Anesthesia for cystotomy in a dog with pancreatitis and a portosystemic shunt

Anesthesie voor cystotomie bij een hond met pancreatitis en een portosystemische shunt

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ABSTRACT

A 21-month-old Cocker spaniel with a portosystemic shunt, a moderate thrombocytopenia and a history of pancreatitis, was anesthetized for a cystotomy to remove bladder polyps and stones. The portosystemic shunt had been treated conservatively with lactulose, ampicillin and a special diet. After premedication with methadone 0.2 mg/kg, by intramuscular (IM) injection, anesthesia was induced with propofol 4 mg/kg intravenously (IV) and maintained with isoflurane in oxygen. Additionally, 2 mL lidocaine 2% and 0.1 mg/kg morphine were injected in the lumbosacral epidural space and 0.1 mg/kg meloxicam was administered intravenously. Except for a moderate decrease in arterial pressure after the epidural injection and the need for intermittent positive pressure ventilation during surgery, anesthesia and recovery were uneventful. Postoperative analgesia was provided with methadone (0.2 mg/kg every 4 hours initially, then 0.1 mg/kg every 6 hours IM) and oral meloxicam (0.1 mg/kg the first day, 0.05 mg/kg during 4 days).

SAMENVATTING

Een 21 maanden oude cocker spaniel met een portosystemische shunt, een matige trombocytopenie en een geschiedenis van pancreatitis onderging onder algemene anesthesie een cystotomie om blaaspoliepen en -stenen te verwijderen. De portosystemische shunt werd reeds een maand medicamenteus behandeld met lactulose, ampicilline en een aangepast dieet. Na premedicatie met methadon (0,2 mg/kg intramusculair (IM)) werd de anesthesie geïnduceerd met propofol 4 mg/kg intraveneus en onderhouden met isofluraan in zuurstof. Bijkomend werden 2 mL lidocaïne 2% en 0,1 mg/kg morfine geïnjecteerd in de lumbosacrale epidurale ruimte. Eveneens werd 0,1 mg/kg meloxicam intraveneus toegediend. Behalve een matige daling van de arteriële bloeddruk na de epidurale injectie en het feit dat artificiële respiratie nodig was tijdens de chirurgie verliep zowel de anesthesie als recovery zonder problemen. Methadon (0,2 mg/kg IM om de 4 uur initieel, daarna 0,1 mg/kg IM om de 6 uur) en meloxicam (0,1 mg/kg de eerste dag, daarna 0,05 mg/kg oraal gedurende 4 dagen) werden toegediend voor postoperatieve analgesie.

INTRODUCTION

In dogs with a portosystemic shunt (PSS), clinical signs are often vague and generally related to the gastrointestinal, nervous or urinary systems(Broome *et al.* 2004). Gastrointestinal symptoms that are often reported include vomiting, diarrhoea, anorexia, ptyalism (Broome *et al.* 2004), weight loss (Greene and Marks 2007b), poor growth and fever (Howe and Boothe 2002). Clinical signs related to the central nervous system result from hepatic encephalopathy and/or hypoglycemia and include ataxia, behavioral changes, lethargy, depression, pacing, wandering, circling, head pressing, blindness, seizures and coma (Howe and Boothe 2002, Broome *et al.* 2004). In a more recent report, multifocal and lateralizing neurological abnormalities were also found in dogs with congenital portosystemic shunts, demonstrating that encephalopathy may be diffuse and associated with gait abnormalities and vestibular signs (Windsor and Olby 2007). Symptoms associated with the urinary tract include hematuria, stranguria, polyuria, dysuria, urethral obstruction and urolithiasis (Howe and Boothe 2002, Broome *et al.* 2004). This vascular anomaly may therefore pose a challenge for the anesthetist, not only with respect to the selection of sedative, analgesic and anesthetic drugs, but also to the overall management and preoperative preparation of the patient.

