Neurogenic Bladder Obstruction

9

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Abbreviations

ALS	Amyotrophic lateral sclerosis
ALPPs	Abdominal leak point pressures
AD	Autonomic dysreflexia
cm	Centimeters
cc	Cubic centimeter
EMG	Electromyography
CNS	Central nervous system
CVA	Cerebrovascular accident
DSD	Detrusor sphincter dyssynergia
H_2O	Water
MRIs	Magnetic resonance imaging studies
mL	Milliliter
mL/s	Milliliters per second
MS	Multiple sclerosis
MM	Myelomeningocele
PD	Parkinson's disease
pDet max	Maximum detrusor pressure on urodynamics
qMax	Maximum urinary flow on urodynamics
UTIs	Recurrent urinary tract infections
SCI	Spinal cord injury
VUR	Vesicoureteral reflux
VUDS	Videourodynamics

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9.1 Introduction

Neurogenic voiding dysfunction refers to disease pathways impacting the function of the afferent and efferent nerve fibers of the somatic and autonomic nervous systems, which innervate the lower genitourinary tract. The term "obstructive voiding" may in and of itself be misleading, as a neurogenic bladder may be unable to empty, not only because of functional obstruction but because of hypocontractility as well. Thus, perhaps a more comprehensive conceptual framework is to think of this as neurogenic urinary retention. From a urological perspective, when managing these patients, we are not actually treating the disease; we are treating their symptoms. The treatment is based on the ability of the bladder and the urethra to store and empty effectively.

The brain stem is responsible for control of coordinated bladder contraction and pelvic floor relaxation. Cortical and subcortical centers can modulate these sacral reflexes as well [1]. Centers mediating micturition are located within the S2 to S4 sacral area of the spinal cord (including parasympathetic innervation). This part of the spinal cord actually sits at the T12 to L1 vertebral level, which is important to know at times of traumatic injury. Thoracolumbar (sympathetic) output from the T9 to L1 area of the spinal cord also participates in regulation of micturition. As mentioned previously, disturbances of the afferent or efferent innervation pathways can cause neurogenic urinary retention with obstruction being one of these manifestations.

Cortical, subcortical, brain stem, and spinal cord (thoracolumbar or sacral) lesions, in addition to peripheral radiculopathy or neuropathy, can all impact function of the lower genitourinary tract. Neurogenic voiding dysfunction can be complete or incomplete, sensory or motor, central or peripheral, acute or chronic, and reversible or irreversible. It impacts bladder compliance, detrusor activity, smooth

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sphincter activity, striated sphincter activity, and sensation in varying fashions [2]. Therefore, neurogenic voiding dysfunction can be exhibited as a result of neurologic insults from a wide range of disease processes and trauma: spinal cord injury (SCI), cerebrovascular accident (CVA), multiple sclerosis (MS), Parkinson's disease (PD), myelomeningocele (MM), amyotrophic lateral sclerosis (ALS), diabetes mellitus, acute transverse myelitis, cervical myelopathy, poliomyelitis, tabes dorsalis, pernicious anemia, and sacral root/pelvic plexus surgery (i.e., radical pelvic surgery and spinal surgery) [3].

Of all the described etiologies, MS patients, with detrusor sphincter dyssynergia (DSD), are perhaps some of the most representative of neurogenic obstruction. MS is an autoimmune disease of the central nervous system (CNS) with an extremely variable clinical course. It is described as relapsing-remitting or progressive and is defined by chronic inflammation, gliosis (scarring), demyelination, and neuronal loss [4]. Lesions occur with temporal variability at different locations throughout the CNS. Physiologically, one of the main effects of MS demyelination is to cause discontinuity in saltatory electrical conduction of nerve impulses from one node of Ranvier, the location of concentrated sodium channels, to the next node, resulting in electrical transmission failure [5]. The clinical patterns of MS include the following:

1. Relapsing-remitting (affecting 55–65 %, sudden neurologic decline that resolves over 4–8 weeks)
2. Secondary progressive (affecting 25 %, develops from relapsing-remitting)
3. Primary-progressive (affecting 10 %, most initial symptoms usually motor and continuous)
4. Progressive-relapsing (affecting 5 %, aggressive onset with rapid worsening of symptoms) [6]

When evaluating patients with possible neurogenic bladder, including patients with MS, although urodynamic tracings can be completed without a video component, fluoroscopy during these studies offers a rich collection of information, including description of a possible functional obstruction (if it exists and where it is in the tract, i.e., bladder neck, urethra), the state of the bladder (severely trabeculated or smooth), and if there is evidence of high pressures contributing to upper tract deterioration (i.e., vesicoureteral reflux (VUR), dilated ureters). In certain instances, performing a urodynamics study without a video component (or least a post-void residual/bladder scan and a cystogram/upper tract imaging) could be misleading; a decompensated neurogenic bladder with hydroureteronephrosis may have a low filling pressure because the body has already enacted "the pop-off valve" of the upper tract, accommodating for chronically high filling/storage pressures. Without the video component, simply using a cystometrogram tracing to interpret low-pressure filling in a neurogenic bladder may not provide all the important information (the patient may have severe VUR, with associated upper tract dilation). Three case studies will now review various patient presentations, with their associated urodynamic studies.

9.2 Case Studies

9.2.1 Patient 1

9.2.1.1 History

The patient is a 55-year-old gentleman with a history of C5–C6 quadriplegia status post a motor vehicle accident with subsequent cervical fusion (1979), with obstructive sleep apnea, gastroesophageal reflux disease, and neurogenic bladder status post a sphincterotomy (1983), recently with recurrent urinary tract infections (UTIs) and more frequent episodes of autonomic dysreflexia (AD), presenting to clinic for follow-up. He currently empties his bladder through a combination of Valsalva and cutaneous trigger (scratching his thigh with his fingertip or lying supine and tapping his suprapubic area), with urine draining into an external condom catheter he wears at all times.

At times of infection, he develops headaches, chills, diaphoresis, flank pain, and rise in his blood pressure (consistent with his usual AD symptoms). He has been treated for symptomatic UTIs every 2–3 months over the last 18 months, including two hospitalizations for pyelonephritis (presented to the emergency department febrile). He also develops AD at times when his bladder is significantly distended or he is experiencing severe constipation. There is no gross hematuria. His every-other-day bowel regimen includes suppositories, fiber, docusate, and senna. For many years, he has been medically managing his baseline AD symptoms with phenoxybenzamine 10 mg by mouth twice daily. He uses baclofen 20 mg by mouth twice daily for muscle spasm relief. He functions independently and is able to use a motorized wheelchair to get around.

