RESEARCH PAPER

Effects of morphine and fentanyl constant rate infusion on urine output in healthy and traumatized dogs

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Abstract

Objective To determine whether healthy and traumatized dogs receiving a constant rate infusion (CRI) of either morphine or fentanyl have decreased urine production.

Study design Prospective randomized controlled study.

Animal population Eighteen privately owned previously healthy dogs that had undergone trauma were included. Twenty-three privately owned healthy dogs were used as the controls.

Methods Traumatized dogs were randomized into one of two groups. Group Tmorphine received a CRI of morphine (0.12 mg kg\(^{-1}\) hour\(^{-1}\)) and group Tfentanyl received a CRI of fentanyl (3 \(\mu g\) kg\(^{-1}\) hour\(^{-1}\)) both administered in lactated Ringer’s solution (LRS) at a rate of 60 mL kg\(^{-1}\) day\(^{-1}\). Control healthy dogs were randomized into one of three groups. The LRS control group (CLRS) \((n = 8)\) received LRS at a rate of 60 mL kg\(^{-1}\) day\(^{-1}\). Group Cmorphine \((n = 8)\) and group Cfentanyl \((n = 7)\) received the same infusions as Tmorphine and Tfentanyl, respectively. Collected data were identical for all groups and consisted of measuring total fluid administered, urine output, and urine specific gravity (USG) for a 24-hour period. An analysis of variance (ANOVA) was used for statistical analysis and a \(p < 0.05\) was considered statistically significant.

Results Urine output was significantly decreased \((p < 0.05)\) in all groups compared with the LRS control group. The end mean USG was significantly lower \((p = 0.003)\) in the LRS control group compared with all other groups.

Conclusions There was a decrease in urine output with a CRI of morphine or fentanyl in both healthy and traumatized dogs.

Clinical relevance Decreased urine output caused by an opioid effect might lead to improper assessments of renal function and urine production.

Keywords fentanyl, morphine, trauma, urine production.

Introduction

Urine output is one clinical indirect measurement that may be used to help assess renal blood flow in the intensive care unit (ICU) (Tonnesen 1990). It can be decreased in dogs secondary to trauma to the urinary system, hypovolemia, hypotension, low cardiac output, and systemic inflammatory response syndrome (Marik 2001; King 2005; DiBartola 2006) but pain can also cause a decrease in urine output (Lamont et al. 2000). While urine output might be helpful in monitoring perfusion, renal function, lower urinary tract patency, and intravenous fluid therapy, interpretation of changes in urine output might be difficult in the critical care setting (Smarick 2006).

Analgesics for traumatized dogs are commonly administered via constant rate infusion (CRI) (Quandt et al. 2005). Opioid agonists, such as morphine and fentanyl, are among the most effective...
analgesics used to provide adequate analgesia (Hellyer & Gaynor 1998; Lamont et al. 2000; Quandt et al. 2005). Opioids are frequently administered as a CRI and one potential side effect observed in humans, mice, and dogs has been a decreased urine output (Bidwai et al. 1975; Fujimoto & Hisada 1978; Liu et al. 1977; Durant & Yaksh 1988; Harukumi et al. 1995; Das & Sasidharan 2001; Robertson et al. 2001; Kokko et al. 2002; Bengtsson et al. 2003; Quandt et al. 2005).

Administration of opioids to traumatized patients raises a dilemma. The veterinarian must decide if a decrease in urine output is a result of trauma-related pathophysiology or opioid effect. Improper interpretation of the cause for a decrease in urine output might lead to inappropriate application of various treatments including fluid therapy. The purpose of this study was to determine whether morphine or fentanyl CRI resulted in a decreased urine output in healthy and traumatized dogs.

