Chapter 16
Bladder Emptying: Contractility

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Definition of Bladder Contractility

Bladder contractility is a term used to describe the strength of bladder detrusor muscle contractions [1, 2]. Since bladder contraction requires parasympathetic stimulation, the detrusor pressure depends on the inherent strength of the detrusor muscle itself, which can be affected by a variety of factors, and intact neural circuitry. Loss of either would clearly impact contractile strength and overall bladder function. *Normal detrusor function* is initiated by a drop in urethral pressure followed by a continuous detrusor contraction which leads to complete bladder emptying within a normal time span. For many women who void primarily by urethral relaxation, this contraction may be modest or even absent. *Detrusor underactivity* implies a detrusor contraction of reduced strength and/or duration resulting in prolonged voiding, and/or a failure to achieve complete bladder emptying. An *acontractile detrusor* identifies the condition where no detrusor contraction is noted, also typically associated with prolonged voids, incomplete emptying, or inability to void altogether. Examples of each of these conditions are demonstrated in urodynamic tracings shown in the remainder of the chapter.

Other factors, such as outlet resistance, can also greatly affect bladder contractility. Enhanced outlet resistance, in the setting of bladder outlet obstruction, will result in increased detrusor force for a variable period of time, followed potentially by a loss of force depending on the severity and duration of obstruction. Similarly diminished outlet resistance, for example in the setting of stress urinary incontinence, has been shown to result in lowered detrusor pressures during voiding.

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The voiding phase of cystometry, also known as the pressure-flow study, measures the relationship between detrusor pressure and flow rate during voiding. While a low flow rate alone may be more likely to be associated with bladder outlet obstruction, this clearly is not always the case. Indeed, the primary purpose of the pressure-flow study is to differentiate bladder outlet obstruction from low flow due to detrusor underactivity or acontractility. Similarly, while uncommon, patients with relatively normal flow may have quite elevated detrusor pressures suggestive of obstruction, a diagnosis which can only be made during pressure-flow analysis [3, 4].

Assessment of Bladder Contractility

Measurement of detrusor contractility is commonly assessed by multichannel urodynamics during the pressure-flow study. Isovolumetric techniques to study detrusor contractility where the urinary stream is interrupted using balloon occlusion, inflatable penile cuffs or occlusive condom catheters are not commonly used as they can be uncomfortable, cumbersome, and may interfere with the normal desire to void. These tests are especially challenging in female patients [1, 2, 5]. Voluntary stopping of the urinary stream by contracting the urinary sphincter may not be reliable in measuring detrusor contractility [6]. Cystoscopic appearance (trabeculations, cellules) and radiological findings (bladder wall thickness) may provide indirect clues regarding bladder function and contractility, but again are non-specific findings.

Several formulas and nomograms based on pressure-flow studies have been developed to better quantify detrusor contractility [1]. Watts Factor (WF) is a calculation used to follow changes in contraction strength as the bladder empties. It is based on detrusor pressure (P_{det}), flow rate (Q), volume in the bladder (V), velocity of shortening of bladder circumference, and constant values ($a = 25$ cm H_2O, $b = 6$ mm/s) based on in vitro studies [7]. WF_{max} is proposed to represent maximal detrusor contractility and is calculated at maximal flow rate (Q_{max}) [8].

$$\mathrm{WF} = \left[\left(P_{det} + a\right)\left(V_{det} + b\right) - ab\right] / 2\pi; \text{where } V_{det} = Q / 2\left[3\left(V + V_t\right) / 4\pi\right]^{2/3}$$

where V_t = volume of noncontracting bladder wall tissue [7].

Although some studies report WF and WF_{max}, no consensus has been made regarding normative values particularly in female patients [6, 8, 9]. A WF_{max} of <12 μW/mm^2 for males and <5 μW/mm^2 for postmenopausal females has been used in some studies to define detrusor underactivity [8, 10, 11]. Use of WF may not be suitable for practical use due to complexity of the calculation, poor reproducibility, and lack of standardized cutoff values [6, 8].

Schafer simplified the approach to classify contractile strength based on relationship of detrusor pressure and urine flow rates in men with bladder outlet obstruction [1, 12]. Bladder contractility index (BCI) is based on detrusor pressure at maximum flow rate (P_{det} @ Q_{max}) and maximum flow rate (Q_{max}):

$$\text{BCI} = P_{det} \text{ @ } Q_{max} + 5Q_{max}$$

In men, BCI > 150 suggests strong contractility; BCI 100–150 suggests normal contractility and BCI < 100 suggests weak contractility [1, 3].

To address the limitation that BCI was based on men with BOO, Tan modified the BCI calculation and proposed a new formula of contraction strength for elderly women (mean age 70.1 years) with urge incontinence, known as the projected isovolumetric detrusor pressure (*PIP*) [2]:

$$\text{PIP} = P_{det} \text{ @ } Q_{max} + Q_{max}.$$

With this modified formula, normal contractility in this cohort was defined as PIP = 30–75 [2].

The relationship between detrusor contractility and urine flow in the form of a pressure versus flow rate (p/Q) plot can be helpful in the understanding of detrusor function and outflow obstruction [12]. In efforts to better clarify this relationship, further calculations such as passive urethral resistance relation (PURR), linear PURR, detrusor-adjusted mean PURR factor (DAMPF) have been proposed and can be helpful in the assessment of patients with outflow obstruction taking into account their detrusor contractility which could otherwise be overlooked [12].

Detrusor Underactivity

Detrusor underactivity is defined by the ICS as "detrusor contraction of reduced strength and/or duration, resulting in prolonged bladder emptying within a normal time span" [13]. It is unclear if bladder contractility diminishes with age, though the most recent data suggests that this may indeed be the case [14–18]. What is clear is that merely the presence of a weak detrusor contraction during voiding itself may not lead to a diagnosis of detrusor underactivity, since the definition implies some sequelae occur (altered or prolonged voiding) as a result of this urodynamic finding. Both myogenic failure and bladder wall ischemia have been proposed as underlying pathophysiological mechanisms responsible for detrusor underactivity [17, 19–21].

Causes and predisposing factors of detrusor underactivity include medications, psychogenic factors, neurologic conditions, end-stage BOO, extensive pelvic surgery, or idiopathic. Urodynamic studies are helpful to diagnosis detrusor underactivity and rule out other causes of voiding dysfunction [17]. Urodynamically, detrusor underactivity is commonly found in a low pressure/low flow state. It also may be diagnosed mistakenly in women who void primarily by urethral relaxation,

