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ROLE OF CYSTOMETRY IN EVALUATING PATIENTS WITH OVERACTIVE BLADDER

ADAM J. FLISSER AND JERRY G. BLAIVAS

ABSTRACT

Overactive bladder (OAB) can be caused by a variety of conditions. We believe that cystometrography (CMG) is an essential part of the diagnostic evaluation, both in defining underlying pathophysiology and directing treatment. Essential to the diagnosis of OAB syndrome is some combination of urinary frequency, urgency, urge incontinence, and pain. CMG can be thought of simply as a provocative test to determine whether bladder filling, involuntary detrusor contractions, or low bladder compliance in fact reproduces any of these symptoms, and whether the symptoms abate when the bladder is empty or when the pressure decreases. At another level, the CMG has been likened to "the reflex hammer" of the lower urinary tract and, as such, provides important neurologic information. Finally, the voiding phase of the CMG is an essential component of the detrusor pressure/uroflow study, which is the only accurate method of diagnosing urethral obstruction and impaired detrusor contractility. Both of these conditions can coexist with detrusor overactivity. The treatment of OAB depends on the underlying cause. We believe that only by understanding the particular cystometric characteristics of patients with OAB can we determine the appropriate treatment. Urodynamic testing should serve as an essential part of therapy and guide future research in diagnosis and management. UROLOGY **60** (Suppl 5A): 33–42, 2002. © 2002, Elsevier Science Inc.

The new International Continence Society (ICS) nomenclature redefines the syndrome of overactive bladder as "Urgency, with or without urge incontinence, usually with frequency and nocturia . . . if there is no proven infection or other obvious pathology."[1] The implication of this definition is that the term *overactive bladder* denotes a syndrome whose etiology is unknown. Strict adherence to this definition leaves no term to describe the same symptoms in patients with common conditions, such as benign prostatic hyperplasia, urethral obstruction, and neurogenic bladder. Accordingly, we recommend that the term *overactive bladder* be used to describe a symptom complex comprising urinary frequency and/or urgency, urge incontinence, and bladder pain, whether or not there is other relevant pathology. If there is other relevant pathology, it should be stated as part of the description, for example, "overactive bladder in a man with benign prostatic hyperplasia." Herein we present our perspective on the utility of urodynamic study and cystometrography (CMG) in the diagnosis and classification of overactive bladder.

From the Department of Obstetrics and Gynecology, New York Presbyterian Hospital, Weill Cornell Medical Center, New York, New York, USA; Department of Urology, New York Presbyterian Hospital, Weill Cornell Medical Center, New York, New York, USA; and Department of UroGynecology, Lenox Hill Hospital, New York, New York, USA.

Reprint requests: Jerry G. Blaivas, MD, The Urocenter of New York, 400 East 56th Street, New York, NY 10022.

URODYNAMICS, CYSTOMETRY, AND THE OVERACTIVE BLADDER

We believe that CMG (particularly as part of a multichannel urodynamic study) is an essential part of the diagnostic evaluation of patients with overactive bladder (OAB), both in its role of defining underlying pathophysiology and for directing treatment. Although the diagnosis of OAB involves some combination of urinary frequency, urgency, urge incontinence, and pain, patients with radically different bladder conditions resulting from a variety of different causes may all present with these nonspecific symptoms.

The belief that urodynamic testing is important in OAB patients, however, is not universal. It has been well documented that only 40% to 60% of patients with signs and symptoms of OAB are actually found to have detrusor overactivity at cystometric examination.[2–6] Although ambulatory monitoring may enhance the detection rate, no

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studies have shown an effect on treatment outcome. Although urodynamic testing is essential for detecting phasic bladder contractions and in classifying them,[7,8] and cystometric examination is held by some to be the standard method of investigating the OAB,[9] the relation between the urodynamic findings in specific OAB conditions and the therapies for these conditions has not yet been clearly elucidated.[10]

As stated by Clarke[11] in 1997, "There is evidence dating back some 20 years to suggest there are inaccuracies in depending on clinical symptoms of the lower urinary tract alone to determine diagnosis and thus management."

In a series of 1000 women who underwent provocative cystometry for symptoms of lower urinary tract dysfunction, Clarke[11] found the symptom of urgency was associated with detrusor instability in 87% of patients. Urge incontinence was associated with the urodynamic finding of detrusor instability in 70% of these patients. Other studies have documented lower detection rates of ≤47% using conventional cystometry.[3,5] Up to 56% of patients with irritative lower urinary tract symptoms and stable bladder at conventional cystometry were found to have detrusor overactivity at ambulatory urodynamics.[5,12,13] In our prior series of 111 patients who were presumed to have detrusor overactivity based on symptoms, 65% had involuntary detrusor contractions (IDCs) at cystometric examination.[14]

CMG can be thought of simply as a provocative test to determine whether any symptoms are, in fact, reproduced by bladder filling, IDCs, or low bladder compliance, and whether the symptoms abate when the bladder is empty or when the pressure decreases. At another level, the CMG has been likened to "the reflex hammer" of the lower urinary tract and, as such, provides important neurologic information. Finally, when combined with synchronous uroflow, it is the most accurate method of diagnosing bladder outlet obstruction and impaired detrusor contractility.

