# Voiding Dysfunction

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Voiding dysfunction in women is a common but poorly characterized problem [1]. The label of voiding dysfunction is not really a "diagnosis" but represents a subset of patients with lower urinary tract dysfunction with either symptoms or evaluation suggestive of a problem with the voiding phase of micturition.

## Terminologies and Evaluation

The ICS-IUGA terminologies document defines voiding dysfunction by symptoms and urodynamic investigations as an "abnormally slow and/or incomplete micturition" [2]. Hence, the condition affects primarily voiding rather than storage although many patients may have associated storage symptoms. Some patients may present exclusively with storage symptoms. Urodynamically, it is a "reduced urine flow rate and/or presence of a raised PVR and an increased detrusor pressure" diagnosed by a simultaneous measure of pressure and flow [2].

Voiding dysfunction broadly speaking can result from poor bladder contractility or outlet obstruction. Abnormal bladder contractility during voiding may be classified as detrusor underactivity or acontractility. ICS-IUGA defines detrusor underactivity as "Detrusor contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span" [2]. While acontractility is defined as a detrusor that "cannot be observed to contract during urodynamic studies resulting in prolonged bladder emptying and/or failure to achieve complete bladder emptying within a normal time span" [2].

Furthermore, the term "detrusor underactivity" or "detrusor acontractility" implies that the failure of the bladder to contract stems from abnormalities of the detrusor alone. This fails to recognize that afferent (sensory), efferent (including myogenic factors), and central CNS factors can all be responsible in varying degrees in different patients. Hence, a more generic term "bladder underactivity" has been proposed [3, 4].

Dysfunctional voiding is defined as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated or levator muscles during voiding in neurologically normal women" [2]. Classical detrusor sphincter dyssynergia that occurs in neurogenic bladder is outside the scope of this discussion.

The limitation of the aforementioned definition is readily apparent on further analysis. While the definition mentions abnormally slow and/or incomplete micturition, cutoffs for what constitutes normal flow or emptying are not well defined. The time duration in which the normal

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bladder should empty itself is also not clearly defined, making it difficult to apply the yardstick of "complete bladder emptying within a normal time span." Moreover, applicability of Western nomograms to Indian patients has pitfalls [5]. There are limitations to the ICS documentation for dysfunctional voiding [6]. Specifically, not all patients with dysfunctional voiding without an underlying neurological problem will show an intermittent flow and the terminologies documents need to make allowance for this finding.

There are no universally accepted criteria for defining bladder outlet obstruction in women [1]. In adult men, prostatic obstruction serves as an excellent model of reversible bladder outlet obstruction and this has helped improve understanding of bladder physiology in men. It is, however, universally accepted that criteria for men cannot be extrapolated to women since many women void with pelvic floor relaxation without a major detrusor contraction. Blaivas and Groutz defined a nomogram for women recommending that women with a maximum flow rate (tube-free) of <12 ml/s and $P_{det.}$ $Q_{max}$ of >20 cm $H_2O$ should be categorized as obstructed [7].

All women with suspected voiding dysfunction must be evaluated in detail by a clinical assessment and judicious investigations (see Table 10.1). Those with high-risk factors such as hydronephrosis, renal dysfunction, or reflux must undergo a full urological evaluation and detailed urodynamics since some of these women might be at risk for long-term renal dysfunction (see Fig. 10.1).

**Table 10.1** Basic clinical evaluation for voiding dysfunction

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|---|
| History |
| *Urinary tract*: details of storage and voiding symptoms, urinary infections |
| *Gastrointestinal tract*: any constipation or encopresis |
| *Neurological evaluation*: neurological screening, lower limb and higher function assessment |
| *Gynecological evaluation*: assess for symptoms of vaginal or uterine disease, obstetric history |
| *Others*: history of relevant surgery |
| Physical examination |
| *Genitourinary*: meatus, labia and vagina, bladder, kidneys |
| *Gastrointestinal*: rectal examination, fecal incontinence |
| *Neurological system*: specific attention to anal sphincter tone, bulbocavernosus reflex, perianal sensation, gait, spine |
| Investigations |
| Bladder diary, urine examination, blood glucose |
| USG for the bladder with prevoid and post-void bladder volume |
| Uroflow with EMG, invasive urodynamics or videourodynamics |
| MRI of the spine, micturating cystourethrogram |

## Etiology of Voiding Dysfunction

There are two broad categories of problems that can present with voiding dysfunction (see Table 10.2). These are detrusor muscle weakness and bladder outlet obstruction. Iatrogenic voiding dysfunction may occur following pelvic surgery such as stress urinary incontinence surgery. Finally, storage abnormalities such as detrusor overactivity may occasionally present with symptoms suggestive of voiding dysfunction.

## Detrusor Underactivity and Acontractility

Detrusor muscle strength can range from normal to acontractility in a continuum. Gross detrusor dysfunction can easily be identified by a constellation of clinical and urodynamic findings including poor flow, large residuals, and obvious detrusor muscle weakness on urodynamics. Unfortunately, many women present with less obvious features.

