**Bladder management of the neurologic cat**
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*Micturition: functional neuroanatomy* - The neuroanatomical structures that control the filling and emptying of the bladder include the cerebral cortex, the brain stem, the cerebellum, the ascending and descending pathways of the spinal cord, the centers of the somatic and autonomic lower motor neurons of the spinal cord, the afferent and efferent peripheral nerves. The forebrain regulates the voluntary emptying and filling of the bladder, modulating the intrinsic reflex activity of the other structures. To understand the functional anatomy of the bladder, it is useful to imagine the bladder like a balloon, where the body and its tie-in point are comparable to the body of the bladder and the bladder sphincter, respectively. During the urine emission, the sphincter is released while the body of the bladder contracts; the opposite occurs during the accumulation phase.

![Bladder is a Balloon](image)

**The bladder: anatomic structure and innervation** – The body of the bladder is composed mainly of the detrusor muscle, which consists of three interwoven layers of smooth muscle. The innervation of the detrusor muscle occurs in specific ways: the nerve endings reach only specific muscle fibers. Excitation of the innervated muscle cell (pacemaker cell) initiates a spread of excitation and contraction through adjacent cells by means of "tight junctions". Loss of anatomic integrity, as following protracted distension, significantly affects regular contraction. The smooth muscle fibers will continue in the proximal urethra, forming the *internal sphincter*. External striated muscle (*external sphincter*) is interwoven with the proximal urethral smooth muscle, but is more prominent in the distal half of the urethra.

The sympathetic and parasympathetic nervous system, with complementary and antagonistic functions, innervates the bladder. The *somatic nervous system* innervates the external sphincter. The sympathetic nervous system through the *hypogastric nerve*, originating from the lumbar segments L2-L5 of the spinal cord, innervates with α-adrenergic fibers the internal sphincter and β-adrenergic fibers the detrusor muscle.

The detrusor muscle is also innervated by parasympathetic fibers, stimulating its contraction. These fibers, originating from the lower motor neurons of the S1-S3 spinal cord segments, run in the *pelvic nerve*. The afferent innervation of the bladder (mechanoreceptors located in the bladder wall) runs in the pelvic nerve.
towards the spinal cord, ascends to the centers of the brain stem and, ultimately, to the cortical structures of the forebrain.

**The neurologic control of urination** – The upper motor neurons centers of the urination are at the level of the brain stem, as neuron pools in the pontine reticular substance. The UMN s receive impulses from the cerebral cortex and convey, depending on the case, an excitatory or inhibitory response activating or inhibiting the micturition reflex. The cerebellum is involved, in turn, in the modulation of the action. **Storage Phase:** the sympathetic nervous system is substantially activated in the storage phase. Sympathetic activation produces, via the activation of α-adrenergic fibers, the contraction of the internal sphincter. At the same time, β-adrenergic fibers maintain relaxed the detrusor muscle. The parasympathetic system is quiescent at this stage. The result is the storage of urine in the bladder up to reaching an adequate volume. In this phase, the internal sphincter is assisted by the contraction of the external sphincter, mediated by the somatic nervous system, through the motor neurons located in the segments S1-S2 spinal cord, whose fibers run in the **pudendal nerve.**

**Voiding phase:** When the bladder reaches an appropriate size, activation of the wall mechanoreceptors occurs and the information is transmitted to the cerebral cortex and pontine centers, which, in turn, inhibit the sympathetic and somatic nervous system, producing the release of the internal and external sphincters, and activate the parasympathetic system, causing the detrusor contraction and release of urine.
Traumatic exogenous and endogenous injuries of the thoracolumbar and lumbosacral segments of the spinal cord produce disorders of urination. Grossly, they produce two typical patterns of bladder dysfunction: the "upper motor neuron bladder" and "lower motor neuron bladder".

**Thoracolumbar lesions and UMN bladder** - Significant thoracolumbar spinal cord lesions cause severe spastic paraparesis or paraplegia, disrupting the UMN descending fibers and causing inability to convey the information for the voluntary movement. Typically, the lower motor neurons innervating the hind limbs are intact (as evidenced by the integrity of the spinal reflexes) and able to maintain normal muscle tone. Muscle tone can be even increased due to the lack of the upper motor neurons modulation. Hence, the definition of spastic paralysis. Bladder function is affected in the same way.