9.2.1.2 Physical Examination

Generally he is in no apparent distress when sitting up in his wheelchair. His upper extremities are contracted, with 3/5 strength and no sensation to light touch (he is not able to hold a pen and squeeze the digits of his hands together). His lower extremities are atrophied. His neck is supple and trachea is midline. Skin is warm and dry. Abdomen is soft, nontender, and nondistended. Genitourinary exam reveals an in-place external condom catheter. The penile skin is intact, with no excoriations. Testes are descended bilaterally, with no pal-

pable masses. Digital rectal exam reveals intact tone, with a 40 g, smooth prostate.

9.2.1.3 Labwork/Other Studies

Post-void residuals as measured by bladder ultrasound were 437 and 397 cc in clinic (additional recent post-void residuals were also documented between 300 and 500 cc). A urine analysis was not checked, secondary to his chronic use of a condom catheter and his lack of symptoms of infection at time of evaluation in clinic. His most recent serum creatinine was 0.4 mg/dL, and estimated glomerular filtration rate (eGFR) was >89 mL/min/1.73 m^2. A CT of his abdomen and pelvis found no evidence of renal mass, hydronephrosis, or nephrolithiasis. Cystoscopy did not reveal any intravesical abnormalities such as stones, tumors, or diverticula.

9.2.1.4 UDS

See Figs. 9.1, 9.2, and 9.3.

A multichannel videourodynamics (VUDS) was performed in the supine position. The condom catheter was carefully removed without any injury to his penile skin. Initial catheterization revealed a 400 cc residual bladder volume. A rectal catheter was placed for intra-abdominal pressure measurements. A separate 7-French dual-lumen catheter was placed in the bladder. Catheters were zeroed, and filling with Cysto-Conray was begun at 30 cc/min. The filling phase of the study revealed a compliant bladder with low filling pressures. He was able to leak with cough, with abdominal leak point pressures (ALPPs) measured at 60–75 cm H_2O. Initial continuous blood pressure monitoring revealed stable blood pressures ranging from 140/65 to 160/55. As he approached a bladder volume of 600 cc, he started to experience sweats and headache, and another check of his blood pressure revealed it was 180/85. Concerned he was developing AD, the volume infusion was halted.

Fluoroscopic images revealed the bladder neck was open, but his external sphincter did not open. There was no VUR at a volume of 600 mL. He was able to empty another 100 mL with strain. His bladder was then drained of 550 cc. His sweats and headache resolved. His blood pressure returned to 140/65.