The specific method of urodynamic study is critically important, because variables in the urodynamic technique can have a substantial impact on the clinical utility of the study. The duration of the study, patient acclimation to urodynamics, patient positioning and provocative maneuvers,[15] learning or instruction,[2,9] and the rate of bladder filling may all influence the presence of clinical signs in a particular group of patients.[16]

Arbitani[17] observed that "the diagnosis of [detrusor instability], its rate of detection, and urodynamic patterns depend on the type of urodynamic test used and the way in which the test is performed. This was already evident for cystometry when the ICS classification was elaborated. Provocative cystometry doubled the rate of detection of unstable detrusor contractions in comparison to supine subtracted cystometry."

In our experience, the detection rate of detrusor instability was markedly influenced by the method of cystometry: 64% versus 48% of the same study population exhibited IDCs when instructed not to inhibit micturition during the cystometric examination.[14] This is in contrast to the cystometry recommendations of the ICS, which state that the patient should be instructed to try to inhibit micturition during bladder filling. This implies that CMG and urodynamics can be performed in a manner that will increase their utility as a tool for diagnosis and classification.

METHODOLOGY FOR PERFORMING MULTICHANNEL VIDEOURODYNAMICS

Our specific methodology for performing videourodynamics begins with the patient in the sitting position with a 7Fr double-lumen transurethral catheter through which room-temperature radiographic contrast (60% iothalamate meglumine mixed with sterile water in a 1:4 ratio) is infused at medium filling rate (75 to 100 mL/sec). Patients are instructed to neither void nor try to inhibit micturition but simply to report their sensations to the examiner. Bladder filling is discontinued (1) if the patient experiences a strong desire to void, uncomfortable fullness, or pain or (2) at the occurrence of incontinence caused by IDCs. An IDC is defined as a sudden increase in detrusor pressure of any magnitude that is involuntary. If neither discomfort nor an IDC occurs, filling is discontinued at the discretion of the examiner in the range of the patient's functional bladder capacity as determined by voiding diary. Patients who experience IDCs are asked if they are aware of the IDC. They are asked to contract the urinary sphincter. The examiner records if the patient successfully contracts the sphincter. If successful, the examiner records whether the urinary stream is interrupted, as well as if the detrusor contraction is aborted. Pressure-flow studies during voiding are taken with simultaneous video fluoroscopy of the bladder outlet and with perineal surface electromyography measurements. Urodynamic tracings are manually reviewed to assess (1) time of first sensation, (2) cystometric capacity, (3) bladder volume at the time of IDC, if present, and (4) detrusor pressure at the time of IDC. The pressure-flow study is plotted on the Shafer nomogram in men and on the Blaivas-Groutz nomogram in women.[18]

URODYNAMIC FINDINGS

Some patients who have overactive bladder symptoms exhibit no IDCs at all, presumably be-

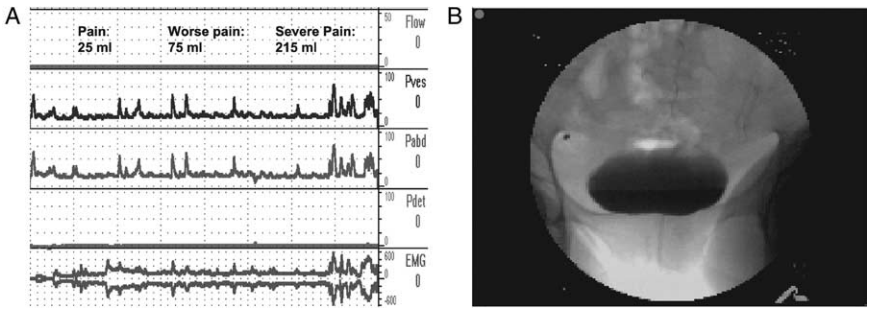


FIGURE 1. *Sensory urgency. The patient is a 43-year-old woman who reported urgency, frequency, and bladder pain, and voided >30 times in 24 hours. (*A*) Urodynamic tracing shows a small capacity, a hypersensitive bladder with a flat detrusor tracing, and the inability to initiate a voluntary detrusor contraction. There are neither voluntary nor involuntary detrusor contractions. (*B*) Fluoroscopic image obtained at a bladder volume of 125 mL, when the patient experienced pain. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O). (Courtesy of Jerry G. Blaivas.)*