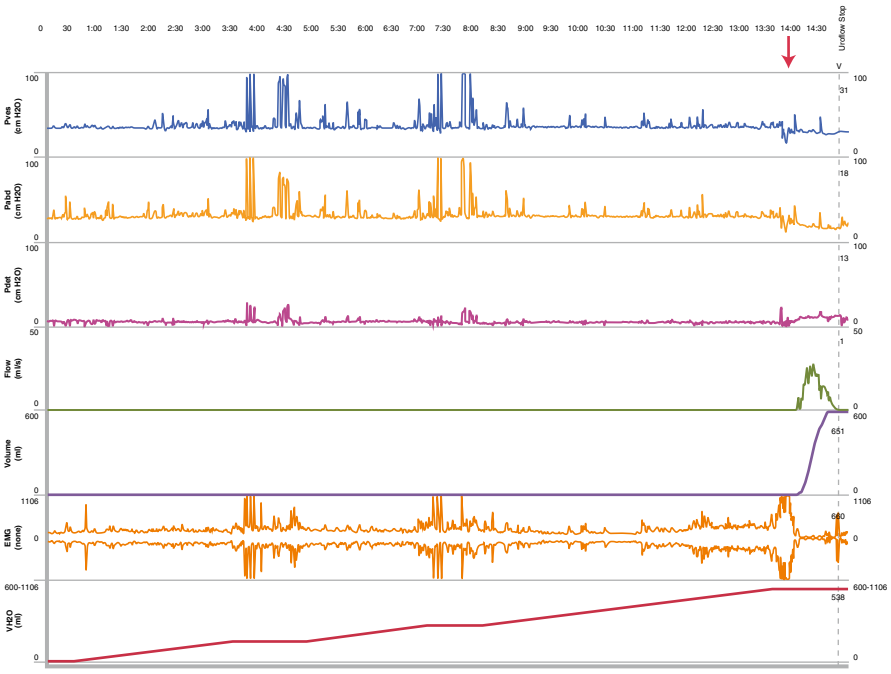

Fig. 16.1 Urodynamic tracing of urethral relaxation in a female. Note decreased EMG activity during voiding. Permission to void denoted by *red arrow*

in which case urinary flow rates may be normal, voiding is not prolonged, and as a result underactivity cannot be truly diagnosed even in the setting of a weak detrusor contraction. In a similar way, a weak detrusor contraction may be associated with intrinsic sphincteric deficiency (ISD) with both men and women, though the presence of relatively normal voiding in these instances argues against the diagnosis of detrusor underactivity, which implies an insufficient contraction. Again, it is important to stress that the urodynamic finding of a minimally contracting detrusor muscle may have no clinical relevance, particularly in women, who void efficiently and effectively by urethral relaxation or have chronically reduced outlet resistance due to ISD. Post-prostatectomy incontinence with compromised bladder outlet is another common situation where a relative detrusor underactivity occurs, in the absence voiding dysfunction or other sequelae.

Clinical scenario 1 (Fig. 16.1):

Urethral relaxation

A 69-year-old female with recurrent urinary tract infections but no bladder storage or emptying complaints. Her PVR is zero. Cystoscopy is unremarkable. She has no prior history of urologic pelvic surgery or neurological disease. Her urodynamics are interesting because the PFS demonstrates urethral relaxation. Note the decrease abdominal pressure and the lack of significant rise in either P_{ves} *or* P_{det}.

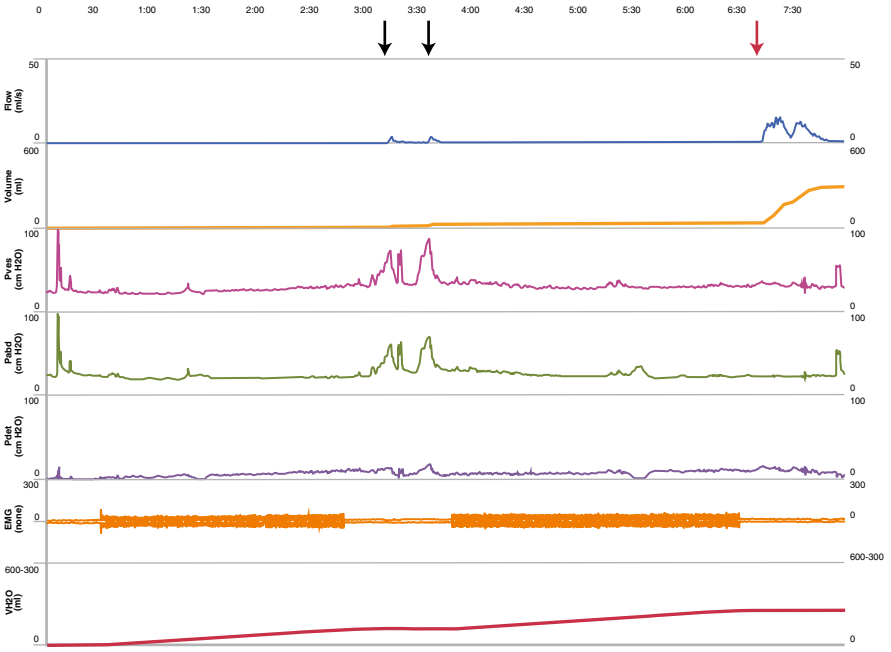

Fig. 16.2 Urodynamic tracing demonstrating low pressure voiding in a female with SUI. Note the presence of a compromised outlet due to SUI can result in lower voiding pressure. Stress urinary incontinence denoted by *black arrows*. Permission to void denoted by *red arrow*

Despite an apparent $P_{det}Q_{max}$ of only 13 cm H_2O (actual P_{det} value may be even lower due to drop in P_{abd}), she completely empties her bladder by urethral relaxation, voiding 651 mL, with a maximum flow rate of 27 mL/s and normal bell-shaped flow curve. This is due to urethral relaxation at the time of voiding—as detected by the diminished sphincteric activity noted on EMG monitoring.

Clinical scenario 2 (Fig. 16.2):

Reduced outlet resistance, low pressure voiding in woman with SUI

A 62-year-old female with symptomatic stress urinary incontinence. She reports voiding with a normal flow. She is noted to have SUI on her study at 114 mL with differential VLPP of approximately 39 cm denoted by first two arrows. The low VLPP is consistent with intrinsic sphincter dysfunction. Bladder compliance is normal and there was no evidence of detrusor overactivity. Her MCC was 251 mL.

During the PFS, the detrusor contraction is relatively weak, but adequate to allow her to empty her bladder to completion with a bimodal flow curve (Q_{max} 15 mL/s). The presence of a compromised bladder outlet has been associated with both lower valsalva leak point pressure (VLPP) and voiding pressures in women with SUI.

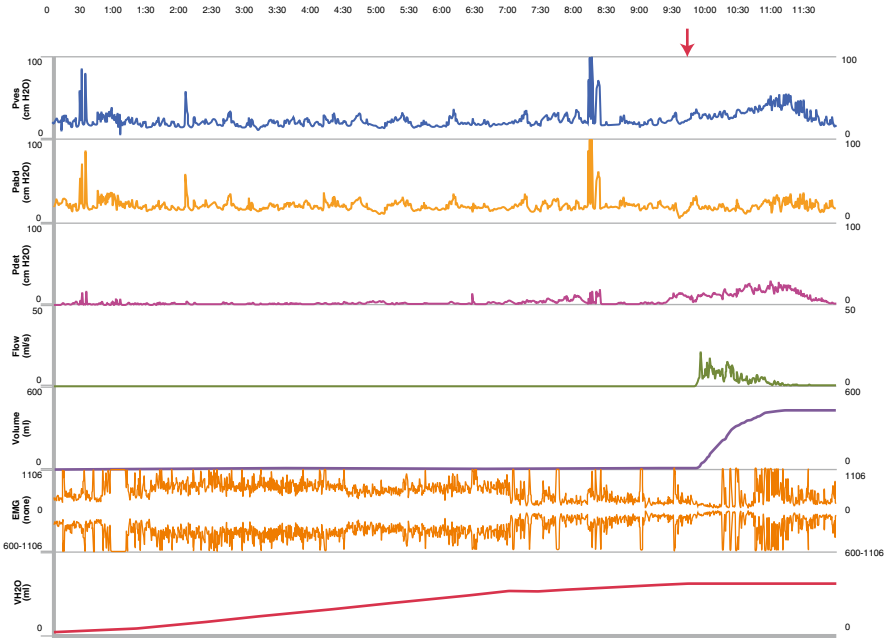

Fig. 16.3 Urodynamic tracing of a female with sensation of difficulty urinating due to detrusor underactivity. Permission to void denoted by *red arrow*

The quality of the UDS is good. A cough at the end of the study confirmed catheters were in appropriate position with adequate zeroing. Her voided volume was not marked on the tracing.