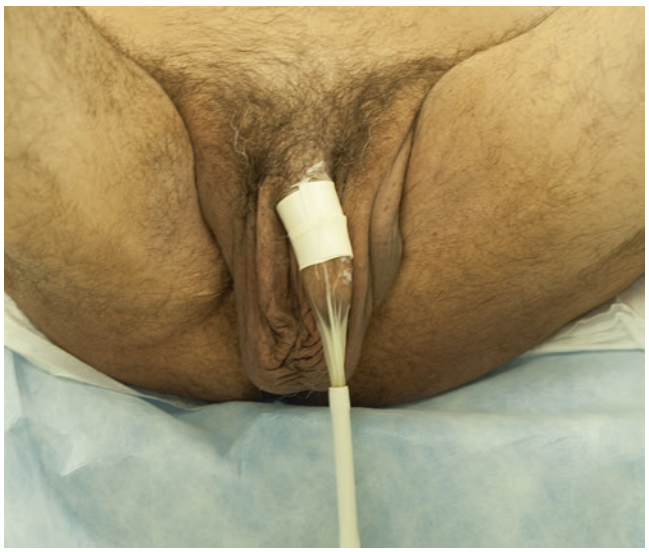

Fig. 9.1 Drainage into an external condom catheter

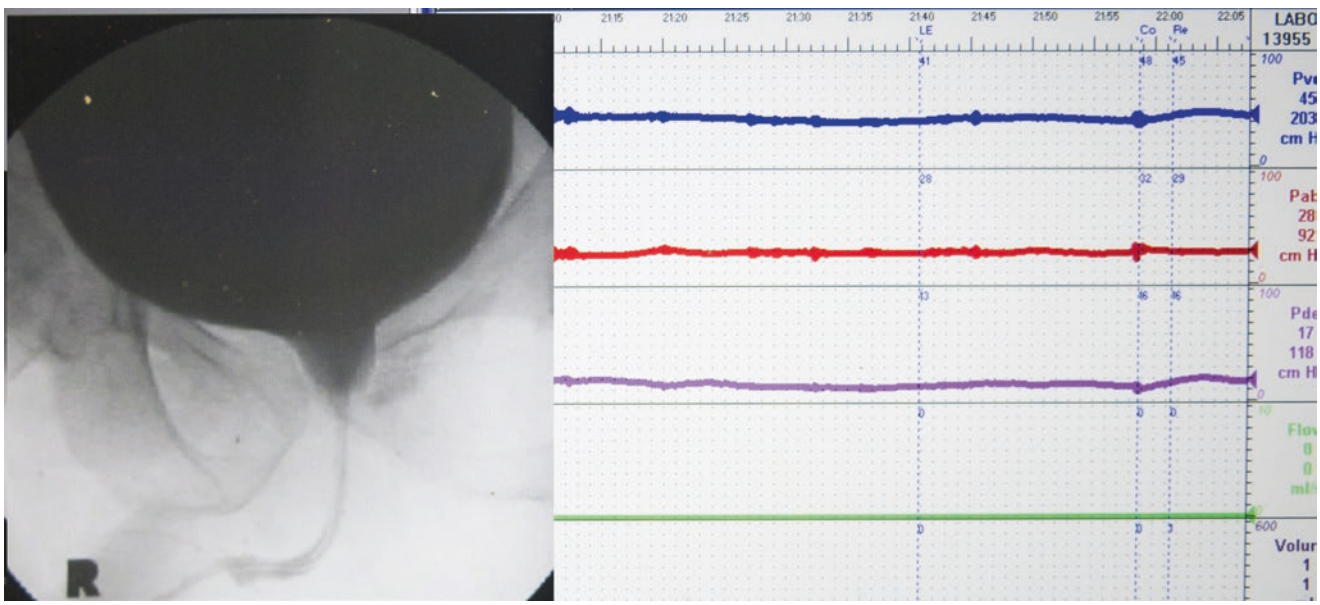

Fig. 9.2 Low pressure filling in a decompensated, hypocontractile bladder

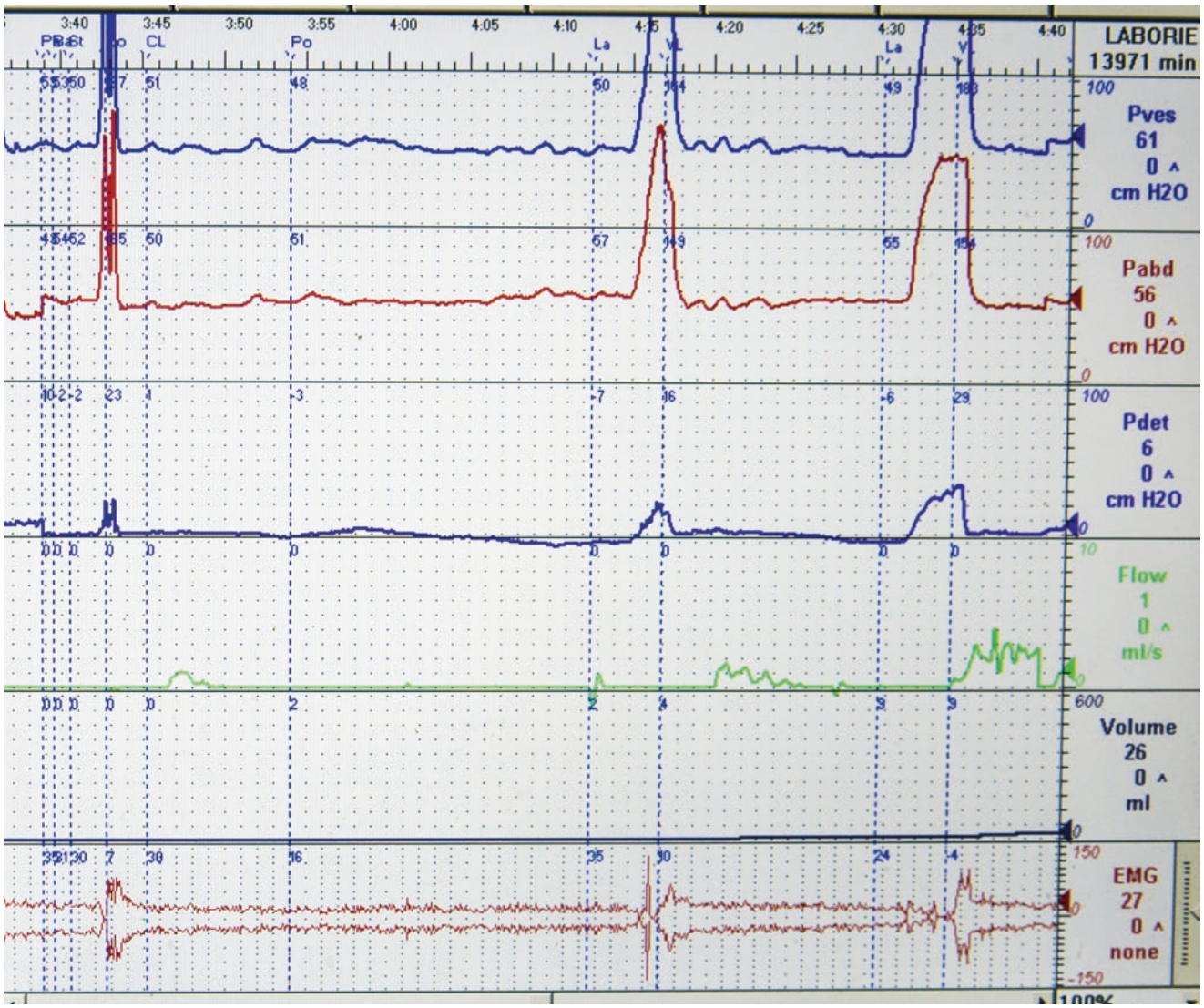

Fig. 9.3 Abdominal leak point pressures

Findings

The patient has normal compliance. Despite previous sphincterotomy, he has evidence of a bladder which has decompensated over time, with hypocontractility, and an external sphincter which does not open. The external sphincter dysfunction is characteristic of a neurological lesion causing lack of relaxation of the pelvic floor. He has no voluntary control over the external sphincter and is not able to completely empty his bladder, with residuals of urine of approximately 300–500 cc at a time. This incomplete emptying puts him at risk for recurrent infection. His AD manifests more frequently, secondary to bladder distension and even more so at times of symptomatic infection. Fortunately, his bladder decompensation and lack of sensation did not impact his upper tract.

9.2.1.5 Treatment Options

He is essentially allowing his bladder to currently empty through overflow incontinence. Management possibilities include the following: commit to intermittent catheterization at least three times a day (but this would require a dedicated caregiver, secondary to his poor dexterity), closure of the bladder neck and creation of an incontinent ileal chimney, another sphincterotomy, or placement of an indwelling catheter (urethral or suprapubic). Considering he is already managing his bladder with urinary leakage into an external condom catheter, he will likely be most effectively served with another sphincterotomy. For now, he has elected to think about his options further; his upper tracts have no evidence of hydronephrosis, renal function is appropriate, he has normal compliance, and there is no VUR. There is not an

acute need for immediate action. While awaiting his decision, he will initiate methenamine hippurate 1 g by mouth twice daily, for UTI prophylaxis.

9.2.2 Patient 2

9.2.2.1 History

The patient is a 43-year-old gentleman with a history of MS, neurogenic bladder, incomplete emptying, and persistent urinary urgency, urge incontinence, and frequency, presenting to clinic for follow-up. He manages his bladder with a mix of self-void and self-catheterization, currently voiding every 1–2 h, with occasional urgency urinary incontinence, and catheterizing three times a day, per his report. He has three to four episodes of nocturia per night as well. He had initially tried oxybutynin (both immediate and extended release formulations) without significant improvement in his urinary symptoms. He saw a mild improvement in his urgency and frequency with the combination of tamsulosin 0.4 mg and fesoterodine fumarate 8 mg daily. He takes baclofen 10 mg by mouth twice daily to aid with baseline muscle spasms. He is treated for a UTI every 3–4 months. He denies gross hematuria.