Materials and methods

Traumatized dogs

Consent to participate in the study was obtained from the clients of 18 dogs that had undergone trauma. The clinic owner approved this project and all ethical guidelines were adhered to. The following parameters were evaluated for determining clinical assessment of hydration: skin turgor (by tenting the skin between the shoulder blades), mucous membrane (MM) moisture, capillary refill time (CRT), packed cell volume (PCV), and total protein (TP). Moist MM, CRT between 1 and 2 seconds, less than 1 second return of a skin pinch, a PCV of 37–55%, and a TP of 5.7–7.6 g dL$^{-1}$ were considered to be within normal limits (Macintire et al. 2005). Dry or tacky MM, a CRT of less than 1 second or greater than 2 seconds, increased skin turgor, or a PCV or TP outside of the normal range were considered abnormal and these patients were excluded from the study. The following parameters were evaluated for determining clinical assessment of intravascular volume status: heart rate (HR) and indirect systolic arterial blood pressure (SAP). Patients with an SAP of 100–160 mmHg, an HR of 80–180 beats minute$^{-1}$ in toy breeds 70–160 beats minute$^{-1}$ in adult dogs, and 60–140 beats minute$^{-1}$ in giant breed dogs were considered within normal limits (Kittleson & Kienle 1998; Macintire et al. 2005). Patients with values outside of the reference ranges listed were excluded from the study.

Traumatic injuries included moderate external lacerations, intervertebral disk rupture, and one or more fractures (Table 1). Exclusion criteria included dogs that had received glucocorticoids, nonsteroidal anti-inflammatory drugs, diuretics, blood pressure-altering medications, or angiotensin-converting enzyme inhibitors. Dogs previously diagnosed with, or having had a history of, clinical signs suggestive of the following diseases were also excluded from the study: hypoadrenocorticism, hyperadrenocorticism, diabetes mellitus, diabetes insipidus, psychogenic polydipsia, hypothyroidism, cardiovascular disease, renal or other primary urinary tract disease, hepatic disease or allergies recently treated with long-acting corticosteroids. Dogs determined to be dehydrated, hypovolemic, or requiring immediate surgery were excluded from the study. Dogs that were anesthetized within the 24-hour study period were also excluded from the study. Traumatized dogs were required to have injuries warranting analgesia and normal intravascular volume using clinical criteria.

Serum chemistries, electrolytes, and PCV/TP were measured and recorded immediately prior to admission in the study. Three mL of whole blood was obtained via jugular venipuncture for sample collection. Of the blood, 0.2 mL was placed in a microhematocrit tube for centrifugation for the PCV and the remaining blood was allowed to clot for 10 minutes. It was then centrifuged and immediately run for analysis. PCV was measured via the hematocrit method and total solids were measured via refractometry. Serum chemistries and electrolytes were measured in house via Idexx

<table>
<thead>
<tr>
<th>Category of trauma</th>
<th>Morphine analgesia</th>
<th>Fentanyl analgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial wounds</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Deep wounds</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Thoracic trauma</td>
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<td>2</td>
</tr>
<tr>
<td>Single fracture</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Multiple fractures</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Neurologic injury</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Polytrauma*</td>
<td>4</td>
<td>3</td>
</tr>
</tbody>
</table>

*Number of patients from the above categories that suffered more than one category of trauma.
chemistry and Vetlyte analyzers (Idexx Corp., Atlanta, GA, USA). Indirect SAP was measured using a Doppler blood pressure monitor (Parks Medical Electronics Inc., Aloha, OR, USA) and cuff measuring approximately 40% circumference of the leg (Macintire et al. 2005). A red rubber catheter was steriley placed into the bladder via the urethra and it was sutured to the skin. The catheter was connected to a closed urine collection system and this was used to measure the volume of urine produced. Rectal temperature, MM color and moisture, HR by cardiac auscultation, respiratory rate and urine specific gravity (USG) were obtained at the start of the study day. Heart rate, MM color and moisture, and CRTs were measured hourly throughout the study in the traumatized patients. Body weight was measured using a Jarvet animal weight scale (Accuracy ± 0.091 kg; Jorgensen Laboratories, Loveland, CO, USA) at the start and at the end of the study period in all dogs. Any feces or vomit production were recorded and weighed during the study.

All traumatized dogs were assessed every 6 hours for pain. Behavioral and physiologic characteristics were evaluated. Patient’s posture, movements in the cage, response to handling, and vocalization were assessed. Patients exhibiting behavioral characteristics such as restlessness, agitation, trembling, or vocalization in combination with physiologic signs of tachypnea or tachycardia were assessed as having inadequate analgesia (Hellyer & Gaynor 1998; Mathews 2000) and were to be removed from the study and their pain medications altered.

Traumatized dogs were randomly assigned to one of two groups. Group Tmorphine (n = 9) received a CRI of morphine (0.12 mg kg⁻¹ hour⁻¹) in lactated Ringer’s solution (LRS) at an intravenous (IV) fluid rate of 60 mL kg⁻¹ day⁻¹ and Group Tfentanyl (n = 9) received a CRI of fentanyl (3 μg kg⁻¹ hour⁻¹) in LRS at an IV fluid rate of 60 mL kg⁻¹ day⁻¹ (Hellyer & Gaynor 1998; Pascoe 2000; Lucas et al. 2001; Wagner et al. 2002; DiBartola 2006).

