

## CHAPTER 2

# URINARY INCONTINENCE AND RETENTION

### INTRODUCTION

Loss of bladder control is a common problem for patients with neurological disease. Incontinence is likely to impose considerable extra burdens on a disabled person and their carers in terms of loss of quality of life, social embarrassment and expense and it is therefore important that the neurologist is aware of what treatments are available and the scientific basis for their effectiveness.

### ANATOMY AND PHYSIOLOGY OF THE LOWER URINARY TRACT

Physiological properties of the bladder enable the urine that is continuously produced to be stored at low pressure until such time as it is convenient and socially acceptable to void. Normal voiding involves reflex detrusor contraction synchronized with voluntary sphincter relaxation to achieve complete bladder emptying.

Any pathology that affects the physiology of these processes will cause symptoms that can be divided into disorders of either storage or voiding (Table 1).

**Storage.** Whether voluntary or not, passage of urine through the urethra occurs when intravesical pressure exceeds intraurethral pressure. Reflex inhibition of the parasympathetic innervation of the detrusor during filling prevents its contraction and facilitates storage. Furthermore the bladder has viscoelastic properties that give it “compliance” so that as it fills, its internal pressure does not rise.

In the female urethra, the voluntary or “external” striated urethral sphincter is situated in the middle third of the urethra, whereas in males the striated sphincter is situated at the most inferior aspect of the prostate around the membranous urethra. Smooth muscle condensations in the region of the male bladder neck form the so-called “internal” sphincter, but the homologous structure

is less well developed in women. The internal and external sphincters remain closed during filling, but even though the internal sphincter is destroyed following transurethral resection of the prostate, incontinence very rarely occurs.

The pelvic floor, its overlying “endopelvic” fascia and the named ligamentous condensations of this fascia that are inserted into the anal sphincter, perineal body and lower vagina or membranous urethra in the male, constitute the mechanism of pelvic organ support. The pelvic floor muscles (*levator ani* and *coccygeus*), act like a hammock to maintain the position of the viscera in the pelvis which is important for continence (Figure 1).

**Voiding.** Voiding is initiated by inhibition of the striated sphincter and pelvic floor, followed some seconds later by a contraction of the detrusor muscle. To achieve complete bladder emptying sphincter activity must be inhibited throughout the cycle of detrusor contraction.

**Innervation of the lower urinary tract.** Figure 2 shows the complex relationship between the somatic and autonomic innervation of the lower urinary tract.

**Somatic.** The motor neurons that innervate the urethral and anal sphincters lie in Onuf’s nucleus within the S2-4 sacral segments of

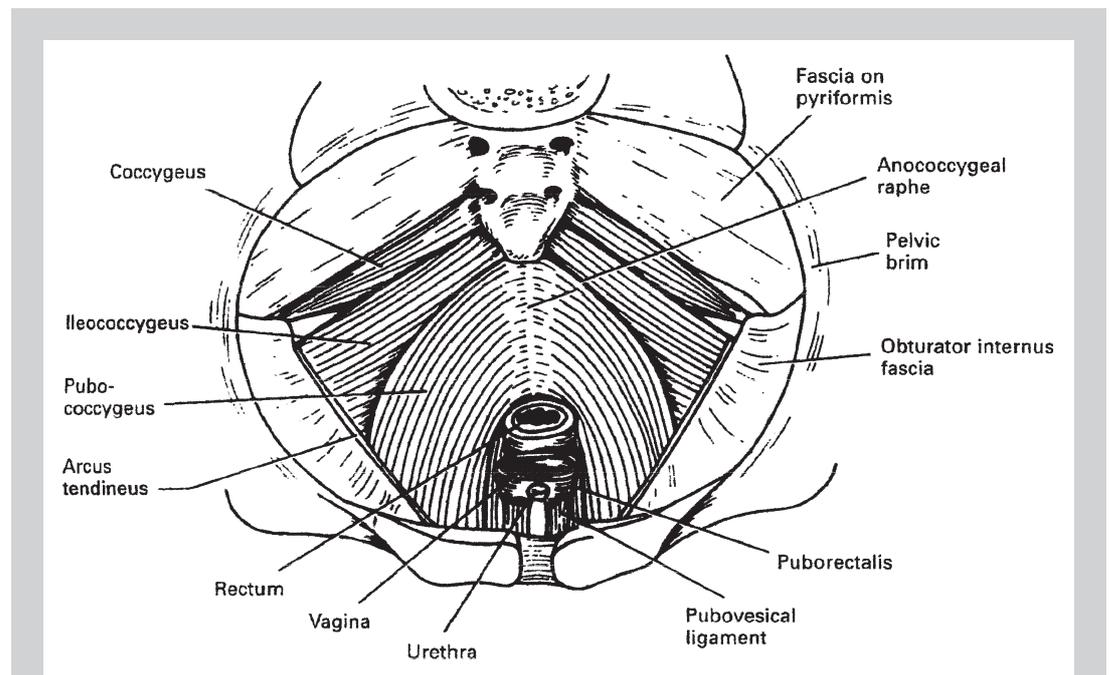
### KEYPOINTS:

- Incontinence is likely to impose considerable extra burdens on a disabled person and their carers in terms of loss of quality of life, social embarrassment and expense and it is therefore important that the neurologist is aware of what treatments are available and the scientific basis for their effectiveness.
- Whether voluntary or not, passage of urine through the urethra occurs when intravesical pressure exceeds intraurethral pressure.

**TABLE 1** Urinary symptoms may be related to either phase of bladder activity (i.e. storage or voiding)

Phase of bladder activity	Symptoms of dysfunction
Urine storage	Frequency, urgency, urge incontinence
Urine voiding	Hesitancy, intermittency, poor stream, terminal dribbling, incomplete emptying (+/- frequency and urgency)

## NEUORLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION



**FIGURE 1** The muscles of the pelvic floor, which are part of the pelvic organ support mechanism in females.

From Dixon JS, Gosling JA. The anatomy of the bladder, urethra and pelvic floor. In: Mundy TP, Stephenson AR, Wein AJ, editors. Urodynamics, principles, practice and application, 2nd ed. Churchill Livingstone; 1994. Reproduced with permission.

the spinal cord. The motor innervation passes through the pelvic and pudendal nerves to innervate the pelvic floor and striated urethral sphincter, respectively.

**Parasympathetic.** The principal autonomic innervation of the bladder is derived from cholinergic parasympathetic fibers that originate from the sacral cord. These pelvic parasympathetic preganglionic fibers are contained in the 2nd, 3rd and 4th sacral spinal nerve roots within the cauda equina, then peripherally in the pelvic nerves.

**Sympathetic.** The sympathetic innervation of the lower urinary tract arises in the lumbosacral sympathetic chain and passes to the bladder via the hypogastric and pelvic nerves. The sympathetic nerves to the bladder interact on parasympathetic ganglia that lie in the pelvic plexus on the bladder wall and may have an inhibitory effect during filling. Activation of  $\alpha$ -adrenoceptors in the smooth muscle of the bladder neck and urethra causes contraction and prevents leakage during filling as well as closing the bladder neck during ejaculation.

**Afferent.** Sensory information concerning bladder filling is thought to arise from unmyelinated free ending nerves which lie in the suburothelium and is conveyed in sensory fibers which run with the parasympathetic, somatic and sympathetic innervation in the pelvic, hypogastric and pudendal nerves. Based on data derived from animal experiments, information regarding detrusor muscle and urothelial derived sensation is thought to be conveyed via finely myelinated A $\delta$  fibers and unmyelinated c-fibers, respectively. Some afferents in the suburothelium are sensitive to changes in the chemical composition of urine, for example changes in pH, osmolality or irritants, such as bacterially derived lipopolysaccharide. Many of the presumed sensory nerves contain vesicles of neurotransmitters, such as neurokinins, ATP or calcitonin gene related peptide which may exert paracrine effects. In addition, some afferent nerves have receptors for these substances resulting in a "chemical dialogue" at the interface of the urothelium, detrusor muscle and afferent

nerves. Afferent discharges that occur during detrusor contraction appear to reinforce the central drive that maintains a detrusor contraction. Afferent pathways from the bladder have been shown to terminate not in the pontine micturition center, but in the periaqueductal gray in cats [1].

**Spinal interneurons.** The afferent fibers synapse on second-order spinal interneurons. These may act on preganglionic or motor nuclei or relay information to other regions of the spinal cord or higher centers. Interneuronal mechanisms play an important role in the integrated regulation of the lower urinary tract.

