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Practical urodynamics

Role of urodynamic studies in the diagnosis and treatment of lower urinary tract symptoms☆

CME Credits

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ABSTRACT

Urodynamic studies (UDSs) encompass a series of clinical tests, such as uroflowmetry, filling cystometry, pressure-flow studies and assessment of urethral closure (including urethral pressure profilometry and measurement of the leak-point pressure). Lower urinary tract symptoms are not always well correlated with the results of UDSs. Such studies are indicated in the following situations: (1) when conservative treatment is ineffective; (2) when invasive treatment is being considered; (3) for protection of the upper urinary tract by creating a low-pressure reservoir and control of urinary incontinence; (4) for evaluation of the effects of treatment; and (5) for biofeedback training. Video urodynamics is described as the gold standard of conventional UDSs. Characteristic features of various types of lower urinary tract symptoms, including urinary incontinence, bladder outlet obstruction and neurogenic bladder, can be identified by UDSs.

1. Introduction

Urodynamic studies (UDSs) involve the investigation of urinary tract function/dysfunction by various methods,[1,2] such as uroflowmetry, filling cystometry (CMG), pressure-flow studies, and assessment of urethral closure [including urethral pressure profilometry and measurement of the leak-point pressure (LPP)]. These tests can be combined with simultaneous electromyography and/or imaging.[3] Video urodynamics are the gold standard investigation for patients with lower urinary tract symptoms (LUTSs).[3]

2. Indications for urodynamic studies

UDSs provide objective information on the occurrence of LUTSs and enable determination of a person's lower urinary tract (LUT) performance in relation to what is known about normal or abnormal urinary tract physiology.[4]

The most important target when treating LUT dysfunction is alleviating LUTSs. LUTSs may be related to a variety of pathophysiological processes, however, and assessment on the basis of symptoms alone often leads to an incorrect diagnosis, especially in patients with a neurogenic bladder. It is therefore necessary to obtain objective data to make an accurate diagnosis, although LUTSs are not always well correlated with the diagnosis provided by UDSs. For example, there is only a weak association between the LPP and the severity of symptomatic or measured urinary incontinence (UI) and a weak correlation between symptoms and results of UDSs (especially cystometry) in patients with UI.[5,6]

Uroflowmetry and measurement of post-voiding residual urine by ultrasonography are noninvasive tests that can be routinely performed in patients with LUTSs. The other UDSs are invasive tests and therefore should only be performed in selected cases.

UDSs are indicated is the following circumstances: (1) when conservative treatment is ineffective; (2) when invasive treatment is being considered; (3) for protection of the upper urinary tract by creating a low-pressure reservoir and for controlling UI; (4) for evaluation of the effects of treatment; and (5) for biofeedback training.[7] It is important for investigators and clinicians to evaluate the results of UDSs in relation to each patient's symptoms, and to interpret the findings obtained by such tests in relation to symptoms, data from the voiding diary, and results of clinical (or other) examinations.[3]

2.1. Conventional urodynamic studies

Conventional UDSs involve artificial filling of the bladder. CMG can be performed using either gas or water. Although the gas method can provide an acceptable alternative to other techniques for measuring pressures, no studies have determined whether

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gas is actually a useful alternative to a fluid-filled catheter for measuring intravesical and intra-abdominal pressures.[1] Accordingly, CMG (including pressure-flow study) with a water-filled catheter is recommended for conventional UDSs. However, it is unknown whether this improves the reproducibility, sensitivity and specificity of UDSs.[3]

Conventional UDSs have been criticized for being non-physiological tests, and there is disagreement between the urodynamic and symptom-based diagnoses in 19–44% of patients.[8,9] Inter-institutional variation and inter-observer variation have been reported for conventional UDSs.[10–14] With regard to short-term (within test), intermediate and long-term reproducibility of UDSs, a test–retest variation of 10–15% for various volume, pressure or flow parameters is regarded as being due to physiological variations.[10]

There are various methods for performing conventional UDSs, and our laboratory uses the following method. After free flowmetry, a thin (e.g., 5–7 Fr) double-lumen catheter is inserted transurethrally for filling and pressure recording, and water CMG is performed at an infusion rate of 50 mL/minute (medium filling) in the supine position. The intra-abdominal pressure (P_{abd}) is simultaneously measured with a balloon catheter that is inserted transrectally. The detrusor pressure (P_{det}) is measured by subtracting P_{abd} from the intravesical pressure (P_{ves}). At maximum cystometric capacity, patients assume a standing position, and provocation tests (including heel bouncing, coughing and exposure to water) are carried out. For females with stress UI, Valsalva or cough LPP is measured by gradually increasing the abdominal pressure (straining) or by coughing, respectively. Patients then void while standing on an elevated platform (males) or sitting (females) behind a screen and pressure-flow studies are carried out (Figure 1).[15]

2.1.1. External sphincter electromyography

External sphincter electromyography is performed simultaneously using surface electrodes or needle electrodes. Surface electrodes are noninvasive and measure the activity of striated muscles near the electrode sites, but they may measure muscles other than the external sphincter. Insertion of needle electrodes may be painful, but such electrodes measure the activity of the target muscle (i.e., the external sphincter). External sphincter electromyography can assess the coordination of detrusor and external sphincter activity, motor unit potency and evoked potentials.[16]

2.1.2. Ice-water cystometry

Ice-water cystometry can be performed to elicit detrusor activity in patients with LUT dysfunction and relevant neurological abnormalities.[17,18] Detection of detrusor contractions during filling with ice water can be interpreted as a sign of pathological C-fiber reflex activity (i.e., only found in patients with the relevant neurologic conditions), but false-negative results can occur.