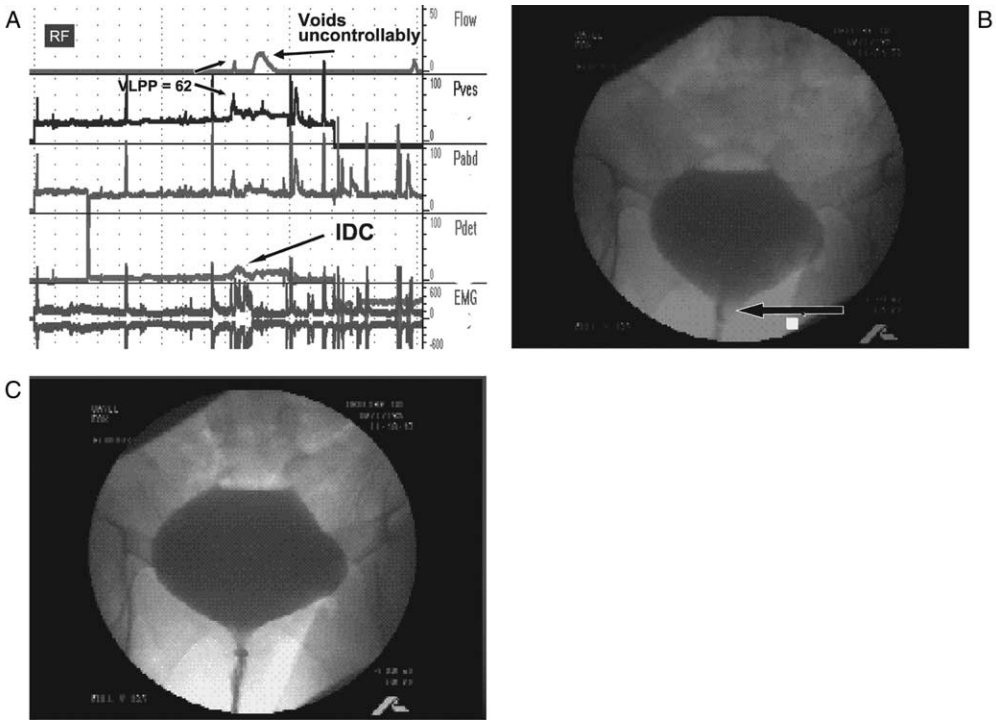


FIGURE 2. *Involuntary detrusor contraction (IDC) and uncontrolled voiding in a woman with stress hyperreflexia. The patient is an 80-year-old woman with frequency, urgency, and urge incontinence. (*A*) Urodynamic tracing. At a bladder volume of 150 mL, she is asked to cough; she has sphincteric incontinence with a vesical leak point pressure of 62 cm H_2O. Immediately afterward, she has an involuntary detrusor contraction and voids involuntarily to completion without any awareness or control. (*B*) Fluoroscopic image obtained during cough at vesical leak point pressure showing sphincteric incontinence (*arrow*). (*C*) Fluoroscopic image obtained during involuntary detrusor contraction and uncontrolled voiding. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O); VLPP = vesical leak point pressure. (Courtesy of Jerry G. Blaivas.)*

cause they retain excellent control (Figure 1). In other patients, an IDC appears to be very much like a neurologic reflex, devoid of voluntary control (Figure 2). Some patients have IDCs, but can contract the sphincter, interrupt the stream, and abort the IDC (Figure 3). Still others have a varying degree of control and awareness of IDC (Figure 4). Finally, the voiding phase of the CMG is an essential component of the detrusor pressure/uroflow study, which is the only accurate method of diagnosing urethral obstruction and impaired detrusor contractility, conditions that often coexist with OAB (Figures 5 to 8).

UTILITY OF URODYNAMICS

The mere presence or absence of an unstable bladder or IDCs comprises only part of the consid-