Detrusor muscle weakness is an important cause of voiding dysfunction which may exist in isolation or in combination with bladder outlet obstruction. Poor detrusor muscle strength varies in a continuum from mild weakness (detrusor underactivity) to a total loss of contractility (acontractility) (see Fig. 10.2). There are several causes for detrusor underactivity (see Table 10.2). Detrusor underactivity is widely prevalent in older women with an estimated prevalence of 12–45 % [4]. It has long been observed that older

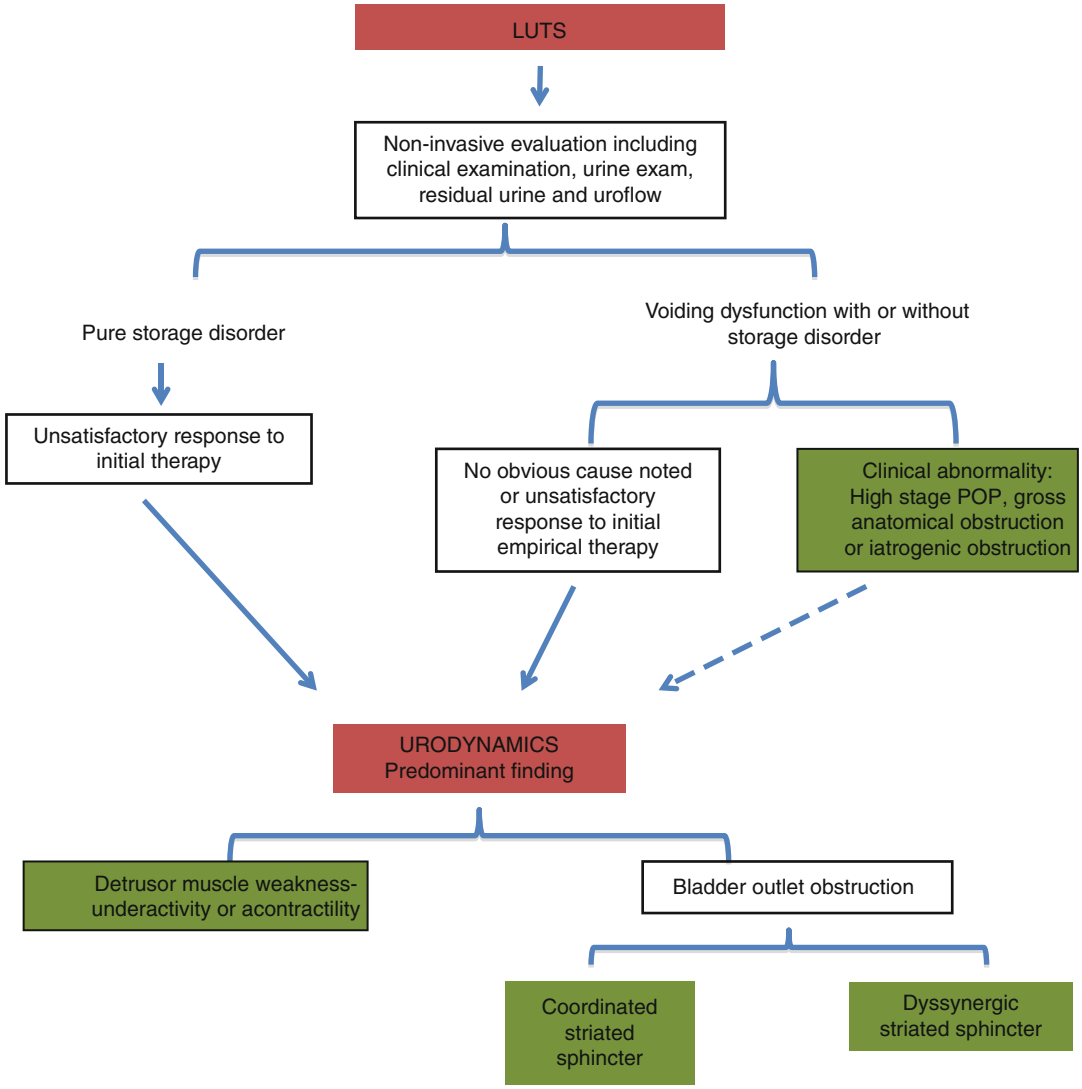


**Fig. 10.1** Algorithm for evaluation of voiding dysfunction in women

individuals are more likely to present with underactivity. Research indicates up to two-thirds of elderly nursing home residents show detrusor underactivity. Bladder biopsy reveals changes in the detrusor muscle including axonal degeneration, loss of muscle fibers, and fibrosis [8].

Detrusor underactivity can also occur due to untreated bladder outlet obstruction or retention due to any other cause. Chronic or severe acute stretch injury can lead to detrusor dysfunction. Postpartum urinary retention is a classic example of such injury.

Diabetes is an important medical cause of underactive detrusor. The classical clinical picture of patients with florid diabetic cystopathy is one of acontractility. Such patients usually have long-standing diabetes of more than 10 years' duration and often have other evidence of diabetic peripheral neuropathy. However, studies have shown a higher prevalence of other patterns such as overactive bladder in patients with diabetic cystopathy [9]. Diabetes impacts all levels of neuromuscular control of the lower urinary

**Table 10.2** Etiological classification and diagnosis of voiding dysfunction in women

| |
|---|
| A. Detrusor muscle weakness (underactive or acontractile detrusor) |
| 1. Idiopathic |
| 2. Aging |
| 3. Drug induced |
| 4. Untreated long-standing obstruction |
| 5. Diabetic cystopathy |
| 6. Neurogenic dysfunction, e.g., after a radical hysterectomy |
| 7. Postpartum retention |
| 8. Severe fibrosis of the bladder wall |
| B. Bladder outflow obstruction |
| I. With coordinated sphincteric activity (presumed or documented) |
| 1. Pelvic organ prolapse |
| 2. Urethral stricture |
| 3. Functional bladder neck obstruction |
| 4. Iatrogenic following urethral or other pelvic surgeries |
| 5. Large fibroid |
| 6. Urethral diverticula |
| 7. Urethral leiomyoma |
| II. With dyssynergic sphincteric activity |
| A. No underlying neurological cause apparent |
| B. Neurological cause present |
| C. Classical detrusor sphincter dyssynergia |

tract. There is altered afferent sensation possibly linked to changes in the urothelium and detrusor. Afferent and efferent nerves demonstrate degenerative changes in function. Depending on the relative impact on different aspects of lower tract dysfunction, one can expect a wide variety of bladder manifestations [10].