2.2. Noninvasive methods

Measurement of pressure and flow in men with a penile cuff or condom catheter have been reported as noninvasive methods that are as clinically useful as conventional pressure-flow studies.[19–21]

Ambulatory urodynamic monitoring (AUM) is a functional urodynamic test that uses natural filling and reproduces the everyday activities of the subject.[2,22,23] AUM has the advantages of natural bladder filling, a longer observation period and a relatively normal study environment, and is therefore more physiological than conventional UDS (Figure 1).[8,24] AUM has been reported to detect detrusor over-activity more accurately than conventional urodynamics, and there is a strong correlation of symptoms with AUM findings.[14]

3. Interpretation of urodynamic study parameters

3.1. Normal subjects

Studies have been performed to obtain normal values of UDS parameters in volunteers, including volumes, compliance and

Figure 1. Video-urodynamic study. Upper left panel, filling phase; upper right panel, voiding phase in females; lower left panel, voiding phase in males; lower right panel, ambulatory urodynamic study.

sensation(s) during filling cystometry.[13,25,26] In the filling phase, detrusor pressure does not usually increase (it is stable) when the first sensation is noted at 150–250 mL or at the maximum cystometric capacity of 300–500 mL in volunteers.[27]

Detrusor over-activity (DO) is defined as involuntary detrusor contractions during the filling phase, which may be spontaneous or provoked (Figure 2).[2] Detrusor over-activity is found in 10–20% of healthy volunteers with conventional UDSs. It has been reported that detrusor over-activity is more common with subjects in the sitting position compared with those in the supine position during cystometry.[25] Furthermore, there is some evidence that entering a toilet and hand washing are strong stimulators of detrusor over-activity.[3] Therefore, the position of the patient during filling cystometry should be taken into account because it can influence the occurrence of detrusor over-activity. The results of provocative cystometry should be interpreted in relation to the patient's symptoms and how reasonable the findings obtained are. The incidence of detrusor over-activity has been reported to increase to 30–70% when using AUM.[22,24,28–31]

During the voiding phase, normal subjects void with good flow (Q_{max} > 15 mL/second with a voided volume of >150 mL) and their detrusor pressure at Q_{max} ($P_{det}Q_{max}$) of 60–90 cm H_2O. According to pressure-flow studies, the International Continence Society (ICS) nomogram or Shäfer nomogram is within the unobstructed range in males. In females, however, detrusor pressure is <40 cmH_2O during voiding, and some elderly subjects or patients with stress incontinence can void without an increase in detrusor pressure (Figure 3).

4. Classification of LUT dysfunction

According to the ICS standards, LUT function is classified into detrusor and urethral function or filling and voiding function. Therefore, LUT dysfunction is classified as detrusor over-activity, an underactive detrusor, or an acontractile detrusor and the presence of an incompetent or obstructive urethra (Table 1).[2]

4.1. Bladder outlet obstruction

Male patients with benign prostatic hyperplasia, bladder neck obstruction or urethral stricture and female patients with bladder neck obstruction or pelvic organ prolapse may have bladder outlet obstruction (BOO). During the voiding phase, BOO may manifest as a high detrusor pressure with decreased urinary flow (obstructed pattern = high pressure/low flow pattern) on an ICS nomogram or Schäfer nomogram obtained by pressure-flow studies (Figure 4).[2] The BOO index, formerly known as the Abrams-Griffith number (=$P_{det}Q_{max}$ − $2Q_{max}$), is >40 in patients with BOO. During the filling phase, detrusor over-activity may be detected in 40–60% of patients with benign prostatic hyperplasia.[15,32–34]

4.2. Patients with UI

There are three main types of UI: urge UI, stress UI and mixed UI. Urge UI is associated with overactive bladder dysfunction (OAB, also known as wet OAB), and is considered to be related to detrusor over-activity. Urodynamic stress UI is defined as the involuntary leakage of urine along with increased abdominal pressure in the absence of detrusor contraction during the filling phase. To assess urethral function during filling, the urethral pressure profilometry or (abdominal or detrusor) LPP is measured. UDSs should be performed in women prior to surgical intervention for stress UI. However, various studies have shown that the results of UDSs do not precisely predict the response to treatment in patients who have OAB with or without detrusor over-activity, or in patients with urodynamic stress UI.[35,36] In fact, the association between symptoms of OAB and detection of detrusor over-activity during UDS is weak, and currently it is not possible to predict the response to treatment on the basis of characterization or quantification of detrusor over-activity during UDS.[37]