Clinical scenario 3 (Fig. 16.3):

Detrusor underactivity/Crede voiding

An 82-year-old female with recurrent UTIs, urinary frequency and sensation of difficulty emptying her bladder. First sensation is at 176 mL. No stress incontinence was seen with cough or Valsalva maneuver. MCC is 365 mL. Compliance is normal. No detrusor overactivity is noted. On pressure-flow component of study, the patient was noted to apply external pressure to her suprapubic region, or crede, to assist with voiding. She voids with a prolonged stream to completion without a residual volume, and a notably weak detrusor contraction.

EMG shows appropriate relaxation as voiding starts though increased EMG activity during void may reflect straining. In this case, due to the presence of abnormal voiding, the term detrusor underactivity should be applied.

The quality of this urodynamic tracing is fair. After permission to void (denoted by red arrow), a subtle rise in the P_{abd} is noted with crede maneuver which is also reflected in P_{ves} tracing. Q_{max} of 21 mL/s is not representative of her flow curve as she does a crede maneuver to achieve that reading. Average flow rate is considerably lower and more representative of her altered voiding function.

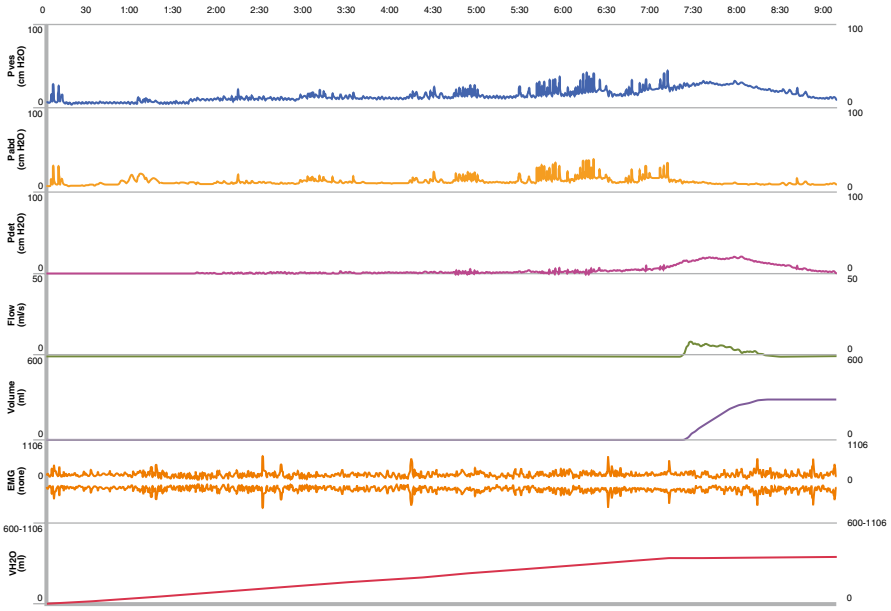

Fig. 16.4 Urodynamic tracing of a male who is 4 years post-radical prostatectomy with incomplete bladder emptying and elevated residual. The BCI is <100 consistent with detrusor underactivity. Permission to void given twice, and noted with increases in both abdominal and vesical catheters and corresponding small volume voids

Clinical scenario 4 (Fig. 16.4):

Idiopathic detrusor underactivity following radical prostatectomy

A 71-year-old man underwent a radical prostatectomy 4 years prior to the study. His PSA is still undetectable. He complains of a weak prolonged stream, urinary frequency, nocturia (3/night), and sensation of incomplete emptying over the past year. Cystoscopy was unremarkable. First sensation occurred at 103 mL. He experiences strong urge at 201 mL but is ultimately able to attain a normal MCC of 348 mL.

P_{det} does not rise substantially with his void, and a prolonged flow curve is noted. Q_{max} is 10 mL/s with $P_{det}Q_{max}$ of 14 cm H_2O. His BCI ($P_{det}Q_{max} + 5(Q_{max})$ is 64 (<100) which represents detrusor underactivity. He also was catheterized for 90 mL at the end of the study.

The quality of this tracing is fair. At the beginning of the study, P_{det} is below zero, which is physically implausible. Vesical and rectal catheter position should be assessed to insure accuracy in situations such as this. Often the catheters may be against the wall of the bladder or rectum and instilling more fluid (or releasing) in the rectal balloon or initiating bladder filling will move the pressure detector from the luminal wall and correct the problem. Prior to voiding, there are corresponding increases of P_{ves} and P_{abd} which represent patient movement and hiccups. It is important to annotate the study with findings such as this to facilitate interpretation.

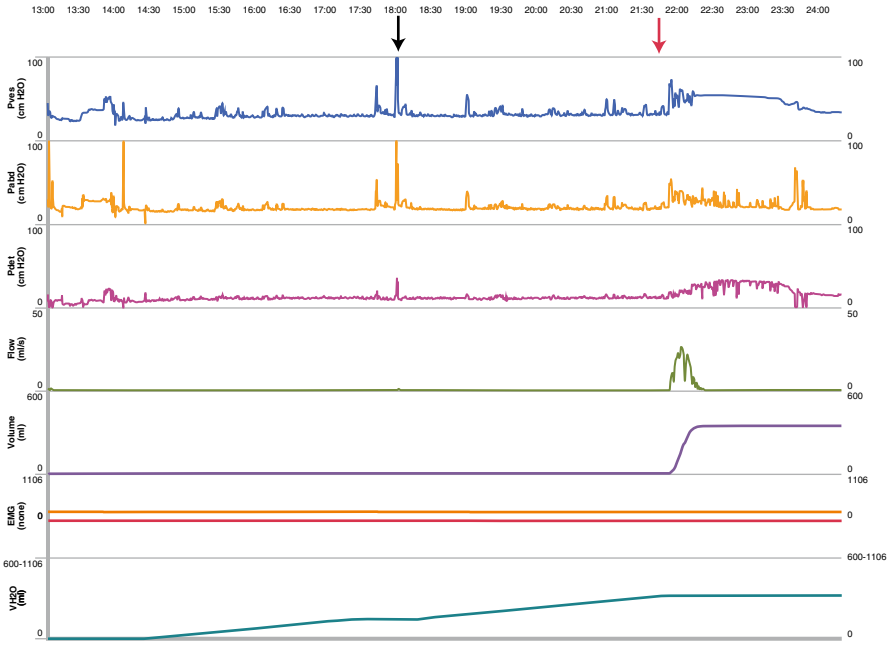

Fig. 16.5 Urodynamic tracing of a male with post-prostatectomy incontinence and relative detrusor hypocontractility. Similar to women with SUI, a compromised bladder outlet in a male can also be associated with low voiding pressures. Stress urinary incontinence denoted by *black arrow*. Permission to void denoted by *red arrow*

Clinical scenario 5 (Fig. 16.5):

Relative detrusor underactivity associated with sphincteric deficiency in man status-post radical prostatectomy.