He continues to have some trouble with memory and attention. He denies any changes with vision. He is taking 100 mg of amantadine daily. He continues disease-modifying therapy with glatiramer given subcutaneously three times a week. He continues to take vitamin D 5000 units daily, and his vitamin D level was recently checked by his primary care provider at his annual physical and is reportedly within normal limits. He continues to walk for exercise. Compared to a year ago, there is nothing that he could do then that he is unable to do now.

9.2.2.2 Physical Examination

Generally he is in no apparent distress when sitting up on the examination table. There is full 5/5 strength throughout. Deep tendon reflexes are symmetric and brisk. Sensation to light touch is intact in all dermatomes. Neck is supple. Trachea is midline. Skin is warm and dry. Abdomen is soft, nontender, and nondistended. His lower extremities are atrophied. Genitourinary exam reveals a circumcised phallus and intact glans and meatus. Testes are descended bilaterally, with no palpable masses. Digital rectal exam reveals intact tone, with a 50 g, smooth prostate.

9.2.2.3 Labwork/Other Studies

Post-void residual was not checked, as he catheterizes three times a day to empty his bladder. A urine analysis was not checked, secondary to his intermittent catheterization and his lack of symptoms of infection at time of evaluation in clinic. His most recent serum creatinine was 0.8 mg/dL and eGFR was >89 mL/min/1.73 m^2. Renal ultrasound found no hydronephrosis, obvious masses, or perinephric fluid collections. Cystoscopy did not reveal any intravesical abnormalities such as stones, tumors, or diverticula. MRI imaging of the brain, cervical spine, and thoracic spine documented numerous non-enhancing T2-hyperintense foci scattered throughout the cerebral white matter, posterior fossa, cervical spinal cord, and thoracic spinal cord. No enhancing lesions identified.

9.2.2.4 UDS

See Figs. 9.4, 9.5, and 9.6.

A multichannel VUDS was performed in the upright position. He was initially catheterized for a 70 cc residual (he had voided 20 min prior to the study and self-catheterized 3.5 h before that). A rectal catheter was placed for intra-abdominal pressure measurements. A separate 7-French dual-lumen catheter was placed in the bladder. Catheters were zeroed and filling with Cysto-Conray was begun at 30 cc/min. The filling phase of the study revealed a compliant bladder with low filling pressures. There were multiple short involuntary detrusor contractions associated with urgency multiple times between 174 and 246 cc. He leaked with these contractions at a pDet max of 64 cm H_2O, at a volume of 237 mL. At a capacity of 246 cc, he attempted to void and mounted a bladder contraction with a Qmax flow of 6 mL/s, with a pDet max during void of 87 cm H_2O, and with a residual of 210 cc. On the fluoroscopic images, there was poor funneling of the bladder neck during attempted void. There was no VUR.

Findings

On the urodynamics, the patient has evidence of a small-capacity bladder, with significant detrusor overactivity associated with urgency urinary incontinence. His bladder neck does not funnel well during voiding, causing inability to empty the bladder. He has normal compliance and no evidence of upper tract damage (i.e., hydronephrosis or vesicoureteral reflux). Considering his underlying MS diagnosis, he may have had chronic obstruction over time from DSD, with subsequent thickening of the bladder wall. A thick, trabeculated bladder wall can contribute to lack of funneling of the bladder neck during attempted void.

9.2.2.5 Treatment Options

His current bladder management of mixed self-void with intermittent catheterization may be yielding a poor quality of life for him. Considering his underlying neurologic dysfunction, any procedure addressing the outlet (i.e., a transurethral incision of his bladder neck or sphincterotomy) would possibly make him even more incontinent. Placement of a suprapubic catheter may create more urinary urgency and urgency incontinence for him. Sacral neuromodulation could be considered, but MS patients often are monitored with MRIs, and