Several drugs can impact lower urinary tract function either by exacerbation of a preexisting voiding dysfunction or by inducing acute-onset symptoms in otherwise normal individuals [11]. Drugs with anticholinergic side effects such as antidepressants and antipsychotic drugs as well as calcium channel blockers are some of the examples. Older women are more likely to be impacted by these side effects due to alterations in neurotransmission, receptor sensitivity, and blood-brain barrier defects. Also, elderly women are more likely to have preexistent lower tract dysfunction such as underactive detrusors that might make them especially susceptible to the adverse effects.

Neurogenic lower urinary tract dysfunction can produce a variety of effects on the lower urinary tract. Classically, lesions above the brainstem do not have an impact on detrusor contractility. Some studies have shown up to 6 % of patients with CVA have acontractile detrusors [12]. It is difficult to judge what proportion of this might be due to preexisting detrusor dysfunction in this elderly age group. Detrusor underactivity may conceivably occur following CVA.

Unrecognized postpartum retention can be a disaster for the bladder with short- or long-term damage. This is now a rare event in the developed world. Pifarotti et al. noted postpartum retention in 105 out of 11,108 (1 %) women undergoing a vaginal delivery. They defined postpartum retention as inability to void within 6 h of delivery. On multivariate analysis, only vacuum-assisted delivery and uterine fundal pressure during the second stage of labor were noted to be independent risk factors [13].

Protracted second stage of labor and vacuum delivery were noted to be risk factors in another large study [14]. In this study, 55 women (0.18 %) developed postpartum retention. Of these, two-thirds recovered by 2 weeks and the remaining by 4 weeks. However, the author has seen anecdotal instances of long-term voiding dysfunction. This is unusual in the Western world where strict labor room protocols are enforced and women are identified before they develop florid bladder damage.

## Bladder Outlet Obstruction with Coordinated Striated Sphincter Activity (Synergic Striated Sphincter)

Pelvic organ prolapse (POP) is an important cause for lower urinary tract dysfunction in women. However, one must remember that the only symptom of feeling a vaginal bulge shows consistent correlation with POP. No urine symptoms have been shown to be consistently associated with POP. The situation might be different in women who primarily present with voiding dysfunction and have severe prolapse. Uncorrected