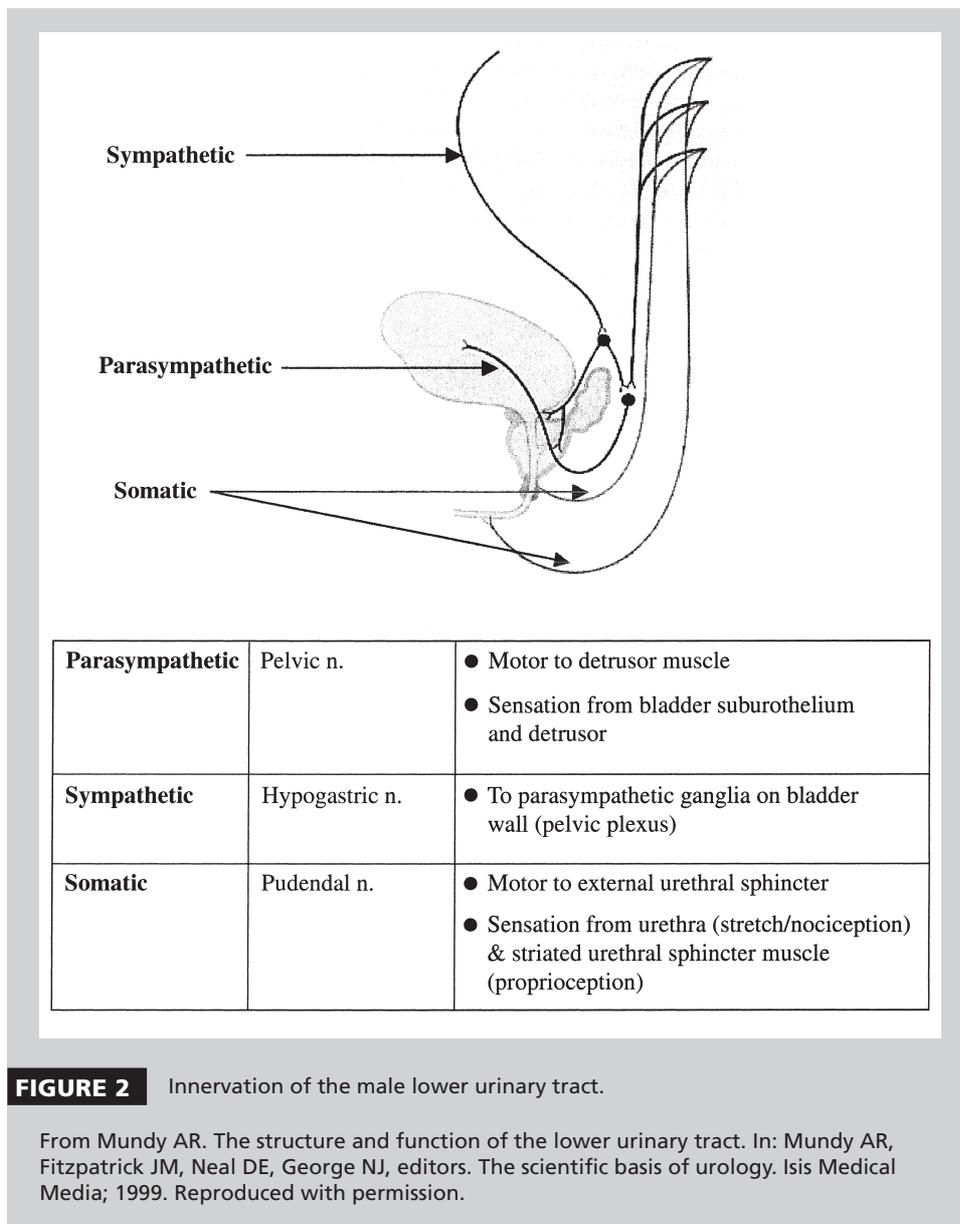
**Neurological control of the bladder.** A modern view of the control of the two mutually exclusive activities that the bladder performs, i.e. storage and voiding, is that neural programs for each process exist in the pons and that suprapontine influences act to switch from one state to the other. In both conditions, a reciprocal relationship is maintained between activity of the sphincter and the detrusor (Figure 3) [2].

In the late 1920s, Barrington demonstrated in the cat that there were areas in the pons that were important for bladder control, such that section of the brainstem below a certain level left the experimental animal unable to void. An area in the pons was identified and subsequently became known as the pontine micturition center (PMC) but is also known as "Barrington's nucleus". More recent animal experiments have shown that there are two separate areas in the PMC. There is a lateral region which when stimulated, results in a powerful contraction of the urethral sphincter, and a more medially placed region which when stimulated results in an immediate decrease in urethral pressure and silence of pelvic floor EMG signal, followed by a rise in detrusor pressure. It has, therefore, been

proposed that the lateral region be regarded as important for continence and the medial region, the site of activation for micturition [3].

There is no direct afferent input from the bladder to the PMC, but instead, connections between the periaqueductal gray are thought to provide information as to the state of fullness of the bladder [1].

Recent studies of voiding using positron emission tomography in male and female subjects have demonstrated that the neurological control of the bladder in man is essentially



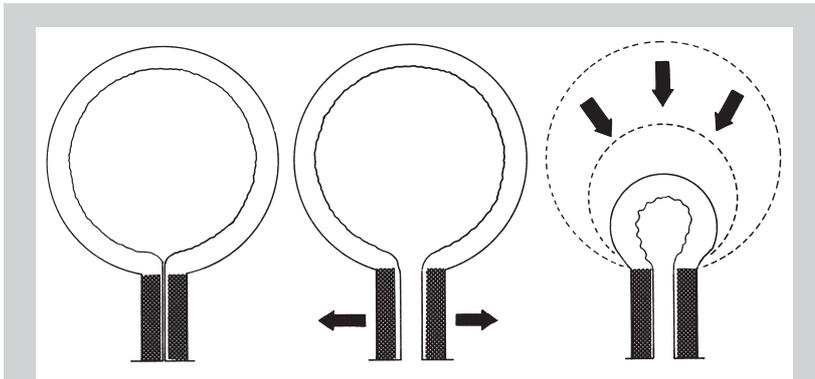
**FIGURE 2** Innervation of the male lower urinary tract.

From Mundy AR. The structure and function of the lower urinary tract. In: Mundy AR, Fitzpatrick JM, Neal DE, George NJ, editors. The scientific basis of urology. Isis Medical Media; 1999. Reproduced with permission.

**KEYPOINTS:**

- A modern view of the control of the two mutually exclusive activities that the bladder performs, i.e. storage and voiding, is that neural programs for each process exist in the pons and that suprapontine influences act to switch from one state to the other.

## NEUROLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION



**FIGURE 3** Urine storage and voiding. During urine storage, the detrusor muscle as it fills accommodates urine at a low pressure and does not contract — this involves the suppression of parasympathetic (motor) activity and the tonic contraction of the sphincters. Just prior to voiding, the intraurethral pressure falls as the sphincters relax and this is followed by detrusor contraction and voiding to completion.

### KEYPOINTS:

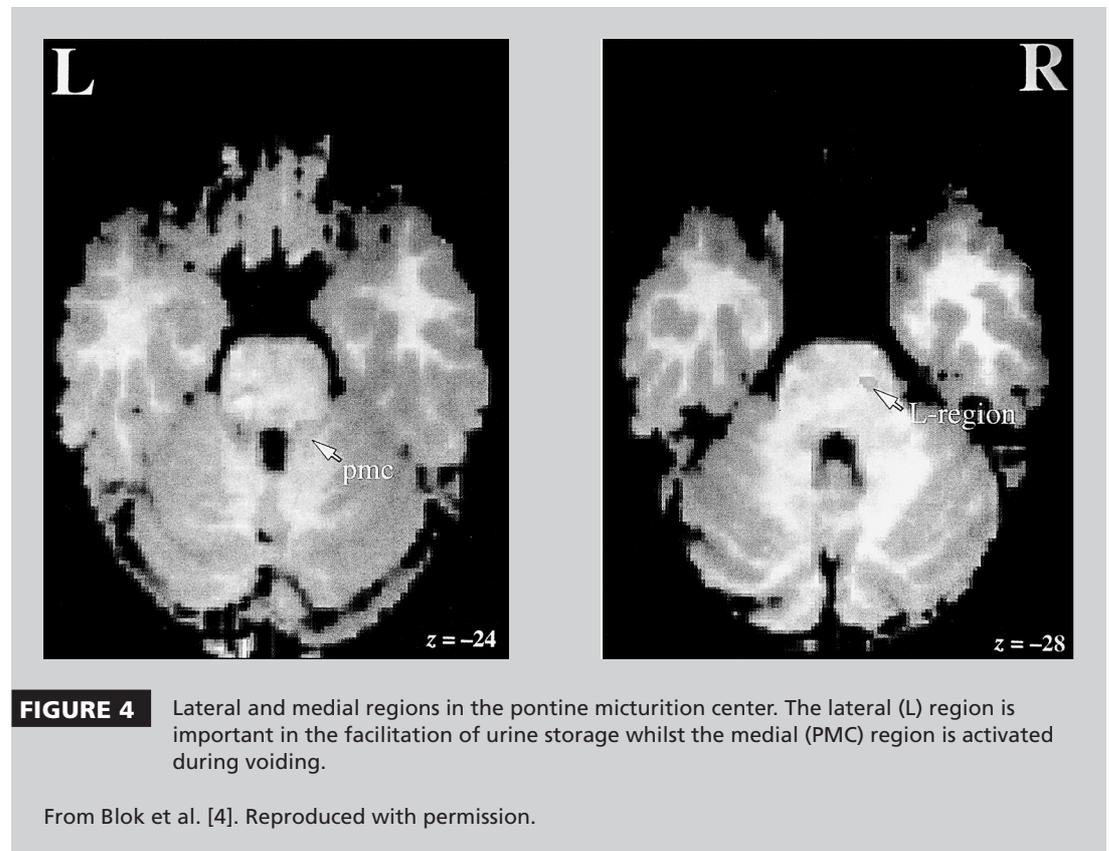
- To effect both storage and voiding, higher centers as well as connections between the pons and the sacral spinal cord must be intact, and also the peripheral innervation that arises from the most caudal segments of the sacral cord. The innervation needed for physiological control of the bladder is extensive, so that urinary incontinence is a likely consequence of neurological disease.

similar to that which had been demonstrated in experimental animals. Right-handed volunteers were trained to void whilst lying in the scanner, but a proportion of the subjects were unable to do so. In those able to void, there was

activity in a region of the medioposterior pons, but in the “unsuccessful” voiders, a region in the ventrolateral pontine tegmentum was seen to be activated (Figure 4). It was proposed that these areas are homologous to those that had been shown to exist in the cat [4].