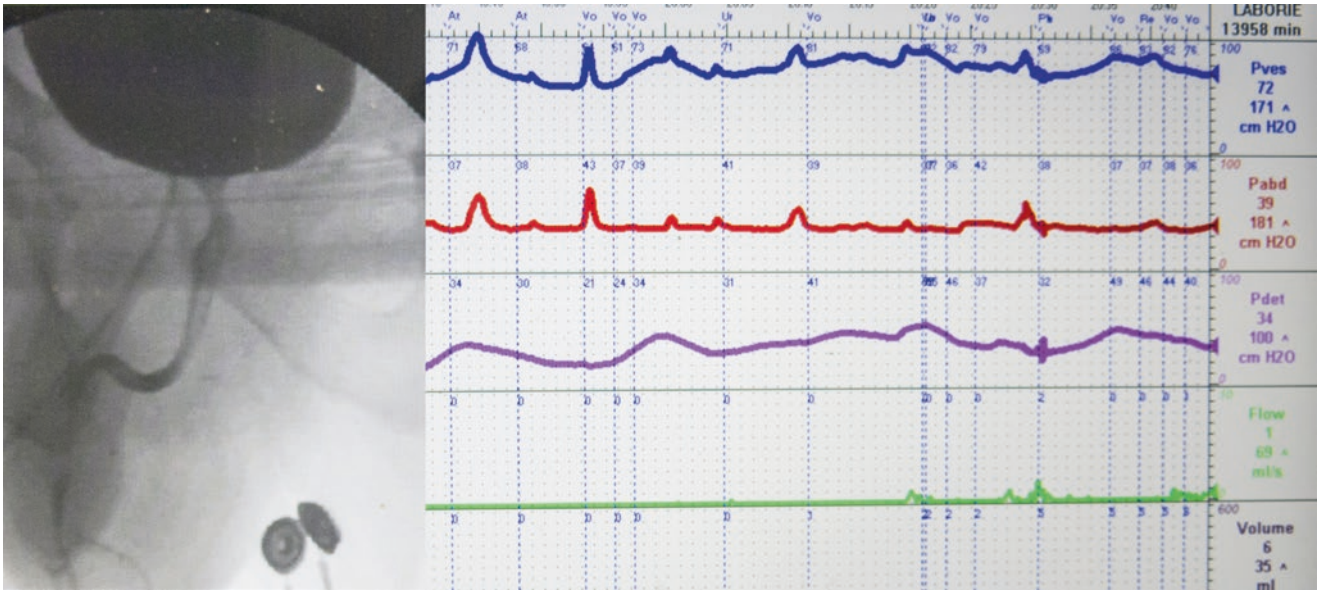

Fig. 9.4 Involuntary detrusor contractions with associated urinary incontinence

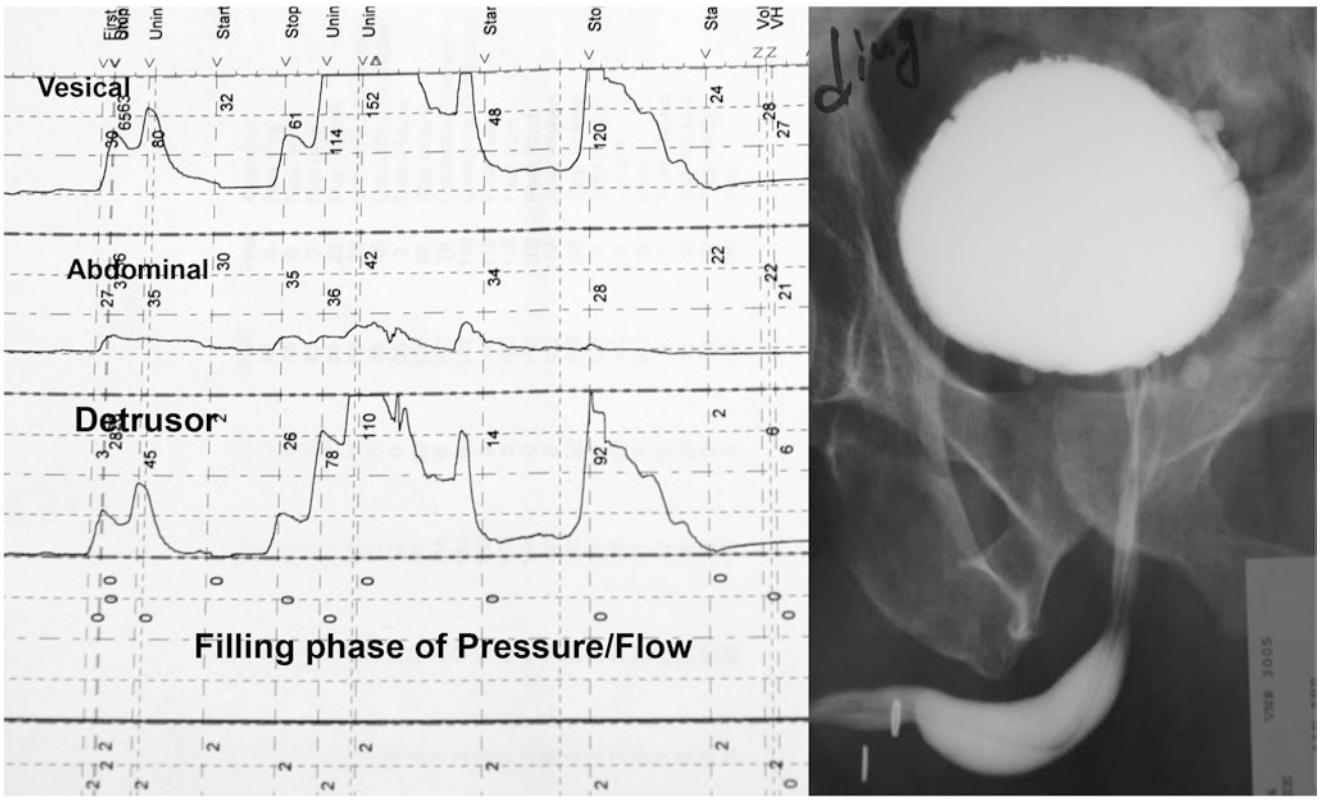

Fig. 9.5 More involuntary detrusor contractions with associated urinary incontinence

the device would prevent him from getting further MRIs. The most reasonable option for him is to try bladder Botox injections to increase the storage capacity of his bladder, with a commitment to increase the frequency of intermittent catheterization to every 3–4 h. If the MS is stable and the Botox fails, augmentation cystoplasty is another reasonable option for him. That would very likely improve his bothersome urgency and frequency. At this time, he has elected to try