A 68-year-old man 3 years out from radical retropubic prostatectomy is noted to have three pads per day leakage. Otherwise, he feels well, has had no bladder infections, and voids with a strong stream. In this urodynamic study, there is a reasonable bladder capacity (360 mL), an absence of detrusor overactivity during filling, stress incontinence (noted at first arrow), and a relatively weak detrusor contraction supplemented by straining. Since he empties to completion, and in the absence of a prolonged flow, this finding would not be consistent with the term "detrusor underactivity" but rather represents a reduced detrusor contraction, possibly related to loss of outlet resistance over time.

Treatments for Detrusor Underactivity

A limited understanding of causes and a dearth of effective treatments for detrusor underactivity suggests that further innovative research and treatment strategies are warranted [22]. Optimization of voiding in patients with detrusor underactivity is attempted by avoiding/treating constipation, discontinuing/limiting medications that can impact detrusor contractility, such as narcotics and medications with anticholinergic properties, and aggressive ambulation. Bethanechol and other parasympathomimetic agents have shown limited efficacy in clinical trials, and thus their use is largely discouraged [23, 24]. TURP in men with detrusor underactivity is controversial with lower success rates compared to those without detrusor underactivity, but may have a role in select patients by reducing outflow resistance or undiagnosed BOO after careful preoperative counseling [24–26]. Intermittent catheterization and indwelling catheterization are the remaining options for those with a symptomatic impaired bladder emptying secondary to underactivity.

Acontractile Detrusor

An acontractile detrusor is defined as "the detrusor cannot be observed to contract during urodynamic studies resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span [13]." Detrusor acontractility may be a more severe form of detrusor underactivity, and is thought to be caused by the similar underlying pathophysiologic mechanisms in many instances. Neurogenic causes (i.e., sacral cord injury), fixed chronic obstruction (prolonged untreated benign prostatic growth), and functional causes (non-neurogenic neurogenic bladder/Hinman syndrome, non-relaxing pelvic floor) may all be associated with detrusor acontractility. As with detrusor underactivity, detrusor acontractility can be transient or permanent depending on the clinical circumstance. Also, the terminology implies not only to the finding of absent detrusor contraction, but to a resultant impact on voiding as well. In certain clinical scenarios, such as voiding by urethral relaxation, the detrusor contraction may be absent, but voiding remains normal.

Treatments for Detrusor Acontractility

The acontractile detrusor is typically managed by intermittent or indwelling catheterization (urethral or suprapubic), especially if due to a transient cause [27]. In cases of persistent detrusor acontractility, sacral neuromodulation (for non-obstructive causes), intravesical electrostimulation, latissimus dorsi detrusor myoplasty, or continent catheterizable channels have been utilized in differing clinical scenarios [27, 28].

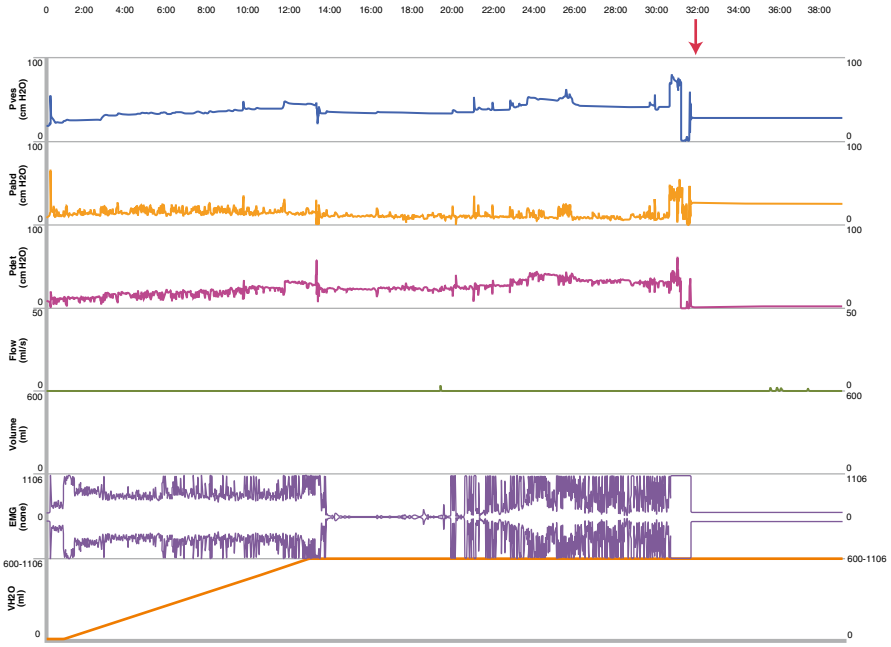

Fig. 16.6 Urodynamic tracing of a female with an acontractile detrusor and inability to void. Permission to void denoted by *red arrow*. Pressure transducers adjusted at this point

Clinical scenario 6 (Fig. 16.6):

Acontractile detrusor in a female

A 26-year-old female presents with complaints of bladder fullness without a typical desire/urge to void. She has noticed that if she tries to urinate the stream only dribbles and may take her several attempts to empty her bladder. She noticed that the stream has worsened with time over the course of 6–8 months. She has also had an increasing number of UTIs. She denies having a known neurological diagnosis but on further questioning reveals that she has experienced some visual changes, and occasional numbness and tingling in her left upper extremity. She has had no prior pregnancy, surgery, or neurological evaluation.

She was subsequently scheduled for urodynamics which revealed delayed first sensation (537 mL) and MCC of 733 mL. No incontinence was observed during filling. Despite having large MCC and desire, with permission, to void (at red arrow), she did not mount any detrusor contraction. An increase in vesical pressure is noted through this long filling curve suggestive of somewhat altered compliance. Given that this occurs over a volume of over 700 mL, the impact of this pressure change can be mitigated by an appropriate catheterization schedule. Urodynamic catheters were removed after the patient was unable to void with reasonable attempts of urination. After the catheter was removed, she only urinated 11 mL.

She has an acontractile bladder on UDS. The differential diagnosis of her acontractile bladder includes an undiagnosed neurologic condition, medication, or idiopathic causes such as Fowler's syndrome. She has been taught and will perform intermittent catheterization to empty her bladder. A neurological investigation revealed demyelinating lesions on CT suggesting multiple sclerosis.

This tracing is relatively poor quality as it is not perfectly zeroed, movement artifact is present, and the side scale has not been recalibrated to show the entire volume of fluid instilled. Since the patient could not void with the catheter in place a PFS could not be performed. However, detrusor acontractility is diagnosed during her attempt to void.

Clinical Scenario 7 (Fig. 16.7)

Acontractile detrusor in elderly male

A 72-year-old male with chronic history of difficulty voiding and post-void dribbling, which was never treated. Noted to have PVR of 650 mL. Urodynamic study shows large bladder capacity, delayed desire to void, a stable bladder during filling, normal compliance, and detrusor acontractility on attempt to void.