The neurological processes in man that determine when to switch from storage to voiding remain to be elucidated. The extent of bladder fullness is obviously a critical factor, as is the sense of urinary urgency that may not necessarily reflect the bladder volume. The importance of areas in the frontal regions have long been recognized [5], and it seems likely that frontal lobe input is involved in the inhibition of micturition until it is socially appropriate.

Thus to effect both storage and voiding, higher centers as well as connections between the pons and the sacral spinal cord must be intact, and also the peripheral innervation that arises from the most caudal segments of the sacral cord. The innervation needed for physiological control of the bladder is extensive, so that urinary incontinence is a likely consequence of neurological disease [6].



**FIGURE 4** Lateral and medial regions in the pontine micturition center. The lateral (L) region is important in the facilitation of urine storage whilst the medial (PMC) region is activated during voiding.

From Blok et al. [4]. Reproduced with permission.

**TABLE 2** General risk factors for incontinence

Woman	Men
Age	Age
Pregnancy	Urinary symptoms
Childbirth	Functional impairment
Menopause	Cognitive impairment
Hysterectomy	Benign prostatic hypertrophy
Obesity	Postoperative
Urinary symptoms	Urinary tract infection
Functional impairment	Medications (e.g. diuretics)
Cognitive impairment	
Urinary tract infection	
Medications (e.g. diuretics)	

**URINARY INCONTINENCE**

Incontinence is defined as the condition where “involuntary loss of urine is objectively demonstrable and is a social or hygienic problem” (International Continence Society).

The prevalence of incontinence in the general population is highly age dependent and in those over the age of 65 years, as many as 10-20% are affected. The recognized risk factors associated with incontinence are listed in Table 2. In the community, many cases of incontinence have a multifactorial etiology.

Several different types of incontinence are recognized.

**Types of incontinence.**

(1) *Urinary urge incontinence* is the involuntary loss of urine that follows an uninitiated, uninhibitable detrusor contraction and is generally associated with a sensation of urinary urgency. There are several different causes of involuntary detrusor contraction, but the most common is the condition called “detrusor instability” (DI). The underlying pathophysiology of DI is as yet unknown, but rival hypotheses suggest that it is either myogenic and due to an abnormality of the detrusor smooth muscle or that it is neurogenic, resulting from abnormali-

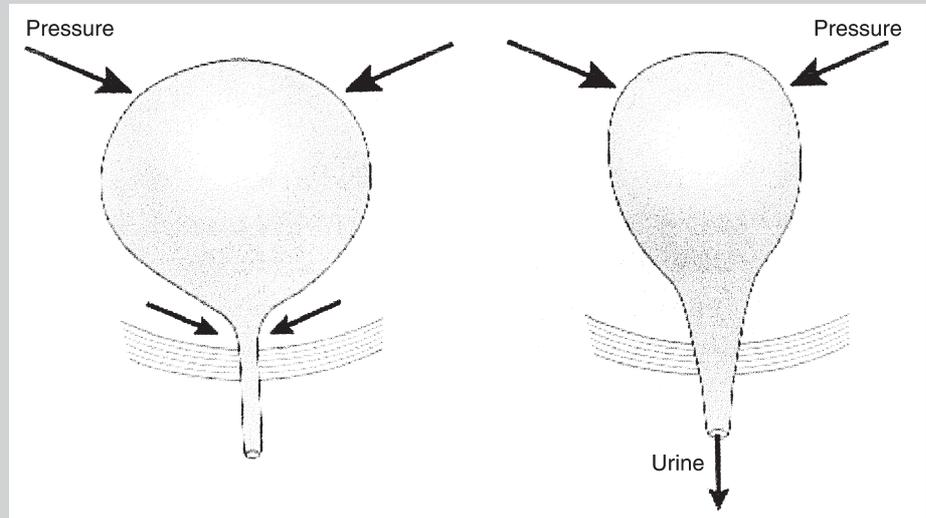
ties of the intrinsic innervation of the bladder [7].

In patients with neurological disease, the same disorder of overactive bladder function is referred to as “detrusor hyperreflexia (DH).” It is not possible to distinguish between DH and DI on cystometry (see Pullout 5) and the correct classification of a patient’s bladder disorder as DH depends on the clinical recognition of its neurological context.

(2) *Genuine stress incontinence (GSI)* is the involuntary loss of urine that follows a rise in intra-abdominal pressure in the absence of a detrusor contraction (Figure 5). Stress incontinence almost exclusively affects women because parity is the most important risk factor for the condition — essentially, the support mechanisms and/or nerve supply of the pelvic floor may be dam-

**KEYPOINTS:**

- Incontinence is defined as the condition where “involuntary loss of urine is objectively demonstrable and is a social or hygienic problem”.
- It is not possible to distinguish between DH and DI on cystometry and the correct classification of a patient’s bladder disorder as DH depends on the clinical recognition of its neurological context.



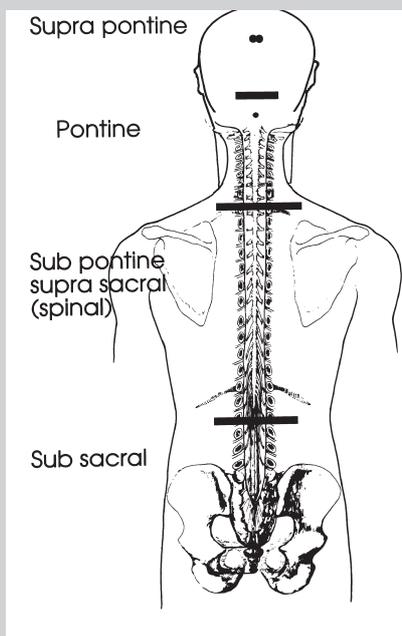
**FIGURE 5** In the bladder on the left, an increase in intraabdominal pressure (large arrows) is transmitted to the bladder. Stress urinary incontinence is prevented by numerous factors such as reflex contraction of the striated urethral sphincter. Pressure increase is also transmitted to the intraabdominal urethra (small arrows) which also has the effect of preventing leakage. In the bladder on the right, the bladder neck is open and the sphincter is deficient resulting in stress urinary incontinence.

From Versi E, Christmas TJ. Bladder. Health Press; 1998. Reproduced with permission.

## NEUROLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION

**FIGURE 6** Neurological causes of incontinence may

be classified according to the level of the disease. The most common site at which damage to the nervous system causes bladder dysfunction is in the spinal cord, interrupting connections between the pons and the sacral cord and resulting in disorders of storage and emptying, together with other features of spinal cord disease. Sub-sacral lesions may also affect both processes and cause dysfunction of other pelvic organs. Suprapontine, i.e. cortical disease can result in failure of appropriate "switching" so that unlike spinal cord disease the process of bladder emptying is normally co-ordinated, but occurs at an inappropriate time.



From Fowler CJ. Classification and investigation of neurovesical dysfunction. In: Whitehead HN, editor. A textbook of genitourinary surgery. Oxford: Blackwell Science; 1998. Reprinted with permission. Redrawn from Comarr AE. Diagnosis of the traumatic cord bladder. In: Boyarsky S, Editor. The neurogenic bladder. Baltimore: Williams and Wilkins; 1967. pp. 147-52. Courtesy of D Slotton.

### KEYPOINTS:

- Although there is a significant prevalence of incontinence in the general population, the prevalence is considerably higher in patients with neurological disease.

aged during pregnancy and parturition. In men, stress incontinence may occur following prostate surgery if there is inadvertent damage to the external sphincter or its innervation, since the bladder neck is incised during prostate resection and continence depends on an intact external urethral sphincter.

Stress incontinence may occur in women and men with neurological disease, such as cauda equina lesions, multiple system atrophy and neuropathies affecting the nerves that supply the continence mechanism [8].

(3) *Mixed urinary incontinence* is not uncommon in women and refers to co-existing stress and urge incontinence. Diagnosis and management can be difficult, but initial therapy is usually focused on the medical treatment of urgency, followed by the conservative management of stress incontinence (e.g. pelvic floor exercises).

(4) *Other causes of urinary incontinence:* Fistulae (e.g. vesicovaginal and uterovaginal and vesicovaginal fistulae) are seen following prolonged obstructed labor which is a problem in

parts of the world where medical resources are limited and transportation is poor [9]. Overflow incontinence may occur when there is an unrecognized disorder of bladder emptying and the capacity of an insensate, hypotonic bladder is exceeded.

Nocturnal enuresis in children rarely has a physical cause, but congenital anomalies (e.g. ectopic ureter) are encountered occasionally and should always be considered.

### Neurogenic incontinence.

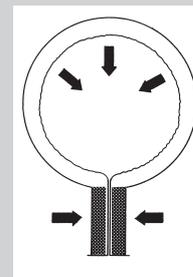
Although there is a significant prevalence of incontinence in the general population, the prevalence is considerably higher in patients with neurological disease. Neurological causes of incontinence are best classified with reference to the level of the pathology as suprapontine, pontine, spinal and subsacral (Figure 6).

*Suprapontine pathology:* The role of suprapontine input is to modulate the switching of storage and voiding. A failure of this will therefore present as a failure of storage and the various neurological causes of this type of bladder dysfunction are discussed in Chapters 8 and 9. Cortical causes of impaired initiation of micturition are rare, but may occur.

