease complex. Moreover, identical OAB symptoms are common in many lower urinary tract disorders; hence, there is a need to understand the differential

diagnosis of OAB so that it can be recognized and treated accordingly.

The significance of this new understanding is that the future treatment of OAB may be highly individualized. For successful subcategorization of patients, it is paramount to have as many clinical and objective data as possible. In addition to a thorough history, physical exam, bladder diary, uroflow (Q) and postvoid residual urine estimation (PVR), inherent to such an approach is the use of multichannel UDS. Its purpose, ideally, is to provide urologists with objective parameters that lead to a clear understanding of underlying pathophysiology and to influence clinical decision making and treatment outcomes. Historically, UDS has been limited in its impact on both clinical decision making and treatment outcomes in patients with OAB, but we believe that this is more a function of the limited treatment options available than of a lack of diagnostic acumen [4]. Moreover, UDS is often time consuming, invasive, costly, and not without associated morbidity. So what is the current role of UDS in the treatment of OAB?

There is an inherent flaw in attempting to correlate UDS findings with OAB symptomatology, because UDS cannot be measured against a "gold standard" reference test but, rather, is measured against clinical diagnosis. If UDS does not reproduce patient's symptoms, the finding is generally considered unreliable. But perhaps UDS findings and clinical symptoms represent different aspects of the disease complex. Although we agree that UDS is not necessary for the diagnosis or the initial treatment of OAB, this diagnostic study has the potential to guide clinicians in individualizing treatment and identifying other conditions associated with OAB. Furthermore, we believe that UDS provides crucial information for managing OAB even when symptoms and detrusor overactivity is not reproduced during the study. In this article, we will discuss our use of UDS in the management of OAB patients and current recommendations for the use of UDS in tailoring treatment of typical patients with OAB.

Detrusor Overactivity and Urgency With/Without Incontinence

So what is the current literature that supports the use of UDS in the treatment of typical patient with OAB? Detrusor overactivity (DO) is defined as involuntary detrusor contractions (IDCs) during the filling phase of cystometry [1]. This is diagnosed exclusively by UDS, and although the presence of DO is thought to result in OAB symptoms, the association has been difficult to establish. A study by Hashim and Abrams [5] showed that only 44 % of women with OAB without incontinence and 58 % of women with OAB with concomitant urge incontinence demonstrated DO on UDS. Of note, in the same study, the association between OAB and DO was 90 % in men with urge incontinence. Nevertheless, when DO is documented by UDS, empiric evidence suggests that treatment directed at abolishing involuntary detrusor contractions should be first-line therapy.

To further determine the role of UDS in the treatment of OAB, we should first investigate the relationship between OAB symptomatology and UDS observations. Our evaluation begins with an initial history with symptom assessment such as severity of primary symptoms, nocturia, and degree of bother. In addition, validated questionnaires such as the ICS male LUTS or the Overactive Bladder Symptom Score (OABSS) should be administered in conjunction with clinical assessment by a bladder diary and pad test. This systematic approach is important for sorting out remediable causes of OAB, such as prostatic obstruction in men and pelvic organ prolapse in women. Information obtained during video UDS (VUDS), especially during the voiding phase, is critical in distinguishing idiopathic OAB from that associated with urethral obstruction

The storage phase is equally important, as was pointed out by Flisser et al. [6], who developed a VUDS classification system for patients with OAB based on the patient's degree of awareness of bladder events, bladder control, and sphincter function. This has the potential to be used as a guide for prognosis and therapy. The authors retrospectively reviewed 132 patients who had chief complaints of urgency, frequency, and/or urge incontinence who underwent VUDS. All patients completed a 24-h voiding diary and a 24-h pad test. Patients were divided into four clinical categories during bladder storage. In type 1, there is no evidence of involuntary detrusor contractions during bladder filling (Fig. 1a, b). In type 2, there are involuntary detrusor contractions present, and the patient is aware of them and is able to contract his sphincter, interrupt the stream, and abort the detrusor contraction, preventing incontinence (Fig. 2a, b). In type 3, there are involuntary detrusor contractions present, and the patient is able to contract the sphincter and interrupt the stream, temporarily preventing incontinence; but the patient is not able to abort the detrusor contraction, and once the sphincter fatigues, the patient is incontinent (Fig. 3a–d). In type 4, there is DO, but the patient has no control at all and is immediately incontinent (Fig. 4a, b). In addition to this filling phase classification, the VUDS is essential in diagnosing (or ruling out) abnormalities of the voiding phase—urethral obstruction, impaired or absent detrusor contractility—and it is the only method that quantifies bladder compliance. Patients with type 4 OAB have DO but are unable to contract sphincter, abort the stream, or abort the detrusor contraction; they have no control whatsoever, and behavioral approaches to controlling the involuntary detrusor contractions are futile. Patients with type 2 OAB have involuntary detrusor contractions, but they can sense their