Acute pancreatitis in dogs is usually associated with abdominal pain, vomiting and anorexia. The preanesthetic preparation of these patients consists of withholding the oral intake of food and water, while admini-

stering intravenous fluids to correct hydration and/or electrolyte imbalances (Greene and Marks 2007a). The choice of anesthetics used in patients with acute or chronic pancreatitis may be influenced not only by the presence of pancreatitis *per se*, but also because many of these dogs suffer from concurrent disease, such as diabetes mellitus, hyperadrenocorticism, chronic renal failure, neoplasia, congestive heart failure or autoimmune disorders (Cook *et al.* 1993).

This case report describes the anesthetic technique and perioperative management for the surgery of a dog with polypoid cystitis, that had both a portosystemic shunt and a history of pancreatitis.

CASE HISTORY

A 21-month-old male, castrated Cocker spaniel, weighing 13.2 kg, was presented to the Queen's Veterinary School Hospital of Cambridge University with problems of hematuria. One month earlier, the dog had already been examined because of vague complaints of fatigue/lethargy, poor appetite and weight loss. A PSS had been diagnosed at that time and had been managed with Hill's UD Diet ® (Hill's Pet Nutrition Ltd, Herts, UK), lactulose and ampicillin. Since then, his condition had improved, although the owner reported the dog to be polyphagic and scavenging at home. The owners preferred a non-surgical treatment of the PSS. Hematology showed a moderate thrombocytopenia (73.10^9/L (reference range 175-500 $.10^9$/L)) with an occasional clump on the smear. Biochemistry indicated a low urea (1.9 mmol/L (reference range 3.3-8.0 mmol/L)) and normal alanine aminotransferase (ALT) and alkaline phosphatase (ALP) levels. Albumin was low (26.3 g/L) but within the reference range (25.0-40.0 g/L). A urine sample taken by cystocentesis was not grossly hematuric, but blood was found on the dipstick and on microscopy. Urinary pH was 9 (compliant with diet) (reference range 5.5-7.0), with a urinary specific gravity of 1.024 (usual range 1.015-1.045). Ultrasonography of the bladder showed a large, amorphous, pedunculated mass in the bladder lumen, originating from the caudal bladder wall and consistent with the appearance of a polyp. Multiple further lesions of similar appearance, but of much smaller size, were present on all aspects of the bladder. Several small urinary stones were also visible. The portosystemic shunting vessels could clearly be followed looping over the caudal *vena cava* and continuing separately through the diaphragm.

The dog was scheduled to undergo a cystotomy for the excision and histopathology of the mass(es) 2 weeks later. The treatment with lactulose and ampicillin was continued. Because the owners mentioned that their dog had suffered from pancreatitis a few months earlier, the diet was changed to one with a somewhat lower fat content (Royal Canin Hepatic Support® (Royal Canin, Somerset, UK), 16.0% fat in dry matter versus 27.0% in Hill's UD Diet®). To allow bacterial culture of bladder biopsies, it was advised to discontinue the ampicillin treatment at least 7 days before surgery.

A clinical pre-anesthetic examination on the day before surgery revealed no further abnormalities. Repeat hematology revealed a low platelet count (91.10^9/L (reference range 175-500 $.10^9$/L)), with a packed cell volume of 0.42 L/L (reference range 0.43-0.59 L/L). Plasma protein was slightly low (56 g/L (reference range 30-45 g/L)). No hemolysis, lipemia or icteremia were present. Liver function tests were not repeated. Mucosal bleeding time, using a standardized test kit (Surgicut®, Sialkot, Pakistan), was 140 seconds and thus well within the reference range (100-250 seconds).

