



Postoperative urinary retention in a dog following morphine with bupivacaine epidural analgesia

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Abstract — Urinary retention, overflow incontinence, and subsequent detrusor atony were observed following surgery in which a morphine with bupivacaine epidural injection was used for perioperative analgesia. The premise that the urinary retention may have been due to the effects of the morphine component of the epidural is discussed, along with other possible causes.

Résumé — Rétention urinaire postopératoire chez un chien suite à une analgésie à la morphine-bupivacaïne. De la rétention urinaire, de l'incontinence de regorgement et une atonie subséquante du détrusor ont été observées, alors que suite à une chirurgie, une injection épidurale de morphine avec bupivacaïne a été utilisée comme analgésique périopératoire. La possibilité que la rétention urinarie ait pu être reliée aux effets de la morphine contenue dans l'injection épidurale est discutée ainsi que d'autres causes possibles.

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A 6-year-old, castrated male, 33.8-kilogram German shepherd cross was admitted to the Western College of Veterinary Medicine's Teaching Hospital for removal of total hip replacement implants.

A preoperative complete blood cell count was within normal limits. Mild cholesterolemia was the sole abnormality of the preoperative blood chemistry test. Urine specific gravity was 1.031. A full urinalysis was not performed. The dog was walked twice during the day but did not urinate or defecate. Medical records did not indicate evidence of urination or defecation in the dog's kennel. Prior to surgery, the dog was fasted for 12 h and deprived of water for 1 h. The next day (Day 0), the dog was premedicated by administering intramuscularly (IM) 0.05 mg/kg body weight (BW) acepromazine maleate (Atravet, Ayerst Laboratories, Montreal, Quebec), mixed with 0.1 mg/kg BW hydromorphone (Hydromorphone HP 10, SRBEX, Boucherville, Quebec). A balanced electrolyte solution (Lactated Ringer's, Abbott Laboratories, Saint-Laurent, Quebec) was given intravenously (IV) at 10 mL/kg BW/h. Anesthesia was induced with 10 mg/kg BW thiopental (Pentothal 5 g/2.5% kit, Abbott Laboratories), administered IV to effect, to allow for

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intubation of the trachea with a 12-millimetre endotracheal tube. Anesthesia was maintained with halothane (Halothane BP, Halocarbon, River Edge, New Jersey, USA) and oxygen by employing a circle system. A presurgical epidural injection of 0.1 mg/kg BW morphine sulfate (Morphine Sulfate Injection USP, SRBEX, Boucherville, Quebec) and 1 mL/5 kg BW bupivacaine hydrochloride (Marcaine 0.50%, Sanofi Winthrop, Markham, Ontario), was administered. A standard anal purse-string suture was placed. Three doses of 22 mg/kg BW cefazolin (Kefzol, Eli Lilly Canada, Toronto, Ontario), 2 doses of glycopyrrolate (Glycopyrrolate Inj USP, SRBEX, Boucherville, Quebec), 0.01 mg/kg BW and 0.006 mg/kg BW, respectively, and approximately 1750 mL of electrolyte fluids were administered IV during the 5-1/4-hour anesthetic period. The volume of fluids administered during surgery approximated the surgical maintenance requirements of 1775 mL. No significant surgical complications were observed. At 8:00 am on postoperative day (POD) 1, 0.05 mg/kg BW oxymorphone (Numorphan, Dupont Merck Pharma, Vaughan, Ontario) was administered IM to alleviate pain. Urine was present in the kennel, but the act of urination was not observed. At 1:00 pm on POD 1, 1.8 mg/kg BW ketoprofen (Anafen, Rhône Mérieux, Victoriaville, Quebec) was administered IM, for analgesia. On POD 2, the dog was discharged to his owner.

On POD 7, the dog was presented at the clinic for failure to urinate for 30 h. The owner reported that the dog had not voluntarily urinated since 4:00 am on POD 6 and had not demonstrated any behaviors consistent

with an urge to urinate on the evening of POD 6; spots of urine on the dog's bedding had been observed daily since POD 2; the dog had urinated a decreased volume of urine each time he was taken outside since POD 2; the dog had exhibited behaviors consistent with an increased urge to urinate each evening from POD 2 to 6. Prior to surgery, the dog had no history of urinary dysfunction.

Physical examination revealed a painful caudal abdomen and a large bladder. Other than the expected physical signs related to the previous surgery, all other parameters were within normal limits. A diagnosis of detrusor atony and overflow incontinence due to bladder distention was suspected. A closed urinary system using an 8 French catheter (Meditron 2.6-millimetre Feeding Tube, Ingram & Bell, Don Mills, Ontario) was placed. Approximately 800 mL of slightly cloudy, amber urine was collected in the 1st hour following urinary catheterization. A midstream urinary sample was collected from the catheter for urinalysis and culture. The only abnormality on urinalysis was a urine specific gravity of 1.014. The blood urea nitrogen (BUN) was normal at this time. Because of the normal hydration and BUN results, the urine specific gravity was not followed beyond POD 7. Urine culture and Gram stain were negative. Urine production volumes, approximately 1500 to 1700 mL/d, were within normal limits during POD 7 to 9. The urinary catheter system was maintained until POD 9, when the dog was able to urinate spontaneously.