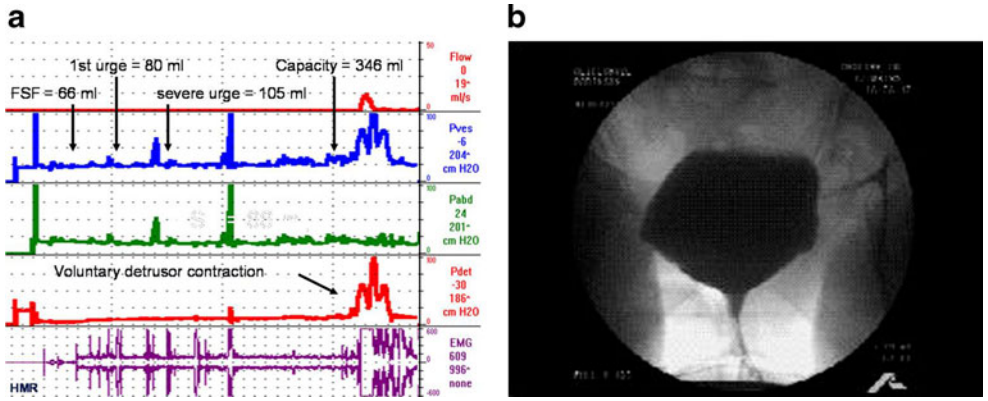

Fig. 1 **a** Type 1 overactive bladder (OAB). The patient, a 54-year-old woman, complains of OAB symptoms—urinary frequency, urgency, and urge incontinence—but the urodynamic study is normal except for a hypersensitive bladder (severe urge to void felt at 105 ml). There is no detrusor overactivity. Urodynamic tracing: FSF=66 ml, first urge=80 ml; severe urge=105 ml; bladder capacity=346 ml. There were no involuntary detrusor contractions, and she had a voluntary detrusor contraction at 346 ml and voided normally. Unintubated Qmax=20 ml/S. Voided volume=346. PVR=0 ml. Pressure flow: pdet@Qmax=25 cm H20; Qmax=14 ml/S; pdetmax=60 cm H2O. (Figure courtesy of Jerry G. Blaivas). **b** X-ray obtained during early voiding shows an irregular border to the bladder consistent with bladder trabeculations. (Figure courtesy of Jerry G. Blaivas)

onset, contract their sphincters to prevent incontinence, and abort the detrusor contraction. Hence, they are ideal candidates for a behavioral approach. In patients with type 1 OAB, there are no involuntary detrusor contractions. Of course, UDS is just a snapshot of a patient's symptomatology, and it is possible that DO is, in fact, the cause of the symptoms. However, it is also possible that it is not, and the clinician should consider other causes more strongly. Perhaps the symptoms are caused by exogenous factors, such as caffeine.