ANESTHETIC TECHNIQUE

The dog was fasted overnight. Forty minutes after premedication with methadone (Physeptone Injection®, Martindale Pharmaceuticals, Romford, UK) 0.2 mg/kg IM), a 20G cephalic catheter was placed in the left limb and anesthesia was induced with propofol (Propoflo®, Abbott Animal Health, Berkshire, UK), 4 mg/kg IV. After intubation, the orotracheal tube was connected to a circle system mounted on a Penlon Prima SP anesthetic unit (Penlon Ltd, Abingdon, UK). Anesthesia was maintained with isoflurane in oxygen. Warmed Hartmann's solution was infused at 10 mL/kg/hour throughout anesthesia. Monitoring (S/5, Datex-Ohmeda Instrumentarium Corporation, Helsinki, Finland) included electrocardiography (limb lead II), pulse oximetry, capnography, side stream spirometry and measurement of oesophageal temperature and Doppler systolic arterial pressure. Meloxicam 0.1 mg/kg (Metacam®, Boehringer Ingelheim Ltd, Berkshire, UK) and amoxicillin/clavulanate 20 mg/kg (Augmentin®, Glaxo-SmithKline, Middlesex, UK) were administered IV. Antibiotic treatment was repeated at 2-hour intervals during surgery. After instrumentation and clipping the surgical area in right lateral recumbency, the dog was positioned in sternal recumbency. Two mL lidocaine 2% (Lidocaine Injection®, B Braun, Melsungen, Germany) and 0.1 mg/kg preservative-free morphine (Morphine Sulphate®, Martindale Pharmaceuticals, Romford, UK) were aseptically injected in the lumbosacral epidural space. Correct needle placement was confirmed using a loss of resistance syringe. The Doppler systolic arterial pressure was 110 mm Hg just before the epidural injection.

The dog was positioned in dorsal recumbency and the surgical area was disinfected and draped. During that period, the Doppler systolic arterial pressure decreased to 89 mm Hg 10 minutes after the epidural injection, but increased again to 100 mm Hg just before the skin incision (30 minutes after the epidural injection) and to 110 mm Hg five minutes later. During the further course of anesthesia, the arterial pressure remained stable (Figure 1). The heart rate followed the same trend, with a decrease from 120 to 100 beats/minute 10 minutes after the epidural injection, followed by an increase to 110 beats/minute at the time of the skin incision. After 10 minutes of surgery, tidal volume gradually decreased from 9.8 mL/kg to 3.8 mL/kg, without a change in respiratory rate (20-22 breaths/minute). $EtCO_2$ increased from 7.6 kPa (57 mm Hg) to 9.6 kPa (72 mm Hg). In-