Control dogs

Twenty-three dogs of various breeds, sex, and size were volunteered by hospital employees for enrollment as controls. Patients were required to be older than 4 months of age and have normal physical examinations. Clinical parameters evaluated and exclusion criteria were identical to those in the traumatized groups except HR, MM color and moisture, and CRTs were measured in the control groups every 4 hours throughout the 24 hour study period.

Healthy control dogs received one of three solutions. The LRS control group (CLRS) (n = 8) received LRS at a rate of 60 mL kg⁻¹ day⁻¹ IV. Group Cmorphine (n = 8) received a CRI of morphine (0.12 mg kg⁻¹ hour⁻¹) in LRS at a fluid rate of 60 mL kg⁻¹ day⁻¹ IV. Group Cfentanyl (n = 7) received a CRI of fentanyl (3 μg kg⁻¹ hour⁻¹) in LRS at a fluid rate of 60 mL kg⁻¹ day⁻¹ IV.

The volume of fluid administered (mL kg⁻¹ hour⁻¹), urine output (mL kg⁻¹ hour⁻¹), and USG were recorded every 4 hours for a 24-hour period in all dogs. An indwelling urethral catheter was used to collect urine from all dogs. The bladder was completely emptied of urine and that urine was discarded after measuring USG via refractometry at the start of the study. Fluid pumps were reset between readings to measure the volume administered in each 4-hour period. Food and water were withheld during the study.

Statistical methods

The data were analyzed using a computer software program (GB-Stat for Macintosh; Dynamic Microsoft Systems, Inc., Silver Spring, MD, USA). An analysis of variance (ANOVA) for repeated measures was used to compare mean rate of total fluid administered (mL kg⁻¹ hour⁻¹), total mean urine output (mL kg⁻¹ hour⁻¹), and initial and end mean USG. Differences in data were considered significant when p < 0.05 and a power analysis was applied to values not statistically significant with a 60% chance of showing a difference with an alpha of 0.05. Body weight and age are reported as mean ± standard deviation.

Results

Initial parameters including physical examination, skin turgor, HR, respiratory rate, MM color and moisture, and SAP measurements were within the reference ranges for all patients admitted to the study. There was no significant difference in serum chemistry and electrolyte values between all five groups. Heart rates remained consistent and within normal ranges for all dogs throughout the study.
period. Mucous membranes remained pink and moist, and CRTs remained between 1.5 and 2 seconds in all dogs throughout the study period. No dogs were recorded as having a bowel movement or emesis during the study. Only one traumatized dog exhibited signs of sedation with fentanyl administration. No other behavioral changes were noted with opioid administration. Pain assessments revealed all dogs as having adequate analgesia throughout data collection.

Age (years) for all groups was: CLRS (3.9 ± 1.8), Cmorphine (4.68 ± 2.6), Cfentanyl (4.19 ± 2.9), Tmorphine (4.99 ± 2.3) and Tfentanyl (4.17 ± 2.3). Mean weights (kg) at the start of the study were: CLRS (65.3 ± 37.9), Cmorphine (55.7 ± 30.3), Cfentanyl (81.6 ± 27.6), Tmorphine (37.3 ± 19.6) and Tfentanyl (34 ± 29.2). Statistical analysis for both age and weight revealed no statistical difference between the groups with \( p = 0.27 \) and 0.19, respectively. Body weight remained unchanged in all dogs throughout the study.

Mean total urine output (mL kg\(^{-1}\) hour\(^{-1}\)) was significantly lower (\( p < 0.050 \)) in groups Tmorphine (0.67 ± 0.32), Tfentanyl (0.76 ± 0.28), Cmorphine (0.70 ± 0.08) and Cfentanyl (0.74 ± 0.06) compared with the CLRS (1.23 ± 0.13) (Fig. 1). There was no statistical difference (\( p = 0.71 \)) in mean total urine output between groups Tmorphine and Cmorphine or between groups Tfentanyl and Cfentanyl, nor when groups Tmorphine and Cmorphine were compared with groups Tfentanyl and Cfentanyl. There was no statistical difference (\( p = 0.26 \)) in the mean rate of total fluid administered (mL kg\(^{-1}\) hour\(^{-1}\)) between the groups: Tmorphine (2.38 ± 0.01), Tfentanyl (2.36 ± 0.04), Cmorphine (2.48 ± 0.04), Cfentanyl (2.48 ± 0.01) and CLRS (2.38 ± 0.04) (Fig. 2).