What is the predictive value of UDS in reproducing clinical findings of urinary frequency, urge urinary incontinence, and/or stress urinary incontinence? In a more recent study, Caruso et al. [7] retrospectively reviewed 537 patients (366 females and 171 males) diagnosed with frequency, urge urinary incontinence, and/or stress urinary incontinence and correlated their clinical findings with UDS findings to determine the predictive value of UDS. Frequency, as measured by setting maximum cystometric capacity (MCC) at 200 mL, did not demonstrate a significant association between a low MCC and a clinical finding of urinary frequency. Out of 278 patients with urge urinary incontinence (UUI), the sensitivity and specificity of DO in confirming the presence of UUI were 59 % and 84 %, respectively. For stress urinary incontinence (SUI), they were 45 % and 99 %, respectively. Despite the significant

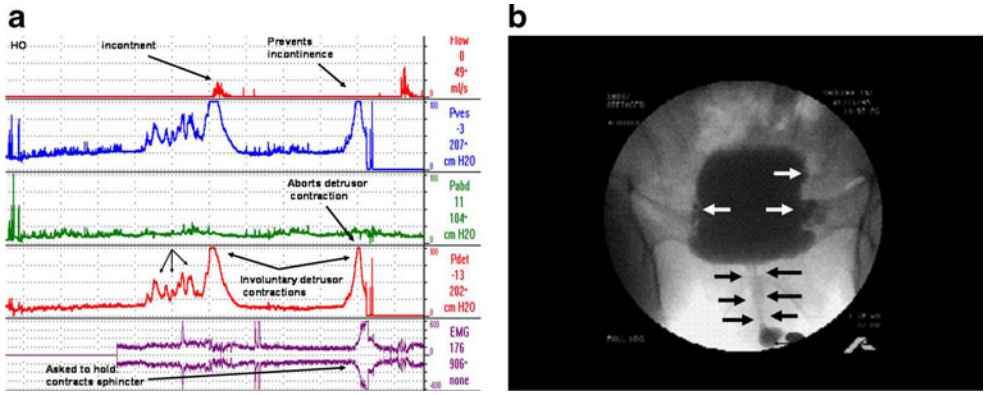

Fig. 2 **a** Type 2 overactive bladder and prostatic obstruction in 53-year-old man with a 20-year history of refractory urgency, urge incontinence, and enuresis. Urodynamic tracing: During bladder filling, he is instructed to neither void nor prevent micturition and to report his sensations to the examiner. There are a series of poorly sustained involuntary detrusor contractions (small arrows) that he perceives as a severe urge to void, and then there is a sustained voiding contraction where he relaxes his sphincter and is incontinent (pdet@Qmax=100 cm H20; Qmax=8 ml/S [Schäfer Grade 5 obstruction]). The bladder is filled again, and there is another involuntary detrusor contraction. This time he is instructed to try to hold. He contracts his sphincter, obstructing the urethra; the detrusor contraction subsides, and he is not incontinent. (Figure courtesy of Jerry G. Blaivas). **b** X-ray obtained at Qmax when he is incontinent shows a narrowed and faintly visualized prostatic urethra (black arrows) characteristic of prostatic obstruction. The bladder is trabeculated, and there are several small- and medium-sized diverticula (white arrows). (Figure courtesy of Jerry G. Blaivas)