*Spinal cord disease:* Disruption of connections between the pontine micturition centers and the sacral cord is likely to result in both disorders of storage and of emptying. As explained earlier, the reciprocal activity of the sphincter and detrusor during voiding which ensures sphincter relaxation and detrusor contraction, is determined by the PMC. With disconnection from the pontine controlling center, the sphincter contracts with the detrusor resulting in the disorder of detrusor sphincter dyssynergia (DSD) (Figure 7).

Detrusor hyperreflexia following disconnection from the pons is thought to be due

**FIGURE 7** Loss of the reciprocal inhibition of the sphincter and activation of the detrusor following disconnection from the pons results in detrusor sphincter dyssynergia (DSD).



to the emergence of a new segmental reflex. In health, the afferent unmyelinated fibers are relatively unimportant in the generation of detrusor contractions and the relevant sensory information is conveyed to the periaqueductal gray in small myelinated fibers. However, with interruption of the physiological afferent spinal pathways, c-fiber afferents become the main initiators of detrusor contractions (Figure 8) [10].

The contractions so generated also may be poorly sustained. The combination of a poorly sustained hyperreflexic bladder contraction and DSD lead to a raised post voiding residual (PVR), which will further exacerbate the urgency associated with DH.

DH with DSD, is the most common abnormality that occurs with a spinal cord lesion, accompanied by a variable degree of spastic paraparesis (see Chapter 5).

*Sub-sacral:* Cauda equina lesions are the main cause of sub-sacral injury to the innervation of the lower urinary tract (see Chapter 6). Damage to the innervation of the bladder within the cauda equina is likely to affect both the anterior and posterior sacral roots and therefore affect both the afferent and efferent innervation of the pelvic organs. The clinical picture is therefore typically of sensory loss because of damage to the S2-S4 roots which innervate the back of the thighs, perianal and perineum region together with loss of voluntary control over both the anal and urethral sphincter, as well as sexual responsiveness. However, the second order parasympathetic innervation running to the detrusor from the spinal cord in the cauda equina terminates on the parasympathetic ganglia that lie in the bladder wall. The detrusor is therefore not denervated, but “decentralized”, and a range of bladder dysfunctions has been described following cauda equina injury.

With spinal dysraphism, there may be both lower and upper motor neuron deficits because of involvement of the conus, leading to a combination of detrusor hyperreflexia,

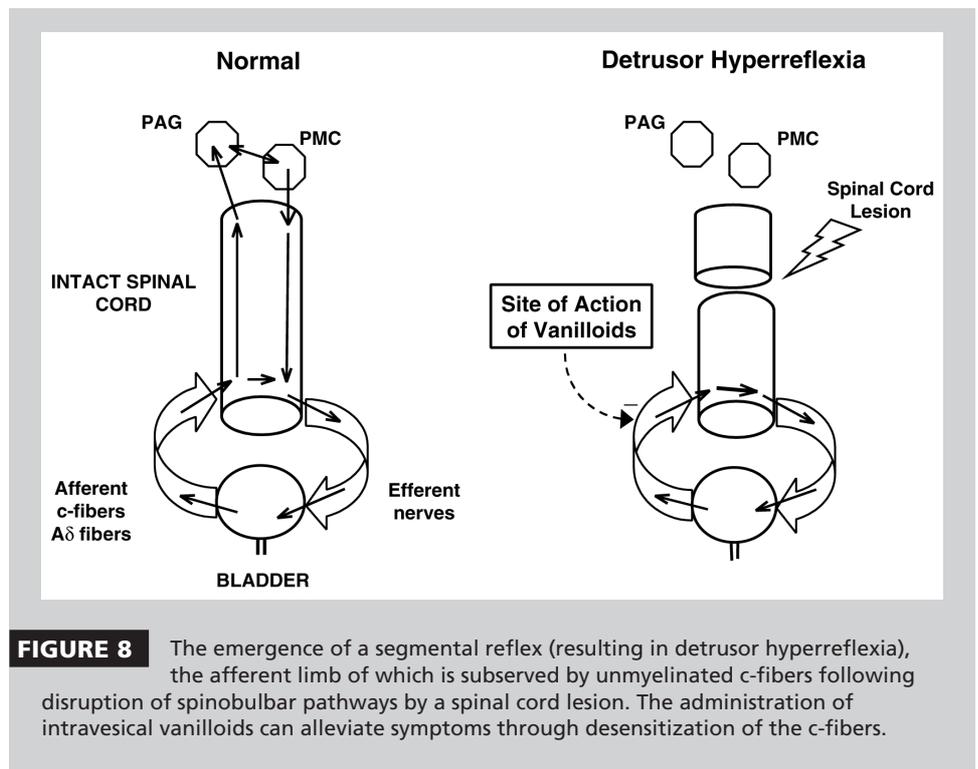
incomplete emptying and pelvic floor weakness which results in severe incontinence.

*Peripheral neuropathy:* The innervation of the bladder may be involved in the same neuropathic process as affects the innervation of the limbs in a peripheral neuropathy. The pelvic plexus can also be damaged by radical surgery within the pelvis resulting in bladder dysfunction, usually impaired emptying, in the absence of somatic sensory loss. This is discussed further in Chapter 7.

#### Investigation of urinary incontinence.

*Patients with neurological disease known to cause bladder dysfunction.* Basic investigations and management of incontinence in patients with established neurological disease can be carried out by the neurologist (Table 3). Of the various tests used in the assessment of incontinence (Pullouts 1-5), the most important is measurement of the PVR.

Although, in most instances, it may be correctly deduced from a patient’s complaint of urinary urgency that there is underlying detrusor hyperreflexia, the extent of incomplete bladder emptying cannot be reliably



**FIGURE 8** The emergence of a segmental reflex (resulting in detrusor hyperreflexia), the afferent limb of which is subserved by unmyelinated c-fibers following disruption of spinobulbar pathways by a spinal cord lesion. The administration of intravesical vanilloids can alleviate symptoms through desensitization of the c-fibers.

## NEUROLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION

### KEYPOINTS:

- Although, in most instances, it may be correctly deduced from a patient's complaint of urinary urgency that there is underlying detrusor hyperreflexia, the extent of incomplete bladder emptying cannot be reliably ascertained from the history.
- Measurement of the PVR is mandatory before commencing treatment with anticholinergic medication in a patient with known neurogenic bladder symptoms.
- When a patient fails to respond to anticholinergics, it is advisable to re-assess the PVR and ensure this has not increased.

**TABLE 3** Minimum urological investigations required

#### Patients with overt neurological disease

- ▶ Typical lower urinary tract symptoms require:
  - Post void residual (PVR) measurement
  - Urinalysis/urine culture
  - Frequency volume chart
- ▶ When atypical lower urinary tract symptoms are present also include:
  - Urine cytology
  - Kidney, ureter, bladder X-ray
  - Urodynamics
  - Cystoscopy
  - Upper tract imaging (ultrasound or intravenous urogram)

#### Patients without overt neurological disease

- ▶ All patients require:
  - Urinalysis/MSU
  - Urine cytology
  - Frequency volume chart
  - Post void residual
  - Urodynamics
- ▶ And refer to a urologist for:
  - Kidney, ureter, bladder X-ray
  - Cystoscopy
  - Upper tract imaging
  - Pad testing

ascertained from the history. If the PVR is raised, persistent urine in the bladder will stimulate hyperreflexic contractions and treatment with increasing doses of anticholinergic medication will not succeed unless some means of improved bladder emptying, namely clean intermittent self-catheterization (CISC) is also used. Furthermore, anticholinergic medication can impair already compromised bladder emptying, further exacerbating symptoms of detrusor hyperreflexia (Figure 9). For this reason, measurement of

the PVR is mandatory before commencing treatment with anticholinergic medication in a patient with known neurogenic bladder symptoms (Figure 10).

Urine microscopy and culture should also be done as recurrent urinary tract infections are common in patients with incomplete emptying, and will exacerbate symptoms considerably.

The need for full cystometry (Pullout 5) in patients with established neurological disease has been questioned. The investigation

### PULLOUT 1 Post void residual (PVR)

After the patient has voided normally, the PVR can be measured using "in-out" catheterization or ultrasound. It is important to ask the patient whether they use any special maneuvers to empty the bladder, such as abdominal straining, bending forward or applying direct pressure to the lower abdominal wall, since, if they do, the figure obtained for the PVR will not be a reflection of detrusor contractility.

When the bladder does not empty completely, the residual urine will exacerbate symptoms of storage dysfunction markedly in two ways. Firstly, it produces a sensation of incomplete bladder emptying, although this may be impaired in patients with neurological disease. Secondly, the time to reach bladder capacity is reduced as the PVR increases. Indeed, patients with neurogenic incontinence will very often have a decreased bladder capacity, so that the time taken to fill the bladder may be very short indeed. The end result is that frequency of micturition, urinary urgency and urge incontinence all worsen with increasing PVR.

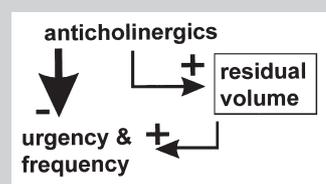
In addition, as the PVR increases, anticholinergic medications become less effective in controlling symptoms, and when the PVR is in excess of 100 ml, patients should be encouraged to do clean intermittent self-catheterization, if possible (Pullout 6). Measurement of the post micturition residual volume is also recommended before starting anticholinergic therapy, because these drugs decrease detrusor contractility, which adversely affects bladder emptying. When a patient fails to respond to anticholinergics, it is advisable to re-assess the PVR and ensure this has not increased (Figure 10).