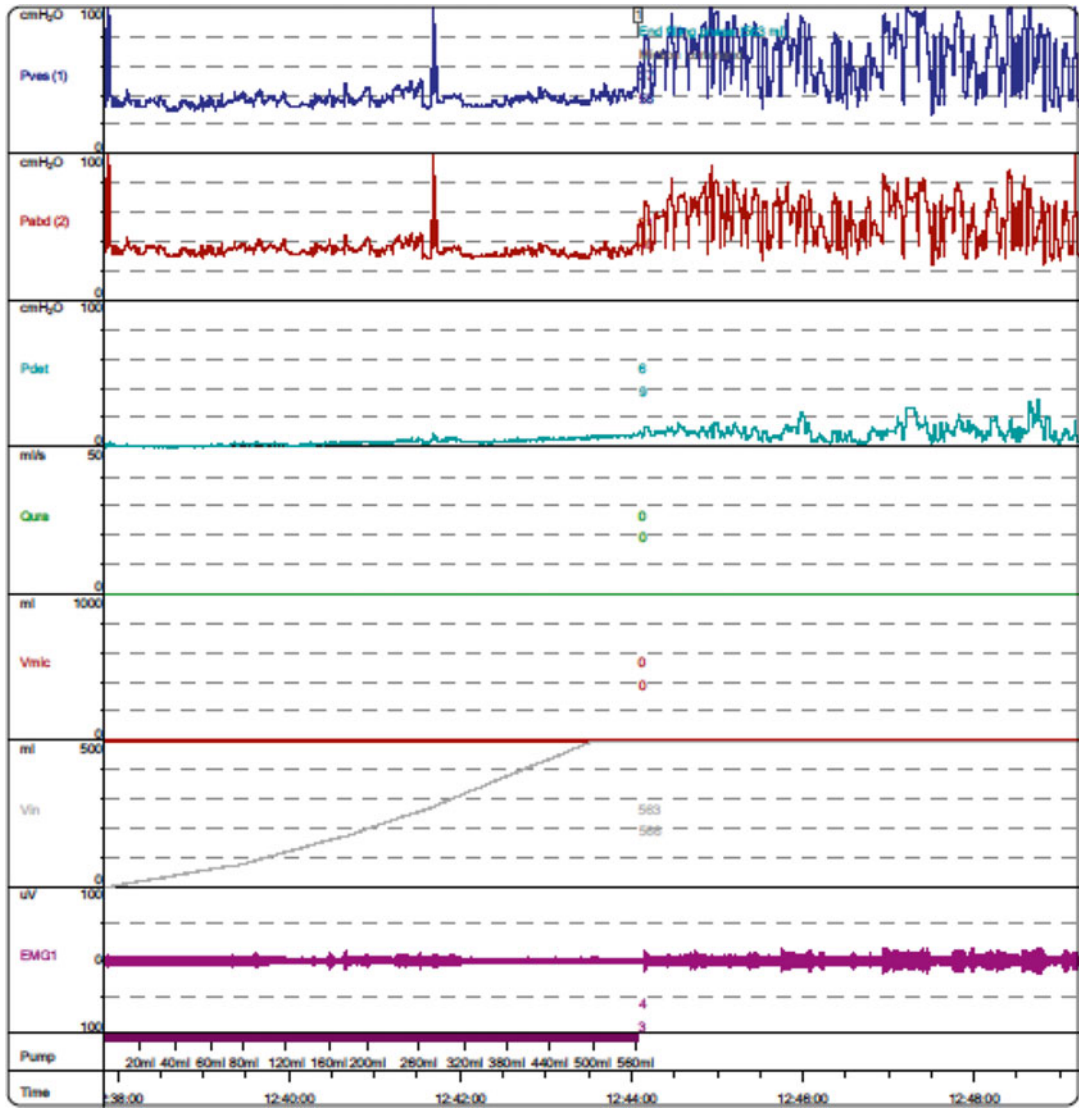


**Fig. 10.2** Acontractile detrusor in a 67-year-old diabetic woman with voiding difficulty and storage symptoms for 2 years. One must exercise caution in making this diagnosis. One may find "acontractility" in an uncooperative (but otherwise normal) woman

prolapse may alter the dynamics of the lower urinary tract and elevated residuals are common in such women. In women with significant POP, which usually implies that the prolapsing organ is beyond the introitus, an important clinical question is whether correcting the prolapse will improve voiding. Such women may habitually reduce the prolapse before voiding [15]. One method of resolving this issue is to use a pessary and perform urodynamic evaluation or flow tests with and without the pessary. Women with obvious improvement in lower urinary tract function are likely to improve with surgery (see Fig. 10.3).

Genuine urethral stricture in women is an uncommon entity. Conversely, a large number of women continue to be subjected to urethral dilatation for various forms of lower urinary tract dysfunction.

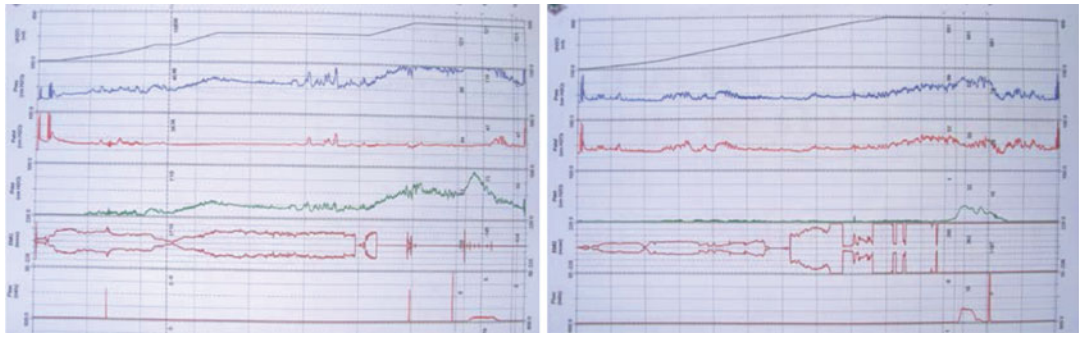

**Fig. 10.3** Urodynamics evaluation without (*left*) and with (*right*) a pessary in a 60-year-old woman with grade III pelvic organ prolapse and voiding dysfunction. Substantial improvement in voiding function (reduced voiding detrusor pressure and better flow rates) was noted upon placement of a pessary. She had complete resolution of voiding dysfunction following surgery for prolapse

Functional bladder neck obstruction is less common in women since the bladder neck smooth muscle is not as well organized into a sphincter in women as compared to men (see Fig. 10.4). As such, in most series only a small number of women have urodynamically confirmed functional bladder neck obstruction [16]. However, in a recent prospective multicenter study from China, the commonest form of voiding dysfunction was noted to be functional bladder neck obstruction. It is unclear whether this result represents methodological differences, variations in diagnostic criteria, or a genuine difference in prevalence [17].

Large uterine fibroids can have an impact on urinary tract function. This is more likely in women, when the uterine size is over 12 weeks of gestation size. These women often complain of voiding difficulty and the symptoms usually improve following surgery [18]. Isolated case reports have found leiomyoma of the urethra as a cause of voiding difficulty [19].

## Dysfunctional Voiding

The ICS-IUGA terminology defines dysfunctional voiding as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the peri-urethral striated or levator muscles during voiding in neurologically normal women" [2]. As discussed in a previous review, this author has made a case for modifying this definition to reflect the two key components of dysfunctional voiding, namely, dyssynergia of the striated urethral sphincter-pelvic floor complex and lack of a clear neurological etiology [6]. In a small but significant proportion of these patients, neurological abnormalities may manifest subsequently [20]. Hence, it is important to evaluate these patients carefully for subtle clues to an underlying neurological abnormality and to perform additional assessment judiciously, including MRI of the spine or other forms of nerve-muscle evaluation. In some women, dysfunctional voiding may represent faulty toilet training in childhood. A related condition in which women with polycystic ovarian disease have urinary retention due to an increased sphincteric tone that has hormonal associations has been termed "Fowler's syndrome" [21].

Most women will have a "staccato flow" with fluctuations in excess of the square root of the maximum flow (see Fig. 10.5). Some women may have slow but non-staccato flow or even normal flow rates [6]. Hence, in suspected dysfunctional voiding, urodynamic evaluation is important for diagnosis and management.