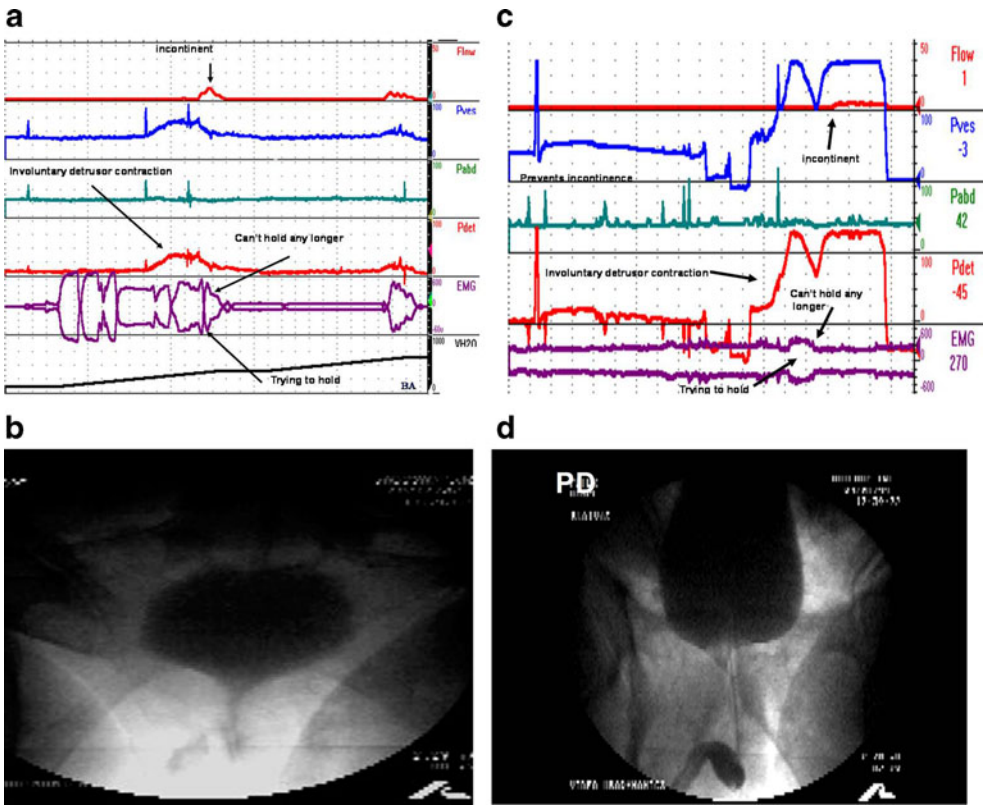

Fig. 3 **a** Type 3 overactive bladder (OAB) in a middle-aged woman with OAB symptoms of urinary frequency, urgency, and urge incontinence. Urodynamic tracing: At approximately 300 ml, patient had an involuntary detrusor contraction. The patient contracts the sphincter and successfully aborts the detrusor contraction. However, the sphincter fatigues, and the patient leaks indicating incontinence. Qmax=10 ml/S, Pdet @ Qmax=20 cm H2O (vertical line), Pdetmax=40 cm H2O. (Figure courtesy of Jerry G. Blaivas). **b** X-ray obtained at Q max shows a normal urethra. (Figure courtesy of Jerry G. Blaivas). **c** Type 3 overactive bladder (OAB) and severe prostatic obstruction (Schäfer Grade 6). The patient is a middle-aged man with severe OAB symptoms of urinary frequency, urgency, nocturia, and urge incontinence who has failed alpha adrenergic blockers and antimuscarinics. Urodynamic tracing: Patient has an involuntary detrusor contraction, which he perceives and contracts his sphincter to prevent incontinence. He temporarily prevents incontinence, and the detrusor contraction starts to abate, but once the sphincter fatigues, the detrusor contraction recurs, and he is incontinent. Qmax=4 ml/S, Pdet @ Qmax=135 cm H2O. (Figure courtesy of Jerry G. Blaivas). **d** X-ray obtained at Qmax shows a severely narrowed prostatic urethra. (Figure courtesy of Jerry G. Blaivas)

positive predictive value of DO for predicting UUI and SUI in patients with OAB symptoms, the authors concluded that UDS has a low predictive value in reproducing the clinical findings of urinary frequency and urinary incontinence. On the basis of these findings, the absence of DO and urodynamic stress incontinence does not rule out the presence of UUI or SUI. However, despite its pitfalls, UDS remains the only objective tool for confirming these clinical complaints. We have found that repeating the filling phases during UDS on select patients can increase the predictive