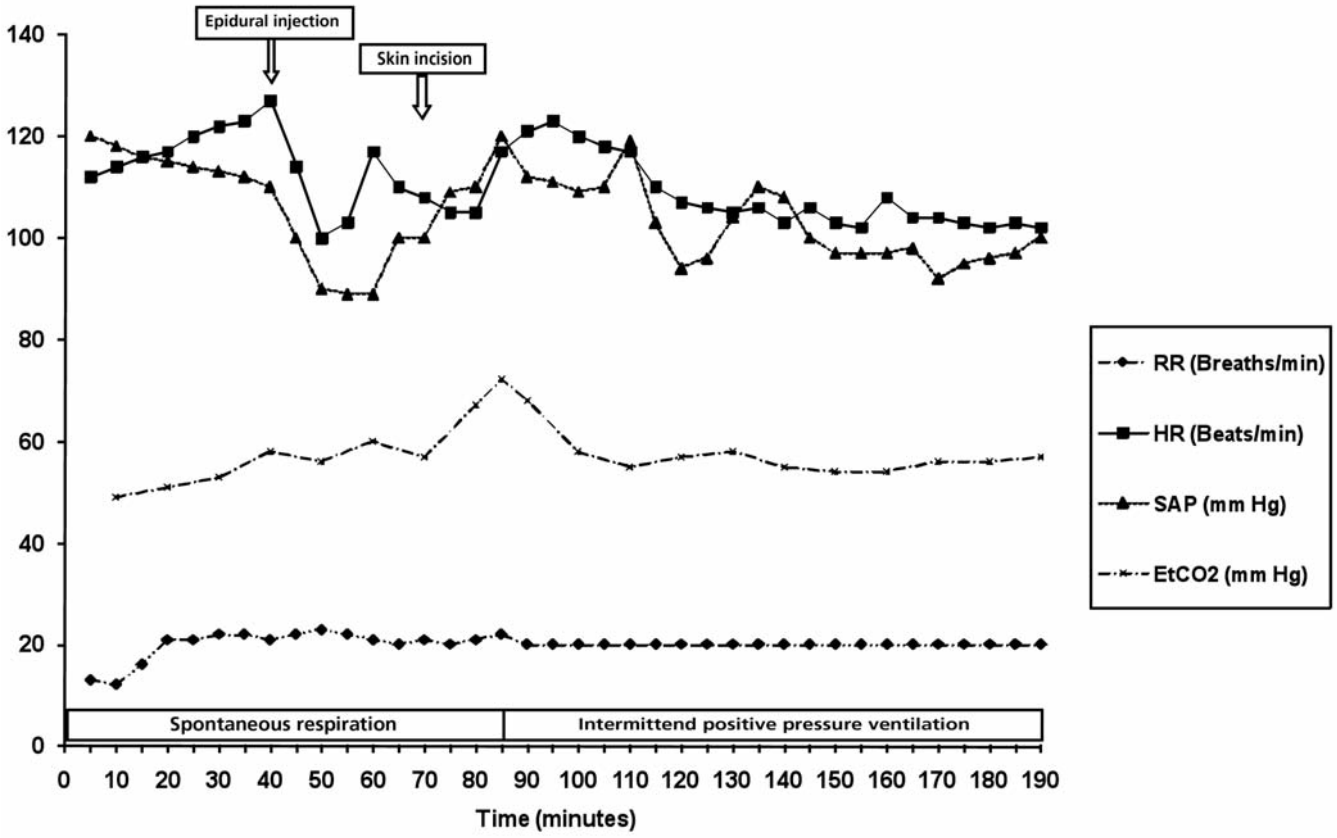

Figure 1. Heart rate (HR, beats/min), respiratory rate (RR, breaths/min), Doppler systolic arterial pressure (SAP, mm Hg) and end-tidal partial pressure of carbon dioxide ($EtCO_2$, mm Hg) in an anesthetized Cocker spaniel with a portosystemic shunt and pancreatitis, undergoing cystotomy. Two mL lidocaine 2% and 0,1 mg/kg morphine were administered epidurally 40 minutes after induction of anesthesia.

termittent positive pressure ventilation was initiated using a Penlon ventilator (Nuffield Anaesthesia Ventilator 200®, Penlon Ltd, Abingdon, UK), delivering a tidal volume of 10 mL/kg at a rate of 20 breaths/minute. $EtCO_2$ decreased and remained between 7.3 and 8.0 kPa (55-60 mm Hg) throughout the remaining period of anesthesia. The anesthesia lasted 190 minutes, and the recovery was quiet and uneventful.

POSTOPERATIVE CARE

The body temperature was 36.6 °C at the end of anesthesia and was normalized during recovery using an infrared heat lamp. Hartmann's solution was administered at 4 mL/kg/hour overnight and 2 mL/kg/hour during the next day. A urinary catheter was placed after surgery to prevent excessive build-up of urine and to monitor urinary output, which remained within normal limits (44 mL/kg/day). This catheter was removed 48 hours later. Four hours after surgery, 0.2 mg/kg methadone was administered IM. This treatment was continued overnight at 4-hour intervals. The following day, the dog was whining and seemed agitated. A general clinical examination did not reveal any abnormalities. The patient did not resent lateral palpation of the abdomen, but disliked direct palpation of the laparotomy wound. Methadone treatment was temporarily discontinued and 0.1 mg/kg meloxicam (Metacam oral suspension ®, Boehringer Ingelheim Ltd, Berkshire, UK) was administered orally. Eight hours later, the dog was calmer but palpation of the wound remained painful. Methadone was administered at a lower dose (0.1 mg/kg IM every 6 hours) until 48 hours after surgery. Meloxicam was administered orally at 0.05 mg/kg, once daily, until 5 days after surgery.

DISCUSSION

Portosystemic shunts may affect anesthetic management, due to reduced hepatic clearance of drugs, hypoalbuminemia (Center and Magne 1990) and increased sensitivity to GABAergic drugs (Schafer and Jones 1982). Most dogs with congenital PSS have smaller livers due to deficient hepatoportal circulation (Grevel *et al.* 1987, Center and Magne 1990). Seven months after surgical induction of portocaval shunts in dogs, mild to extensive atrophy of hepatocytes, hepatic fibrosis and collapsed portal veins were observed (Schaeffer *et al.* 1986). Reduced hepatic drug clearance manifests as poor tolerance of anesthetic or sedative agents in some affected patients (Center and Magne 1990). Anesthetics that require minimal biotransformation and which produce minimal cardiopulmonary depression are preferred (Broome *et al.* 2004).