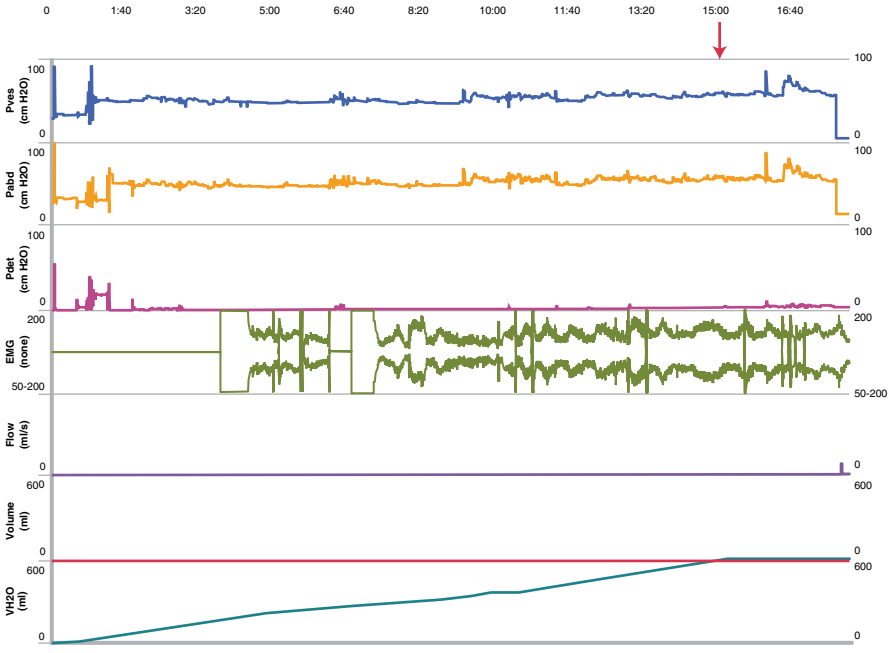

Fig. 16.7 Urodynamic tracing in an elderly male with large capacity bladder, delayed bladder sensation, and inability to urinate. Note no significant increase in detrusor pressure during voiding attempt. Permission to void denoted by *red arrow*

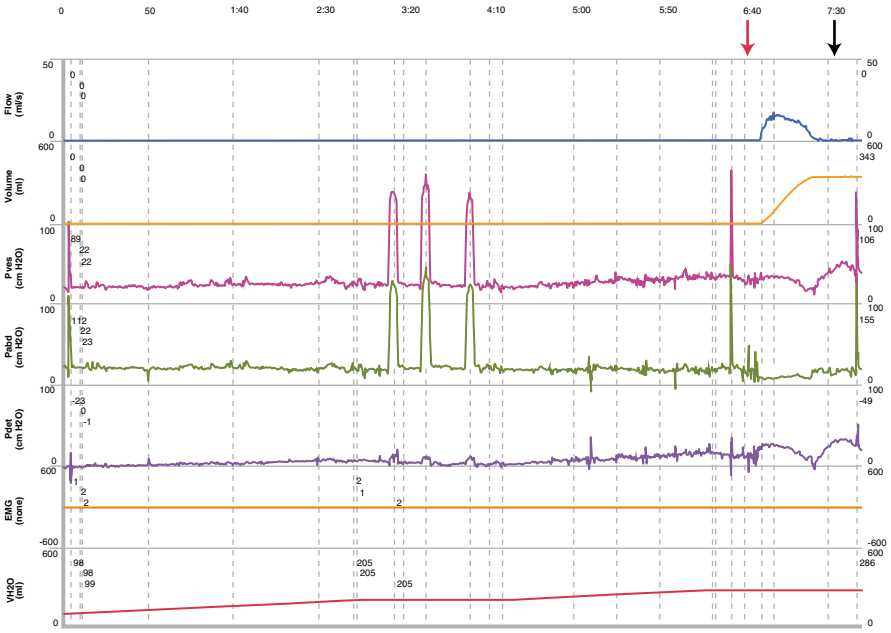

Fig. 16.8 Urodynamic tracing of a male with post-prostatectomy incontinence and low voiding detrusor pressure. Note the sustained detrusor contraction after he urinates. Permission to void denoted by *red arrow*. After contraction denoted by *black arrow*

Abnormal Detrusor Contractility

Clinical Scenario 8 (Fig. 16.8)

Detrusor after contraction

A 59-year-old man with post-prostatectomy incontinence. In this urodynamic study, note the sustained detrusor contraction after he urinates, denoted by second arrow. The detrusor after contractions may be an expression of sphincter contraction interrupting an incomplete detrusor contraction. It has been reported mostly in connection with urge incontinence and urge symptoms.

Clinical Scenario 9 (Fig. 16.9)

"Super Voider"

A 37-year-old male complains of dysuria and urinary frequency. His UDS tracing demonstrates high detrusor pressure (P_{det} 78 cm H_2O) while voiding and a high peak flow rate (Q_{max} 24 mL/s).

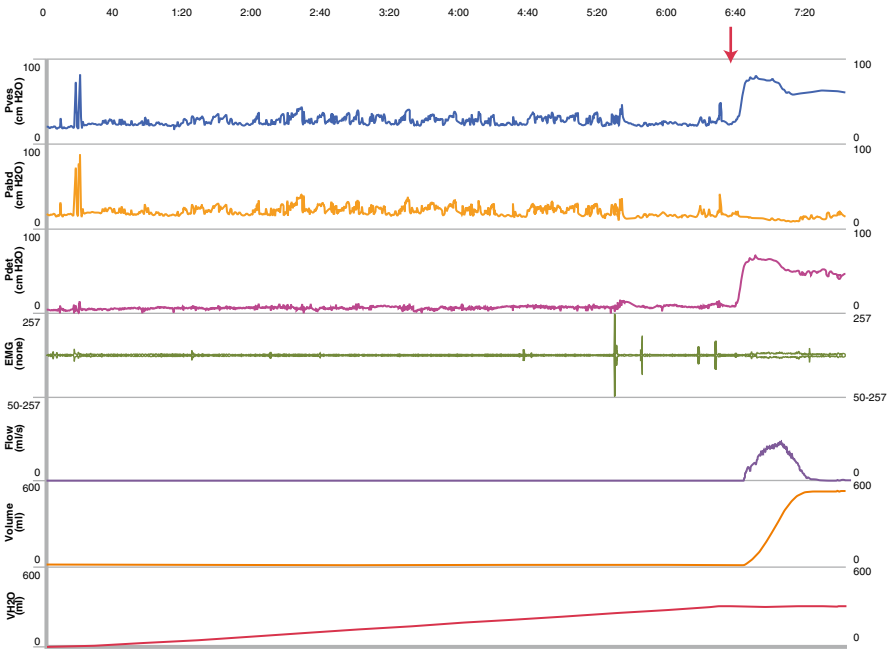

Fig. 16.9 Urodynamic tracing of a male voiding at high detrusor pressure and high flow rate. Permission to void denoted by *red arrow*

Detrusor Hyperactivity and Impaired Contractility

First described in 1987 [29], detrusor hyperactivity with impaired contractility (DHIC) is characterized on urodynamics by low-amplitude involuntary detrusor contractions during filling that are often associated with urethral relaxation, as well as impaired bladder emptying, and occurs predominantly in the elderly [30]. Presentation of DHIC can be variable and is often misdiagnosed. Females may be misdiagnosed with intrinsic sphincter deficiency (ISD), whereas males are more often misdiagnosed with bladder outlet obstruction (BOO); both of which may result in unnecessary surgery [30].