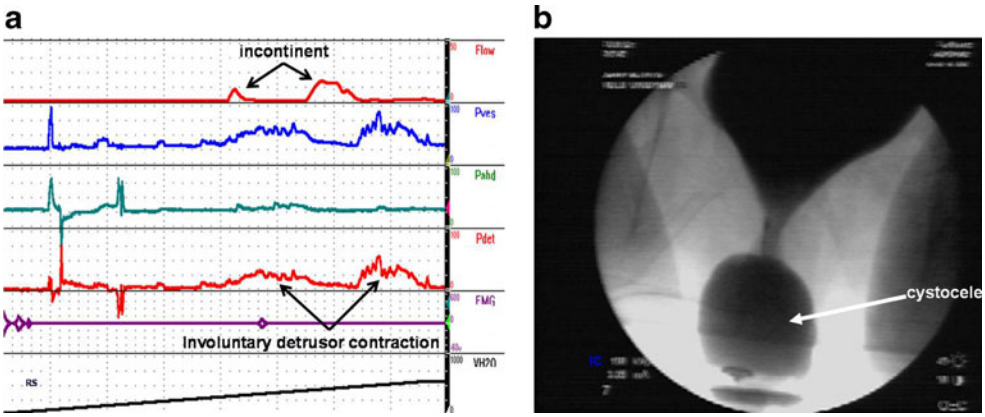

Fig. 4 **a** Type 4 overactive bladder (OAB) in an elderly woman with OAB and grade 3 prolapse. Urodynamic tracing: At approximately 200 ml, she had two involuntary detrusor contractions and was immediately incontinent. She had no voluntary control at all. Qmax=25 ml/S, Pdet @ Qmax=20 cm H2O. (Figure courtesy of Jerry G. Blaivas). **b** During voiding, the urethra is obscured by the cystocele. (Figure courtesy of Jerry G. Blaivas)

value of the study in reproducing symptoms. Having the patient cough, changing positions of catheters, running water, and hand washing are all maneuvers to aid in reducing artifacts during UDS.

What is the correlation between DO and urgency without associated incontinence? Urgency is the sine-qua-non OAB symptom, but it is difficult to measure due to its subjective nature. Although some urinary symptoms, such as frequency and nocturia, are more objectively quantifiable, it is entirely possible that DO is simply missed by standard UDS. Recent studies have reported increased diagnostic accuracy with ambulatory UDS over conventional UDS [8, 9]. Therefore, it can be a timing issue when the relationship between DO and urgency is assessed to establish cause and effect. It was initially hypothesized that the clinical sensation of urgency is the result of an involuntary detrusor contraction, but this has been shown to be not true. Lowenstein et al. [10] demonstrated this in their continuous recording of patient-reported urgency during a UDS study of 33 patients who demonstrated DO. The authors showed that substantial variability exists between the patient-reported sensations of urinary urgency and the onset of the DO episode. Moreover, episodes of reported urgency were present either before an episode of DO or after an episode of DO, and 29 % of DO episodes were not associated with an increase in urgency at all. The discordance between central and peripheral viewers also raises more doubts concerning the validity of DO in predicting OAB symptoms [11]. The presence of DO in asymptomatic patients is also another confounding factor in the use of DO as the point of initiation of anticholinergic treatment. After all, UDS is performed with a supraphysiologic fill rate using a catheter instilling fluid, and this can be a very awkward setting for many patients. Each of these can contribute to the presence of IDCs in a patient without OAB symptoms. Heslington and Hilton [12] recruited 22 asymptomatic patients and saw DO in asymptomatic patients.

But it is true that certain objective differences do exist between OAB with DO and OAB without DO, and this needs to be further elucidated. Perhaps patients who have DO have an altogether different pathology. Fan et al. [13] sought to identify the difference between patients with OAB and DO and those with OAB but without DO. They evaluated 133 patients (76 women and 57 men) using the International Prostate Symptom Score (IPSS), Overactive Bladder Symptom Score (OABSS), and UDS, and all total scores and subscores were compared. While the rest of the comparisons were similar between the two groups, the nighttime urinary frequency subscore on the OABSS differed significantly between OAB patients with DO and those without DO. Furthermore, Guralnick et al. [14•] demonstrated that despite a similar symptomatology, there are objective differences between OAB patients with and without DO. On UDS, patients with DO were more likely to have abnormal sensations, with strong desire and urgency occurring at significantly lower bladder volumes.