In more than 60 % of dogs with portosystemic vascular anomalies, the mean corpuscular volume, corpuscular hemoglobin and corpuscular hemoglobin concentrations are decreased (Simpson *et al.* 1997). Se-

rum biochemical abnormalities include increased resting/fasting ammonia concentrations, high bile acid concentration and ALT and ALP activities, low urea, creatinine, cholesterol, albumin and/or total protein concentrations (Grevel *et al.* 1987, Maddison 1988, Center and Magne 1990, Simpson *et al.* 1997, White 1998). In dogs with experimentally induced portocaval shunts, significant decreases were also observed in serum calcium and glucose concentrations (Schaeffer *et al.* 1986).

Decreased plasma protein concentrations enhance drug availability due to reduced protein binding, which is of particular concern if the anesthetic agent is highly protein bound and has a low therapeutic index (Broome *et al.* 2004). Hypoalbuminemia in dogs with PSS may result from decreased hepatic synthesis (insufficient hepatic mass), anorexia and intestinal blood loss due to bowel inflammation (Center and Magne 1990). Blood volume expansion due to sodium and water retention was also stipulated as a possible cause of hypoalbuminemia (Center and Magne 1990), but other authors reported increased glomerular filtration rate and renal volume in dogs with congenital PSS, which would partly explain the low blood urea nitrogen and serum creatinine concentrations (Deppe *et al.* 1999). Increased diuresis was also observed in rats with portocaval shunts (Lauterburg and Bircher 1976).

Certain hepatic diseases may compromise hepatic function to the extent that coagulopathies develop (Badylak 1988). In a study by Niles *et al.* (2001) dogs with congenital PSS tended to have a prolonged partial thromboplastin time (PTT), but the prothrombin (PT) value (the most powerful predictor of intraoperative and postoperative bleeding) was within the normal range for 92.3 % of the dogs and in none of the dogs with elevated PTT was a bleeding diathesis noted during or after surgery. PT and PTT are helpful screening tests for the detection of hemostatic abnormalities, but their sensitivity is limited. More specific investigation requires assays of individual clotting factors, which are not routinely available to veterinary practitioners. In the dog of the present report, a moderate thrombocytopenia was found, but because the packed cell volume and the mucosal bleeding time were within the normal range, no further assays were performed. Cocker spaniels may develop immune mediated thrombocytopenia, but a much lower platelet count is expected in such cases (Botsch *et al.* 2009). Although not a typical finding, Niles *et al.* (2001) reported a mild thrombocytopenia in 7.7% of the cases with PSS. Thrombocytopenia may also occur in dogs with inflammatory or infectious diseases, e.g. of the pancreas or the urinary tract (Botsch *et al.* 2009), which may have been the case in the present report. Although none of the dogs with PSS in the report of Niles *et al.* (2001) showed evidence of a bleeding diathesis, the evaluation of the coagulation profile in these dogs before surgery is recommended. The administration of fresh frozen plasma may also be useful because it provides clotting factors, while increasing both serum albumin and globulin concentrations.

Most of the clinical signs associated with PSS are related to hepatic encephalopathy (Broome *et al.* 2004). In a rabbit model the development of hepatic encephalopathy was associated with increased levels of gamma-amino butyric acid (GABA) in plasma, increased permeability of the blood-brain barrier, increased numbers of binding sites for GABA and benzodiazepines in the brain and a pattern of neural activity similar to that induced by drugs which activate the GABA neurotransmitter system (Schafer and Jones 1982). Endogenous benzodiazepine receptor ligand activity has also been detected in the peripheral and portal blood of dogs with congenital PSS. The gastrointestinal tract may be a source for these compounds, which are thought to produce central nervous system depression by interacting with binding sites on the GABA-benzodiazepine receptor complex (Aronson *et al.* 1997). An increased GABA-ergic tone in dogs with PSS was confirmed by the observation that the administration of sarmazenil, a benzodiazepine antidote, resulted in a significant improvement of portosystemic encephalopathy (Meyer *et al.* 1998). As a result of the increased number of drug-binding sites, the sensitivity to GABA-ergic agonist drugs, such as benzodiazepines and barbiturates, may be increased in patients with liver failure (Schafer and Jones 1982, Broome *et al.* 2004).