Finally, residual urine is likely to become infected — a further reason for measuring the PVR and instituting CISC.

is indicated if there is uncertainty as to the pathophysiological basis of a patient's bladder symptoms, but this is unlikely to be the case in a patient with, for example MS, in whom investigations should be focussed on symptom management. However, if the patient fails to respond to first-lines measures, cystometry may provide useful information regarding the properties of the bladder during filling and voiding.

Cystometry is essential following traumatic spinal cord injury and spina bifida where high resting intravesical pressure

**FIGURE 9** Although anticholinergic medication can suppress detrusor hyperreflexia, it may also impair already compromised bladder emptying leading to an increasing post-void residual volume, which, in turn, may exacerbate symptoms of urgency and frequency.



might be expected. In these cases, there is a risk of silent chronic upper tract damage as the ureters are unable to empty into a

**PULLOUT 2** Frequency-volume chart

A frequency-volume chart is a self-administered fluid-balance chart that provides an objective record of micturition pattern and is useful in the interpretation of symptoms of storage dysfunction.

Information recorded includes the time and volume of each drink and void and whether the void was preceded by urinary urgency or associated with incontinence (graded as "drops, moderate loss or flooding incontinence"). Pad or catheter usage should also be recorded along with the time of relevant medication (e.g. diuretics or anticholinergics).

Day: \_\_\_\_\_ Date: \_\_\_/\_\_\_/\_\_\_

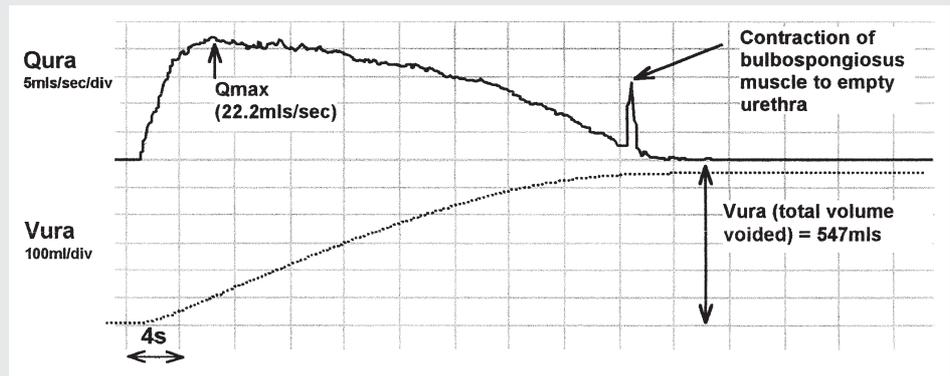
Time hr:min	Drink	Urge?	Leak? +, ++ +++	Urine Passed Void/ ISC (mls)	Residual Urine? (if ISC) (mls)	Pad change	Tabs?
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Time to Bed:

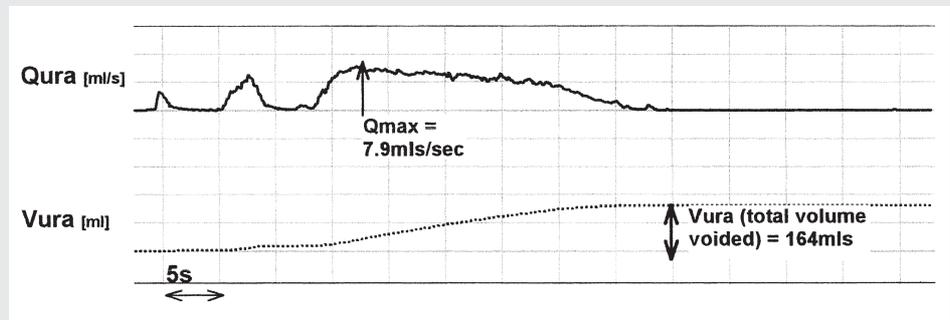
**PULLOUT 3 Uroflowmetry**

Uroflowmetry is a noninvasive test indicated in the assessment of voiding disorders. The patient is asked to void into a modified toilet and urine flow slows the speed of a motor driven spinning wheel in the base, from which the volume of urine voided per unit time (ml/s) is measured. In health, the normal void gives a bell-shaped curve on the uroflow graph (Figure A). The maximum flow rate ( $Q_{max}$ ), mean flow ( $Q_{ave}$ ) rate and volume voided ( $V_{ura}$ ) are assessed in conjunction the uroflow curve. It is not possible to distinguish between low flow secondary to detrusor failure and bladder outlet obstruction. To interpret a uroflow trace meaningfully, the total voided volume should be greater than 150 ml.

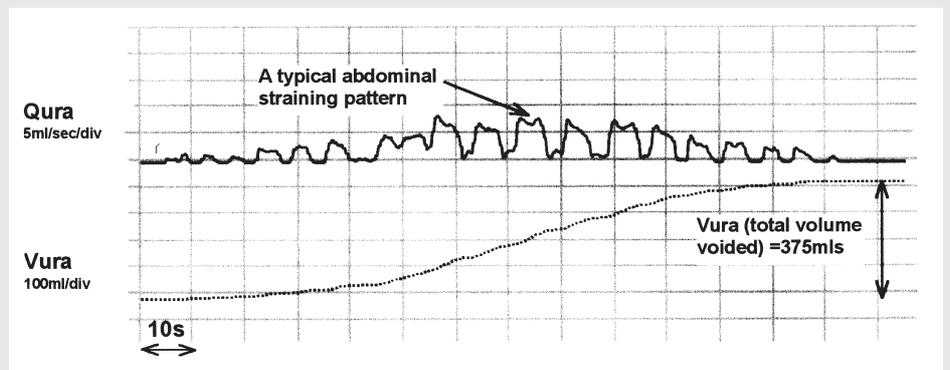
(A) Normal uroflow and complete emptying. The detrusor, like all smooth muscle, is unable to contract as rapidly as skeletal muscle, resulting in a smooth bell-shaped uroflow curve. Therefore, it is generally possible to distinguish between a detrusor generated uroflow curve and one produced by abdominal straining, which undulates characteristically. The abrupt transient increase in flow recorded towards the end of the flow represents the voluntary contraction of the bulbospongiosus muscle, which empties the urethra of urine at the end of a void.  $Q_{ura}$  is the rate of urine flow in milliliters per second,  $V_{ura}$  is the volume voided (547 ml),  $Q_{max}$  is the maximum urine flow in this trace (22.2 ml/s),  $Q_{ave}$  is the average flow rate (14.5 ml/s).

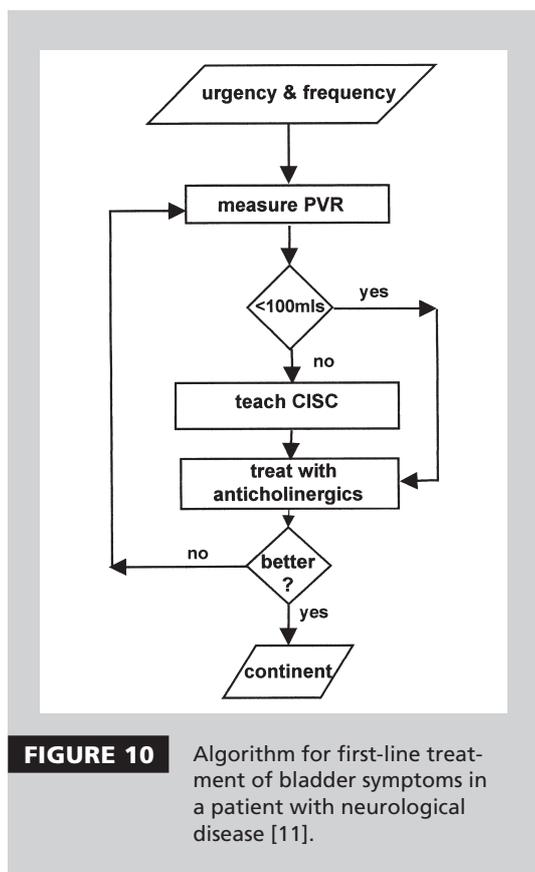


(B) This figure illustrates a uroflow trace and low urinary flow rate. It is not possible to distinguish whether the low flow rate is secondary to bladder outlet obstruction or detrusor failure. Bladder outlet obstruction can be caused by sphincteric dysfunction (e.g. DSD), benign prostatic obstruction or a urethral stricture. Maximum urinary flow rate ( $Q_{max}$ ) is 7.9 ml/s, average urinary flow rate ( $Q_{ave}$ ) is 3.8 ml/s in this patient. The total voided volume ( $V_{ura}$ ) is 164 ml.



(C) Intermittent and low uroflow trace due to abdominal straining. Note the typical abdominal straining pattern, generated by repeated voluntary contractions of skeletal muscle.  $V_{ura}$  (total volume voided) is 415 ml,  $Q_{max}$  is 9.3 ml/s,  $Q_{ave}$  is 3.7 ml/s.





high-pressure bladder, resulting in obstructive uropathy. Cystometry combined with fluoroscopy (“video urodynamics”) would be the investigation of choice in the diagnosis of DSD and ureteric reflux. It is unknown why obstructive uropathy in patients with MS is rare despite the fact that DSD, DH, a reduced bladder capacity and incomplete emptying are common findings in these patients.

Patients who do not respond to medical treatment or who have recurrent urinary tract infections should also have a urological evaluation to rule out conditions such as bladder calculi, ureteric reflux or carcinoma in situ.