There are conflicting results regarding the association between the severity of UI and the results of urethral function tests (LPP and urethral closing pressure). Measurements of urethral function (LPP and urethral pressure profilometry) are not useful for accurately predicting the likelihood of success after surgical treatment for stress UI. However, there is some evidence that a low urethral (closing) pressure is associated with a lower suburethral sling procedure success rate.[38–40]

5. Neurogenic bladder (Table 2)

Lesions in the cerebral cortex, the basal ganglia and brain stem, the spinal cord, conus, or cauda equina and peripheral lesions can cause various types of neurogenic bladder. Neurogenic detrusor over-activity is frequently associated with upper motor neuron

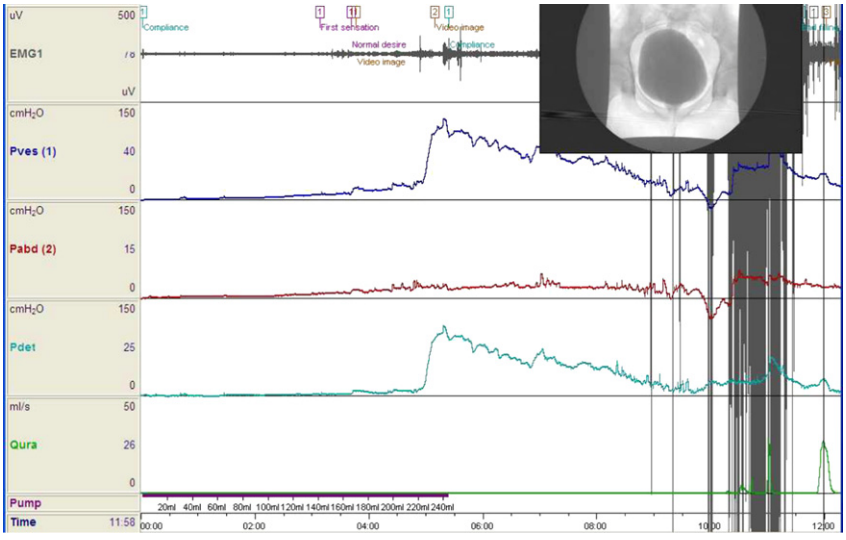

Figure 2. Urodynamic findings of detrusor overactivity.