The use of benzodiazepines in patients with PSS therefore remains controversial (especially in cases with hepatic encephalopathy). Although benzodiazepines tend to produce more pronounced sedation in these patients than in healthy animals, they have minimal cardiopulmonary side effects and markedly reduce the dose requirements of more depressant injectable agents (Broome *et al.* 2004). Acepromazine should be avoided in patients with PSS: reduced hepatic metabolism may prolong its duration of action and hypotension can occur due to peripheral vasodilation. Acepromazine might also lower seizure threshold (Broome *et al.* 2004) and coagulopathies associated with hepatopathy may be exacerbated (Greene and Marks 2007b). Alpha 2 agonists are not often used in animals with PSS due to their well-known cardiovascular effects (Bennett 2007). The dog in the present report was premedicated with methadone. Opioids are potent analgesics, produce sedation in dogs and can generally be used in patients with liver disease, provided that typical side effects such as bradycardia and respiratory depression are treated (Greene and Marks 2007b).

For the induction of anesthesia, reduced hepatic clearance and the presence of hypoproteinemia may affect the agent choice (Broome *et al.* 2004). Thiopental is generally not recommended: the dose requirements may be reduced and its duration of action prolonged (especially with repeated dosing), since this drug is highly protein bound and extensively metabolized by the liver (Calvey and Williams 2001a). Additionally, increased permeability of the blood-brain barrier and higher sensitivity to GABA-ergic agonist drugs, as mentioned earlier, may potentiate the effects of barbiturates in patients with liver disease. Although ketamine is also eliminated mainly by hepatic metabolism, plasma levels initially fall rapidly due to redistribution (Calvey and Williams

2001a) so a single bolus of ketamine would be unlikely to result in prolonged recovery times (Broome *et al.* 2004). However, ketamine can induce seizures and should be avoided in cases with hepatic encephalopathy (Greene and Marks 2007b). To the authors' knowledge, no studies have described the effects of alfaxalone in dogs with PSS, but its action may be more pronounced as neurosteroids have been shown to act at GABA-A receptors (Weir *et al.* 2004).

Propofol undergoes rapid and extensive hepatic metabolization (glucuronidation), but other methods of metabolism have also been suggested, since the total body clearance was shown to exceed hepatic blood flow (Adam *et al.* 1983). Because propofol is highly protein bound, it is advisable to start with a lower induction dose and slowly administer the drug to effect in patients with hypoalbuminemia (Broome *et al.* 2007b). In the dog of the present report, where the plasma albumin level was within the normal range for dogs, the propofol dose required for intubation was not reduced.