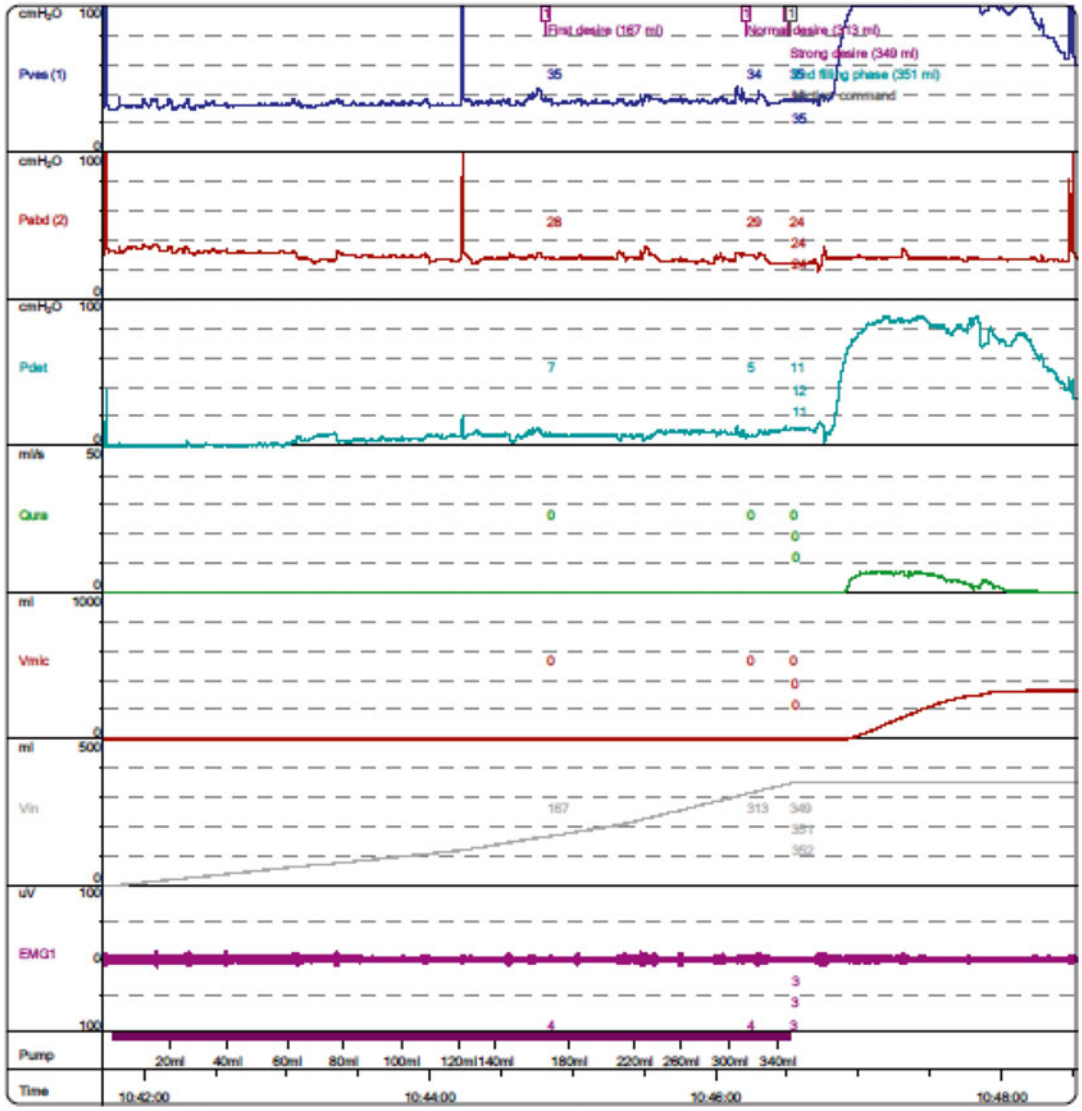


**Fig. 10.4** Functional bladder neck obstruction in a 46-year-old nondiabetic woman with voiding difficulty, frequency, and nocturia. It is important to search for consistent findings across multiple cycles of testing preferably including a tube-free uroflow with EMG

## Detrusor Sphincter Dyssynergia

Classical detrusor sphincter dyssynergia that occurs in women with neurological disease represents a significant urological problem and a full discussion on this condition is outside the scope of this chapter. A brief summary of the usual neurological features of patients with classical neurogenic bladder follows.

Patients with lesions above the brainstem typically present with detrusor overactivity with coordinated sphincters. However, patients with Parkinsonism may show a delayed ability to relax the striated sphincter presenting with a form of voiding dysfunction. This delayed relaxation has also been termed "bradykinesia" of the striated sphincter [22]. Parkinsonism occurs in 3 % of both men and women above the age of 65