There was no statistical difference (\( p = 0.13 \)) in the initial mean USG (g mL\(^{-1}\)) between groups: Tmorphine (1.039 ± 0.001), Tfentanyl (1.038 ± 0.004), Cmorphine (1.038 ± 0.001) and Cfentanyl (1.038 ± 0.001) and CLRS (1.037 ± 0.004) (Fig. 3). The end mean USG was significantly lower (\( p = 0.003 \)) in the CLRS group (1.023 ± 0.011) compared with all other groups: Tmorphine (1.034 ± 0.008), Tfentanyl (1.036 ± 0.005), Cmorphine (1.033 ± 0.001) and Cfentanyl (1.036 ± 0.001) (Fig. 4). There was no statistical difference (\( p = 0.42 \)) in the end mean USG between groups Tmorphine, Tfentanyl, Cmorphine and Cfentanyl.

**Figure 1** Urine output (mean ± SD) in traumatized dogs receiving a CRI of morphine (Tmorphine) or fentanyl (Tfentanyl) and in control dogs receiving a CRI of lactated Ringer’s (CLRS), morphine (Cmorphine), or fentanyl (Cfentanyl).

**Figure 2** Rate of fluid administration (mean ± SD) in traumatized dogs receiving a CRI of morphine (Tmorphine) or fentanyl (Tfentanyl) and in control dogs receiving a CRI of lactated Ringer’s (CLRS), morphine (Cmorphine), or fentanyl (Cfentanyl).

**Figure 3** Initial urine specific gravity (mean ± SD) in traumatized dogs receiving a CRI of morphine (Tmorphine) or fentanyl (Tfentanyl) and in control dogs receiving a CRI of lactated Ringer’s (CLRS), morphine (Cmorphine), or fentanyl (Cfentanyl).
All nonstatistically significant comparisons revealed a power >60%.

**Discussion**

Urine output in this study was reduced during opioid administration in both healthy and traumatized dogs when compared with dogs receiving LRS alone. Normal urine output in the dog varies from 1 to 2 mL kg$^{-1}$ hour$^{-1}$ and is determined by the glomerular filtration rate (GFR) and the rate of tubular fluid reabsorption (Tonnesen 1990; DiBartola 2006). It is important when assessing volume status, renal excretory function, and tissue perfusion (Smarick 2006). A decrease in urine output during opioid administration, as observed in this study, might lead to improper interpretations of this value in traumatized dogs in the ICU.

Decreases in urine output related to trauma might be multifactorial. Pain has been shown to cause an increase in vasopressin in both dogs and laboratory animals (Wright & Woodson 1990; Hauptman et al. 2000). It has also been shown to cause an increase in catecholamines, renin and angiotensin II, resulting in vasoconstriction and decreased GFR (Wright & Woodson 1990; Quandt et al. 2005). These circulating hormones and their effects can result in a decrease in urine output. Trauma might also cause blood loss and/or renal ischemia and vasoconstriction with resulting hypovolemia and hypotension, both of which can result in an increase in vasopressin release (DiBartola 2006; Guyton & Hall 2006). Vasopressin binds to receptors in the renal interstitial cells initiating conservation of free water to help restore intravascular volume and blood pressure.

Mu-receptor opioid agonists, as used in these dogs, have also been implicated in decreasing urine output. Several explanations for the mechanism of decreased urine output observed during opioid administration have been described in the literature. A vasopressin-related mechanism in the dog, mouse, and rat has been proposed (De Bodo 1944; Duke et al. 1951; Papper & Papper 1964; Bidwai 1976; Huidobro & Huidobro-Toro 1979; Harukumi et al. 1995). In dogs, morphine caused an increase in urine osmolality and a decrease in free-water clearance (Bidwai et al. 1975). Fentanyl has been reported to have a vasopressin-mediated effect in humans but no studies have been performed to show a similar effect in dogs (Harukumi et al. 1995; Kokko et al. 2002). This vasopressin-mediated effect might be due to a direct interaction with vasopressin receptors in the central nervous system. Fujimoto & Hisada (1978) observed that even a small dose injected intercerebroventricularly inhibited the diuretic effects of prostaglandin E2 in the hypothalamus of rats. A single study observed a decrease in urine output during opioid administration but reported no correlation with changes in vasopressin concentrations (Philbin et al. 1976). Further studies in the literature have reported a decrease in urine output in dogs, rats, and humans; however, the mechanism was not elaborated (Liu et al. 1977; Fujimoto & Hisada 1978; Ishihara et al. 1978; Lejus et al. 1994; Robertson et al. 2001).