First Sensation/First Desire—Sensory Dysfunction

The traditional hypothesis concerning the approach of utilizing antimuscaric therapy for OAB is that the symptoms are due to DO and that one can block parasympathetic input to muscarinic receptors and, subsequently, inhibit detrusor contractility. Although the association with DO was not as clear, the mere fact that detrusor contractility could be objectively displayed further bolstered this notion in the early understanding of OAB. Over time, however, more data have been accumulating suggesting that the primary etiology of OAB in some, perhaps most, patients may be sensory in nature. To further validate this pathophysiologic model, studies have shown that antimuscarinics have been shown to affect bladder afferent neural pathways, including C fibers and A-δ fibers [15]. Finney et al. [16] reported that at clinical treatment doses, antimuscarinics have a minor impact on bladder contractility and a much greater impact on sensory parameters such as urgency, time to first sensation and void, and urinary frequency. In other words, while an afferent component likely contributes to the etiology of OAB, it could be suggested that involuntary detrusor contractions may not be the primary etiology of OAB, as has traditionally been believed. It can also be hypothesized that the association between OAB symptoms and DO may be afferent signals producing motor dysfunction.

Does the time of first desire or sensation during cystometry correlate with OAB symptoms? Rapp et al. [17] explored the hypothesis that certain sensory UDS parameters may correlate strongly with bladder sensation. The authors initially compared bladder sensation questionnaire scores and UDS variables to assess a statistical correlation, suggesting that these markers can be used to further delineate a subgroup of OAB patients with clinical symptoms of sensory dysfunction. Seventy patients were evaluated prospectively with UDS, and several questionnaires, including validated urgency (Urgency Perception Score), general overactive bladder (Urogenital Distress Inventory), and quality of life (Incontinence Impact Questionnaire) questionnaires, were used. A weak negative correlation was noted between the Urgency Perception Score and bladder capacity, while bladder urgency velocity statistically significantly correlated with Urgency Perception Score, despite the lesser or absent correlation associated with the individual components of these parameters. The lack of a significant correlation for standard bladder sensation variables such as first sensation and first desire was noted. The authors concluded that patients with identical first-urge

sensations might experience a different symptomatology, given different bladder capacities. These data suggest the relevance of this sensory response rate and the possibility that a processing defect inhibits the gradual intensification of the bladder sensation characteristic of normal patients. The authors also noted a negative correlation between bladder urge velocity and urge perception score, showing that patients with more rapid progression from first sensation through capacity had more severe symptoms.

Bladder Outlet Obstruction

Bladder outlet obstruction in both sexes is an important remediable cause of OAB symptoms. From an empiric standpoint, Qmax and PVR are essential components of the diagnostic workup. In men, a Qmax of less than 10 mL/s is utilized as a cutoff for establishing the diagnosis of bladder outlet obstruction (BOO) as per the 6th IC Guidelines [18]. Postvoid residual, although inaccurate at predicting obstruction, is also of importance for the management of male OAB, since there is a potential risk for development of acute urinary retention upon initiating antimuscarinics. Although a PVR greater than 50 mL has been correlated with increased risk to progression to acute urinary retention, we are willing to tolerate much higher volumes when empirically treating OAB patients with antimuscarinics, provided that the Q and voided volumes are reasonable [19]. In men with OAB symptoms, UDS can substantially change the treatment approach. Van Vernrooij et al. [20] reported that 50 % of men with concomitant OAB and BOO with DO had resolution of their OAB symptoms after treatment for obstruction. In fact, we believe that prostatic obstruction is the commonest cause of OAB symptoms in men and that, in the vast majority, successful treatment of the obstruction alleviates the OAB symptoms.

In men with impaired bladder contractility who are treated with antimuscarinics, there is concern about the risk of urinary retention. Ronchi et al. [21] evaluated 49 neurologically intact OAB men with a bladder contractility index (BCI) <100 on 5 mg of solifenacin once daily for 120 days. A complete UDS study was carried out on the day before the first dose of solifenacin and repeated at day 120. The parameters that decreased were Qmax, detrusor pressure at maximum flow (PdetQmax), the BOO index (BOOI), and the BCI. On the other hand, PVR and maximum cystometric capacity both increased, as would be expected. However, no significant change in subjective perception of voiding difficulties was found. The incidence of acute urinary retention was only 2.2 %, and OAB symptoms improved after solifenacin treatment. In men with LUTS/OAB and equivocal obstruction on UDS, it is unknown whether the symptomatic benefits achieved by antimuscarinics therapy outweigh the risks of urinary retention. Te et al. [22] proposed that a nomogram be used to predict the risk of urinary retention in patients with LUTS and OAB. The study included 944 consecutive men evaluated for LUTS during a 2-year period. The nomogram was based on the observation that urodynamically obstructed patients in acute retention or those who had a history of urinary retention had higher detrusor pressures at maximum flow rates and longer detrusor contraction durations, as compared with patients with symptoms of obstruction but without a history of retention. The authors found that patients with BOO and a pdet@Qmax of >79 cm/H_2O and a detrusor contraction duration of >99 s have a 50 % risk of developing urinary retention. On the basisi of this observation, the authors recommended surgical treatment to relieve BOO in men with a >50 % chance of urinary retention on UDS, rather than empiric antimuscarinic.