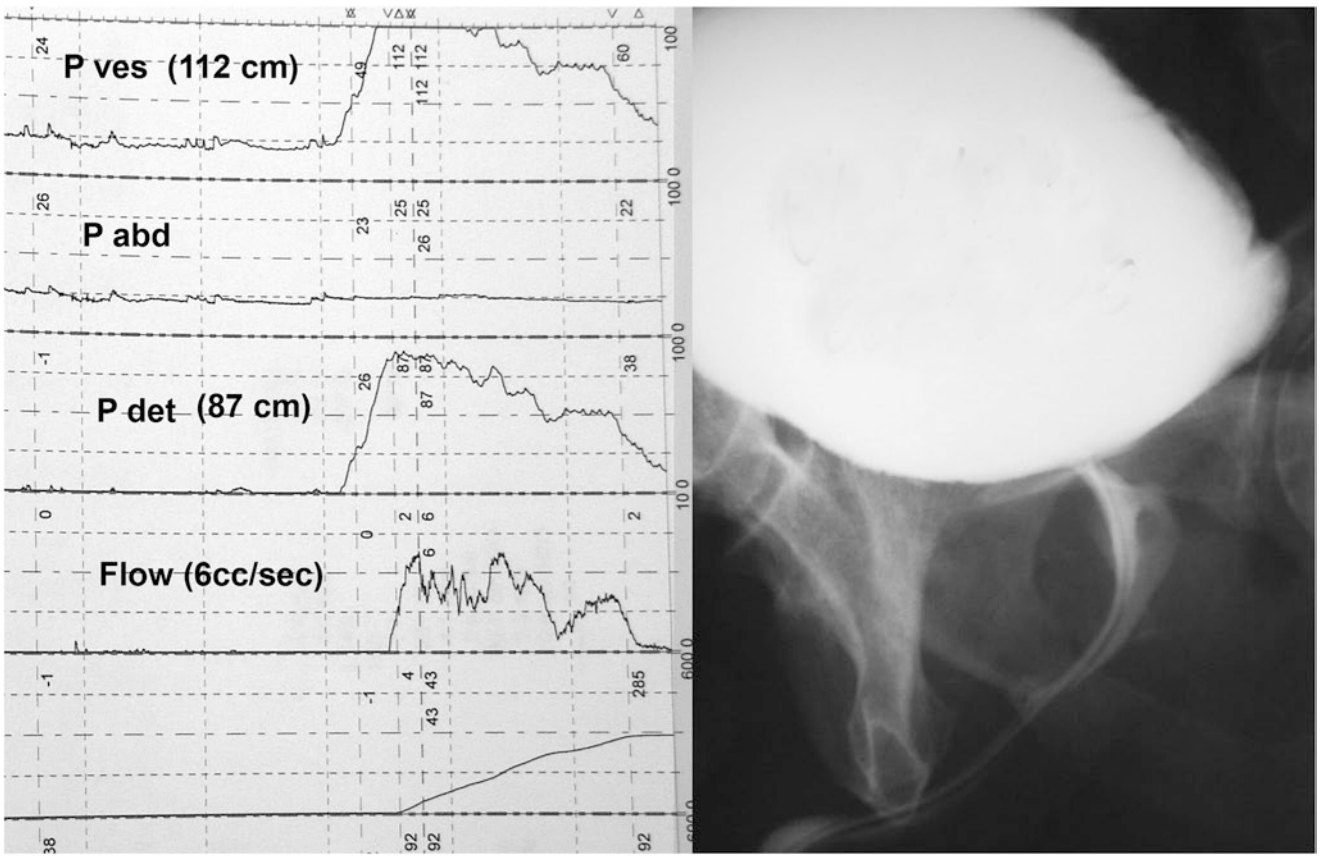

Fig. 9.6 High-pressure, low-flow voiding, with a poorly funneling bladder neck

bladder Botox injections. He will also initiate methenamine hippurate 1 g by mouth twice daily for UTI prophylaxis.

9.2.3 Patient 3

9.2.3.1 History

The patient is a 51-year-old woman with a history of hypothyroidism and a C6–C7 spinal cord injury status post a traumatic fall with subsequent cervical fusion (2013), with neurogenic bladder and urinary incontinence, presenting for evaluation. She is intermittently catheterized twice a day by a caregiver, as she has very poor manual dexterity herself. She cannot catheterize independently. She needs to be transferred to a bed for the catheterization. The availability of her caregiver only allows for the catheterization twice daily. She reports urinary incontinence throughout the day, without sensation of when she is leaking urine. Her urinary incontinence is such that she wears two to three diapers a day. There are no UTIs or gross hematuria. She uses suppositories, docusate, senna, and digital stimulation to aid with chronic constipation. She is very unhappy secondary to her continued dependence on others for her bladder and bowel care. She is able to move about with a motorized wheelchair.

9.2.3.2 Physical Examination

Generally she is in no apparent distress when sitting up in a wheelchair. She has 4/5 strength in her upper extremities, with decreased sensation to light touch in both upper extremities. She is able to hold a pen and squeeze the digits of her hands together. Her lower extremities are atrophied with no sensation and no motor strength. Her neck is supple. Trachea is midline. Skin is warm, dry. Abdomen is soft, nontender, and nondistended. Genitourinary exam reveals normal appearing external female genitalia, with no evidence of significant vaginal prolapse. Digital rectal exam reveals intact tone.

9.2.3.3 Labwork/Other Studies

Post-void residual was not checked, as she catheterizes two times a day to empty her bladder. A urine analysis was not checked, secondary to her intermittent catheterization and her lack of symptoms of infection at the time of evaluation in the clinic. Her most recent serum creatinine was 0.9 mg/dL and estimated glomerular filtration rate (eGFR) was >89 mL/min/1.73 m^2. Renal ultrasound found no hydronephrosis, obvious masses, or perinephric fluid collections. Cystoscopy did not reveal any intravesical abnormalities such as stones, tumors, or diverticula.

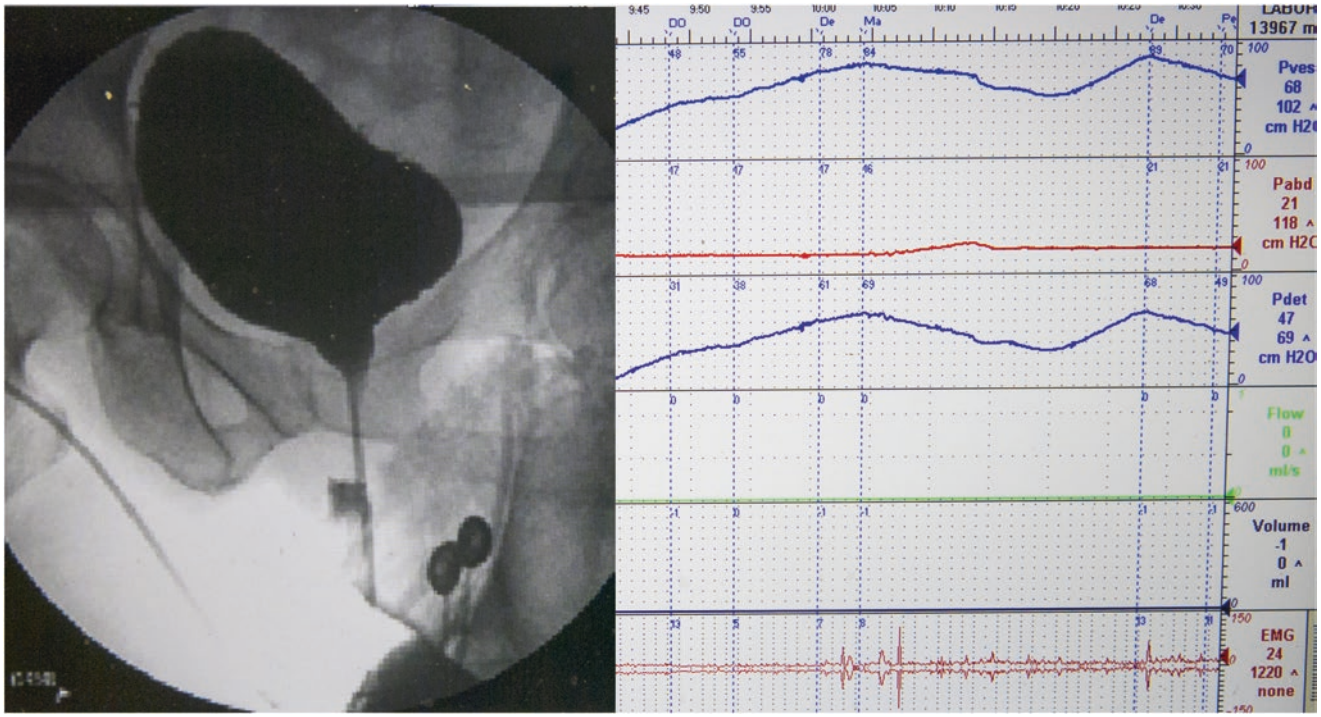

Fig. 9.7 Involuntary detrusor contraction, with subsequent permission to void, in the setting of a closed external sphincter (DSD)