For the maintenance of anesthesia, inhalants are the best choice in patients with severe liver disease (Greene and Marks 2007b). Because isoflurane is metabolized to a lesser extent than other inhalants and better preserves or even increases hepatic artery blood flow (Gelman *et al.* 1984), it is considered the drug of choice in patients with liver disease (Bennett 2007). Sevoflurane would also be an appropriate choice (Bennett 2007). However, since all volatile anesthetics induce cardiovascular depression (Klide 1976), balanced anesthetic techniques are still preferable. In the Cocker spaniel of this report, an epidural injection of lidocaine and morphine was performed. It may be argued that the presence of thrombocytopenia in this dog formed a contraindication to epidural anesthesia, since epidural injections should not be performed in dogs with bleeding disorders (Jones 2001). Instead, a balanced anesthetic protocol, e.g. using an inhalant combined with systemic opioids could have been used. However, the degree of thrombocytopenia was mild, no clinical evidence of a bleeding disorder was present, the buccal mucosal bleeding time (which specifically tests primary hemostasis) was well within normal limits and increased tendency to bleed usually does not occur until platelet counts are lower than approximately 40.10^9/L (Hackner 1999). A recent review study in human medicine examined the safety of neuraxial techniques in patients with common bleeding diatheses (Choi and Brull 2009). In 326 of these patients, idiopathic thrombocytopenic purpura was present and usually not treated before epidural or intrathecal injection, unless the platelet counts were lower than 50.10^9/L. Hemorrhagic complications were not encountered in any of these cases. The authors concluded that hemorrhagic complications after neuraxial techniques appear to be infrequent when the platelet count is $>50.10^9$/L. Furthermore, pre-emptive epidural morphine administration has been shown to induce long-lasting analgesia in small animals, superior to standard management of postoperative pain. In the same study, the end tidal isoflurane level was significantly lower in patients which received a combination of morphine and bupivacaine epidurally, than in animals given epidural morphine alone (Troncy *et al.* 2002). A non steroidal anti-inflammatory agent was also administered to the dog of the present report, since a combination of meloxicam and epidural morphine plus mepivacaine was shown to improve the level of postoperative analgesia compared to epidural injection alone (Fowler *et al.* 2003). In general, non steroidal anti-inflammatory agents (NSAID's) are contra-indicated in patients with hepatic insufficiency or thrombocytopenia. However, meloxicam is a cyclo-oxygenase (COX)-2 preferential NSAID which was shown to be quite safe and effective for controlling post-operative pain in dogs undergoing abdominal surgery (Matthews *et al.* 2001). Furthermore, the dose of meloxicam was deliberately kept low (0.1 mg/kg) to avoid side effects in this patient.

An epidural dose of 1 mL 2% lidocaine per 4.5 kg is usually recommended in dogs to produce anesthesia of the body caudal to the first lumbar vertebra (Jones 2001). In the present report, a somewhat lower dose was administered because of the caudal localization of the surgery and because a combination of lidocaine and morphine has been shown to produce synergistic antinociceptive effects (Akerman *et al.* 1988, Maves and Gebhart 1992, Penning and Yaksh 1992). Despite this low dose of lidocaine, hypotension was observed following epidural injection, which was transient and resolved spontaneously. Hypotension occurs frequently after subarachnoid or epidural injections in humans and is caused by sympathetic blockade, with a decrease in peripheral vascular resistance (Nolte 1978). In small animals, respiratory depression has also been observed after the epidural administration of morphine and bupivacaine (Troncy *et al.* 2002). The administration of morphine into the fourth ventricle or cisterna magna in awake dogs produced a reduction in tidal volume but not respiratory rate (Pelligrino *et al.* 1989), much like the dog in the present report, where intermittent positive pressure ventilation was needed to normalize $EtCO_2$ values. Urinary retention has also been described after epidural anesthesia in dogs (Jones 2001), but this was avoided in the present case by the placement of a urinary catheter.

Adequate monitoring during the postoperative period is needed in dogs with portosystemic shunts. These patients are prone to develop hypothermia and hypoglycemia (Bennett 2007). Body temperature can be maintained intraoperatively by assuring adequate room temperature, infusing warmed fluids and/or using warm water or hot air blankets. In the immediate postoperative period, hypothermia should be treated promptly, e.g. with the use of warm water gloves, infrared heat lamps or radiant-heat warmers. In the present report, using an appropriate room temperature, warmed infusion fluids and an infrared heat lamp during recovery, the body temperature was maintained at an acceptable level. Because the dog was 21 months old, eating properly and generally doing well with treatment, glycaemia was not monitored perioperatively, although it may have been more cautious to regularly assess blood glucose concentrations. Mild hypoglycaemia in these cases can be treated using

5% glucose solutions IV (Bennett 2007). After shunt ligation, the patient should also be monitored for signs of portal hypertension and/or seizures, but in the present case, where only cystotomy was performed, these complications were less likely. On the first postoperative day, the dog did seem somewhat agitated. A general clinical examination did not reveal any abnormalities. Because the dog had recently received methadone and did not show clear signs of pain other than disliking direct palpation of the wound, it was decided to reduce the dose and frequency of the administration of methadone, since higher doses of opioids may induce some agitation in dogs (Pascoe 2000). No further episodes of agitation were observed.