Decreased urine output observed with morphine might be an indirect effect seen with IV morphine secondary to hypotension (Handley & Moyer 1952). Morphine has been shown to cause histamine release in dogs when injected intravenously that could result in hypotension secondary to vasodilation (Robinson et al. 1988; Wagner 2002). However, higher infusion rates of morphine in normal dogs have not been associated with significant increases in plasma histamine concentrations and there were no significant differences in SAP in that or this study (Guedes et al. 2006), so it is unlikely that this altered urine output.

Not all studies report a decrease in urine output with mu-opioid agonists. Rathburn et al. (1983) observed an increase in urine output in mice during morphine administration and little to no effect on urine output during fentanyl administration. In rats morphine has also been shown to increase concentrations of atrial natriuretic factor and resulted

![Figure 4](image-url)
in diuresis (Gutkowska et al. 1993). Kono et al. (1981) showed in humans that high-dose fentanyl anesthesia attenuated the hormonal stress response and improved urine output during surgery. Another study in humans showed that fentanyl abolished the vasopressin response during anesthesia (Ecoffey et al. 1984).

The results of this study might support an opioid effect for the decrease in urine output as this change was observed in both healthy control dogs and traumatized dogs receiving opioids. However, the exact mechanism of decreased urine output as a result of an opioid effect remains elusive. Further studies investigating the mechanisms for the decrease in urine output observed during opioid administration are warranted.

As an opioid vasopressin-related mechanism for decreased urine output has been supported, it is interesting to note that there were changes in USG during the course of the study in dogs receiving opioids. Urine specific gravity was recorded to evaluate whether an appropriate decrease was observed during IV fluid administration. Although USG was measured every 4 hours, only the initial and end USG were evaluated as each dog followed a trend throughout the study. Urine specific gravity was additionally evaluated, as it has been reported that a high USG correlates with a high urine osmolality, which might result from increased vasopressin concentrations (Waldrop 2005; Guyton & Hall 2006). The relationship between osmolality and specific gravity is heavily dependent on the solutes present and various sites in the kidney are responsible for their concentration (Tonnesen 1990; Musilamani et al. 2000). As an animal becomes rehydrated and extracellular volume is replaced, urine output is expected to increase while USG decreases (DiBartola 2006). Although all dogs receiving IV fluids maintained a USG above that of isosthenuria, both the control dogs and traumatized dogs receiving either morphine or fentanyl maintained a USG statistically higher than the control dogs receiving LRS alone. This observation only weakly supports a vasopressin-related mechanism for a decrease in urine output during opioid administration and other causes of an increased USG should be considered.

One mechanism responsible might be an increase in solutes, as such an increase might significantly increase USG while only negligibly altering urine osmolality (Tonnesen 1990). Measurements of urine electrolytes and actual urine osmolality might help support these changes related to an opioid effect. Glucose is also a solute and a hyperglycemic effect has been reported with both morphine (Radosevich et al. 1984) and fentanyl (Ambrisko et al. 2005) in the dog, although at much higher doses than those used in the present study. Blood glucose concentrations did not exceed thresholds for spillover into the urine in these studies, but neither study measured actual glucose concentrations in the urine. Serial blood glucose and urine glucose concentrations might have helped evaluate this as a contributing factor.

Variability might have been induced by the same rate of IV fluids administered to all dogs regardless of weight as water turnover scales allometrically. Larger dogs would have received higher than normal fluid rates compared with smaller dogs. Inadequately low rates might result in higher concentrations of solutes in the urine and therefore, a higher USG (DiBartola 2006). Intravenous fluids were also not adjusted for insensible losses, as there is no way to measure these losses in a clinical environment. Although respiratory depression has been more frequently cited as a potential side effect for opioids, panting is often observed with opioid administration and could have contributed to increased insensible losses in this study (Lucas et al. 2001). In dogs that were panting because of heat stress, water loss can increase from 0.4–1.2 to 2–5 mL kg\(^{-1}\) (O’Connor 1977). Respiratory rates were not measured consistently throughout the study and so we do not know how much this could have contributed to the difference in urine output.