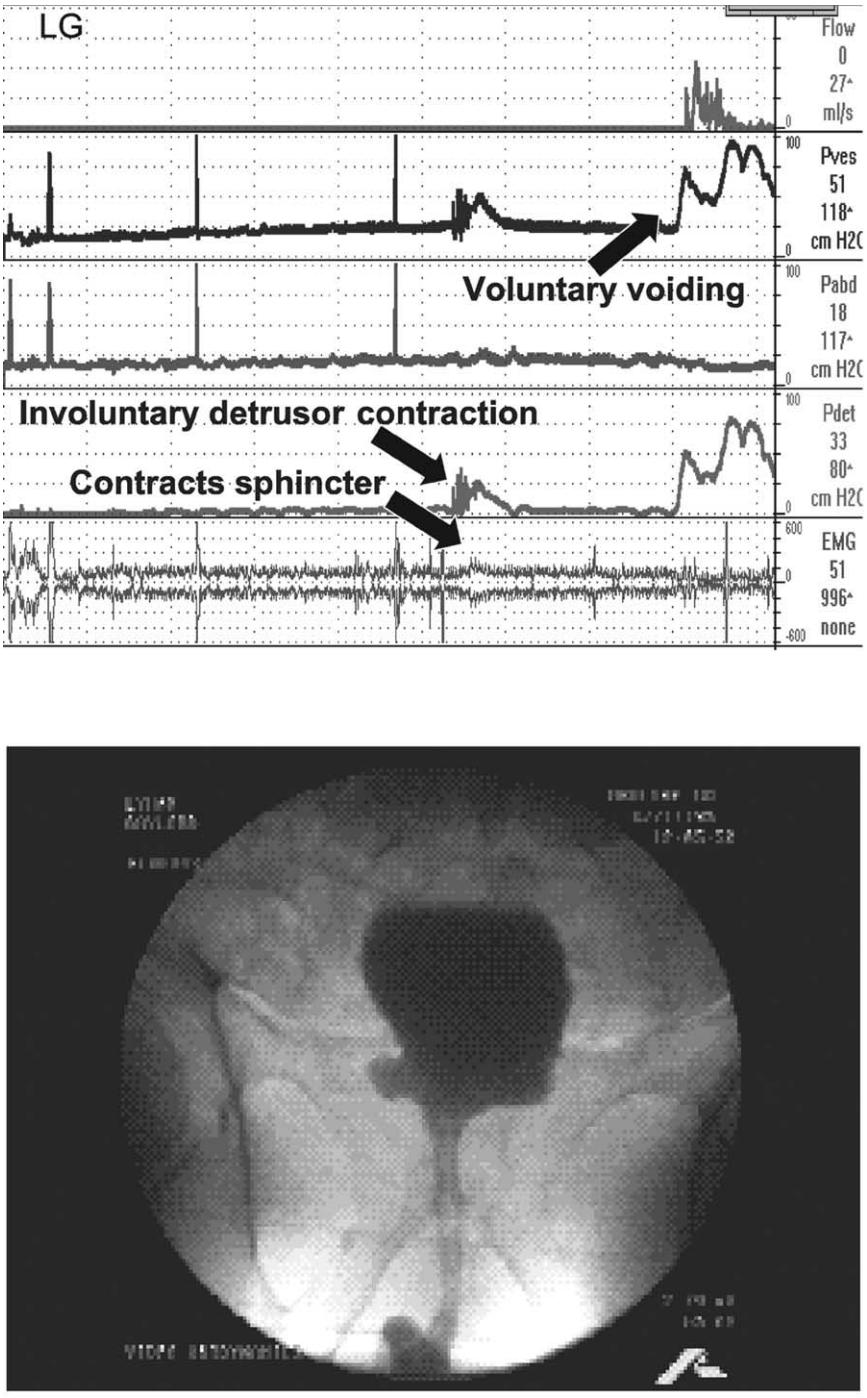


FIGURE 3. *Involuntary detrusor contractions and normal control in a man with benign prostatic hyperplasia and prostate cancer without urethral obstruction. The patient (LG) is a 71-year-old man with urinary frequency, urgency, and urge incontinence. Subsequently, 3 months after this study, he was found to have stage T1c prostate cancer. (*Top*) Urodynamic tracing. During bladder filling there is an involuntary detrusor contraction. He perceives this as an urge to void and voluntarily contracts his sphincter, and the detrusor contraction is aborted, preventing incontinence. With a comfortably full bladder, he is instructed to void and has a voluntary detrusor contraction. Unobstructed micturition ensues. (*Bottom*) Fluoroscopic image obtained during voluntary micturition. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O). (Provided courtesy of Jerry G. Blaivas.)*