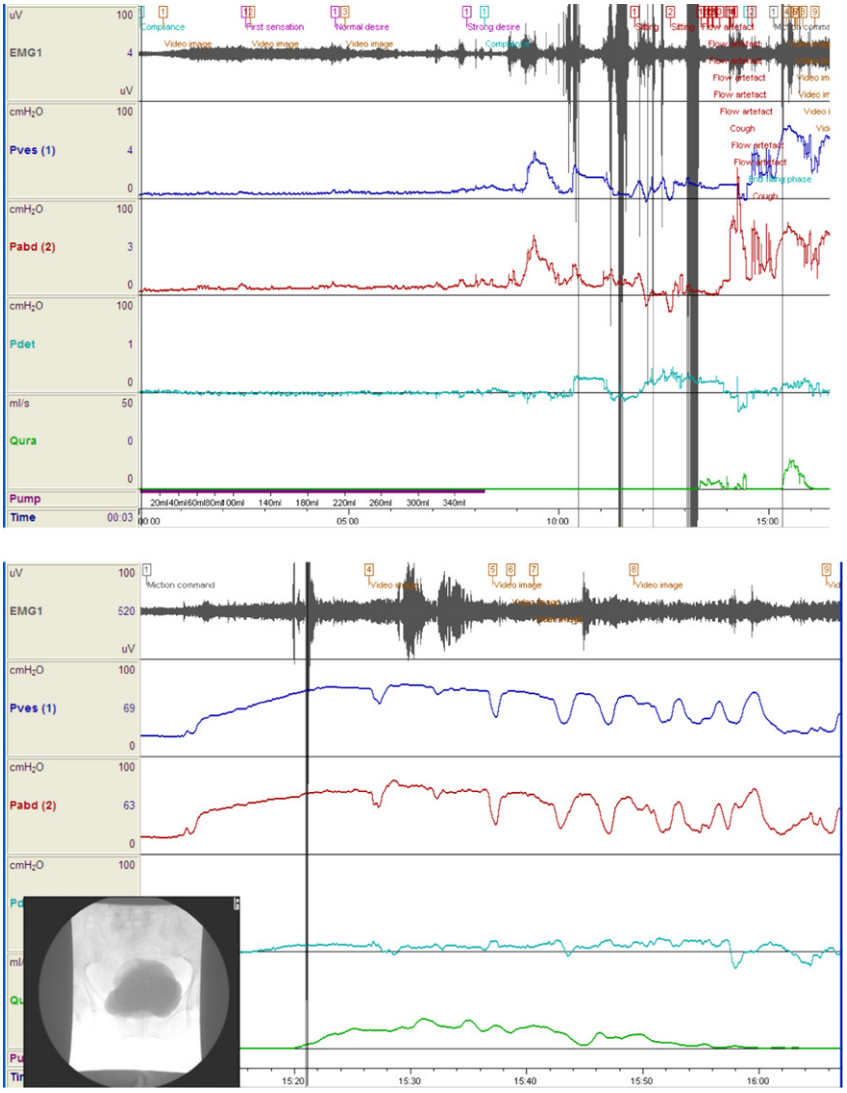

Figure 3. Urodynamic findings in a normal female who voided without an increase in detrusor pressure. Upper panel, entire study; lower panel, voiding phase.

lesions (above the sacral cord). Detrusor-sphincter dyssynergia (DSD) is frequently found in patients with spinal cord lesions and less frequently in those with brain stem lesions, but is rare in patients with cerebral lesions. Underactive or acontractile detrusor may be present in patients with lower motor neuron lesions (affecting the lower lumbar cord, conus or cauda equina lesions and peripheral nerves).[41,42]

Table 1
Classification of urinary tract dysfunction (ICS 2002)

	Filling phase	Voiding phase
Detrusor function	DO	UAD, ACD
Bladder sensation	increased (hypersensitive), reduced (hyposensitive)	
Urethral function	Incompetent	Obstructive: BOO Dysfunctional voiding DSD Non-relaxing

DO = detrusor overactivity; UAD = underactive detrusor; ACD = acontractile detrusor; BOO = bladder outlet obstruction; DSD = detrusor sphincter dyssynergia.

5.1. Urodynamic study findings in patients with cortical lesions

In general, the most common cystometric finding is detrusor over-activity, and a few patients with cortical lesions show DSD. In our series of 72 patients with acute hemispheric stroke, 53% were found to have significant urinary complaints.[43] The most common problem was nocturnal urinary frequency (36%), followed by UI (29%) and difficulty in voiding (25%). Urinary retention was observed in 6% of patients during the acute phase of the stroke. Brain Imaging confirmed a more anterior location of brain lesions in the former group. UDS showed detrusor over-activity in 79%, a non-relaxing urethra in 16% and uninhibited sphincter relaxation in 42% of patients. There was no preponderance of right-sided lesions.[43] In some patients with a particular brain lesion (e.g. small cortical infarcts in the caudal part of the right anterior cingulate gyrus), retention of urine probably occurs because of injury to the facilitatory region of the brain.[44]

The classical triad of idiopathic normal pressure hydrocephalus consists of gait disturbance, memory deficit and UI. In a series of 42 patients with idiopathic normal pressure hydrocephalus, LUTSs were present in 93% of patients, with storage symptoms (93%) being more common than voiding symptoms (71%). UDSs showed

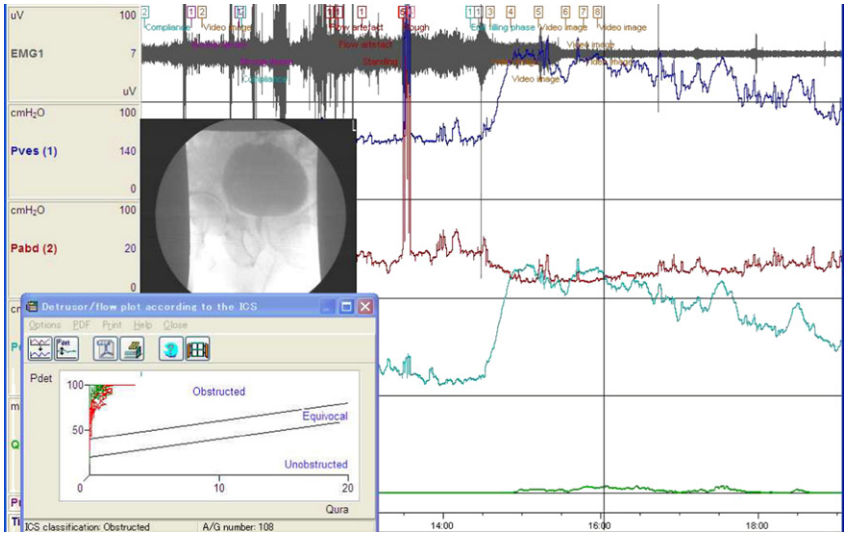

Figure 4. Urodynamic findings and ICS nomogram in bladder outlet obstruction.

that the bladder capacity was small (with a mean of 200 mL), and detrusor over-activity was found in 95% of the patients.[45]