*Patients with neurological disease in whom the neurogenic basis for bladder dysfunction is uncertain.* In patients with established neurological disease who develop urinary symptoms, the question may arise as to whether these new symptoms are part of the neurological condition or indicate a co-existing urological pathology. This can be particularly difficult if the neurological condition

is one that is known to be associated with a bladder disorder. The prime example of this situation is a man with long standing Parkinson’s disease who develops urinary frequency and urgency. It can be difficult to distinguish between a neurogenic and urological cause of bladder dysfunction. Voiding cystometry is indicated to attempt to identify the extent to which outflow obstruction is contributing to the bladder disorder, and referral to a urologist is therefore indicated (see Table 4).

Cystometry may also identify “sensory urgency” where symptoms of storage disorder are associated with bladder “hypersensitivity”. This is a disorder of bladder function which is not neurogenic, the etiology of which remains unknown, but can have symptoms somewhat similar to the neurogenic disorder of detrusor hyperreflexia.

*Patients without known neurological disease and normal urological investigations.* Patients with marked urinary symptoms will have been thoroughly investigated by a urologist who, on discovering no structural cause, may refer the patient to a neurologist asking if there is an occult neurological basis for the bladder disorder. The clinical examination of such a case should focus particularly on whether or not there is an early spinal cord lesion — since most other causes of a neurogenic bladder are likely to be associated with clinically apparent neurological deficits.

Lower limb sensory evoked potentials are helpful in this respect, more so than the pudendal evoked potential. MR scanning of the cord is likely to be performed in these circumstances, although an abnormality is rarely found if the clinical examination and evoked potentials are all normal.

**KEYPOINTS:**

- It is not possible to distinguish between low flow secondary to detrusor failure and bladder outlet obstruction on uroflowmetry.
- Patients who do not respond to medical treatment or who have recurrent urinary tract infections should also have a urological evaluation to rule out conditions such as bladder calculi, ureteric reflux or carcinoma in situ.
- Patients with marked urinary symptoms will have been thoroughly investigated by a urologist who, on discovering no structural cause, may refer the patient to a neurologist asking if there is an occult neurological basis for the bladder disorder.

**PULLOUT 4 Pad testing**

A pad test may be performed to quantify objectively urine loss in cases of stress or urge incontinence. It is also useful in the pre-operative assessment of GSI and in assessing the response to anticholinergic therapy in mixed incontinence.

Pre-weighed incontinence pads are worn for intervals — the time over which data is collected varying from 20 minutes to 48 hours, depending on the protocol, during which patients may be asked to perform various exercises. The pads are then reweighed.

**TABLE 4** Investigations used in urological assessment of incontinence

- Pelvic floor assessment/digital rectal examination of the prostate
- Frequency-volume chart
- Pad testing
- Uroflowmetry and postmicturition ultrasound
- Urodynamic studies (cystometry +/- fluoroscopy, urethral pressure profilometry)
- Cystoscopy
- Urine cytology

**KEYPOINTS:**

- M3 receptors appear to be more important in bladder contraction.

**MANAGEMENT OF NEUROGENIC INCONTINENCE (TABLE 5)**

**Oral therapy.** Detrusor muscle contraction, whether physiological or pathological is mediated largely through parasympathetic cholinergic activation of muscarinic receptors. Thus the treatment of those symptoms resulting from detrusor hyperreflexia, such as urgency and frequency, are appropriately treated by antimuscarinic, anticholinergics.

There are five different subtypes of muscarinic receptors (M1-M5) present in different proportions in the end organs innervated by the parasympathetic nervous system, namely the smooth muscle of the cardiovascular, gastrointestinal, genitourinary and respiratory systems, the ciliary muscles and the salivary glands. Unwanted blockade of the cholinergic receptors in these target organs accounts for the side effects experienced with all these drugs. M2 and M3 receptors have been identified using molecular biological techniques in the detrusor, with M2 receptors predominating (M2:M3 = 3:1). However, M3 receptors appear to be more important in bladder contraction. It is likely that muscarinic receptor subtypes M1, M4 and M5 are also present in the bladder. Contraction of the detrusor muscle is inhibited and bladder capacity increases following the

administration of anticholinergics, resulting in reduced frequency and urgency.

Oxybutynin is a tertiary amine with mixed pharmacological activity. In addition to its anticholinergic properties, oxybutynin also has smooth muscle relaxing (antispasmodic or musculotropic) effects. It also has anesthetic properties that may play a role in its reported intravesical action, but it is likely that when administered orally, its clinical effect is based upon its anticholinergic properties alone.

Dose-related side effects include a dry mouth, constipation and rarely blurring of vision and drowsiness. Dry mouth due to blockade of muscarinic receptors in the salivary glands resulting in decreased salivation, is the commonest reason for poor patient compliance. A sustained-release once-daily oral preparation of oxybutynin has recently become available which has a lower incidence of dry mouth, whilst maintaining stable and therapeutic levels of the medication.

Oxybutynin can be directly instilled into the bladder, which seems to lessen side effects, but this therapy has not yet gained universal acceptance, largely because of the inconvenience involved in preparing instillations regularly [13]. However, an intravesical slow-release drug-delivery system has recently been developed which releases a constant dose of oxybutynin over a period of 1 month. Adequate plasma concentrations are

**TABLE 5** Management of neurogenic incontinence

**First-line treatment options**

- Oral anticholinergics
- CISC (when PVR >100 ml)

**Second-line treatment options**

- DDAVP
- Treatments under evaluation (intravesical oxybutynin, resiniferatoxin, botulinum toxin)

**Late treatment options**

- Indwelling catheters (suprapubic vs. urethral drainage)
- Pads
- Surgery (e.g. augmentation cystoplasty or urinary diversion) in selected cases

achieved, but the side effects are less than those that occur with comparable oral dosages, thought possibly to be due to lack of intestinal enzymatic conversion which is responsible for the formation of the metabolite causing side effects.

Oxybutynin has been found to be safe and effective for long-term use.

Tolterodine is an anticholinergic with similar efficacy to oxybutynin, but has less in the

way of side effects, dry mouth, in particular. This is thought to be because it has a greater affinity for detrusor than salivary gland muscarinic receptors.

Darafenacin is a highly selective M3 blocker currently undergoing clinical trials.

The activation of muscarinic receptors in the detrusor muscle results in both the influx of calcium and its release from intracellular stores — calcium channel blockade is, there-

#### KEYPOINTS:

- The detrusor pressure ( $P_{det}$ ) is derived by subtracting the intra-abdominal pressure ( $P_{abd}$ ) from the intravesical pressure ( $P_{ves}$ ).

### PULLOUT 5 Cystometry

Cystometry [12] is an invasive test requiring urethral catheterization to measure intravesical pressure as well as measurement of intra-abdominal pressure via a fine intrarectal pressure line.

The test is divided into two phases: filling and voiding. After the patient has voided normally, a filling catheter and pressure recording line is passed into the bladder through the urethra and the PVR volume is recorded. The catheter is then connected to a manometer so that intravesical pressure can be measured (cm water). A catheter is also inserted into the rectum and connected to a manometer to measure the intra abdominal pressure. The detrusor pressure ( $P_{det}$ ) is derived by subtracting the intra-abdominal pressure ( $P_{abd}$ ) from the intravesical pressure ( $P_{ves}$ ).

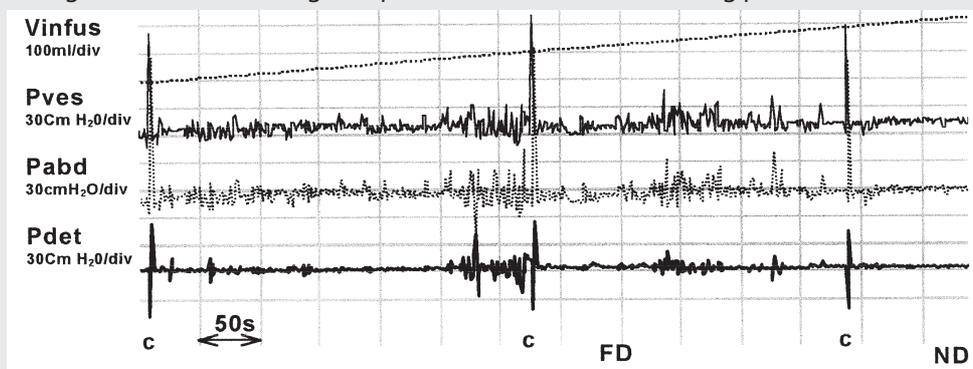
Cystometry may be performed in the lying, sitting or standing position. At the beginning of the test resting  $P_{det}$  should be between 0 and 5 cm H<sub>2</sub>O and  $P_{abd}$  and  $P_{ves}$  should be between 20 and 50 cm H<sub>2</sub>O. The  $P_{abd}$  and  $P_{ves}$  will vary in relation to patient position — sitting or standing increases the weight of the abdominal viscera on the bladder. Ideally, the filling phase should be performed with the patient in the lying position, and prior to voiding, the patient is repositioned into either the standing or sitting position, although this is often difficult in patients with poor mobility and detrusor hyperreflexia.

The bladder is filled with warmed saline at slow-, medium- or fast-fill rates. Slow-fill rates (i.e. less than 20 ml/min) are preferred in patients with neurogenic bladder problems because unstable contractions can be provoked by fast-fill rates. The patient is instructed to communicate any bladder sensations to the investigator. Valsalva straining and coughing at regular intervals during filling is used to assess urethral competency. Whenever the patient feels as if he or she would normally void, the pump is stopped, the filling catheter is removed and the patient voids into a uroflowmeter with the pressure lines in situ. Thus information on bladder storage functions, namely sensation, capacity, detrusor compliance and stability as well as urethral competency is provided in the filling phase.