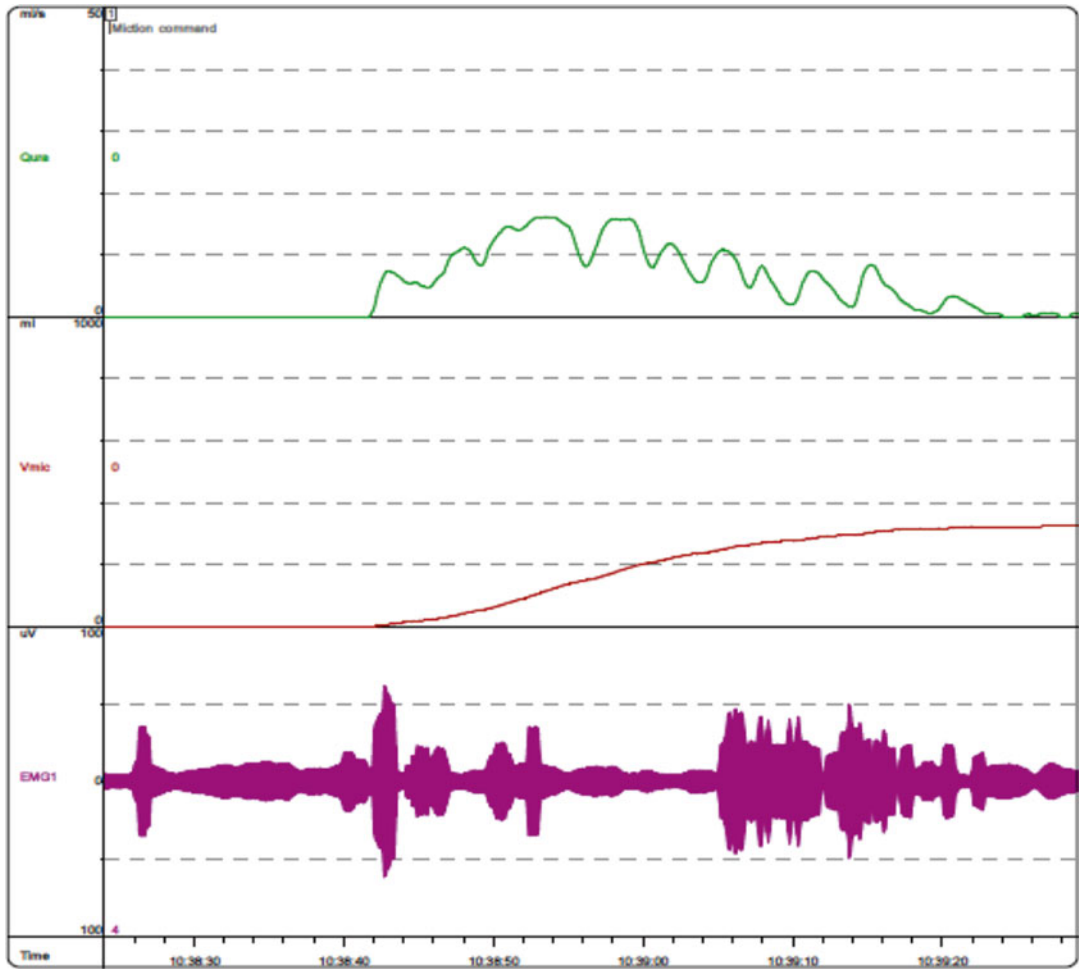


**Fig. 10.5** Staccato voiding in a 40-year-old woman with voiding difficulty. Note the EMG contractions during voiding. This finding is only significant if it is clinically consistent and documented on repeated assessment

years. Patients with cerebrovascular accidents have synergic sphincteric activity. In some patients with severe detrusor overactivity, the sphincteric response to phasic contractions may be confused as dyssynergia, and this has been termed "pseudo-dyssynergia." True dyssynergia does not occur in patients with cerebrovascular accidents. Multiple sclerosis patients usually show detrusor overactivity but about half of these patients with storage abnormalities have dyssynergic voiding [23, 24].

Patients with spinal cord injuries most often show both storage and voiding phase abnormalities (see Fig. 10.6). In lesions above T6 level, there is usually detrusor overactivity with dyssynergia of both the smooth and striated sphincter. In lesions between T6 and S2, the striated sphincter alone is most often dyssynergic. In patients with sacral lesions, the sphincter classically demonstrates a fixed and increased tone (non-relaxing sphincter) along with acontractility of the detrusor and an incompetent smooth sphincter. One must however remember the dictum that the level of neurological lesion is not a reliable guide for management. Bladder management in patients with neurogenic bladder is based on urodynamic findings rather than neuroimaging or neurodiagnosis [24].