5.2. Urodynamic study findings in patients with multiple system atrophy

In patients who have severe LUTSs despite relatively mild parkinsonism, multiple system atrophy should be considered.[41] The onset of LUTSs in multiple system atrophy may precede overt neurological involvement by a number of years, with LUTS and erectile dysfunction occurring 4–5 years prior to a diagnosis and an average of 2 years before more obvious neurological symptoms appear.[41] Detrusor over-activity and hyperactivity with impaired contraction are the most common initial UDS findings.[46–49] Over ensuing months or years, a failure of bladder emptying may develop due to underactive detrusor/acontractile detrusor or DSD, where the post-voiding residual urine gradually increases and urinary retention may occur.[48] It has been suggested that detrusor over-activity is due to loss of cells in the brainstem, whereas incomplete bladder emptying is due to loss of parasympathetic stimulation of the detrusor following atrophy of cells in the intermediolateral cell column. In addition to this, anterior horn cell loss in Onuf's nucleus results in denervation of the urethral sphincter, resulting in patients having a combination of detrusor over-activity/hyperactivity with impaired contraction and a weak sphincter.[41,48] A previous study showed a decrease in cerebellar vermis activation during urinary storage and micturition, which was detected by single-photon emission computed tomographic brain imaging.[50]

5.3. Urodynamic study findings in patients with Parkinson's disease

LUTSs usually arise after many years of treating Parkinson's disease and patients often show long-term side-effects of levodopa.[41] The most common UDS finding in Parkinson's disease patients is detrusor over-activity, and these patients complain of OAB symptoms.[51,52] In anaesthetized cats, the dopamine D1 receptor is responsible for the main inhibitory effect on the micturition reflex.[53] BOO may occur in Parkinson's disease because of impaired relaxation or "bradykinesia" of the urethral sphincter.[54] When administration of domperidone was followed by subcutaneous apomorphine in 10 patients who had Parkinson's disease and urinary symptoms, it was found that apomorphine reduced bladder outflow resistance and improved voiding in all 10 patients.[54]

Genitourinary autonomic dysfunction often fails to respond to levodopa treatment. The neural pathology causing bladder dysfunction (over-activity) involves alteration of the dopamine circuit in the basal ganglia, which normally suppresses the micturition reflex. In contrast, hypothalamic involvement is mainly responsible for sexual dysfunction in Parkinson's disease (decrease in libido and erections) due to changes in the dopamine–oxytocin pathway that normally promotes libido and erection. The pathophysiology of genitourinary dysfunction in Parkinson's disease differs from that in multiple system atrophy, and therefore investigations might be helpful in making a differential diagnosis.[55]

5.4. Urodynamic study findings in patients with brainstem lesions

In a study of 39 patients with brainstem strokes, 50% had LUTSs, with nocturnal frequency and voiding difficulty in 28%, urinary retention in 21% and UI in 8%. LUTSs were more common following cerebral hemorrhage.[56] LUTSs did not occur in patients with midbrain lesions, but were found in 35% of those with pontine lesions and 18% of those with medullary stroke. Among 11 symptomatic patients, UDS showed detrusor over-activity in 73%, low-compliance bladder in 9%, acontractile detrusor in 27% and DSD in 45%.[56]

5.5. Urodynamic study findings in patients with spinal cord disease

Immediately following spinal cord injury and during the phase of spinal shock, the bladder is acontractile. In patients with spinal cord disease above the sacral cord (upper motor neuron involvement), new reflexes (detrusor over-activity) develop without bladder sensation over the course of several weeks. Disconnection of the pontine micturition centers from the sacral cord means that synergy between the detrusor and sphincter is lost, resulting in DSD and detrusor bladder neck dyssynergia (Figure 5).[32,57]

With regard to the mechanism of recovery, it was reported that C fibers emerge as the major afferents in a spinal segmental reflex.[41] In patients with lower lumbar spinal cord or cauda equina lesions (lower motor neuron involvement), the bladder is acontractile, and the urethra is inactive.