Treatment of DHIC can be challenging given the paradoxical nature of the voiding disorder [29]. Management strategies depend on the symptoms, degree of bother, and general health status in addition to urodynamic findings. Conservative measures of fluid restriction, pelvic floor exercises, and bladder retraining can provide satisfactory results in some patients [30]. In the presence of OAB predominant symptoms despite trial of conservative measures, low dose anticholinergics or a beta-3 agonist may be initiated with careful attention to post-void residuals (PVR) and related side effects for each type of medication; particularly cognitive

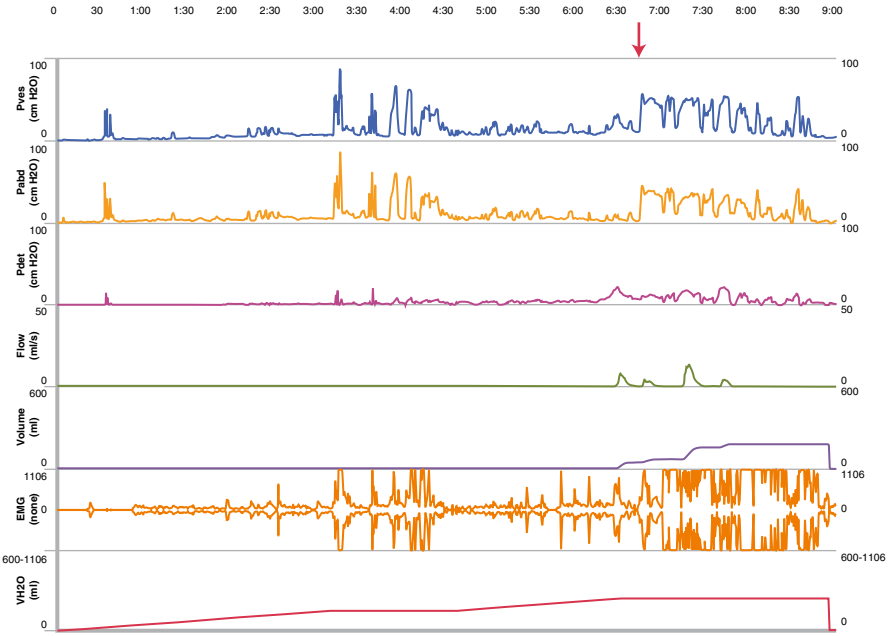

Fig. 16.10 Urodynamic tracing in an elderly female with detrusor hyperactivity and impaired contractility consistent with DHIC. Note terminal DO just prior to permission to void. Permission to void denoted by *red arrow*

impairment with anticholinergic use [30, 31]. If predominant symptoms are due to impaired bladder emptying, alpha blockers may help reduce outlet resistance and improve emptying particularly in male patients. Mixed symptoms can be safely managed with combination therapy of alpha blockers with anticholinergics or a beta-3 agonist. Intermittent catheterization (IC) is commonly employed to manage elevated PVR in DHIC [30]. Prompted voiding every 2 h can be beneficial for cognitively impaired and frail elderly. When all other approaches fail in the setting of high PVRs or large volume incontinence, protective incontinence devices and indwelling catheterization may be the only option.

Clinical Scenario 10 (Fig. 16.10):

DHIC in a female

A 73-year-old female complains of frequency, urge incontinence, and sensation of incomplete emptying. She has no prior history of GU surgery. Her past medical history is significant for diabetes and hypertension. She wears three thick incontinence pads per day.

Her urodynamics demonstrate reduced MCC of 245 mL. SUI was not seen with cough or valsalva during filling. She has urgency incontinence with terminal detrusor overactivity at 245 mL. After permission (denoted by red arrow), she voids primarily by straining and does not generate very high detrusor pressures. Q_{max} *is*

14 mL/s with an intermittent stream. Determination of P_{det} is especially challenging in the setting of valsalva voiding. She has a residual of 60 mL. This picture is suggestive of detrusor hyperactivity with impaired contractility.

The quality of this study is fair but the increases in P_{abd} from movement make interpretation challenging as it is not perfectly clear if incontinence episode is purely associated with an uninhibited detrusor contraction. It is best to have the clinician and an experienced urodynamics technician present at the time of urodynamics to increase the accuracy of diagnosis particularly when there is significant artifact present.

Clinical Scenario 11 (Fig. 16.11)

DHIC in a male

An 82-year-old male with history of prostate cancer treated by external beam radiation 10 years ago presents with urgency and urge incontinence. He has also had bladder cancer previously treated with intravesical instillations. He has been on tamsulosin for many years and finds mild improvement with it. He has tried multiple anticholinergics without any benefit. He wears eight pads per day and leaks throughout the day and night.

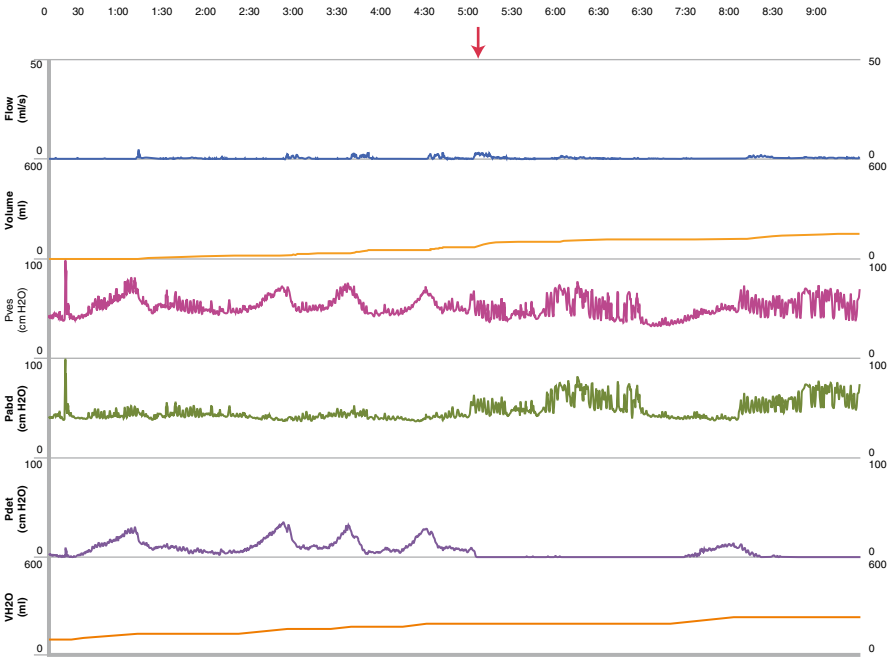

Fig. 16.11 Urodynamic tracing in an elderly male with combination of detrusor overactivity and impaired bladder emptying with no significant rise in detrusor pressure during voiding. This pattern is suggestive of DHIC. Permission to void denoted by *red arrow*

Urodynamics were performed as part of his investigation with a fill rate of 50 mL/s. He has phasic detrusor overactivity with small volume incontinence starting at 133 mL. MCC is estimated at 235 mL although may be less given his episodes of incontinence during the filling phase.

After permission (denoted by red arrow), he voided by straining with very a low-amplitude detrusor contraction. Flow curve is flattened and prolonged. PVR was 177 mL. This pattern is suggestive of detrusor hyperactivity impaired contractility and is consistent with his history.