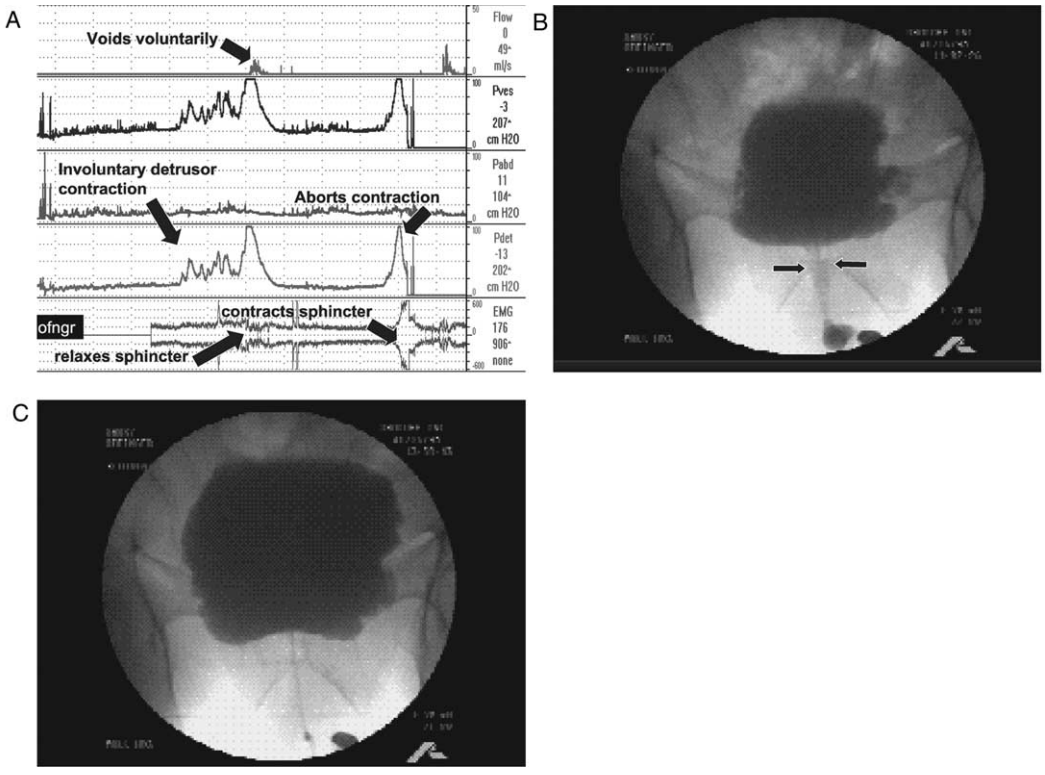


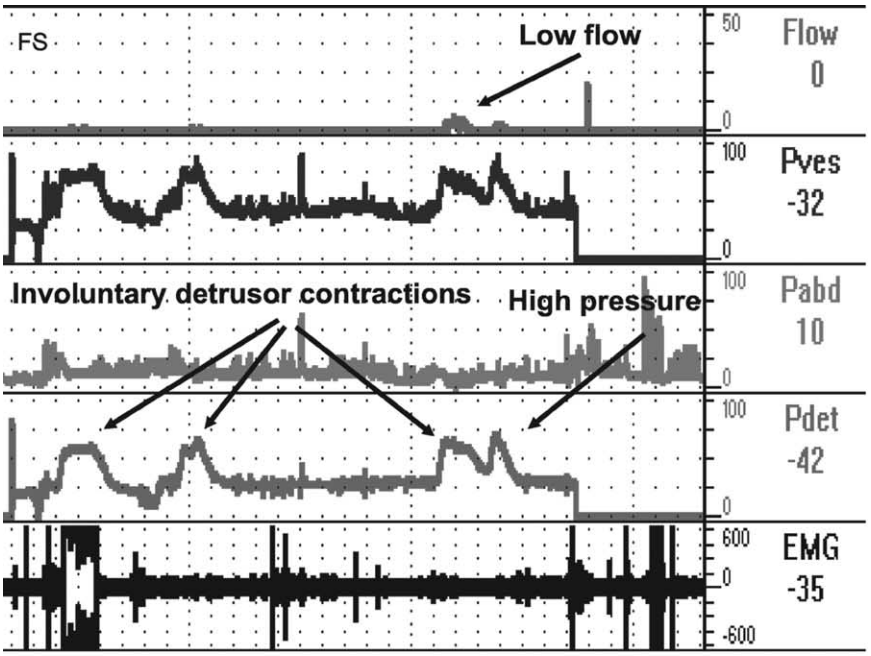
FIGURE 4. *Involuntary detrusor contractions and normal control in a man with benign prostatic hyperplasia and prostatic obstruction. The patient is a 59-year-old man with benign prostatic hyperplasia, frequency, urgency, and urge incontinence. (*A*) Urodynamic tracing. During bladder filling there is an involuntary detrusor contraction. He perceives it as urgency and voluntarily contracts his sphincter, preventing incontinence. He is instructed to void and relaxes his sphincter (*arrow*). This results in high detrusor pressure (detrusor pressure at Q_{max} >100 cm H_2O) and low flow (Q_{max} = 9 mL/sec) diagnostic of urethral obstruction. The bladder is refilled, and he has another involuntary detrusor contraction. This time he is instructed to try to hold and he contracts his sphincter, aborts the detrusor contraction, and prevents incontinence. (*B*) Fluoroscopic image obtained during voluntary voiding showing the point of obstruction in the prostatic urethra (*arrows*). (*C*) Fluoroscopic image obtained during voluntary contraction of the sphincter. There is no contrast in the urethra. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O). (Provided courtesy of Jerry G. Blaivas.)*

eration of how to treat a patient. The underlying causative or associated conditions are equally important. Consider the urodynamic studies cited above: bladder pain without IDC (Figure 1), uncontrollable IDCs in a woman with stress incontinence (Figure 2), urethral diverticulum (Figure 5), obstructing or unobstructing benign prostatic hyperplasia (Figures 3 and 4), and bladder tumor (Figure 8). All of these patients presented with symptoms of OAB, but are they all treated the same? Without performing urodynamic testing and in the presence of identical symptoms, how can you reliably distinguish among them?

Patients who have any of these etiologies can also have a variety of findings at urodynamic examination. Some have perfectly normal control, others have no awareness or control at all; there are many grades between these 2 extremes. The information gained at urodynamics, therefore, is not merely of diagnostic value (identifying detrusor overactivity, low bladder compliance, urethral obstruction, impaired detrusor contractility, sensory urgency, etc.), but it may also prove useful in directing therapy based on the patients' degree of awareness, concern, and control. For example, men with OAB and benign prostatic hyperplasia without urethral obstruction may respond well to behavioral therapy, whereas men with the same symptoms, benign prostatic hyperplasia, and prostatic obstruction might not respond well and would be better treated with medications or surgery.

CONCLUSION

In conclusion, we believe that urodynamics is extremely important in the diagnosis and treatment of lower urinary tract symptoms in general, and OAB in particular. If all patients with OAB are treated the same way, according to some kind of OAB treatment algorithm, it can be argued that there is no need for urodynamic testing. However, if urodynamics are not done, it is likely that the