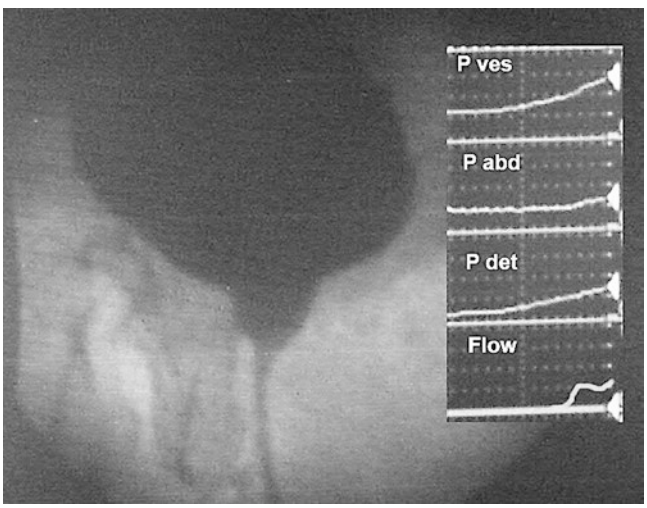

Fig. 9.8 Attempted void, in the setting of DSD

9.2.3.4 UDS

See Figs. 9.7 and 9.8.

A multichannel VUDS was performed in the supine position. She was initially catheterized for a volume of 400 cc. A rectal catheter was placed for intra-abdominal pressure measurements. A separate 7-French dual-lumen catheter was placed in the bladder. Catheters were zeroed, and filling with Cysto-Conray was begun at 30 cc/min. The filling phase of the study revealed a compliant bladder with low filling pressures. An involuntary detrusor contraction at 216 mL, with a pDet max of 61 cm H_2O, was associated with incontinence. During the detrusor contraction, her bladder neck funneled. She was also deemed to have reached capacity at this infusion volume. Fluoroscopic views obtained during the filling phase showed a smooth contoured bladder. There was no cystocele. There was no urethral hypermobility with Valsalva. At rest the bladder neck was closed. With Valsalva, there was no funneling of the bladder neck or incontinence. EMG was performed using surface perineal electrodes. The EMG showed normal activity during filling and increased activity during attempted void; however, during attempted void, she also had lower extremity spasms, and the sensors were wet by this point in the study.

At a capacity of 216 mL, she was already having an involuntary detrusor contraction and was given permission to void; she attempted to void, in the supine position, with the catheter in place at a pressure of 65 cm H_2O with no documented flow. There was no component of abdominal straining. On fluoroscopic images during attempted void, her bladder neck funneled, but her external sphincter appeared to remain closed, consistent with DSD; no urinary stream was visible. Fluoroscopic residual was 300 cc. There was no VUR.

Findings

The patient has a compliant bladder with detrusor overactivity associated with urinary incontinence and DSD. She is unable to empty her bladder. She does not feel the loss of urine with urgency in between intermittent catheterizations, as her neurologic lesion leaves her with no bladder sensation. This is an example of obstruction with severe bladder overactivity.

9.2.3.5 Treatment Options

Catheterizing in a wheelchair can be very challenging for a woman and often requires the patient to transfer to a supine position. She cannot do it without assistance from a caregiver. Although she has DSD, her outlet is still incompetent enough (likely from prior vaginal delivery) that she has urinary incontinence. Management options include a trial of an anticholinergic medication or bladder Botox injections to increase capacity and decrease overactivity, bladder augmentation with creation of a continent catheterizable stoma and closure of the outlet (with either a pubovaginal sling or actual bladder neck closure), an incontinent urinary diversion (a Bricker ileal conduit or an ileal chimney), or placement of a SPT. The option that offers her the greatest opportunity for independence would be the augmentation with creation of a continent catheterizable stoma and closure of the outlet. Her upper extremity dexterity (she can hold a pen and squeeze the digits of her hand together) would be enough for her to catheterize the stoma. This would allow her to avoid diapers and the need for an aide with urethral catheterization. For now, she has elected to think about her options further; her upper tracts have no evidence of hydronephrosis, renal function is appropriate, she has normal compliance, and there is no VUR.

9.3 Summary

When evaluating patients with neurogenic bladder, urodynamics provides important insight, but must be contextualized with additional information, to fully understand the patient's clinical condition. Fluoroscopy images taken during the study (VUDS) allow the provider a window into the upper tracts, to determine if there is VUR or hydronephrosis, and also give the provider the opportunity to assess where a point of obstruction may be (in cases of incomplete emptying). Without imaging the upper tracts, one may evaluate a cystometrogram tracing with low filling pressures and mistakenly presume that the bladder is of reasonable capacity, when in fact there is severe bilateral reflux compensating for a bladder with poor compliance. Without imaging to assess the attempted voiding phase, one may not be able to determine if it is smooth or striated sphincter dyssynergia contributing to obstruction (or perhaps another anatomical finding, such as a urethral stricture). See Figs. 9.9 and 9.10. If not obtaining videofluoroscopic images during the urodynamics study, one should consider at least obtaining a voiding cystourethrogram (VCUG) and upper tract imaging (i.e., renal ultrasound) to contextualize tracing findings.