The quality of this study is relatively poor. It could have been more appropriately zeroed so the P_{det} line would not be less than zero (negative) for the majority of the study. The clinician should have also decreased the rate to try to reduce the phasic detrusor contractions. Another flaw with this tracing is that the initial filling cystometrogram is missing. The interpreter recognizes this as the instilled volume does not start at zero.

Management of this patient is challenging as his predominant complaint is due to urgency/urge incontinence but he also empties his bladder very poorly. A TURP may not provide significant symptom relief in this setting, and generally should be avoided. Having failed medical management of urge incontinence, onabotulinumtoxinA injections could be considered and may be of some benefit to reduce his overactivity and leakage, though he would very likely require intermittent catheterization after such a treatment.

Valsalva Voiding

Valsalva voiding typically refers to the presence of straining or "muscular effort to initiate, maintain or improve the urinary stream [13]." Some series have further specified that valsalva voiding occurs when detrusor pressure (P_{det}) at time of bladder contraction is ≤10–15 cm H_2O when accompanied by abdominal straining to initiate micturition [32–34]. The flow pattern on uroflowmetry has a sawtooth pattern with a corresponding sawtooth pattern in the abdominal (P_{abd}) and vesical (P_{ves}) pressure tracers. Urodynamic studies of men with post-prostatectomy incontinence have found valsalva voiding in 30–53 % [35–37]. Valsalva voiding may occur in conjunction with BOO, detrusor underactivity, SUI, post-prostatectomy incontinence, and even in normal individuals [38]. In series of SUI females undergoing urodynamics preoperatively, 12–30 % had valsalva voiding [32, 34].

Clinical scenario 12 (Fig. 16.12):

Post-prostatectomy valsalva voider

A 63-year-old man post-radical prostatectomy complains of stress urinary incontinence and prolonged, weak stream. He does not have any UTIs. His PSA remains undetectable. Cystoscopy shows no bladder neck contracture.

He was assessed for SUI which was present at several points during filling (denoted by first arrow). His compliance appears to decrease slightly at the end of fill. He also has a low-amplitude detrusor contraction with urge incontinence at the

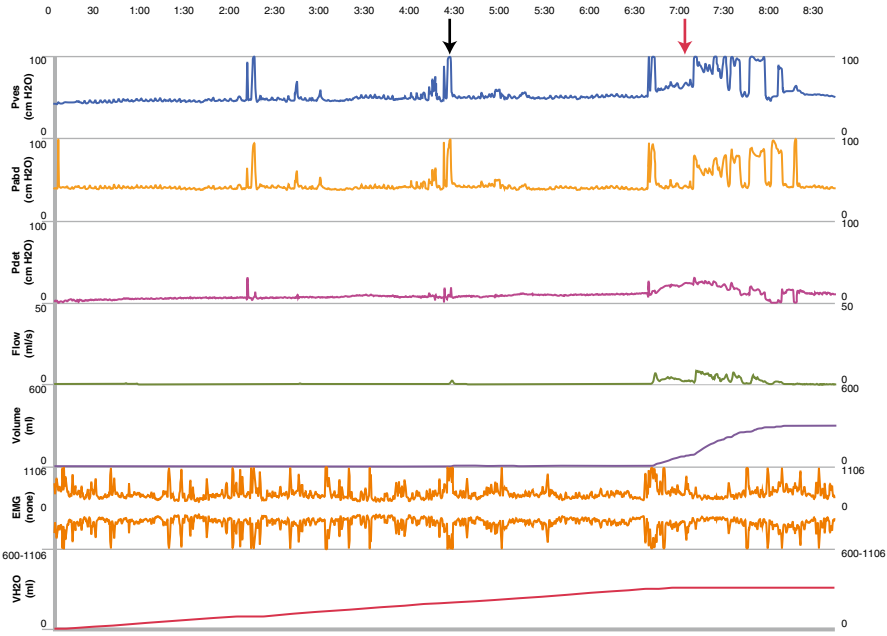

Fig. 16.12 Urodynamic tracing in a male who urinates with a valsalva voiding pattern. Stress urinary incontinence denoted by *black arrow*. Permission to void denoted by *red arrow*

end of fill (perhaps induced by valsalva). He then valsalva voids to empty his bladder (denoted by second arrow). This sawtooth pattern in P_{abd} *and* P_{ves} *with an intermittent flow is characteristic of the valsalva voiding pattern. He voids to completion with no PVR. The quality of this tracing is good, free from significant artifact.*

Clinical scenario 13 (Fig. 16.13):

Female who strains to empty

A 53-year-old female complains of frequency, a weak stream, and sensation of incomplete emptying. She has had a hysterectomy but denies other surgery. She denies history of UTIs or any known neurological disease.

Her urodynamics demonstrate first sensation at 99 mL. SUI was assessed and not demonstrated at 220 mL. Her MCC is slightly reduced at 272 mL. Compliance is normal. When she voids, she Valsalva voids with a characteristic sawtooth pattern. Her overall voiding time is prolonged and flow is intermittent. True P_{det} *cannot be reliably assessed in the presence of valsalva voiding but it is clear that her detrusor pressures are not elevated. She voids 304 mL and does not have a PVR. EMG was not performed in this study. The quality of this study is adequate as it is nicely zeroed and free from significant artifact. The apparent and artifactual rises in detrusor pressure during voiding are a result of slightly unequal transmission in vesical and abdominal pressures (artifact).*

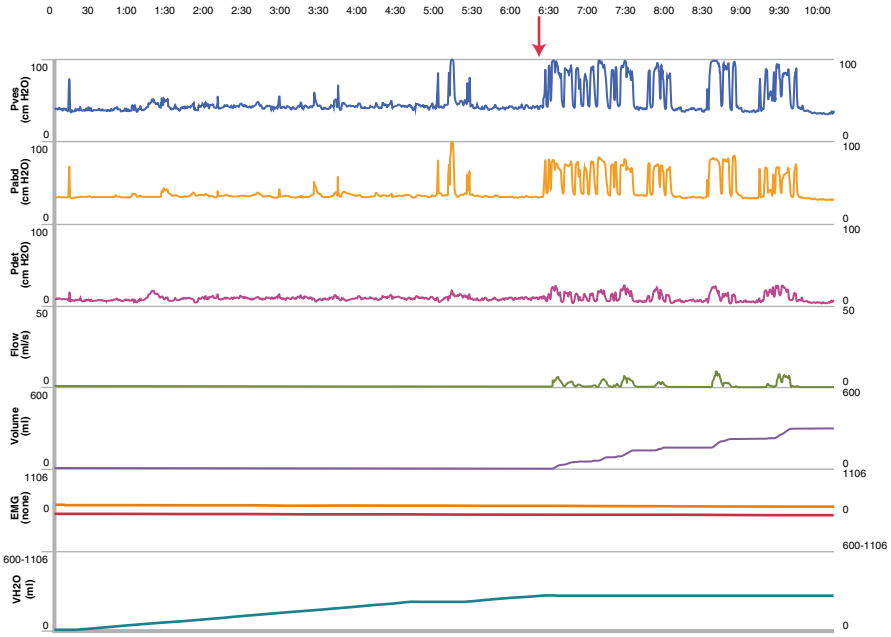

Fig. 16.13 Urodynamic tracing in a female with valsalva voiding. Note the characteristic saw-tooth pattern in P_{abd} and P_{ves}. Permission to void denoted by *red arrow*.