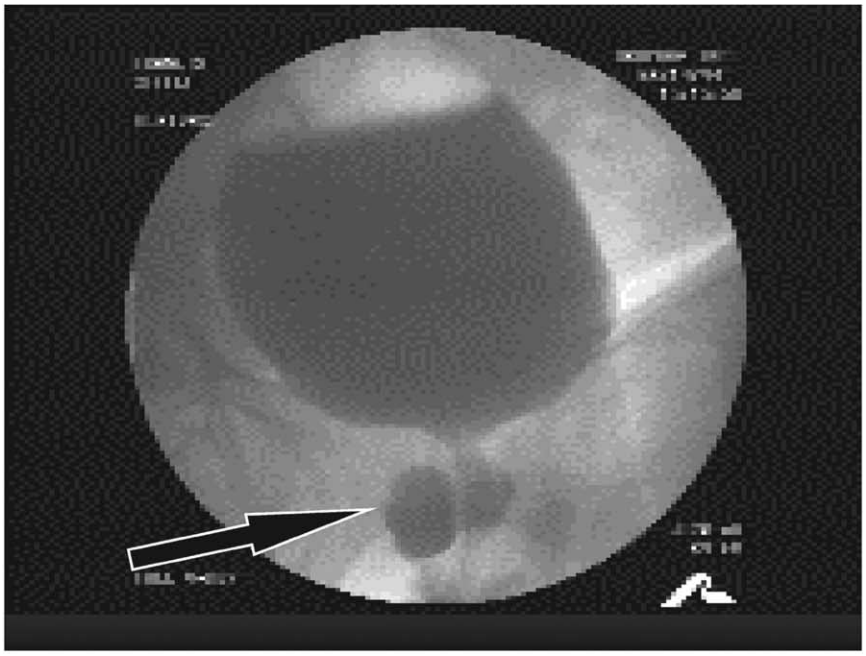


FIGURE 5. *Involuntary detrusor contractions in a woman with obstruction caused by a large, multiclocular urethral diverticulum. The patient (FS) is a 67-year-old woman with urinary frequency, urgency, and dysuria. (*Top*) Urodynamic tracing. There are multiple involuntary detrusor contractions during filling. She perceives them as urgency and is able to contract her sphincter but can neither interrupt the stream nor abort the detrusor contractions. She voids involuntarily. Even when she voluntarily relaxes her sphincter, there is high pressure (detrusor pressure at* Q_{max} *= 75 cm* H_2O*) and low flow (*Q_{max} *= 6 mL/sec), documenting urethral obstruction. (*Bottom*) Fluoroscopic image obtained during voluntary voiding showing the obstruction caused by a large multilocular urethral diverticulum (*arrows*). EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm* H_2O*). (Courtesy of Jerry G. Blaivas.)*

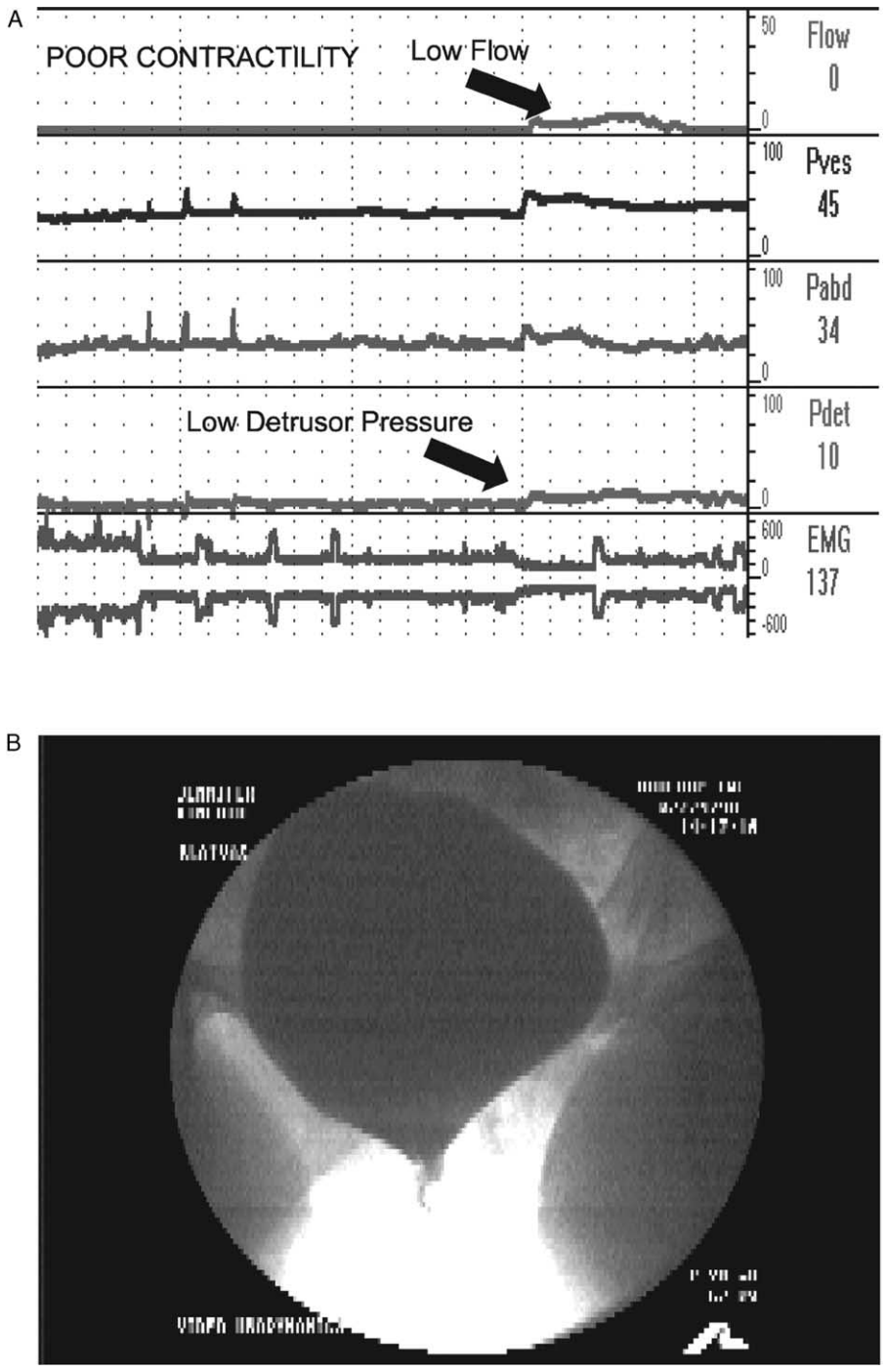


FIGURE 6. *Involuntary detrusor contraction in a woman with impaired detrusor contractility. There is an involuntary detrusor contraction that she perceives as urge to void, but she is unable to contract her sphincter, and she voids uncontrollably. Pdet (detrusor pressure) at Q_{max} >6 cm H_2O, Q_{max} = 9 mL/sec, indicating impaired detrusor contractility.* Arrows *indicate low urinary flow and low maximum detrusor pressure. (*B*) Fluoroscopic image obtained at Q_{max}. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure. Pves = vesical pressure (cm H_2O).*