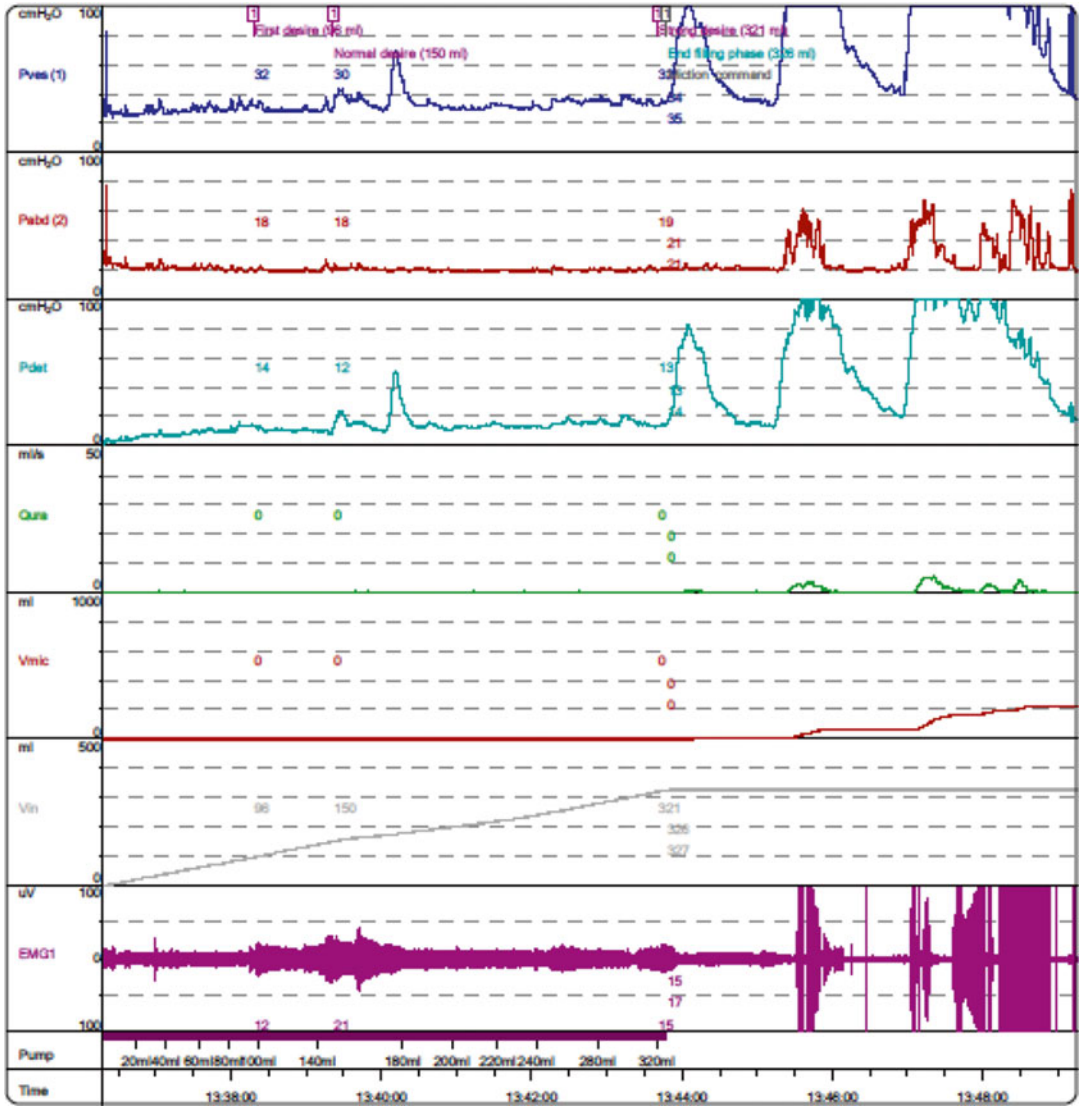


**Fig. 10.6** Classical neurogenic bladder in a 16-year-old girl operated for tethered cord at the age of 7 years. Note the detrusor overactivity and reduced compliance during storage and the severe bladder outlet obstruction with classical detrusor sphincter dyssynergia during voiding

## Management of Voiding Dysfunction

Treatment of voiding dysfunction is dependent on the clinical setting, the type of abnormality noted, and the risk for consequential damage such as back-pressure effects or infection (see Table 10.3).

## Detrusor Underactivity and Acontractility

In patients with an underactive detrusor, lifestyle changes can be quite useful [25]. Avoiding overdistension of the bladder is the key. Fluid restriction and reduction in intake of dietary diuretics

**Table 10.3** Treatment of female voiding dysfunction

| |
|---|
| Timed voiding |
| Lifestyle changes including fluid restriction |
| Pharmacotherapy |
| Surgery for pelvic organ prolapse |
| Clean intermittent self-catheterization |
| Urethral dilatation |
| Optical urethrotomy |
| Intraurethral botulinum toxin |
| Neuromodulation |
| Surgical reconstruction including urinary diversion, augmentation cystoplasty, or other major lower tract reconstructive surgeries |

such as tea, coffee, and alcohol can reduce the overall urine volume. This should be combined with timed voiding every 2–3 h during the day. Patients should be instructed to void "by the clock" rather than waiting for a desire to void. Many of these patients have impaired bladder sensation and their strong desire to void may be delayed. Waiting for bladder sensations to trigger a void can lead to progressive overdistension of the bladder and ongoing detrusor damage. The bladder may contract best at a fill of about 300–350 ml and hence limiting the bladder volume to this level can help. Double and triple voiding 20 min after the initial void can also help in minimizing residual urine [25]. There are no medications that can reliably increase the bladder's ability for a coordinated contraction. Although bethanechol is a popular drug, there is no objective evidence to suggest benefit and the drug should not be used for this purpose [26]. Diabetic patients should observe good glycemic control to reduce the progressive neuromuscular damage due to hyperglycemia. In patients with severe underactivity or acontractility, clean intermittent self-catheterization is the solution [25, 27]. Patients are instructed to empty their bladder five times a day using a small catheter that they are taught to self-insert by a clean (as contrasted to a sterile) technique. Catheters can safely be reused several times.

## Bladder Outlet Obstruction with Coordinated Sphincters

In women with bladder outlet obstruction with coordinated sphincters, one must address the primary cause of obstruction. Those with obvious anatomical narrowing of the urethra (in extreme cases, a urodynamics study will, in fact, not be possible), the urethra can be dilated with a successful and often durable outcome. However, urethral dilatation is a much misused therapy, and women with all and sundry urine symptoms, are offered urethral dilatation without rationale. Used as such, this therapy is either ineffective or has limited or short-term efficacy at best and can lead to long-term urethral incompetence and stress incontinence at worst. In women with true functional bladder neck obstruction, alpha blockers are often effective and should be tried [28]. Not all women with a functional bladder neck obstruction on urodynamics respond to alpha blockers and this begs the question whether the diagnosis is correct (in other words, has a dyssynergic void been missed) or is the obstruction too severe for oral medication to work (just as alpha blockers do not relieve obstruction in all men with severe functional bladder neck obstruction). A clear answer is currently not forthcoming from literature since most urologists are reluctant to perform a surgical correction for functional bladder neck obstruction for the fear of producing iatrogenic incontinence. There are sporadic reports in literature regarding bladder neck incision in women either by use of a "cold" knife of some kind (under vision or blind) or by the use of electrocautery or laser. A pediatric scope has been recommended by some authors in view of the shorter length of the female urethra and the challenges in obtaining good vision and precise incision using regular instruments designed for adult males [29].