The owners of the dog also mentioned an episode of pancreatitis in the past, although the dog was doing well and had not shown further signs of pancreatitis ever since. Nevertheless, it remains difficult to rule out the presence of chronic pancreatitis in such cases. Chronic pancreatitis is often subclinical or associated with mild, nonspecific clinical signs and most cases therefore likely remain undiagnosed (Steiner 2003). Besides a careful evaluation of the animal's history, physical examination and routine clinical pathology findings, highly specific and sensitive tests (pancreatic lipase immunoreactivity, ultrasonography, histopathology) can be useful to correctly diagnose pancreatitis (Xenoulis *et al.* 2008). In the present case however, they were not carried out, but some considerations were made regarding the choice of anesthetic and analgesic drugs used, since anesthesia and surgery may increase the risk of a new episode of acute pancreatitis. A pronounced increase in serum lipase concentrations has indeed been demonstrated in healthy dogs undergoing exploratory laparotomy (Bellah and Bell 1989). It has classically been described that morphine is contra-indicated in pancreatitis because it can induce spasm of the sphincter of Oddi, with reflux of bile into the pancreatic duct (Calvey and Williams 2001b). Other opioids including buprenorphine and pentazocine also produce contractions of this sphincter (Shintani *et al.* 1982). Traditional teaching dictates that pethidine is the analgesic of choice in humans with acute pancreatitis, because it believed not to elevate pressure in the sphincter of Oddi. However, there are no good arguments to assume that the spasmogenic effect of pethidine on the sphincter of Oddi is less than that of other opioids (van Voorthuizen *et al.* 2000, Thompson 2001), nor is there any good evidence to suggest that the spasmogenic effect of opioids would influence the course of pancreatitis in an unfavorable way (van Voorthuizen *et al.* 2000). Based on a literature review, Thompson (2001) concluded that morphine is probably not contra-indicated in acute pancreatitis. It may even be of more benefit than pethidine by offering longer pain relief with less risk of seizures. The epidural or intrathecal use of opioids (Niesel *et al.* 1991, Verheijen *et al.* 1996) has also been described in the management of both acute and chronic pancreatitis.

The dog in the present report was premedicated with methadone. Opioids are often used as premedication in dogs with pancreatitis (Greene and Marks 2007a). Alpha2 agonists inhibit insulin release by the ß-cells in the islets of Langerhans in the pancreas and are therefore often avoided in patients with pancreatitis, although it is not known whether the effects on the pancreas are of clinical significance (Greene and Marks 2007a), especially since pancreatitis often only affects the exocrine part of the pancreas. Nevertheless, chronic pancreatitis may be associated with impaired glucose tolerance in dogs and a high proportion of dogs with chronic pancreatitis may have a reduced β-cell response (Watson and Herrtage 2004). Avoiding drugs that contain a lipid emulsion, such as propofol and diazepam, may also be wise (Bennett 2007). Propofol has been associated with elevated serum lipase and amylase levels, hypertriglyceridemia and increased risk of developing pancreatitis in intensive care patients (Possidente *et al.* 1998, Devlin *et al.* 2005). In dogs, a single propofol dose, used for the induction of anesthesia, increased triglyceride levels but did not significantly affect pancreatic enzyme levels (Watson and East 2002). In the latter report, one dog, which may already have suffered from sub-clinical pancreatitis before the anesthetic, did show increased serum lipase and trypsin-like immunoreactivity after the administration of propofol and although no clinical signs were observed, the authors advocated caution when using propofol in dogs with uncertain pancreatic health. Nevertheless, the induction of anesthesia in the present case was performed with propofol, because this drug may have some advantages over other drugs in dogs with PSS (Greene and Marks 2007b), while experiments in healthy rats could not confirm a direct relationship between propofol administration and pancreatitis (Dönmez *et al.* 1999).

In conclusion, after conservative treatment of PSS with a special diet combined with lactulose and ampicillin, only minor complications occurred during the anesthesia in the present case in which methadone, propofol and isoflurane and epidural administration of morphine and lidocaine were used for the anesthetic management.

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