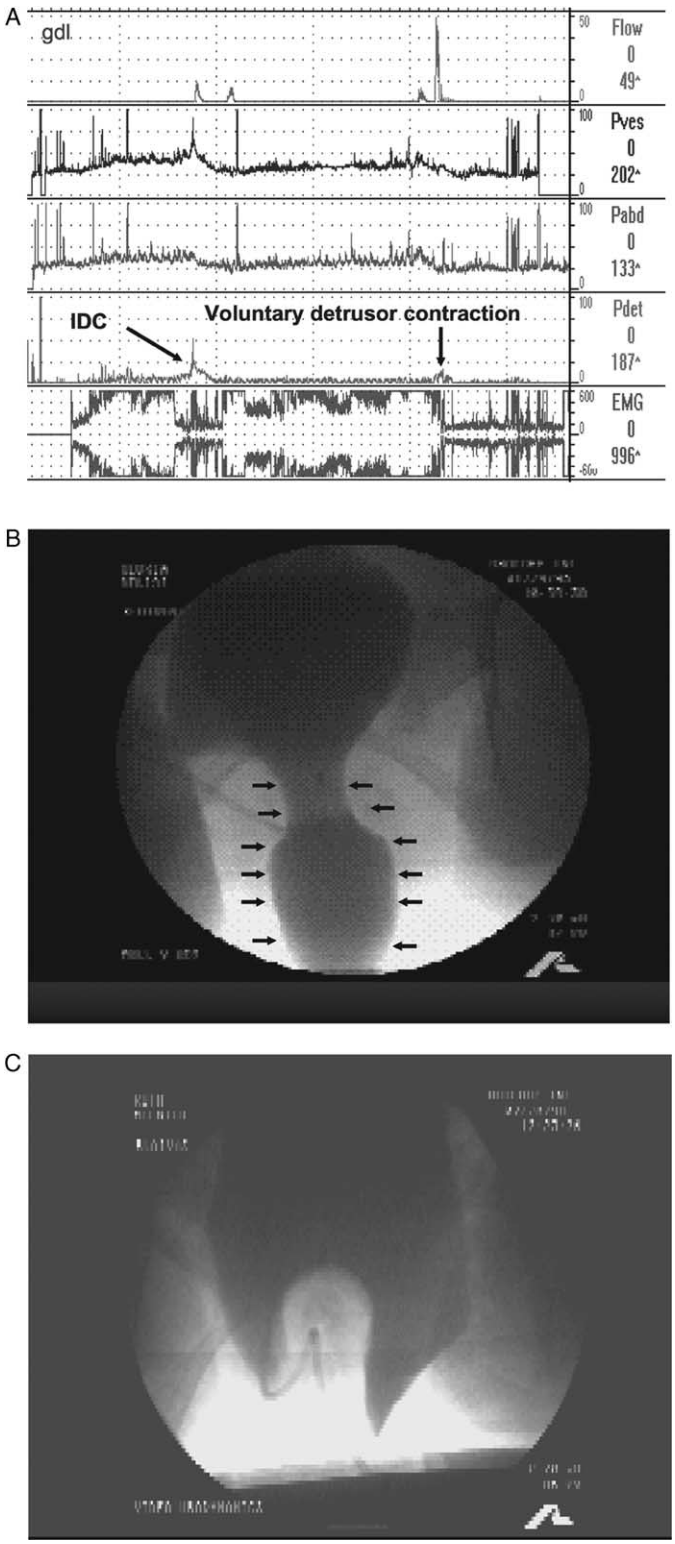


FIGURE 7. *Detrusor instability and urethral obstruction due to large cystocele. The patient (gdl) is a 75-year-old woman with urinary frequency, urgency, urge incontinence, and prolapse. (*A*) During bladder filling with the prolapse out, there was an involuntary detrusor contraction, of which she was aware but was unable to abort. She voided with a detrusor pressure at Q_{max} = 50 cm H_2O and Q_{max} = 10 mL/sec, documenting urethral obstruction. Reduction of the cystocele with a pessary relieves the obstruction, and she voids voluntarily with a detrusor pressure at Q_{max} = 12 cm H_2O and Q_{max} = 48 mL/sec. (*B*) Fluoroscopic image obtained during the involuntary detrusor contraction shows a large cystocele that obscures the urethra. The arrows point to the obstructing cystocele. (*C*) Fluoroscopic image obtained during voluntary detrusor contraction with the prolapse reduced by a pessary. EMG = electromyography; Flow = urinary flow (mL/sec); IDC = involuntary detrusor contraction; Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O). (Courtesy of Jerry G. Blaivas.)*