In women with high-grade prolapse, surgical repair of prolapse may resolve urinary symptoms. However, one must counsel such

women carefully. A preoperative urodynamics done with and without vaginal pessary can sometimes predict the resolution of urinary symptoms. One must recollect that the only symptom that reliably correlates with pelvic organ prolapse is a feeling of bulge in the vagina. There is limited evidence that women with large fibroids may benefit from surgery for the fibroid. Again, one must be careful in prognosticating such women since it is by no means certain that removing a fibroid will resolve urinary symptoms. A better approach is to tackle the fibroid on merits and observe for any benefit in urinary symptoms [30].

Iatrogenic urethral obstruction following surgery for stress urinary incontinence is a subject in itself and beyond the scope of this chapter. Briefly, the diagnosis of such an obstruction is challenging since women can present with an array of symptoms, not all of which are voiding symptoms. Many women have storage symptoms alone and are noted to have poor voiding efficiency only on investigations. Certainly, any woman with new-onset voiding difficulty following stress incontinence surgery needs to be carefully assessed for iatrogenic obstruction. No single test can prove obstruction (unless the obstruction is "gross," which is unusual). A constellation of suggestive symptoms and signs along with poor uroflow, increase in residuals, worsening of voiding dynamics (in those who had a preoperative urodynamics study), and cystoscopic evidence of anatomical obstruction can suggest iatrogenic obstruction. Intervention in such women is controversial. In those with gross obstruction, the tape can be divided, usually to one side of the midline. Early intervention at or before 3 weeks might reduce the odds of long-term persistent storage symptoms [25]. Satisfactory voiding is restored in about 75 % and about 40 % have recurrent stress urinary incontinence following tape division [31]. Hence, recurrence of stress incontinence is not a certainty following tape division. In most women, a formal urethrolysis is not necessary and the entire tape need not be removed, unless there is associated sepsis.

## Dysfunctional Voiding

Patients with dysfunctional voiding need to be triaged on the basis of their presentation. Those patients with high-risk markers such as reflux, hydronephrosis, marked voiding difficulty, or renal dysfunction should undergo urodynamic evaluation at the outset [6]. Patients with primarily storage symptoms can undergo initial management based on uroflow with surface electromyography recording. However, one must have a low threshold for offering urodynamics in those who fail to respond satisfactorily. All patients with dysfunctional voiding must undergo a basic neurological evaluation and select patients who need more detailed assessment including MRI to search for underlying neurological abnormality [20].

All patients need to be educated about their problem. Some patients may benefit from biofeedback and aggressive constipation care. Biofeedback consists of repeated voiding with simultaneous auditory or visual feedback to the patient regarding striated sphincter activity. In some women, this may help to resolve a "habitual" striated sphincter dyssynergia [32]. Alpha-adrenergic blockers are usually not useful but can be tried in patients with safe voiding pressures [6]. Patients with very high voiding pressures can potentially damage their upper tracts and such patients should not be offered alpha-adrenergic blockers since it is unlikely to work and a more effective treatment option must be chosen.

Botulinum toxin injection into the sphincter has been tried in dysfunctional voiding with mixed results. The injection is usually given periurethrally in women. Fifty to hundred units are diluted in saline and injected into four sites representing four quadrants of a circle [33]. The effect of the injection starts in about 1–2 weeks and can last for several months.

Sacral neuromodulation can also be useful in select patients. In women with the Fowler's syndrome, 78 % of women continued to void spontaneously 10 years following InterStim sacral neuromodulation implantation [34]. However, the procedure carries a high revision rate of about 30–50 % and failure rate of about 30 %.

Clean intermittent self-catheterization is the mainstay of therapy and helps bypass the sphincteric mechanism.

## Detrusor Sphincter Dyssynergia

There is no oral medication for classical detrusor sphincter dyssynergia. Treatment revolves around clean intermittent self-catheterization [25]. Additional urodynamic and clinical findings need to be addressed individually. Many patients have concomitant storage pressure abnormalities which need to be managed. Storage abnormalities may consist of various combinations of neurogenic detrusor overactivity, reduced compliance and reduced capacity. Anticholinergics are the first-line treatment with intra-detrusor botulinum toxin and augmentation cystoplasty reserved for nonresponders. Sphincterotomy and stents are not a good option in women. Isolated reports suggest that intrasphincteric botulinum toxin might help in reducing voiding pressures but experience with this is not uniformly good [35]. In women with limited ambulation or other severe comorbidities, an indwelling catheter may represent an acceptable salvage option.

### Conclusion

Managing voiding dysfunction in women is complex as identification of the causative factor itself is composite, varying from anatomical abnormalities to neurological (either peripheral or central). The clinical evaluation including urodynamic assessment has a major role in the management algorithm. The management options are varied and not necessarily aimed at addressing the pathology but rather the symptom mostly. In a significant proportion of patients' catheterization, either clean intermittent or continuous drainage forms the mainstay of treatment.

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