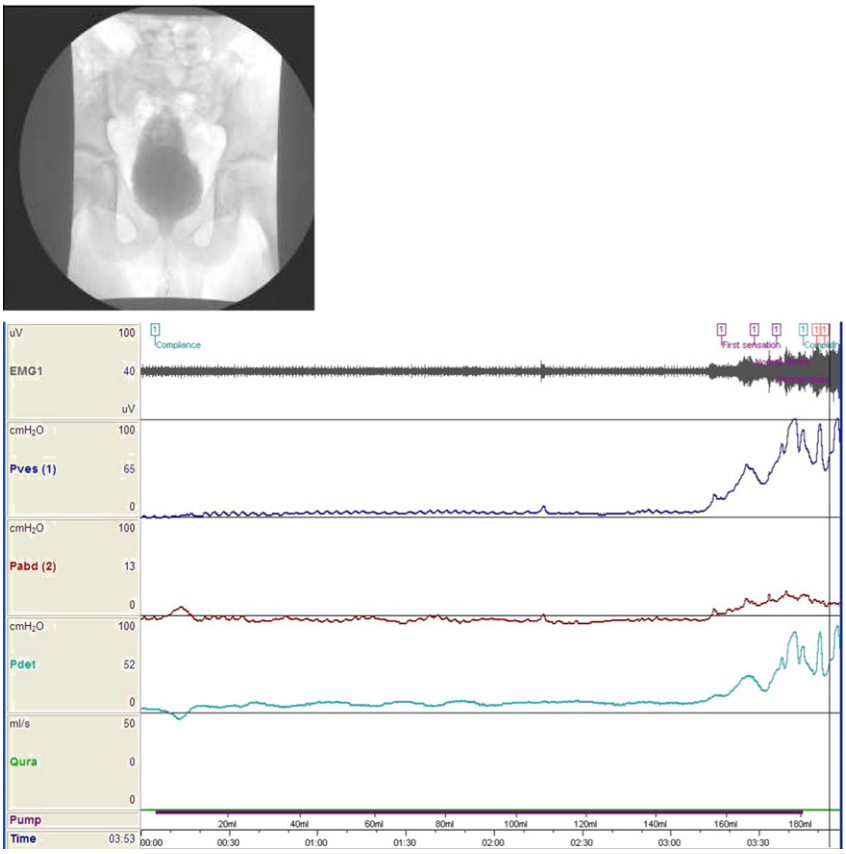

Figure 5. Urodynamic findings in a patient with neurogenic detrusor overactivity and detrusor-sphincter dyssynergia.

5.6. Spinal cord injury

A typical spinal cord disorder is a spinal cord injury. If a patient has a cord injury above the sacral micturition center (upper motor neuron involvement), detrusor over-activity occurs with DSD, while sacral or lower lesions lead to underactive/acontractile detrusor. Neurogenic detrusor over-activity, loss of compliance and DSD may cause vesicoureteric reflux, hydronephrosis and upper urinary tract damage.

5.7. Nontraumatic spinal cord diseases

UDS findings in patients with nontraumatic spinal cord diseases are similar to those due to traumatic spinal cord injuries, but the findings may vary if there is partial spinal involvement (Table 2). In patients with upper motor neuron involvement, detrusor over-activity and DSD are the most common urodynamic findings and the majority of patients with these abnormalities have long tract symptoms and signs. In 128 patients with cervical myelopathy, including 82 with spondylotic myelopathy and 46 with ossification of the posterior longitudinal ligament, storage symptoms were observed in 64% and voiding symptoms in 75%. In addition, UI was detected in 26% of patients and post-voiding residual urine of >100 mL or retention was found in 23%, while UDS revealed detrusor over-activity in 64% and DSD in 23% of these patients. Furthermore, a relationship between detrusor over-activity and pyramidal signs was found in patients with cervical myelopathy.[58,59]

Among patients with anterior spinal artery syndrome, detrusor over-activity was observed in 80%, a normal bladder was found in 10% and acontractile detrusor was found in 10% of patients.[60] Vesicomotor dysfunction in this syndrome is similar to that of

Table 2
Urodynamic findings in neurogenic diseases in our study

Disease	DO	DO	UAD/ACD
	DSD (−)	DSD (+)	
Brain lesions			
Acute hemispheric stroke ($n = 72$)[58]	79	0	
iNPH ($n = 42$)[45]	95	0	0
Multiple system atrophy ($n = 121$)[46]	14	45	36
Parkinson's disease ($n = 39$)[51]	46	3	0
Brainstem stroke ($n = 39$)[56]	28	45	36
Non-traumatic spinal cord lesions			
Cervical myelopathies ($n = 128$)[58]	64	23	
Cervical/thoracic disc prolapse ($n = 14$)[59]	22	14	7
Anterior spinal artery syndrome ($n = 10$)[60]	40	40	10
Tabes dorsalis ($n = 8$)[61]	46		36
Amyotrophic lateral sclerosis ($n = 36$)[62]	25	22	0
Syringomyelia ($n = 14$)[63]	21	29	14
Transverse myelitis ($n = 10$)[64]	0	40	0
HAM ($n = 5$)[65]	60	40	0
Arteriovenous malformation[66]	7	33	40
Lumbar disc prolapse ($n = 19$)[67]	5	5	26
Lumbar spinal canal stenosis ($n = 29$)[67]	31	3	7
Radiation myelopathy ($n = 5$)[69]	0	90	10
Brown-Sequard syndrome ($n = 8$)[70]	12	38	12
Peripheral lesions			
Diabetic mellitus ($n = 84$)[73]	42	0	48
CIDP ($n = 32$)[74]	50	0	25
Guillain-Barré syndrome ($n = 28$)[75]	50	0	25

Values are shown as %.
DO = detrusor overactivity; UAD/ACD = underactive detrusor or acontractile detrusor; DSD = detrusor external sphincter dyssynergia; iNPH = idiopathic normal pressure hydrocephalus; HAM = human T-lymphotropic virus type 1 (HTLV-1)-associated myelopathy; CIDP = chronic inflammatory demyelinating polyradiculoneuropathy.