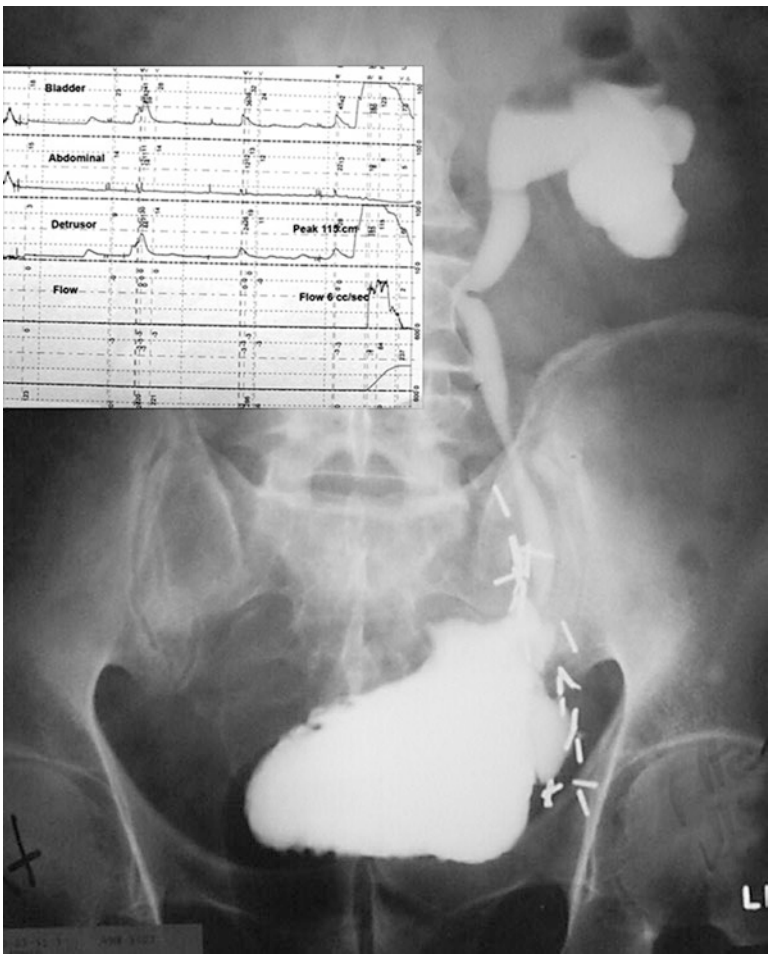

Fig. 9.9 Paraplegic patient with low-pressure filling, but significant left vesicoureteral reflux; during voiding phase, with high pressure, low flow, consistent with obstruction, possibly in the setting of smooth sphincter dyssynergia

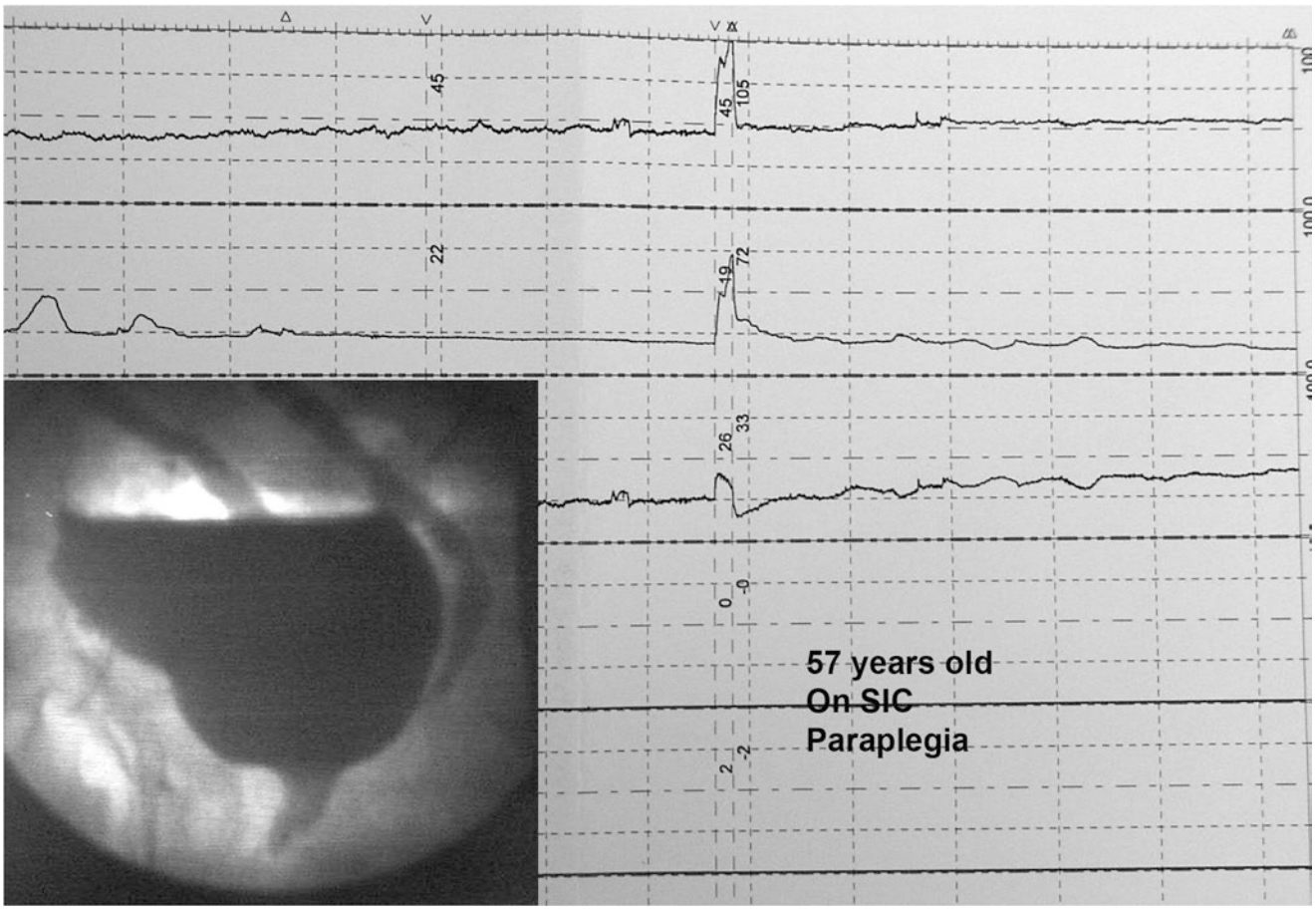

Fig. 9.10 Paraplegic patient, normally performing SIC (self-intermittent catheterization), with low-pressure filling but bilateral significant vesicoureteral reflux

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