NORMAL urinary continence depends on the ability of the bladder and urethra to store urine without leakage (filling phase) and to completely empty the bladder when it is full (voiding phase).

The causes of abnormal micturition can be divided into neurogenic and non-neurogenic types (Figure 1).

The most common neurogenic causes are:

• spinal cord diseases (sacral or suprasacral);

• pelvic nerve or sacral plexus diseases; and

• functional urinary obstruction (formerly known as detrusorurethral dyssinergia).

Myogenic causes of bladder dysfunction can include:

• over-distention (due to obstruction, inactivity, pain, confinement and polyuria);
• disorders causing muscle weakness (hypocalcaemia and hypokalemia); and

• pharmacologic agents (tricyclic antidepressants, calcium channel blockers, opioids and anticholinergic agents).

Micturition can also be altered by structural anatomic disorders involving the bladder, by bladder masses or by replacement of the detrusor muscle by fibrous or inflammatory tissue. The outlet resistance can be altered by functional myogenic disorders, urethral sphincter disorders (due to inflammation, pain haemorrhage and oedema, or following urethral disease), urinary tract infection, urolithiasis or prostate enlargement.

A basic knowledge of the anatomo-physiological processes involved in micturition is important to judge its neurogenic dysfunctions and to decide the appropriate treatment. Micturition is the reaction that occurs with an integration of autonomic (sympathetic and parasympathetic) and somatic pathways, as a response to the filling of the bladder, so it can empty its contents. The integration of these complex pathways extends from the cerebral cortex to the sacral segments of the spinal cord. The muscles of the bladder involved in normal micturition (filling and voiding) are:

• detrusor muscle (smooth);

• the external skeletal muscle sphincter of the urethra; and

• the internal smooth muscle sphincter of the urethra.

The muscles of the bladder are innervated by:

• the hypogastric nerve (sympathetic nerve, arising from L1-L4 spinal cord segments);

• the pelvic nerve (parasympathetic nerve, arising from S1-S3 spinal cord segments); and

• the pudendal nerve (somatic nerve, arising from S1-S2 spinal cord segments).

These nerves are under the control of the pontine micturition centre in the brain stem, which comprises a storage centre and a voiding centre, and of other supraspinal areas (the cerebral cortex, thalamus, cerebellum, basal ganglia and hypothalamus).

**Normal function**

**Filling phase**

The filling phase is characterised by a progressive filling of the bladder with the urine that the
ureters carry from the kidneys. During this phase, the bladder muscle is relaxed to allow the bladder wall to expand, and the urethra muscles are contracted to avoid urine leakage.

During normal filling, the sympathetic tone (hypogastric nerve) predominates and the detrusor muscle remains relaxed. Meanwhile, the urethral sphincters contract the bladder neck and urethra. The hypogastric nerve innervates the beta receptors of the detrusor muscle (to give relaxation) and the alpha receptors of the bladder, bladder neck and urethra (internal smooth muscle sphincter of the urethra) to give contraction. The contraction of the external skeletal muscle of the urethra and neck is stimulated by the somatic tone via the pudendal nerve. Stretch receptors on the bladder wall project afferent sensory information to the pontine micturition centre via the pelvic nerve in regards to the increased pressure and volume in the bladder.

• Voiding phase

When the bladder is completely full, stretch and pain stimuli inform the brain about the need to empty it. A voluntary release of inhibition starts the voiding phase in the adult animal. This phase is characterised by a contraction of the bladder detrusor muscle, with a simultaneous relaxation of the external skeletal muscle sphincter and of the internal smooth muscle sphincter of the urethra.

After receiving the information from the stretch receptors via the pelvic nerve, the pontine micturition centre starts the coordinated relaxation of the urethral sphincter and contraction of the detrusor muscle.

The pelvic nerve (parasympathetic) is stimulated to contract the detrusor muscle. At the same time, the hypogastric nerve (sympathetic) is inhibited. Therefore, the detrusor muscle is free of inhibition and the internal sphincter is relaxed. The pudendal nerve (somatic) is also inhibited and the external sphincter is released.

Bladder contraction and flow through the urethra generates secondary reflexes that help to continue the contraction until the bladder is totally empty.

Diagnosis

It is important to collect a database to reach the diagnosis of a micturition disorder.

• History. It is important to know the animal’s micturition habits, the presence of previous surgeries and details about the presenting micturition abnormalities (including if the patient is consciously controlling the urination). Behavioural changes should always be considered.

• Physical examination. It is useful to measure residual urine volume (it should be less than 10 per cent of the normal volume), palpate the bladder volume (before and after urination) and tone, check the difficulty to express the bladder and to assess the urethra with a catheter for
obstructions.

• **Neurological examination.** A full neurological examination is important, paying special attention to the anal tone and perineal sensation and reflex, as it is important to assess the pudendal nerve.

• **Clinical pathology.** This includes haematology and biochemistry (blood urea and/or nitrogen or creatinine), urinalysis and urine culture.

• **Diagnostic imaging.** It is always useful, as part of the basic database, to perform radiographs and abdominal ultrasound to rule out non-neurogenic causes of micturition disorders.

Radiography can include contrast-enhanced cystourethrography. Abdominal ultrasound is used to assess the kidneys, ureters, bladder, bladder neck and urethra.

• **Urodynamic testing.** This can be performed if a dynamic disorder of the filling or voiding phases is suspected. The tests are:

  – cystometrogram, to assess intravesical pressure during filling and voiding;
  
  – urethral pressure profile, to assess urethral tone along its length; and
  
  – electromyogram, which includes taking an image of the perineum and anal sphincter to assess the pudendal nerve.