In a severe thoracolumbar lesion, the bladder competence is lost for the disruption of the UMN descending fibers. In this clinical setting, all the lower motor neurons are intact and fire without any UMN inhibition, resulting in hypertonic internal and external sphincter and in the inability of the detrusor muscle to empty the bladder. The animal is unable to void and the bladder becomes greatly distended, and expressing the bladder manually is difficult or impossible. The so-called UMN bladder is characterized by an increased size with hard and tonic walls, easily palpable in the abdomen and very difficult to squeeze.

**Lumbosacral lesions and LMN bladder** - The opposite situation occurs in the case of severe lumbosacral intumescence lesion. In this situation, the LMNs are damaged and there is disruption of the muscle innervation. The patient is paraplegic because voluntary movement cannot take place but, differently form the UMN lesion, the muscle tone is flaccid for the lower motor neurons damage. This condition is defined flaccid paralysis. The clinical setting of the LMN bladder is opposite to what above described: the lower motor neurons innervating the internal and external sphincters and detrusor muscle are damaged, resulting in atonic sphincters and flaccid paralysis of the detrusor. Affected animals experience true urinary incontinence, characterized by continuous urine leakage. On palpation, the bladder is relatively small, loose and easy to squeeze.

**Sacroccigeal injury and bladder dysfunction: always LMN bladder?** – For cats with access to the outdoors, road traffic accidents (RTAs) represent a significant hazard. During an RTA, the tail may become trapped underneath a car wheel; the resultant traction can cause subluxation/luxation or fracture at the tail base and stretching of the sacrococcygeal nerve roots/cauda equina, colloquially known as a ‘tail pull injury’. Depending on the extent of damage to the nerves, affected cats may present with a flaccid tail, bladder paralysis and/or urinary incontinence. Cats with tail pull injuries are difficult cases to manage: the severity of nerve damage cannot be ‘measured’ and, therefore, the likelihood and extent of recovery is difficult to predict. While cats with neuropraxia (nerve bruising) should have a good prognosis for full recovery, those with completely severed nerves (neurotmesis) clearly will not. In a recent study, of 51 cats with sacrococcygeal injuries, every cat that could not urinate normally within 1 month remained incontinent during the 2–36 month follow-up period.

In the event of sacrococcygeal injury, the clinician is expected to face a LMN bladder, since the nerve roots merging from the lumbosacral intumescence are injured. Surprisingly, is not rare to find cats showing an
enlarged bladder very difficult to express, a clinical scenario resembling an “UMN bladder”. This finding is almost not reported in dogs, while it’s quite well known in cats. Possible reasons include the fact that the internal sphincter in the cat is more pronounced than in the dog. This paradoxical situation may be explained considering that the Hypogastric nerve, spared by the sacrococcygeal lesion (its roots originate from the lumbar segment of the spinal cord) maintains a good/exaggerated tone of the sphincter in presence of a flaccid detrusor muscle.

Figure 2. Latero-lateral x-ray of a cat with sacral fracture and tail, perineal and bladder dysfunction.

**Therapeutic management of the bladder with neurologic dysfunction** – In the neurologically injured patient, the management of the bladder is extremely important not only to prevent further deterioration of the general condition but also to avoid that the lack of recovery of the bladder function may compromise healing. The expression of the bladder can be manual or instrumental, and is possibly associated to drug treatment to facilitate this process. All manual and pharmacological interventions are focused on a major objective: prevent bladder overdistention.

**Therapeutic management of the UMN bladder** – UMN bladder in dogs is the most common type of bladder clinical presentation, resulting from lesions of the thoracolumbar spinal cord. Normally, recovery of the spinal cord injury is associated with spontaneous restoration of bladder function. In parallel to general rehabilitation treatment, special care must be focused on the bladder and on its regular emptying, goal not always easy to achieve. **The bladder should be expressed not less than four times per day.**

**Manual expression** of the bladder is often difficult, especially in the first days after the injury and, in most instances, results in the expression of not more than short urine spurts. After a few days, sphincter hypertonus usually spontaneously decrease, facilitating manual expression.

If manual expression is impossible or dangerous (i.e. in case of polytraumatized cats), it is appropriate to consider catheterization with mobile or fixed catheters. The latter must be the least traumatic. Polyurethane catheters are ideal: they are inserted using a stylet and are fixed with stitches. It should be remembered that fixed catheters produce greater risk of infection. Rigid mobile catheters (polypropylene catheters), especially if the procedures are repeated over time, may create local trauma and urethral spasms that contribute to worsen the recovery of spontaneous micturition.