Voiding Dysfunction and Pelvic Floor Dysfunction

Voiding dysfunction secondary to pelvic floor dysfunction (PFD) (also referred to as dysfunctional voiding) can affect both men and women. Its exact incidence is unknown but is estimated to affect at least 2 % of adults [39]. It may manifest by symptoms of urgency, frequency, hesitancy, weak or intermittent stream, incomplete emptying, double voiding, incontinence, dysuria, constipation, and even recurrent urinary tract infections [39, 40]. Although dysfunctional voiding is most commonly seen in children, a number of patients with a long standing history of mixed lower urinary tract symptoms may not be diagnosed until adulthood [39–41]. PFD is thought to be a learned behavior which may date back to childhood toilet training [39, 40]. It can occur in combination with defecatory dysfunction/constipation, pelvic or genital pain, and sexual dysfunction [42]. Neurologic causes of dysfunctional voiding should often be excluded in patients given this diagnosis. Adults with dysfunctional voiding tend to have predominantly obstructive and irritative LUTS compared to children who more frequently present with recurrent UTI and incontinence [39].

The International Continence Society has attempted to standardize terminology and defined dysfunctional voiding as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated muscle during voiding in neurologically normal individuals" [13, 43, 44]. Clues suggestive of dysfunctional voiding based on a clinical history of LUTS may include difficulty urinating in public places, needing special techniques to relax or concentrate in order to urinate, listening to running water or touching the genitalia to prompt voiding; intermittent voiding pattern on non-invasive flow; and free of neurologic or anatomic causes for voiding abnormality [39, 45]. In patients with dysfunctional voiding, the external urinary sphincter may contract during voiding on EMG or during fluoroscopy generally as a sign that the pelvic floor is non-relaxing. Unlike neurogenic patients who have true detrusor-external sphincter dyssynergia (DESD), EMG activity of the external urethral sphincter in non-neurogenic patients decreases just prior to the detrusor contraction followed by an increased EMG activity during detrusor contraction [39]. Behavior modification, physical therapy using techniques such as myofascial release or pelvic floor biofeedback and addressing other symptoms such as constipation may help alleviate symptoms [46]. Hinman's syndrome (non-neurogenic neurogenic bladder) is a severe form of dysfunctional voiding in which the external urinary sphincter contracts during voiding. It is usually diagnosed in childhood and if unrecognized, can result in a severely trabeculated, decompensated bladder and irreparable upper tract damage [39].

Fowler's Syndrome

Fowler's syndrome is a special category of voiding dysfunction characterized by urinary retention and abnormal inability of external urinary sphincter relaxation in young women without neurologic disease or clear attributable cause [47, 48]. This condition has most often been reported in women under 30 years of age with development of urinary retention of nearly or greater than 1 L urine without a strong urge to urinate [47–49]. The condition is diagnosed by needle electromyogram (EMG) of the external urinary sphincter which demonstrates repetitive complex discharges and decelerating bursts of EMG activity during voiding. Increased urethral closure pressure is also often seen [47]. The underlying etiology of Fowler's syndrome is unclear [48, 50]. A current hypothesized cause of Fowler's syndrome is a "hormone-sensitive channelopathy" resulting in increased activity of the external urethral sphincter [47]. Nearly 50 % of Fowler's syndrome patients also have polycystic ovaries [47, 49, 51]. Neurologic examination and investigation including MRI of the central nervous system is unremarkable. Attempts to identify predisposing psychological factors have been inconclusive [50]. In roughly 70 % of patients, the first episode of urinary retention may occur following a surgical procedure.

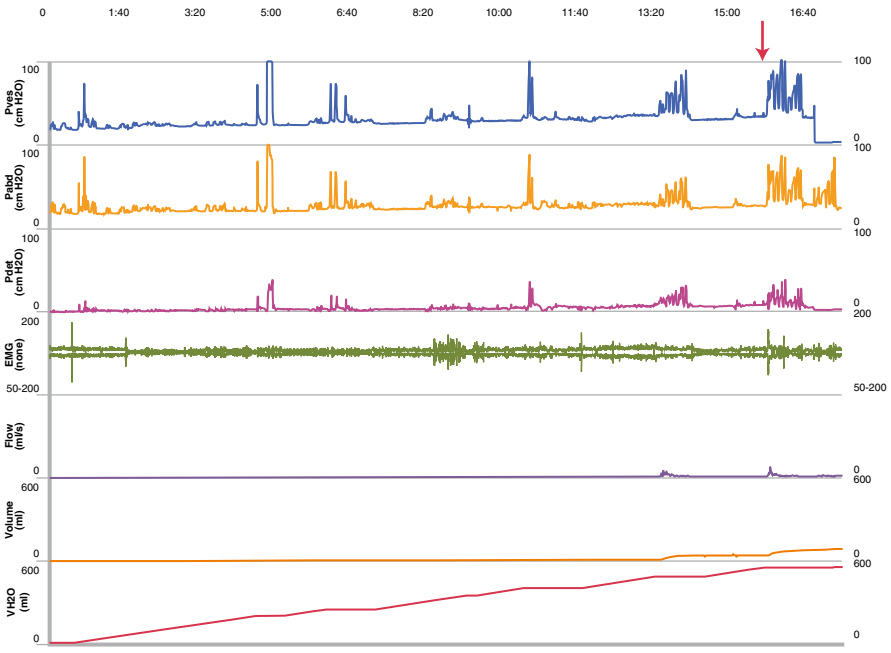

Fig. 16.14 Urodynamic tracing in a female with dysfunctional voiding. Note the presence of abdominal straining, elevated post-void residual, and increased EMG activity. It is not possible to determine if the increased EMG activity is due to the artifact of straining or due to pelvic floor contraction. Permission to void denoted by *red arrow*

Clinical scenario 14 (Fig. 16.14):

Difficulty voiding in female

A 28-year-old female noted to be in urinary retention of 1,500 mL 3 days after non-GU related surgery. She has no known neurological disease or symptoms. Her urodynamics demonstrated a MCC of 600 mL. She had normal compliance. She attempted to void (at red arrow) by abdominal straining and was unsuccessful leaving significant post-void residual. There was increased EMG activity in her attempts to void which could be either due to her straining or inadvertent recruitment of her pelvic floor muscles.

In patients who do not spontaneously recover their ability to void after first initiating intermittent catheterization, sacral neuromodulation can be offered. Women with urinary retention and diagnosis of Fowler's syndrome may have a higher likelihood of success with sacral neuromodulation compared to females retention [50]. Treatment of Fowler's syndrome using medications and onabotulinum toxin A injection has been largely unsuccessful [50].

Conclusion

Bladder contractility is a measure of detrusor muscle contractile strength and it can be affected by a variety of factors. Urodynamics is the only established tool used to evaluate bladder contractility and thus the overall bladder function. During assessment of the bladder contractility, flow rate must also be taken into consideration as outlet resistance can greatly affect bladder contractility. Understanding the relationship between bladder contractility and outlet resistance relation is of paramount importance. A careful analysis of the urodynamic tracing along with a thorough understanding of the clinical scenario can aid tremendously in a better understanding of the pathophysiology and help guide treatment recommendations.

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