### Neurogenic disorders

Neurogenic disorders of micturition can be the cause of abnormal function (increased or decreased) in the detrusor muscle and/or of the sphincter. The consequences can be abnormal storage or abnormal elimination of the urine.

• **Upper motor neuron bladder disorders**

These disorders are the consequence of severe cranial spinal cord damage to the sacral segments (commonly seen with thoracolumbar spinal cord lesions). Often, the signs are associated with other neurological deficits (postural reaction abnormalities, cutaneous trunci cut off and spinal pain) and gait abnormalities (paresis and/or plegia or ataxia). At the beginning of the voiding phase, the hypogastric nerve is not inhibited by the pontine micturition centre. Therefore, there is a continuous storage phase, and as a result, an absence of voluntary micturition – the bladder is distended and the sphincter muscles are contracted. The bladder is clinically full, has a flaccid wall and is resistant to manual expression.

Treatment involves drugs to decrease internal sphincter tone (alpha-adrenergic antagonist).
quantities for phenoxybenzamine are 0.25mg/kg to 0.5mg/kg orally q12-24h. For prazosin in dogs, the treatment is 1mg/15kg orally q12-24h, and in cats it is 0.25mg to 0.5mg orally q12-24h. The drugs to decrease external sphincter tone (skeletal muscle relaxant) include diazepam or dantrolone. When using diazepam in dogs, use 2mg to 10mg q6-8h – it is not recommended for use with cats. For dantrolene, in dogs, use 1mg to 5mg orally q8-12h. For cats, use 0.5mg/kg to 2mg/kg orally q8h.

Other drugs can stimulate detrusor activity, such as bethanechol (cholinergic stimulation). Administer 2.5mg to 25mg orally q8h, or use manual expression. Such drugs shouldn’t be given alone when the sphincter tone is increased – drugs to decrease the sphincter muscle tone should always be associated. It normally takes three to five days for bethanechol and phenoxybenzamine to be effective. Indwelling catheters might be required if the bladder disorder is associated with prolonged recumbence. It is important to empty the bladder regularly (every two to four hours) with catheterisation or manual expression to prevent overstretching of the detrusor muscle, as this will cause irreversible atonia, urine retention and cystitis. Antibiotics can be added if necessary.

• Lower motor neuron bladder disorders

Disorders are the consequence of severe sacral spinal cord (Figures 4 and 5) or sacral plexus disease. The bladder is distended and atonic, and the external sphincter is relaxed. The smooth sphincter is intact because it is innervated by the hypogastric nerve. There is incontinence from urine overflow and the bladder is easily expressed. In some cases, the internal sphincter offers some resistance to bladder expression. The signs are often associated with loss of the perineal reflex and sensation. Myopathic, neuropathic and neuromuscular junction disorders, in addition to dysautonomia, can also be associated with a distended and easy to express bladder.

Treatment involves phenoxybenzamine 0.25mg/kg to 0.5mg/kg orally q12-24h to overcome any residual smooth sphincter resistance and bethanechol (cholinergic stimulation) at 2.5mg to 25mg orally q8h, to stimulate detrusor activity. It is important to empty the bladder regularly (every two to four hours) with catheterisation or manual expression to prevent overstretching of the detrusor muscle, which will cause irreversible atonia, urine retention and cystitis. Antibiotics can be added if necessary.

• Pelvic nerve damage

This damage can be the consequence of trauma (external or iatrogenic) to the pelvic nerve. The bladder is distended, and there is a lack of a conscious sensation of filling. The external sphincter is constricted by the pudendal nerve and the internal sphincter by the hypogastric nerve. The anal tone and perineal reflex are normal.

Treatment involves drugs to decrease internal sphincter tone (alpha-adrenergic antagonist). Phenoxybenzamine is administered at 0.25mg/kg to 0.5mg/ kg orally q12-24h. With prazosin, it is
administered in dogs at 1mg/15kg orally q12-24h. In cats, it is used at 0.25mg to 0.5mg orally q12-24h. To decrease external sphincter tone (skeletal muscle relaxant), one method is to use diazepam. In dogs, it is administered at 2mg to 10mg q6-8h. It is, however, not recommended for use in cats. Dantrolene can be used in both species. In dogs, it is used at a rate of 1mg to 5mg orally q8-12h, and in cats, it is administered at 0.5mg/kg to 2mg/kg orally q8h. To stimulate detrusor activity, use bethanechol (cholinergic stimulation) at 2.5mg to 25mg orally q8h.

In animals affected by micturition disorders, it is always important to rule out non-neurogenic causes with standard diagnosis procedures (history, physical examination, haematology, biochemistry, urine analysis, abdominal radiographs and ultrasound), since these are more common than the neurogenic ones. The prognosis for animals with neurogenic causes of micturition disorders depends on the severity of the underlying cause.

As a general rule, animals with upper motor neuron bladder problems tend to recover urinary function and are able to walk. Animals with lower motor neuron bladder or pelvic nerve damage have a more guarded prognosis, and might need longer to recover the ability to control the micturition (which could take weeks or months).

**Further reading**

Figure 1. Causes of abnormal micturition can be divided into neurogenic and non-neurogenic types. Therefore, gaining a knowledge of the cat or dog’s micturition habits is important.
Figure 2. A basic knowledge of the anatomo-physiological processes involved in micturition is important to judge a dog or cat’s neurogenic dysfunctions and to decide the appropriate treatment – some drugs should only be used for one species.
Figure 3. The neuromuscular control process of the bladder.
Figure 4. MRI of a lumbosacral disc herniation causing signs of a lower motor neuron bladder disorder.
Figure 5. A traumatic fracture or luxation of the caudal vertebrae with suspected traction injury to the cauda equina, causing signs of a lower motor neuron bladder disorder.