During cystometry, the patient must not try to suppress an urge or the pump stopped in order to allow the urge to settle. It must be accepted that certain artifacts will limit the result, but the investigator should aim to reproduce the patient's troublesome symptoms.

(A) A cystometric trace demonstrating normal bladder filling. The patient was filled in the standing position

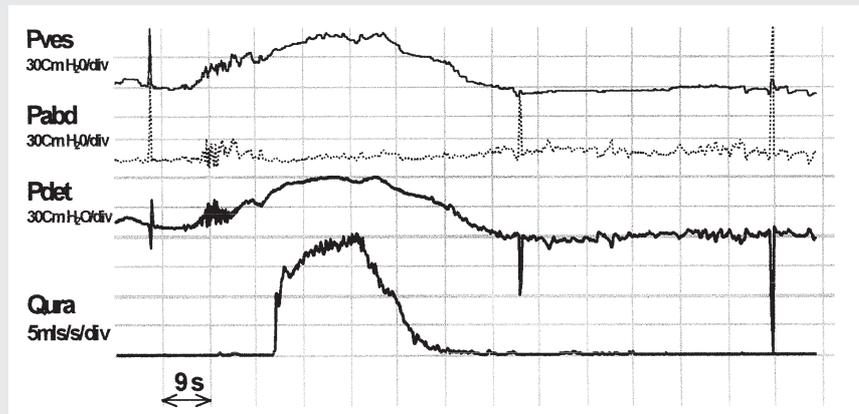
with 0.9% saline at a rate of 30 ml/s and the point of first sensation of filling (FD) is marked on the trace. The pump was stopped when he had a normal desire to void (ND) and the total volume infused ( $V_{infus}$ ) was 254 ml. The patient was asked to cough at regular intervals to check the accuracy of the pressure recordings (c = cough).



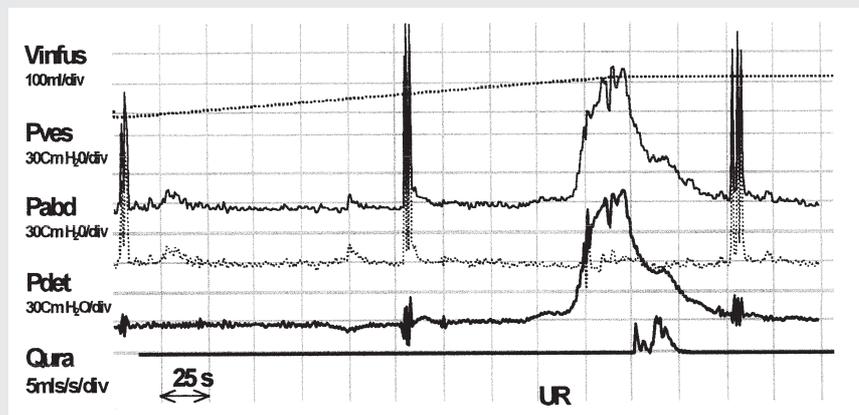
## NEUORLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION

Pullout 5 continued ►

(B) The patient (in A) is now ready to void. The bladder filling catheter is removed prior to voiding, but both pressure recording catheters are left in place. Urine flow ( $Q_{ura}$ ) is recorded by a uroflowmeter and during the flow, the detrusor pressure ( $P_{det}$ ) is observed. Any abdominal straining during the flow can be identified and quantified.  $Q_{max}$  was 20.5 ml/s,  $P_{det}$  at  $Q_{max}$  was 49 cmH<sub>2</sub>O in cases of bladder outlet obstruction this pressure would be much higher, until decompensation of the detrusor occurs. This is a normal study.



(C) Cystometrogram trace demonstrating detrusor hyperreflexia (DH) and urge incontinence. After 118 ml had been instilled ( $V_{infus}$ ), the patient experienced urgency (UR) that was associated with an uninitiated, uninhibitable detrusor contraction ( $P_{det}$ ) and involuntary leakage of urine occurred ( $Q_{ura}$ ).  $P_{ves}$ , intravesical pressure;  $P_{abd}$ , intra-abdominal pressure;  $P_{det}$ , detrusor pressure;  $Q_{ura}$ , urine flow.



### KEYPOINTS:

- CISC has transformed the uro-neurological care of patients with all types of neurogenic bladder dysfunction.

fore, another potential target for pharmacological intervention in the treatment of DH.

Propiverine is a recently introduced drug that has both anticholinergic and calcium channel blocking properties.

In patients with typical symptoms of storage dysfunction and a PVR less than 100 ml, anticholinergic drug therapy may be commenced see Figure 10 [14].

**Clean intermittent self-catheterization.** CISC was introduced in 1972 and has been shown to be safe and effective method of emptying the bladder in patients who have a raised PVR. Efforts to treat detrusor hyperreflexia with anticholinergics or other treatments are unlikely to succeed when the PVR is in excess of 100 ml, since residual urine reduces the interval between unstable bladder contractions. Draining a residual will therefore help to reduce symptoms of frequency

and urgency (see Pullout 1). It is true to say that CISC has transformed the uro-neurological care of patients with all types of neurogenic bladder dysfunction (see Pullout 6 and Table 6) as formerly these patients would have been offered continuous catheter drainage.

The commencement of a CISC regimen is a significant intrusion into a patient's daily routine, but many patients adapt well to it because of the improvement in symptoms it may afford. The required frequency of catheterization is largely determined by symptoms. For example, many patients will not empty well after the first void in the morning and will benefit from catheterization at this time. Likewise, they should perform CISC before going to bed as nocturnal frequency may so be greatly reduced. Catheterization at variable times during the day, such as prior to leaving

home or before a meeting or social event, may also help control symptoms.

Importantly, in addition to alleviating bladder symptoms, CISC may help reduce the risk of urinary tract infection, acquired ureteric reflux and obstructive uropathy, all of which are associated with high PVR (see Pullouts 1 and 6). There is, however, an accepted risk of urinary tract infection from using the technique [15].

**Desmopressin.** A common problem is nocturnal frequency and nocturnal incontinence secondary to severe DH. Despite use of anticholinergic agents, some patients may continue to be woken by urinary urgency. An effective agent for treatment of nocturia is desmopressin (DDAVP), which is an analog of the posterior pituitary hormone, antidiuretic hormone (ADH) (see Pullout 7).

**Future treatments.** Animal experimental studies on the mechanism of DH following spinal cord injury have shown that the neural mechanism for this appears some weeks after acute spinal cord transection and is due to the emergence of a new functional pathway which operates at a sacral segmental level rather

**TABLE 6** Advantages of CISC over indwelling catheterization

Bladder capacity can be better preserved
The patient is independent to empty the bladder
There is no permanent catheter (which requires continuous care)
Bladder neck erosion and prostatitis are avoided
Interference with sexual function is avoided
Urethral trauma/strictures/diverticuli and fistulae are avoided
Catheter blockage and bypassing (leakage around the catheter) are avoided
Catheter-related calculi are avoided
The incidence of urinary tract infection/epididymo-orchitis and septic episodes is less than in those on CISC
There is probably a decreased risk of long-term, catheter associated bladder cancer

than trans-spinaly (Figure 8). This led to the therapeutic use of the selective c-fiber neurotoxin, capsaicin to “de-afferent” the detrusor muscle in cases of spinal hyperreflexia [16]. Early reports of the success of resiniferatoxin (RTX), an analogue of capsaicin to treat detru-

## **PULLOUT 6** Clean intermittent self-catheterization (CISC)

The rationale of CISC should be explained and the importance of adhering to proper hygienic (but *not* sterile) technique. Patients must also be assured that there is no risk of damage to the external urethral sphincter by passing a catheter or ultimate “dependence” on the catheter to void — two common patient concerns. Urethral false passages and bladder perforation are extremely rare complications. Introducing infection may also be a problem but the benefits of regular CISC far outweigh the complications of its use.

A dedicated nurse who will teach CISC and provide follow-up for advice and assistance is critical to success with teaching CISC. The patient must also be motivated to perform it regularly. Hygiene is especially important — the patient must have access to a supply of clean tap water for hand washing and preparation and cleansing of the catheters before and after use.

The patient gently cleanses the external urethral meatus and slowly advances the lubricated catheter into the bladder to drain the urine. Patients get used to passing catheters, although the time this takes depends on many factors including the patient’s manual dexterity, eyesight and also attitude.

Catheter size varies in length (according to sex) and diameter (10-14 Ch are most commonly used). Catheters suitable for children are also available. Smaller sized catheters will drain urine more slowly and some patients therefore prefer a larger diameter. Catheters may be single-use or reusable: reusable catheters are rinsed under a tap before use and coated with a water-based lubricant (e.g. “KY Jelly”). Single-use catheters have a hydrophilic coating that is activated on contact with tap water and which acts as a lubricant. There is no evidence to suggest that there is an increased risk of infection when reusable catheters are used instead of the single-use variety.

Many patients with neurological disease will simply be unable to catheterize due to decreased manual dexterity, leg spasms or mobility and their carer may then be taught the technique. Some patients will not have carers who are prepared to do this and some patients may be unwilling to do self-catheterization because of a dislike of the principal. Attitude is clearly very important in determining the success of this form of management.