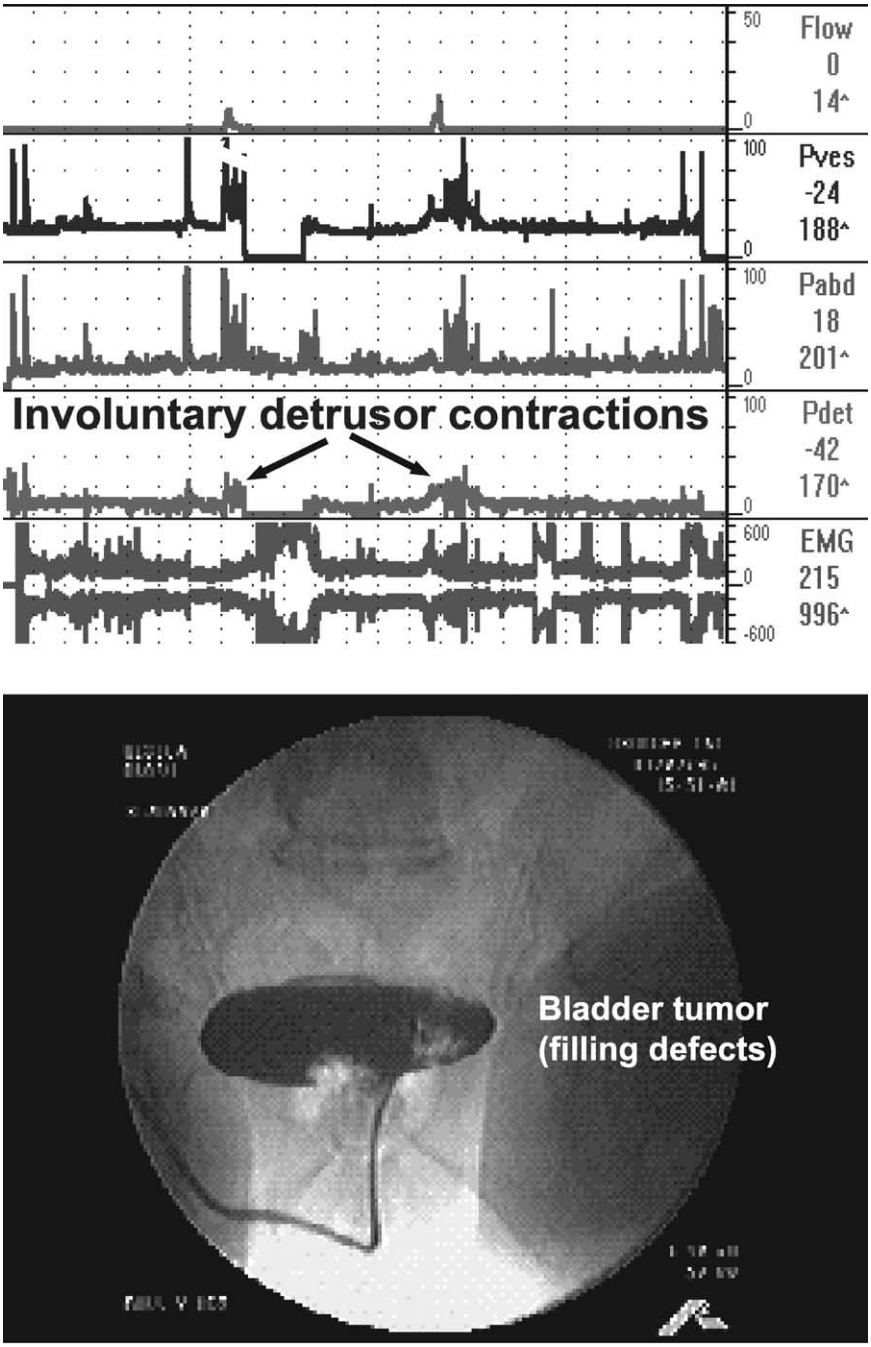


FIGURE 8. *Involuntary detrusor contractions in a woman with multiple transitional cell bladder tumors. (*Top*) A 72-year-old woman with urgency and frequency, but no hematuria. There are multiple involuntary detrusor contractions that she perceives as urge to void. She contracts her sphincter but is unable to abort the stream or prevent incontinence. (*Bottom*) Fluoroscopic image shows multiple filling defects. EMG = electromyography; Flow = urinary flow (mL/sec); Pabd = abdominal pressure; Pdet = detrusor pressure; Pves = vesical pressure (cm H_2O). (Courtesy of Jerry G. Blaivas.)*

correct diagnosis will be missed in many patients. Without urodynamics, the patient is denied the opportunity to receive a precise diagnosis and, therefore, treatment based on the underlying cause. Further, the physician is denied the opportunity to appreciate the subtle differences among patients. If the physician does not do urodynamics, he or she will be denied the opportunity to understand pathophysiology. Without that understanding, he or she is denied the opportunity to make the intelligent hypotheses that direct future research into diagnosis and treatment.

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DISCUSSION FOLLOWING DR. BLAIVAS' PRESENTATION

Roger R. Dmochowski, MD (Nashville, TN): When you did your initial stratification and looked at the urodynamic criteria, you obviously also had diary data and quality-of-life data. Was there a rough parallelism or what was the magnitude?

Jerry G. Blaivas, MD (New York, NY): There was no correlation between the characteristic of the cystometrogram and either diary data or clinical diagnosis.

Dr. Dmochowski: Regarding hyperreflexive stress-induced incontinence, is it your impression that this is rare, and what do you do for it?

Dr. Blaivas: I think it is rare.

Stuart B. Bauer, MD (Boston, MA): What is the rate of flow in your cystometrograms?

Dr. Blaivas: Within the range of medium fill in adults, ie, about 60 to 100 mL/min.