traumatic spinal cord injuries, except that bladder sensation is preserved.[60] In contrast, a previous study observed disturbed bladder sensation with various types of voiding dysfunction due to tabes dorsalis.[61] From these findings, the pathway subserving detrusor function appears to mainly be located in the lateral column of the spinal cord. The majority of the descending pathway subserving coordination of the bladder and urethral sphincter appears be located in the lateral column. Moreover, the path subserving bladder sensation is thought to be located in the dorsal column of the spinal cord.[58–61]

Amyotrophic lateral sclerosis is a degenerative neurological disease in which limb weakness, amyotrophy, dysarthria, dysphagia and respiratory failure develop because of degeneration of motor neurons. It is thought that amyotrophic lateral sclerosis patients rarely have LUT dysfunction, and that the anterior horn nucleus (Onuf's nucleus) is selectively preserved in this disease. In a previous study, however, LUTSs were found in 47% of amyotrophic lateral sclerosis patients, with voiding symptoms in 14%, storage symptoms in 22% and both types of symptoms in 11% of patients. Detrusor over-activity was noted in 25% of patients.[62]

Syringomyelia is cavitation of the spinal cord parenchyma that can cause progressive spinal cord dysfunction due to expansion of the central canal. In patients with Arnold-Chiari malformation, syringomyelia often occurs and LUTSs are found in 9–25% of patients. In our series of patients, 35% had detrusor over-activity, including DSD in 14%.[63]

In patients with transverse myelitis, there may be good clinical recovery from tetraparesis of such severity that artificial ventilation is necessary, and bladder dysfunction may be the sole residual neurological sequela.[64]

Human T-lymphotrophic virus type 1-associated myelopathy is also known as tropical spastic paraparesis and progressive myelopathy, because of viral infection being observed in British patients of Caribbean origin and in the Japanese.[65] This disease is a neurological manifestation of infection by the human T-lymphotrophic virus type 1 retrovirus. Myelopathy slowly progresses over the course of a decade or more, with its onset usually being before the age of 40 years. UI due to detrusor over-activity is an early feature found in most of these patients. A low-compliance bladder or acontractile detrusor is not found. External sphincter electromyography shows DSD in 40% of patients, but neurogenic changes in the motor unit potential are not observed.[65]

Arteriovenous malformations of the spinal cord may be difficult to recognize clinically, but disturbance of bladder function is a prominent early feature in many cases. Although the majority of arteriovenous malformations occur in the thoracolumbar region, alterations in cord blood flow and subsequent conus ischemia mean that the patient may present with what appears to be a conus or cauda equina lesion.[41] Voiding difficulties are common at an early stage and are followed later by urinary retention.[66] In 80% of these patients, abnormalities are revealed by UDSs before treatment, including detrusor over-activity with or without DSD in 40% and underactive detrusor in 40% of patients. LUT function only recovers in 25% of patients after treatment.[66]

The common urodynamic findings in patients with lower lumbar spine lesions, such as intervertebral disc prolapse and spinal canal stenosis, are underactive or acontractile detrusor, but detrusor over-activity is also found in 10% of patients with lumbar disc prolapse and 29% of those with lumbar canal stenosis. In these patients, urgency, urge UI and, in males, erectile dysfunction are usually observed in association with intermittent claudication.[67]

Clinical features of cauda equina syndrome include low back pain, bilateral sciatica, saddle anesthesia and acute urinary retention. It typically occurs with central lumbar disc prolapse (the incidence is 1–5% of all prolapsed lumbar discs). Clinically, there is perineal sensory loss caused by damage to the S2–S4 roots together with loss of voluntary control of both the anal and urethral sphincters and sexual dysfunction. The detrusor is not denervated, but is decentralized by cauda equina injury, and sympathetic innervation of the bladder neck may be preserved.[41] In our study of eight patients, all of them still demonstrated acontractile detrusor up to 6 years after undergoing emergency surgery within 48 hours. Bladder function was irreversibly lost despite spinal surgery, while urethral function showed better recovery in patients with acute retention because of central lumbar disc prolapse. However, most of those patients could only empty their bladder by straining or changing the voiding posture postoperatively.[68]

LUTSs have been observed in patients with radiation myelopathy and Brown-Séquard syndrome (Table 2).[69,70] LUTSs have also been described as a result of brainstem encephalitis and viral sacral myeloradiculitis following herpetic infection.[71,72]

5.8. Urodynamic study findings in patients with peripheral lesions

According to the number of nerves affected, peripheral neuropathies can be classified as mononeuropathy, multiple mononeuropathy and polyneuropathy. The major lesions that cause peripheral neuropathies affect the axon (degeneration due to toxicity, metabolic disease, etc.), the myelin sheath (demyelination due to inflammatory diseases, hereditary diseases, etc.), or both the axon and sheath (diabetes, etc.). Peripheral neuropathies can also be classified into motor neuropathies (axonal type of Guillain-Barré syndrome, etc.), sensory neuropathies (chronic sensory neuropathy, familial amyloid neuropathy, or Sjögren's syndrome presenting as dorsal root ganglionitis), autonomic neuropathies (acute pandysautonomia, etc.), and a combination of these.