Normal urination is a spinal reflex function with mediation from the caudal brain stem. As stretch signals are received in the detrusor muscle, sensory information is passed through the pelvic nerve to the sacral spinal cord and the brainstem. After data integration at the pons level, detrusor contraction signals are relayed through parasympathetic efferent axons in the pelvic nerve. Urination is facilitated by relaxation of the bladder neck and proximal urethra, through inhibition of alpha-adrenergic sympathetic neurons (7).

The administration of morphine via the epidural route for relief of postoperative pain in dogs has become increasingly popular in veterinary medicine, particularly for orthopedic surgery involving the hindlimbs. Common postoperative complications associated with epidural morphine in humans are nausea, vomiting, respiratory depression, pruritus, and urinary retention (1,2,3), but these complications have not proven to be clinically significant in dogs (1,2). Urinary retention has been mentioned as a potential problem in dogs (2), but its occurrence has not been documented in the veterinary literature.

Studies in humans have shown that urinary bladder function generally resolves spontaneously within 16 h, as the effects of the morphine dissipate (3,4). Although this dog was not presented at the clinic for lack of urination until 1 wk following administration of the epidural analgesic, his history indicated that there were signs of urinary dysfunction immediately following the surgery, so it is possible that the urinary retention occurred due to the morphine component in the analgesic. Prolonged effects of bladder hypotonicity combined with urinary retention can lead to excessive detrusor expansion and damage to the elasticity of the urinary bladder wall

(5,6). The eventual detrusor atony and lack of urination experienced by this dog may have been directly related to the initial urinary retention.

Three possible mechanisms have been suggested for morphine epidural-induced urinary retention (3). The first is local detrusor inhibition through opiate receptors. This theory has been refuted in human studies due to the lack of decreased urinary bladder wall tone following parenteral morphine administration. The second is inhibitory action on the primary micturition center in the caudal brain stem. However, experimental results have shown that bladder relaxation and analgesia following morphine epidural occur within approximately 15–30 min. This is too short of a time for morphine to travel in the cerebrospinal fluid to the level of the pons. Third, and most likely, is through a spinal mechanism. Opiate receptors are present in the dorsal horn of the spinal cord (5). Canine cystometric studies have shown detrusor relaxation and increased bladder capacity following the spinal injection of morphine (3,5), so it may be that morphine binds these receptors and interrupts the sacral parasympathetic flow, resulting in detrusor relaxation and increased bladder filling. This mechanism is favored because of the rapidity with which bladder relaxation and analgesia occur following epidural administration. Support for this spinal mechanism comes from the inhibition of bladder motility with μ - and δ -opioid receptor agonists but not with a k-agonist (8).

The dog's urinary dysfunction may have been caused by multiple factors. In humans, intrathecal bupivacaine hydrochloride has resulted in urinary retention and voiding difficulties (9). Halothane is not directly nephrotoxic, but decreased renal function can occur secondary to decreased renal blood flow and glomerular filtration rate (10). The subject dog received 2 doses of glycopyrrolate during surgery. Anticholinergic drugs relax the smooth muscle of the urinary tract by occupying muscarinic receptors of effector cells, thereby preventing acetylcholine from binding to the receptor area. This competitive binding can result in urinary retention by attenuating the physiological responses to parasympathetic nerve impulses (4,11). Patients receiving large volumes of IV fluids may have an increased risk of urinary retention, due to the effect of excessive stretching of the urinary bladder wall (4); however, in the subject dog, the IV fluids administered approximated his maintenance requirements. Pain, stress, and increased bladder distention may stimulate sympathetic receptors and cause increased urethral resistance (4,5,12,13). Anal stimulation, packing, and pain may also produce postsurgical urinary retention, due to activation of the vesicoanal reflex (4). Although urinary retention has been associated with total hip and knee arthroplasties in a significant number of humans, particularly men, a specific cause has not been identified (14).

While multiple factors, such as halothane anesthesia, glycopyrrolate administration, anal purse-string suture, and surgical pain may have abetted urinary retention in the subject dog, an inciting stimulus would still be required. Better observation of any urination behaviors or acts postoperatively would have provided additional insight as to why the problem occurred in this dog. Although correlation of history and clinical signs with

human medical facts does not prove that the effect was due to the morphine, it is a plausible explanation.

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