Many men with OAB have other coexisting urological problems [23•]. Table 1 summarizes these potential comorbidities. UDS can help stratify the patients and direct appropriate treatment to the most bothersome issue. Their data show that the most common diagnoses were benign prostatic enlargement (32 % of cases), BOO (22 %), and complications following prostate cancer treatment (20 %). Idiopathic OAB was diagnosed in only 5 % of their study population.

In women with OAB, urethral obstruction is much less common, accounting for about 10 % of cases. The commonest causes are grade 3 and 4 pelvic organ prolapse and prior incontinence surgery. Relief of the obstruction usually ameliorates the OAB symptoms. The diagnosis of bladder

Table 1 Possible coexisting medical condition with overactive bladder

Urinary retention/bladder outlet obstruction
BPH
Prostatitis
Vaginitis
Pelvic organ prolapse
Interstitial cystitis
Radiation cystitis
Dehydration
Urinary tract infection
Pelvic or abdominal surgery (APR, pelvic organ prolapse repair, radical prostatectomy, TURP)
Sexually transmitted diseases (gonorrhea, chlamydia, syphilis)
CVA
Neurologic diseases (multiple sclerosis, CVA)
Bladder stones
Kidney stones
Genitourinary cancers (bladder cancer, prostate cancer, etc.)
Urinary incontinence (stress, urge, mixed)
Diabetes
CHF

outlet obstruction in women can be made only by VUDS. The pressure flow relationship defines obstruction, and the narrowest point in the urethra during the voiding cystourethrogram defines the point of obstruction [24]. The potential risks of worsened urge incontinence or recurrent SUI must be explained to patients undergoing urethrolysis for relief of obstruction, especially for those who have undergone prior anti-incontinence surgery [25].

Can Urodynamics Predict Response to Therapy?

Insofar as UDS is the only objective measure of BOO and relief of BOO has at least a 50 % (or greater) chance of alleviating OAB symptoms, UDS is a useful prognostic indicator. However, as is discussed below, in the absence of BOO, UDS has not been shown to be a good predictor of therapeutic response. Nevertheless, we believe that UDS is of the utmost importance in diagnosing OAB and directing treatment in patients who have failed a 1- or 2-month trial of empiric therapy. We further believe that the reasons why UDS has not been shown to be useful in this regard are twofold. First, most studies have been flawed from the outset because they have taken too simplistic a view—characterizing OAB by whether or not DO was demonstrated, rather than looking at a more detailed analysis, such as the OAB classification system. Second, therapeutic alternatives are limited and not very effective. So, no matter what UDS shows, the patient is treated with an antimuscarinic. If UDS is increasingly used for diagnosis and directing treatment, we believe that new treatments will emerge based, in part, on UDS findings and that, once that occurs, UDS will be shown to be of greater prognostic value.

From a clinical standpoint, the purpose of UDS is to measure and record various physiologic variables while the patient is experiencing the symptoms that constitute his or her usual complaints. In this context, UDS may be considered to be a provocative test of vesico-urethral function. Therefore, it is the responsibility of the examiner to ensure that the patient's symptoms are, in fact, reproduced in the study.

Trials evaluating the effects of antimuscarinics on OAB provide insight into whether the diagnosis of DO has any effect on response to treatment. Nitti et al. [26] found that the response to fesoterodine in patients with OAB and UUI was independent of the UDS findings of DO. Patients with and without DO had similar changes in voids per 24 h and UUI episodes. This study supports prior work showing the lack of a clear relationship between DO and response to pharmacologic or behavioral therapy. In a situation where planned therapy is expensive or invasive, such as with botulinum toxin or sacral neuromodulation, UDS evaluation may provide some guidance or prognostic information. The data on botulinum toxin are mixed in this regard. Cohen et al. [27] reported in their study that PVR was increased at 12 weeks but all other UDS parameters, including DO, were unchanged despite improved quality of life.

Indications for Urodynamics in Overactive Bladder

Most authorities and most guidelines agree that empiric treatment of OAB, after the workup delineated above, can usually be initiated without further diagnostic workup. Given the discordance between patients' clinical symptoms and certain UDS parameters, the need for UDS in OAB may seem even less relevant. However, we feel strongly that once empiric therapies have failed (after a month or two), UDS is the best diagnostic tool that is currently in our armamentarium.

Bladder outlet obstruction, detrusor overactivity, low bladder compliance, dysfunctional voiding, and sphincteric and mixed incontinence can be demonstrated only with UDS.

In addition, we recommend UDS in patients with clinical suspicion of upper tract deterioration in the setting of neurogenic voiding dysfunction, history of radical pelvic surgery (APR, radical hysterectomy, etc.), and history of pelvic radiation. Patients with UDS evidence of mixed incontinence (with a bothersome stress component) who have failed initial management should likely have their stress incontinence treated prior to proceeding to more invasive OAB treatment. Studies have demonstrated that between 50 % and 75 % of patients with mixed incontinence will have resolution of urge incontinence after surgical treatment of stress urinary incontinence [28, 29].

Currently, antimuscarinic agents are the mainstay treatment for typical OAB patients. While additional data are necessary to assess the outcomes of surgical management in terms of presence/absence of DO, there are data supporting the use of standard OAB treatments regardless of the findings of DO on UDS. As such, UDS documentation of DO is not needed before instituting OAB treatment. However, some studies have demonstrated that patients with UDS-proven DO may have a more severe form of OAB [14•]. In a more fundamental way, though, the OAB classification system may prove to be of even greater prognostic utility when and if new treatments are developed based on the degree of neuromuscular control exhibited during VUDS [6].

The American Urological Association has recently published guidelines regarding the utility of UDS in the setting of OAB [30••]. Despite an evidence strength of grade "C," the panel currently recommends UDS in clinical situations in which initial conservative and/or pharmacologic therapies fail in patients who desire more invasive treatment options for OAB. They concluded that patients with OAB may have other, simultaneous findings on UDS that may affect course of treatment. For example, a patient with urgency incontinence may have concomitant UDS diagnoses of SUI or BOO. Such

UDS parameters must be taken into consideration when considering treatment options for refractory urgency incontinence, since addressing these parameters may greatly improve the overall symptoms related to urinary urgency. Similarly, in mixed urinary incontinence, UDS can provide objective evidence of bladder and urethral abnormalities of urine storage and can reveal that other complicating factors are present that may affect treatment decisions. The panel also recognizes the technical and clinical limitations of UDS. Urologists should advise their patients with urgency incontinence and mixed incontinence that the absence of DO on a single UDS study does not invalidate their symptoms. UDS should always be interpreted in the context of a thorough physical examination, voiding diaries, and postvoid residual, as well as other clinical information.

Conclusion

The rationale for UDS in diagnosis and treatment is still evolving. Balancing the cost, invasiveness, and potential morbidity against potential diagnostic and prognostic information has been evaluated with particular scrutiny in recent series due to rising healthcare costs, as well as newfound knowledge of the disease process of OAB. Despite a weak correlation between DO and UDS, there are other UDS parameters that may potentially provide additional information regarding diagnosis and prognosis of certain treatments in OAB. When patients fail first-line therapy or when patients present with complex symptomatology, UDS plays an invaluable role in formulating second- or third-line management options. The current theory is that OAB is a constellation of both sensory and motor dysfunction. As more information regarding the exact pathophysiologic mechanism is elucidated, we propose a judicious use of UDS in the treatment of OAB.

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- •• Of major importance

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