Variability in urine output might have been introduced into our traumatized groups because of lack of a standardized pain assessment scoring system. Traumatized dogs in this study suffered from a variety of injuries resulting in pain ranging from mild to severe (Rudloff 2004). Although all dogs were assessed as having adequate analgesia with a morphine or fentanyl infusion, mild pain could have persisted. This mild pain could have contributed to a decrease in urine output due to pain rather than to an opioid effect (Wright & Woodson 1990; Hauptman et al. 2000). Control dogs receiving LRS alone were assumed to be pain free. However, these dogs might have experienced mild discomfort from the indwelling urethral catheter and this cannot be excluded in contributing to a decrease in urine output seen in this group.

Although the authors attempted to eliminate variability in fluid intake by withholding food and
water and providing consistent IV fluid volume during data collection, this might have actually introduced more variability in urine output. While normal dogs can produce a minimum of 1 mL kg\(^{-1}\) hour\(^{-1}\) if not under stress of acute study (Smith et al. 1964; Bentinck-Smith & French 1989), dogs suddenly caged and with no access to food might only produce approximately half this volume (DiBartola et al. 1980). However, dogs on IV fluids and healthy kidneys with a urine production of 1–2 mL kg\(^{-1}\) hour\(^{-1}\) are considered to have relative oliguria and the kidney should produce a diuresis with a urine output of at least 2 mL kg\(^{-1}\) hour\(^{-1}\) (Ross 1989). While the withholding of food and water from all groups was less likely to create variability between the groups, it could have created variability in interpreting the mechanism for the decrease in urine output observed.

Although similar results were found between the healthy dogs and traumatized dogs receiving either a morphine or fentanyl CRI, limitations exist with our traumatized dogs that could have contributed to the decreased urine output. Dogs were assessed to have clinically normal intravascular volumes by physical examination, vital signs, indirect SAP measurements, baseline serum chemistries, and electrolytes. These parameters, including PCV/TS, indirect SAP measurement and HR, do not always correlate with changes in intravascular volumes however, and some of our dogs could have had undetected hypovolemia (Hansen & DeFrancesco 2002; Wingfield & Raffe 2002; DiBartola 2006). Further ongoing blood loss in dogs with long bone or pelvic fractures could have occurred. More frequent monitoring including serial PCV, TS and indirect SAP measurements as well as more invasive monitoring, including central venous pressure, pulmonary capillary wedge pressure, and direct arterial blood pressure measurements, could have provided more information on intravascular volume and its effects on urine output.

Additional limitations including differences in the accuracy of fluid pumps as well as many different veterinary assistants recording volume of fluid administered and urine output could have contributed to variability. Although some bias was eliminated by assigning traumatized dogs to either morphine or fentanyl infusions based on a predetermined random order rather than on the extent of their trauma and by having only one veterinarian evaluate all control dogs, bias could not be completely eliminated as several veterinarians were involved in the assessment of analgesia. Furthermore, the study was not blinded and assessments of analgesia could have been influenced by the opioid administered. While only one veterinarian (M.K.A.) evaluated all the healthy dogs that were admitted to participate, several veterinarians evaluated the traumatized dogs that were admitted. The small size of our study groups might not be representative of all healthy and traumatized dog populations. However, after evaluating the statistically significant changes observed in this study, the small number of dogs in each group does not limit the findings. Power calculations for nonsignificant values were high enough to provide validity to the findings. Underlying disease processes that could have resulted in decreased urine output were eliminated by history, physical examination, baseline serum chemistries, electrolytes, and indirect SAP measurements. Additional diagnostics, such as thyroid and cortisol concentrations, were not performed, and dogs with subclinical disease could have been included and statistically altered the findings.

Although there are limitations in comparing traumatized dogs with healthy dogs, the findings were similar to those found in the healthy dogs receiving opioids. The decrease in urine output was clinically and statistically relevant in both groups. A decrease in urine output in traumatized dogs might result from trauma-related mechanisms or an opioid effect. Interpretation of decreased urine output in dogs receiving a CRI of either morphine or fentanyl should be made in light of these findings and further information should be gathered prior to therapeutic intervention.

**References**


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