Diabetes mellitus is the most common cause of small-fiber neuropathies. Small-fiber involvement is usually part of a generalized distal sensory neuropathy, but relatively independent autonomic neuropathies do less commonly occur.[41] The onset is insidious over the course of several years, with progressive loss of bladder sensation and impairment of bladder emptying due to over-distention. UDSs demonstrate impaired detrusor contractility, a reduced urinary flow rate, increased post-voiding residual urine and reduced bladder sensation (diabetic cystopathy). In a series of 84 patients with diabetes (58 males and 26 females with a mean age of 60.8 years), diabetic cystopathy was observed in 4% (average duration of diabetes of 143.5 months; hemoglobin A_{1C} of 7.7%). In addition to large post-voiding residual urine and decreased sensations, OAB symptoms, detrusor over-activity and increased bladder sensation were observed in 55%, 42% and 14% of patients, respectively. On brain magnetic resonance imaging of 32 patients, multiple cerebral infarcts were observed in 76.5% of those with detrusor over-activity. Therefore, both central and peripheral mechanisms are involved in patients with diabetes.[73] Although the most common finding on UDSs may be underactive detrusor, detrusor over-activity is also reported to occur in patients with peripheral neuropathies such as Guillain-Barré syndrome and chronic inflammatory demyelinating polyradiculoneuropathy, probably due to peripheral nerve irritation or ephaptic transmission.[74,75]

Peripheral nerves to the pelvic organs can be damaged by pelvic surgery, such as resection of rectal carcinoma, radical prostatectomy or radical hysterectomy. Damage to parasympathetic innervation of the detrusor results in painless retention, poor urine stream, loss of normal bladder sensation during filling and near loss of detrusor contraction pressure (Figure 6). The primary cause of UI after radical prostatectomy is sphincter insufficiency, but detrusor over-activity, reduced bladder compliance and decreased contractility may also be relevant factors.[76]

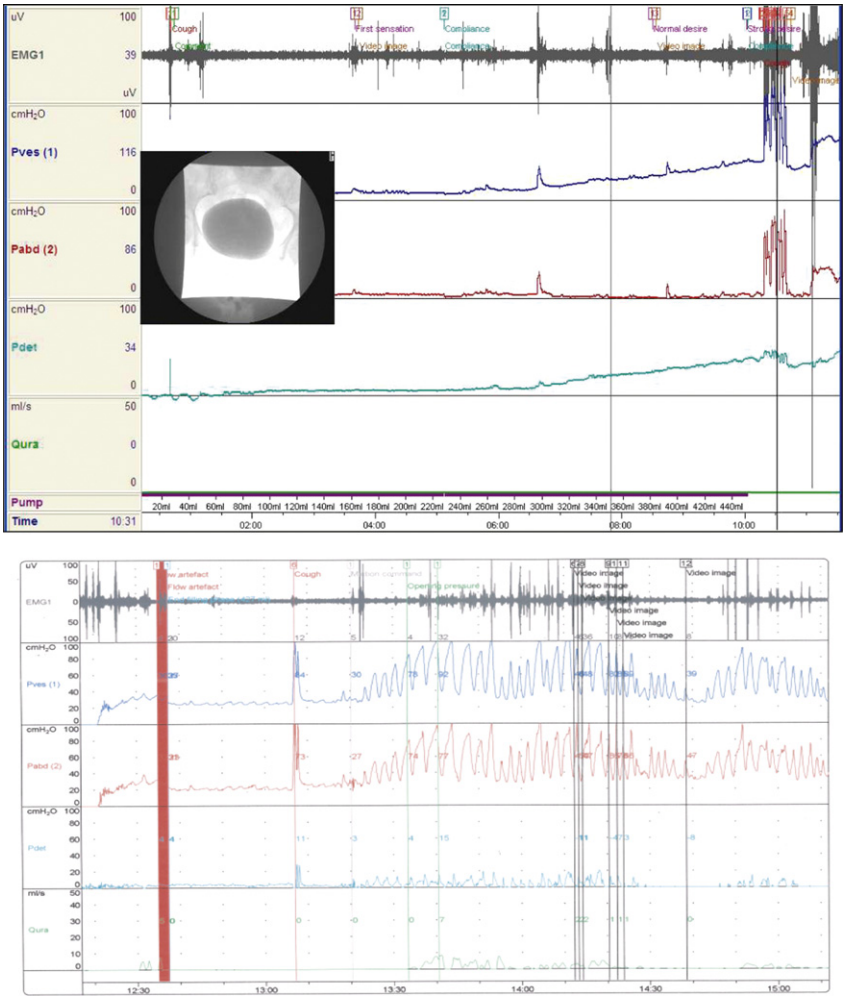

Figure 6. Urodynamic findings in a patient after a radical hysterectomy who showed a low-compliance bladder (upper panel) and an acontractile detrusor (lower panel).

6. Conclusions

Characteristic features of various types of lower urinary tract symptoms, including urinary incontinence, bladder outlet obstruction and neurogenic bladder, can be identified by UDSs.

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