### **PULLOUT 7**    **Desmopressin (DDAVP)**

Desmopressin promotes increased water reabsorption at the collecting tubule of the kidney, resulting in reduced nocturnal urine production. The use of DDAVP can result in hyponatremia and should be used with great caution in the elderly. All patients should be warned that if they develop headache, general malaise or swelling of the face or ankles they should discontinue the medication and serum sodium should be checked. DDAVP is suitable for use in younger patients, including children with DH.

Whilst DDAVP is not intended for use during the day, patients with DH may occasionally use the drug prior to meetings or social events, when they wish to have the added security of being free from urinary urgency. It is, however, important to stress that DDAVP should only be used once in 24 hours.

essor hyperreflexia have promising implications for development of therapies in the future (see Pullout 8). It may well be that treatments based on this principle of “de-afferentation” will only be effective in detrusor hyperreflexia of spinal origin and not for DH of suprapontine origin or in cases of detrusor instability [17].

Reports of intravesical injections of botulinum toxin in patients with severe DH are showing interesting results.

**Indwelling catheters.** With progression of a disabling, neurological disease, decreased mobility and cognitive decline may make the combination of anticholinergics and CISC no longer possible. At this point, long-term catheterization is unavoidable.

Patients may have either a urethral catheter or suprapubic catheter. It is not unreasonable to insert a urethral catheter initially and if and when any of the common complications of urethral catheterization arise, arrange for a urologist to insert a suprapubic catheter. Problems associated with indwelling urethral catheters include catheter blockage and “bypassing” (leakage around the catheter secondary to a detrusor contraction, which generally occurs when the catheter is blocked). Catheter blockage is caused by encrustation of the catheter by crystals present in the urine, that adhere to any foreign body (the catheter). In addition, solutes in the urine can precipitate out of solution forming calculi, given certain physiological circumstances (e.g. urine pH or osmolality); and the presence of a foreign body is an important factor in this process. Bypassing is best managed by changing the catheter more frequently and may require the prescription of anticholinergics.

Urethral catheters may cause bladder neck erosion, sphincter damage and urethral trauma, which are difficult problems to manage in debilitated women.

A further disadvantage of urethral catheters is that they interfere with sexual function which is especially important for younger patients, and this must be taken into consideration.

Suprapubic catheterization involves the insertion of a catheter into the bladder directly through a small surgical incision in the lower

### **PULLOUT 8**    **Intravesical vanilloids**

The intravesical administration of vanilloids (capsaicin and RTX) in patients with spinal DH results in a reduction of suburothelial nerve densities and ultrastructural changes in unmyelinated C-fibers of the bladder. The vanilloid receptor (VR) is a non-selective cation channel present on unmyelinated afferent nerves. The VR integrates noxious stimuli at a cellular level and thus, in vitro, the action of the VR is facilitated by changes in acidity, rapid changes in temperature, bacterial lipopolysaccharide and the vanillyl-containing molecules, including capsaicin and RTX. RTX has 1000 times greater affinity for the vanilloid receptor and has less initial side effects compared to capsaicin (e.g. suprapubic pain or burning urethra).

Capsaicin is derived from the *capsicum* red hot chilli pepper and RTX from the dried resin of the cactus *Euphorbia resinifera*.

Clinically, intravesical instillation of vanilloids is associated with an increase in bladder capacity and an improvement in the symptoms of spinal DH — frequency, urgency and urge incontinence. The effect of intravesical vanilloid treatment lasts for a variable length of time, usually 3-6 months. Early phase clinical trials of RTX are currently in progress.

abdominal wall. This short procedure is performed under cystoscopic control and requires a brief general, spinal or local anesthetic. Patients often find suprapubic catheters easier to manage and many of the long-term complications seem less. The incidence of urine infection is probably equal with both methods of drainage.

In those where catheterization has been used for 5 years or more, annual cystoscopy is advised to exclude squamous cell carcinoma [18].

**Surgical options.** A urinary diversion procedure may be required in certain patients. An incontinent vesicostomy involves the construction of a stoma on the lower abdomen from a segment of ileum that is connected to the bladder, enabling free drainage of urine into a urostomy bag. Catheterizable stomas can be surgically fashioned in patients who are able to perform catheterization. This procedure has the advantage in that it preserves the bladder and the ureterovesical junction. When the bladder cannot be used (e.g. the small shrunken bladder as may occur in spina bifida or co-existing genuine stress urinary incontinence), an ileal conduit urinary diversion is an alternative. This involves the construction of a stoma and intra-abdominal reservoir made of ileum, into which the ureters drain directly [19]. A urostomy bag is required to collect the free draining urine.

Where surgery is being considered, a number of factors including disability, urological status and life expectancy should be carefully considered.

In those with outlet obstruction from DSD who cannot perform CISC, surgical sphincterotomy or the placement of sphincteric stents are options. However, these procedures require adequate detrusor contraction to facilitate emptying. Urethral sphincterotomy involves the endoscopic ablation of the voluntary urethral sphincter, which allows enhanced emptying and lower bladder pressures, but is rarely performed nowadays as CISC should be possible in most cases. A sphincteric stent maintains urethral patency and adequate urine flow. Male patients will subsequently require the use of a condom catheter because of persistent leakage, whereas women will require pads or diapers.

Artificial urinary sphincters (AUS) are used in selected patients with incontinence secondary to sphincteric weakness (e.g. spina bifida). Following either a trans perineal or suprapubic surgical approach, an inflatable cuff is placed around the urethra. A reservoir, containing fluid and a pump are attached to the cuff, which is then filled and drained to maintain continence and empty the bladder as required. The complications of these devices include infection, erosion and mechanical failure. It is, however, contraindicated to insert an artificial sphincter in the presence of marked vesicoureteric reflux, which is a common problem in those with spinal dysraphism [20]. Whilst the AUS is a frequently used surgical option in patients with spina bifida, the procedure has not gained much popularity in the adult neurogenic bladder population.

Carefully selected patients with non-progressive neurological disease and low-capacity, neurogenic bladders may benefit from augmentation cystoplasty both symptomatically and with regard to preservation of upper tract function. This procedure involves increasing the bladder capacity using a patch made from ileocecum, ileum, colon, or stomach and thus decreasing intravesical pressure. This is a major operation with significant short- and long-term complications and should not be considered in those who are deteriorating neurologically. Because impaired emptying is likely to ensue, the patient must be warned that CISC is likely to be necessary [21].

## URINARY RETENTION

Urinary retention may be due either to mechanical obstruction, failure of the bladder neck or sphincter to relax, or failure of the detrusor to contract. Urinary retention may be either complete or partial and may be acute or chronic in onset.

**Complete urinary retention.** The most common causes of complete urinary retention are urological and it is unlikely a neurologist will be asked to see a patient in complete urinary retention who has not undergone urethrocystoscopy to exclude a local urological lesion. Acute urinary retention from a urological cause is usually painful, whereas a neurological lesion should be suspected if acute complete retention is painless. The common gen-

**TABLE 7 Causes of complete urinary retention**

**General**

- Constipation
- Postoperative — (general and epidural anesthesia)
- Drug-induced (tricyclic antidepressants, anticholinergics, ganglionic blocking agents)

**Urological**

- Benign prostatic hyperplasia
- Carcinoma of the prostate
- Urethral stricture
- Clot retention Calculus

**Neurological**

- Acute spinal shock phase — spinal cord injury or transverse myelitis
- Spina bifida, myelodysplasia, sacral agenesis
- Cauda equina lesion
- Herpes zoster
- Diabetic neuropathy and other small fiber neuropathies
- Tabes dorsalis
- Pontine pathology (rare)
- Cerebral disease (rare)
- Multiple system atrophy
- Fowler's syndrome (isolated urinary retention in young women)

eral, non-neurological and neurological causes of complete retention are listed in Table 7.

It will be evident from Table 7 that other accompanying neurological symptoms and signs would be expected if the problem were neurogenic. The exception to this is the acute onset of urinary retention in a young women. Fowler and colleagues recognized a syndrome affecting young women who presented with massive, painless urinary retention in whom sphincter EMG showed there was a myotonic-like activity [22]. It was postulated that the disorder caused a primary failure of sphincter relaxation and many of

the young women also have polycystic ovaries. The onset of retention may follow an operation, not necessarily performed in the pelvis, or childbirth, and spontaneous recovery is not common. The diagnosis necessitates examining the striated urethral sphincter with a concentric needle electrode (see Chapter 1) although other investigations, such as a raised urethral pressure profile, may also indicate a local sphincter abnormality.

Other causes of urinary retention have yet to be explained and non-urological retention in young men who have preserved sexual function (and some young women who do not have the sphincter EMG abnormality) remains an enigma. Imaging and neurophysiological investigations fail to reveal an abnormality in these patients and it is assumed the problem must be either in the detrusor muscle or its innervation.

**Partial urinary retention (or incomplete emptying).** Partial urinary retention or incomplete bladder emptying, is a common feature of the neurogenic bladder and Table 8 lists those neurological conditions in which a raised post-micturition residual volume is often found. The importance of recognizing incomplete emptying has already been stressed (see Pull-out 1) since, if this occurs in combination with detrusor hyperreflexia, the symptoms of urgency and urge incontinence can be resistant to treatment until some means of effectively emptying the bladder is found.

**TABLE 8 Neurological disorders commonly associated with incomplete bladder emptying**

- Post-traumatic spinal cord disease
- Progressive spinal cord disease, e.g. multiple sclerosis
- Peripheral neuropathy (small fiber)
- Cauda equina lesion
- Multiple system atrophy

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## NEUORLOGIC BLADDER, BOWEL AND SEXUAL DYSFUNCTION

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