**Pharmacologic therapy** of the bladder is aimed to reduce the tone of the internal and external sphincters and, eventually, stimulate the contractile activity of the detrusor muscle. Before going into the details of the administration of drugs designed to achieve these effects, it must not be forgotten to ensure adequate antibiotic, non-steroidal anti-inflammatory (NSAID) and, possibly, analgesic therapy.

α-adrenergic antagonists are used to reduce the tone of the internal sphincter. The most commonly used drug is phenoxybenzamine (0.25-0.5 mg / kg PO BID in the cat). Another possibility is represented by prazosin, α1-antagonist drug administered in the dog at the "extrapolated" dosage of 1 mg if less than 15kg (if weighing more, administer 2mg) every 8-12 hours. In cats, the dosage is 0.25-0.5 mg PO every 12/24 hours. Administration of α-antagonists drugs is associated to major side effects, given their non-specific action, mostly consistent in systemic hypotension, which can lead to collapses. This is the main reason why it is preferred starting therapy with lower doses and, if necessary, gradually increase them. 

**Diazepam** at a dose of 0.2-1 mg/kg every 8/12 hours is administered to reduce the external sphincter tone (usually administered half an hour before the manipulation of the bladder). With the same purpose, **Dantrolene** can be used at a dose of 0.5-2 mg / kg PO TID in cats.
The most widely used drug in the stimulation of the detrusor muscle is betanechol, a parasympathomimetic drug with selective action on muscarinic receptors. The reported dosage for cat is of 1.25 to 5 mg/cat. Betanechol has dose-dependent side effects, characterized by excessive stimulation of the parasympathetic nervous system: nausea, vomiting, diarrhea, drooling, and bronchospasm. During the first week of treatment, is mandatory to start betanechol treatment after permanent catheterization.

**Therapeutic management of the LMN bladder** – This presentation is typical in case of a lesion of the lumbosacral intumescence or, more frequently, sacrococcygeal trauma. These LMN lesions produces both flaccid sphincters and detrusor muscle. The recovery of the function is mainly linked to the severity of the damage rather than to the action of different drugs. In this context, betanechol can be used similarly to what previously reported for UMN lesions. In the "paradoxical" LMN bladder of the cat, namely those maintaining a hypertonic internal sphincter, is suggested the association with phenoxybenzamine, in the dosages described above.

**Figure 3.** Manual expression of the bladder in a paraplegic cat with a chronic lumbosacral spinal cord lesion and LMN bladder (the cat developed some reflex spinal movements; “spinal walking”). Bladder expression, very easy, is obtained with pressure of one hand. Notice the flaccid hind limbs.

**Cystostomy** – Cystostomy can be useful in case of aggressive cats (or, more generally, difficult to manage) and need of prolonged treatment. Cystostomy ensures proper and easy emptying of the bladder for long periods avoiding repeated difficult or invasive manual procedures (i.e. manual expression or repeated catheterization). In this type of surgery, the bladder is put in contact to the left abdominal wall and is fixed to a catheter (with mushroom-shaped end, or better, a silicone Foley catheter) connecting the bladder externally. The catheter can stay in situ for several months allowing easy emptying of the bladder, without stress for the animal. Complications related to the use of this technique are mostly represented by urinary tract infection and, seldom, by the dislocation of the catheter itself.

**Urine tract infections** – In cats with spinal cord injuries, urinary output (1–2 ml/kg/hour), urine SG and sediment cytology as well as changes in color and odor should be monitored daily. Prophylactic antibiosis for urinary tract infection is not recommended when an indwelling urinary catheter is placed, as it significantly increases the chance of drug-resistant bacterial disease. A recent study on dogs showed that UTI is an important complication in dogs that had thoracolumbar surgery. These dogs should be routinely monitored for UTI with urine culture regardless of urinalysis results. Most probably, similar suggestion can be extrapolated for cats.

**Urine scalding** - Urine scalding arises from prolonged contact of urine with the skin, appearing in the beginning as erythematous areas. Paraplegic cats with urinary incontinence should be monitored daily and efforts should be taken to prevent the problem. Urine scalding can be relieved by:

- regular cleansing with a mild antiseptic shampoo,
• clipping the hair around the perineal/inguinal area,
• applying a barrier cream
• catheterization
• using non-retentive bedding.

The most important aspects of the bedding are that it is non-retentive and will allow urine to drain away from the patient. Paraplegic patients require a well padded non retentive bed to prevent pressure sores. This can be provided by a waterproof foam mattress or multiple thick blankets covered by a non-retentive bed. Incontinence pads can be placed under the patient’s hind end and, once soiled, immediately removed.

REFERENCES: