Hypotension is a common complication of general anesthesia in small animal patients. Anesthetic agents may decrease vascular resistance as a result of vasodilation and reduced cardiac contractility, which leads to decreased systemic arterial blood pressure. Systemic hypotension can then decrease perfusion of various tissues, especially the kidneys and brain. Other consequences of systemic hypotension include decreased coronary artery perfusion, increased bacterial translocation in the gastrointestinal tract, impaired hepatic function, and activation of the coagulation cascade. The reported incidence of anesthesia-associated hypotension in small animal patients is between 7% and 74%. Under normal physiologic conditions, autoregulation maintains spinal cord perfusion despite wide fluctuations in MAP, as has been demonstrated in multiple mammalian species, including sheep, rats, cats, and dogs. However, following injury, autoregulation may no longer be effective, potentially resulting in severely decreased spinal cord blood flow. Neurogenic shock is a type of distributive shock affecting patients with spinal cord injuries that is characterized by prolonged and persistent hypotension. Acute spinal cord injury has 2 phases: primary injury and secondary injury. In cases of IVDH, primary spinal cord injury occurs when the extruded disk material causes direct contusion and compression of

**Effect of anesthesia-associated hypotension on neurologic outcome in dogs undergoing hemilaminectomy because of acute, severe thoracolumbar intervertebral disk herniation: 56 cases (2007–2013)**

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**OBJECTIVE**
To evaluate the effect of anesthesia-associated hypotension on final motor and urinary function in paraplegic dogs without nociception that underwent hemilaminectomy because of acute, severe thoracolumbar intervertebral disk herniation (IVDH).

**DESIGN**
Retrospective case series.

**ANIMALS**
56 paraplegic dogs with acute thoracolumbar IVDH and absent nociception.

**PROCEDURES**
Medical records were reviewed, and signalment, history, anesthetic details, and results of serial neurologic assessments performed for at least 4 weeks after surgery were recorded. Motor function was retrospectively scored with a 5-point scale, and urinary function was scored with a 3-point scale. Hypotension was defined as MAP ≤ 60 mm Hg or SAP ≤ 80 mm Hg for at least 2 consecutive readings 5 minutes apart. Associations between hypotension and outcome were assessed by use of the Fisher exact test.

**RESULTS**
Thirty-three (59%) patients experienced hypotension during anesthesia. Thirty-four (61%) patients (20/33 with and 14/23 without hypotension) regained ambulation. Whether dogs regained motor or urinary function was not significantly associated with the occurrence of hypotension (P = 0.35 and P = 0.86, respectively), the duration of hypotension (P = 0.213 and P = 0.274), or the lowest blood pressure recorded (P = 0.556 and P = 0.699).

**CONCLUSIONS AND CLINICAL RELEVANCE**
For this group of dogs undergoing hemilaminectomy because of acute, severe thoracolumbar IVDH, anesthesia-associated hypotension was not significantly associated with whether dogs regained motor or urinary function after surgery. However, normotension should be the goal in all patients with spinal cord injuries, especially patients undergoing general anesthesia. (J Am Vet Med Assoc 2017;250:417–423)
the spinal cord. Secondary injury occurs when spinal cord ischemia leads to activation of inflammatory pathways. These pathways result in cytokine production, infiltration by inflammatory cells, production of radical oxygen species, and fluctuations in ion concentrations that continue to cause damage to the spinal cord for up to a week after the primary injury. Systemic hypotension after acute spinal cord injury can worsen spinal cord ischemia and exacerbate secondary injury.

We are unaware of previous studies examining the relationship between anesthesia-associated hypotension and neurologic recovery in dogs undergoing surgery for treatment of acute, severe thoracolumbar IVDH. As such, the purpose of the study reported here was to determine whether hypotension during general anesthesia was associated with a poorer prognosis for regaining motor and urinary function in dogs undergoing hemilaminectomy because of acute thoracolumbar IVDH and absent nociception. We hypothesized that dogs that lacked nociception as a result of spinal cord trauma associated with thoracolumbar IVDH and experienced hypotension during anesthesia for diagnostic imaging and surgery would be less likely to regain motor function (ie, the ability to ambulate) or urinary function than would dogs with absent nociception that did not experience hypotension during anesthesia. We also hypothesized that duration of hypotension during anesthesia and lowest blood pressure measured during the anesthetic period would be associated with the likelihood of regaining motor and urinary function.

Materials and Methods

Case selection criteria

Medical records of dogs examined because of thoracolumbar IVDH and treated by means of hemilaminectomy between 2007 and 2013 at the Purdue University Small Animal Hospital were retrospectively reviewed. Dogs were included in the study if they met all of the following criteria: absence of nociception prior to surgery (as assessed by pinching the digits of the hind limbs), nociception absent for ≤ 24 hours, a diagnosis of thoracolumbar IVDH made by means of CT or MRI with no other spinal cord abnormalities noted, a diagnosis of Hansen type 1 thoracolumbar IVDH confirmed intraoperatively, treatment of thoracolumbar IVDH by means of hemilaminectomy, complete anesthetic and surgical records available, and at least 4 weeks of follow-up information available, including details of motor function (ie, ability to ambulate) and bladder function. Dogs with concurrent neurologic disorders were excluded.

Medical records review

Information obtained from medical records of dogs included in the study consisted of history, signalment, results of physical examination, duration of clinical signs prior to surgery, duration of absence of nociception prior to surgery, site of disk herniation, type and results of preoperative diagnostic imaging, details of surgery (including whether a durotomy was performed), anesthetic protocol, duration of anesthesia, occurrence of intraoperative hypotension, lowest blood pressure recorded during anesthesia (in dogs with hypotension), duration of intraoperative hypotension (if applicable), treatments for intraoperative hypotension (if applicable), whether a clinical diagnosis of myelomalacia was made after surgery (ie, development of an ascending cutaneous trunci reflex, lower motor neuron signs in the pelvic limbs and anus, tetraparesis, or hypoventilation and respiratory distress), and neurologic status at the time of discharge and at all follow-up examinations. For patients that did not return to our hospital within the first 4 weeks after surgery, referring veterinarians and owners were contacted by telephone to obtain follow-up information.

Intraoperative hypotension was defined as previously reported. Specifically, hypotension was defined as a MAP ≤ 60 mm Hg or an indirectly measured SAP ≤ 80 mm Hg on ≥ 2 consecutive readings obtained 5 minutes apart. The severity of hypotension was classified as moderate or severe on the basis of the lowest blood pressure recorded, with moderate hypotension defined as a lowest MAP of 50 to 60 mm Hg or lowest SAP of 70 to 80 mm Hg and severe hypotension defined as a lowest MAP < 50 mm Hg or lowest SAP < 70 mm Hg.

Duration of intraoperative hypotension was determined from review of the anesthetic record and was categorized as mild, moderate, or severe. Mild duration of intraoperative hypotension was defined as a 10- to 15-minute period of hypotension (ie, 2 or 3 consecutive readings) or < 30 minutes of hypotension in total during the anesthetic period, moderate duration of hypotension was defined as a 15- to 25-minute period of hypotension (ie, 3 to 5 consecutive readings) or 30 to ≤ 60 minutes of hypotension in total, and severe duration of hypotension was defined as a > 25-minute period of hypotension (ie, > 5 consecutive readings) or > 60 minutes of hypotension in total. When available, values for MAP were used to classify severity and duration of intraoperative hypotension. If MAP values were not available, then indirect SAP values were used to classify severity and duration of intraoperative hypotension. If direct blood pressure measurements were available, those values were recorded; otherwise, indirect measurements of blood pressure were recorded.

Motor function was graded on the basis of an adapted version of a previously described canine spinal cord injury scale. This modification was made to increase sensitivity for detecting small changes in the degree of motor function. For the present study, grade 1 was defined as ambulatory, grade 2 was defined as nonambulatory paraparesis with movement of > 1 joint (including hip, stifle, and tarsal joints), grade 3 was defined as nonambulatory paraparesis with movement of only the hip joint, grade 4 was
defined as nonambulatory paraplegia with nociception, and grade 5 was defined as paraplegia without nociception.

Urinary function was graded on a scale from 1 to 3, with grade 1 defined as normal urinary function, grade 2 defined as the ability to begin urination voluntarily but without completely voiding the bladder or the inability to begin urination but with the ability to maintain a urine stream once started through manual bladder expression, and grade 3 was defined as complete urinary incontinence.

**Statistical analysis**

A \( \chi^2 \) test or Fisher exact test was used to determine whether the presence of hypotension (yes vs no), severity of hypotension (none, moderate, or severe), duration of hypotension (none, mild, moderate, or severe), acepromazine administration (yes vs no), or administration of various specific treatments for hypotension (yes vs no) was associated with final motor or urinary function grade after surgery. For all analyses, a value of \( P < 0.05 \) was considered significant. All statistical analyses were performed with commercially available statistical software.

**Results**

Fifty-six dogs met the criteria for inclusion in the study. There were 31 spayed females, 22 castrated males, and 3 sexually intact males. There were 32 Dachshunds and 8 mixed-breed dogs. The remaining dogs consisted of Cocker Spaniels (n = 3), Pekingese (3), Shih Tzus (3), French Bulldogs (2), a Beagle (1), a Chihuahua (1), a Welsh Corgi (1), a Doberman Pinscher (1), and a Miniature Poodle (1). Median age was 4.8 years (range, 1.6 to 12.1 years). Median weight was 7.3 kg (16.1 lb; range, 3.4 to 56.0 kg [6.8 to 123.2 lb]). Median duration of clinical signs of thoracolumbar myelopathy prior to initial examination was 48 hours (range, 3 to 480 hours), and median duration of loss of hind limb nociception prior to surgery was 12 hours (range, 3 to 24 hours).

Prior to surgery, dogs were premedicated with various combinations of acepromazine (0.001 to 0.05 mg/kg [0.0045 to 0.023 mg/lb], IM or IV), hydromorphone (0.05 to 0.1 mg/kg [0.23 to 0.45 mg/lb], IM or IV), and dexmedetomidine (0.02 to 0.03 mg/kg [0.009 to 0.014 mg/lb], IM). One dog received acepromazine alone; 28 dogs received acepromazine and hydromorphone; 1 dog received acepromazine and dexmedetomidine; 1 dog received acepromazine, dexmedetomidine, and hydromorphone; and 23 dogs received hydromorphone alone. Two dogs did not receive premedications because they had already been administered analgesics prior to initial examination by the anesthetic department. Anesthesia was induced with propofol (1.3 to 9.2 mg/kg [0.6 to 4.2 mg/lb], IV) in 55 dogs and with ketamine (3.8 mg/kg [1.7 mg/lb], IM) and butorphanol (0.4 mg/kg [0.18 mg/lb], IM) in 1 dog. General anesthesia was maintained in all dogs with isoflurane in oxygen after endotracheal intubation. Median duration of anesthesia was 192 minutes (range, 100 to 300 minutes). In 53 of the 56 dogs, noninvasive blood pressure monitoring was used. In 5 of those patients, blood pressure was monitored indirectly with a Doppler ultrasonic flow detector; therefore, only SAP values were available. In the other 48 patients, indirect blood pressure was monitored with an oscillometric method. In 2 of 56 patients, blood pressure was monitored directly (via an arterial catheter and pressure transducer), and in 1 patient, the method was not recorded, but both MAP and SAP data were available.

Thirty-three of the 56 (59%) dogs developed hypotension during the anesthetic period. Hypotension was treated by decreasing the delivered isoflurane concentration in 17 dogs, administering vasopressor medications in 11 dogs, administering an IV crystalloid fluid bolus in 5 dogs, and administering IV colloidal fluids in 3 dogs. Eleven of the 33 dogs had a mild duration of hypotension, 12 had a moderate duration, and 10 had a severe duration. Seventeen dogs had moderate hypotension, and 16 had severe hypotension.

Incidence of hypotension was not significantly different (14) between dogs that received a single premedication (21/31) and those that did not (12/25). Final motor and urinary function grades were not significantly different between dogs in which hypotension was treated by decreasing the delivered isoflurane concentration (\( P = 0.12 \) and \( P = 0.31 \), respectively), administering vasopressors (\( P = 0.22 \) and \( P = 1.00 \)), or administering an IV bolus of crystalloid fluids (\( P = 0.54 \) and \( P = 0.40 \)).

Overall, 34 of the 56 dogs had a final motor function grade of 1, 3 had a final motor function grade of 2, 4 had a final motor function grade of 3, and 15 had a final motor function grade of 5. Of the 23 dogs that did not develop hypotension, 14 had a final motor function grade of 1, 1 had a final motor function grade of 2, and 8 had a final motor function grade of 5. Of the 33 dogs that developed hypotension, 20 had a final motor function grade of 1, 2 had a final motor function grade of 2, 4 had a final motor function grade of 3, and 7 had a final motor function grade of 5. Final motor function grade was not significantly associated with whether dogs developed hypotension (yes vs no).

All 56 dogs had complete urinary incontinence (grade 3) prior to undergoing hemilaminectomy. Overall, 42 dogs had a final urinary function grade of 1, 1 had a final urinary function grade of 2, and 13 had a final urinary function grade of 3. Of the 23 dogs that did not develop hypotension, 17 had a final urinary function grade of 1 and 6 had a final urinary function grade of 3. Of the 33 dogs that developed hypotension, 25 had a final urinary function grade of 1, 1 had a final urinary function grade of 2, and 7 had...
a final urinary function grade of 3. Final urinary function grade was not significantly (P = 0.86) associated with whether dogs developed hypotension.

Duration of hypotension was not significantly associated with final motor function grade (P = 0.21; Table 1) or final urinary function grade (P = 0.27; Table 2). Similarly, severity of hypotension was not significantly associated with final motor function grade (P = 0.49; Table 3) or final urinary function grade (P = 0.51; Table 4).

Five of the 56 (9%) dogs were euthanized after surgery because of clinical signs consistent with myelomalacia. The diagnosis was not confirmed via necropsy in any of these dogs; however, all 5 had at least 2 of the following signs: ascending cutaneous trunci reflex, lower motor neuron signs in the pelvic limbs and anus, tetraparesis, or hypoventilation and respiratory distress: Three of these 5 dogs developed hypotension while anesthetized and 2 did not. One dog had a mild duration of severe hypotension, 1 had a moderate duration of moderate hypotension, and 1 had a severe

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**Table 1**—Cross-classification of duration of hypotension during the anesthetic period versus final motor function grade for 56 dogs that underwent hemilaminectomy because of acute thoracolumbar IVDH and absent nociception.

<table>
<thead>
<tr>
<th>Duration of hypotension</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
<th>Grade 5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotension</td>
<td>14 (41)</td>
<td>1 (33)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>8 (53)</td>
<td>23 (41)</td>
</tr>
<tr>
<td>Mild</td>
<td>5 (15)</td>
<td>0 (0)</td>
<td>3 (75)</td>
<td>0 (0)</td>
<td>3 (20)</td>
<td>11 (20)</td>
</tr>
<tr>
<td>Moderate</td>
<td>7 (21)</td>
<td>1 (33)</td>
<td>1 (25)</td>
<td>0 (0)</td>
<td>3 (20)</td>
<td>12 (21)</td>
</tr>
<tr>
<td>Severe</td>
<td>8 (24)</td>
<td>1 (33)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>1 (7)</td>
<td>10 (18)</td>
</tr>
</tbody>
</table>

Data represent number (%) of dogs. Motor function was assessed for 4 weeks after surgery and graded on a scale from 1 (ambulatory) to 5 (paraplegia without nociception). Hypotension was defined as an MAP ≤ 60 mm Hg or SAP ≤ 80 mm Hg on ≥ 2 consecutive readings obtained 5 minutes apart. Duration of hypotension was graded as mild, moderate, or severe. Duration of hypotension was not significantly (P = 0.21) associated with final motor function grade.

**Table 2**—Cross-classification of duration of hypotension during the anesthetic period versus final urinary function grade for the dogs in Table 1.

<table>
<thead>
<tr>
<th>Duration of hypotension</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotension</td>
<td>17 (40)</td>
<td>0 (0)</td>
<td>6 (46)</td>
<td>23 (41)</td>
</tr>
<tr>
<td>Mild</td>
<td>7 (17)</td>
<td>1 (100)</td>
<td>3 (23)</td>
<td>11 (20)</td>
</tr>
<tr>
<td>Moderate</td>
<td>10 (24)</td>
<td>0 (0)</td>
<td>2 (15)</td>
<td>12 (21)</td>
</tr>
<tr>
<td>Severe</td>
<td>8 (19)</td>
<td>0 (0)</td>
<td>2 (15)</td>
<td>10 (18)</td>
</tr>
</tbody>
</table>

Data represent number (%) of dogs. Urinary function was assessed for 4 weeks after surgery and graded on a scale from 1 (normal) to 3 (complete urinary incontinence). Duration of hypotension was not significantly (P = 0.27) associated with final urinary function grade. See Table 1 for remainder of key.

**Table 3**—Cross-classification of severity of hypotension during the anesthetic period versus final motor function grade for the dogs in Table 1.

<table>
<thead>
<tr>
<th>Severity of hypotension</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
<th>Grade 5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotension</td>
<td>14 (41)</td>
<td>1 (33)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>8 (53)</td>
<td>23 (41)</td>
</tr>
<tr>
<td>Moderate</td>
<td>10 (29)</td>
<td>1 (33)</td>
<td>3 (75)</td>
<td>0 (0)</td>
<td>3 (20)</td>
<td>17 (30)</td>
</tr>
<tr>
<td>Severe</td>
<td>10 (29)</td>
<td>1 (33)</td>
<td>1 (25)</td>
<td>0 (0)</td>
<td>4 (27)</td>
<td>16 (29)</td>
</tr>
</tbody>
</table>

Data represent number (%) of dogs. Severity of hypotension was graded as moderate (lowest MAP of 50 to 60 mm Hg or lowest SAP of 70 to 80 mm Hg) or severe (lowest MAP < 50 mm Hg or lowest SAP < 70 mm Hg). Severity of hypotension was not significantly (P = 0.49) associated with final motor function grade. See Table 1 for remainder of key.

**Table 4**—Cross-classification of severity of hypotension during the anesthetic period versus final urinary function grade for the dogs in Table 1.

<table>
<thead>
<tr>
<th>Severity of hypotension</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
<th>Grade 4</th>
<th>Grade 5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotension</td>
<td>17 (40)</td>
<td>0 (0)</td>
<td>7 (54)</td>
<td>23 (41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>13 (31)</td>
<td>1 (100)</td>
<td>2 (15)</td>
<td>17 (30)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>12 (29)</td>
<td>0 (0)</td>
<td>4 (31)</td>
<td>16 (29)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data represent number (%) of dogs. Severity of hypotension was not significantly (P = 0.51) associated with final urinary function grade. See Tables 2 and 3 for remainder of key.
duration of severe hypotension. One other dog died at home 9 days after surgery. Death was suspected to be a result of myelomalacia; however, no clinical signs or physical examination findings immediately prior to death were available.

Discussion

The present single-center retrospective case series conducted over a 6-year (2007–2013) period did not identify any significant associations between anesthesia-associated hypotension and motor or urinary function 1 month after surgery in dogs with severe thoracolumbar IVDH treated by means of hemilaminectomy. Overall, 41 of 56 (73%) dogs in the present study recovered some motor function, and 34 (61%) were ambulatory (motor function grade of 1) within 4 weeks after surgery. Previous studies of dogs with thoracolumbar IVDH and absent nociception have reported that 41% to 69% of patients treated with decompressive hemilaminectomy will recover the ability to ambulate, which is consistent with our results. Olby et al reported that for 87 dogs with absent nociception, return of ambulation required a mean of 7.5 weeks (range, <1 to 36 weeks); however, 62% of patients were ambulatory within 4 weeks. We speculate, therefore, that at least some of the 7 dogs in the present study that had regained motor function but were nonambulatory at the time of the final follow-up examination may have improved even further if the follow-up time had been longer, which may have affected our results.

Five of the 56 (9%) dogs in the present study were euthanized after surgery because of clinical signs consistent with myelomalacia. Although the diagnosis was not confirmed via necropsy, all 5 had typical clinical signs, including development of an ascending cutaneous trunci reflex, lower motor neuron signs in the pelvic limbs and anus, tetraparesis, or hypoventilation and respiratory distress. Three of these patients experienced hypotension while anesthetized, and the other 2 did not. Previous studies have reported similar findings, with suspected ascending or descending myelomalacia occurring postoperatively in 3.5% to 11% of dogs with thoracolumbar IVDH and absent nociception before surgery. We suggest that the possibility of myelomalacia is an important factor when counseling owners regarding the prognosis for dogs with absent nociception prior to surgery.

The percentage of patients that experienced hypotension in the present study (33/56 [59%]) was similar to findings for previous studies in which 7% to 74% of dogs undergoing anesthesia for a variety of conditions reportedly developed hypotension. In the present study, all patients underwent surgery because of acute, severe thoracolumbar IVDH. Experimental studies have demonstrated that persistent hypotension for up to 5 hours can be seen following severe injury to the spinal cord in several mammalian species. This phenomenon is referred to as neurogenic shock and has also been clinically reported in human patients with acute spinal cord injury. However, we are not aware of reports of neurogenic shock in dogs with naturally occurring IVDH.

For the patients of the present study, there was no significant association between development of hypotension during anesthesia and final motor or urinary function grade. There was also no significant association between final motor or urinary function grade and severity of hypotension or duration of hypotension. These findings contradict findings of previous reports which suggested that it is important to maintain normotension in dogs with acute spinal cord injury or IVDH to improve clinical outcome. However, hypotension in any patient under general anesthesia should be immediately addressed to prevent potential damage to other organs. To our knowledge, there has only been 1 previous report that found a significant effect of perioperative hypotension during neurosurgery on development of postoperative complications. Results of that report indicate that 12% (60/508) of patients undergoing a ventral slot procedure for cervical IVDH experienced hypotension during anesthesia and that these dogs were 23 times as likely to develop complications, including intraoperative hemorrhage, worsening pain after surgery, worsening of neurologic function after surgery, and aspiration pneumonia. Many of the most severely affected dogs required a second surgery within the first 5 days after surgery. Dogs experiencing complications were significantly more likely to have a poor outcome; however, neurologic grade at discharge was used as the outcome measure in that study; therefore, long-term motor and urinary function was not evaluated.

One of the contributing factors to spinal cord ischemia is loss of spinal cord autoregulatory capacity. Blood flow is dependent on multiple factors, the most important of which are MAP and systemic vascular resistance. In healthy individuals, spinal cord blood flow will typically be maintained within physiologic limits despite fluctuations in MAP by means of autoregulation and local control of vascular resistance. After severe injury, spinal cord autoregulatory capacity is lost and blood flow is therefore directly affected by MAP. The loss of autoregulation combined with systemic hypotension can result in further secondary ischemic injury to the spinal cord. Because of this concern, the American Association of Neurologic Surgeons’ 2013 treatment guidelines for human patients with acute spinal cord injury include the recommendation to maintain MAP in the upper range of normotension (85 to 90 mm Hg) for the first 7 days after injury. There have been no randomized controlled trials examining this recommendation because of the obvious ethical implications. In veterinary medicine, the recommendation has also been made to maintain normotension in patients with acute spinal cord injuries to similarly decrease the potential for worsening spinal cord ischemia and secondary injury.
Multiple studies\textsuperscript{9–11,29–31} in animals and humans have demonstrated that decreased spinal cord perfusion and ischemic injury begin within minutes to hours after severe spinal cord injury. In experimental studies\textsuperscript{9,29,30} of spinal cord injury in animals, spinal cord blood flow and tissue oxygenation decreased within the first hour after injury and remained persistently decreased for the first 4 hours after injury in both white and gray matter. Spinal cord hemorrhage was evident within the first 15 minutes after injury, and edema and necrosis, especially affecting the gray matter and central white matter, reportedly developed within the first 2 hours after injury. By 5 hours after injury, edema and necrosis were present throughout the white matter. In a recent review, Martirosyan et al\textsuperscript{8} suggested that if treatments to improve spinal cord blood flow after injury could be implemented within the first 3 to 4 hours, this may improve outcome for some patients. In the present study, the median duration of clinical signs prior to initial evaluation was 48 hours (range, 3 to 480 hours) and the median duration of loss of nociception was 12 hours (range, 3 to 24 hours). As such, we suggest that by the time most patients were examined, severe spinal cord ischemia had already occurred following by secondary injury. It is possible that no significant association between intraoperative hypotension and neurologic outcome was found in this study because our patients were initially examined too late after the primary injury had occurred. Therefore, even if normotension was maintained, it may have been too late to prevent ischemia or to limit secondary spinal cord injury.

In experimental studies of spinal cord ischemia in animals, it has been reported that the longer the duration of ischemia, the more severe the secondary injury and the worse the neurologic outcome is. In mice, when spinal cord ischemia was induced by cross clamping the aorta for 9 minutes, motor function was recovered within 48 hours; however, when cross clamping occurred for 11 minutes, 80% of the animals remained paralyzed and hemorrhage and parenchymal edema were more severe.\textsuperscript{35} The authors of another study\textsuperscript{35} reported that when hypertension was artificially maintained during periods of spinal cord ischemia in rats, the neurologic outcome was significantly improved versus the outcome for animals that were normotensive. Because hypertension can be deleterious, Wallace and Tator\textsuperscript{34} reported that when rats with an experimentally induced spinal cord injury received low molecular weight dextran to reverse posttraumatic hypotension, neurologic outcome was improved. These studies demonstrate that maintaining normotension at the time of injury and in the immediate period afterwards improves outcome. As such, we suggest that if dogs were hospitalized immediately after IVDH and if blood pressure management as well as decompressive surgery were initiated within 3 to 5 hours after injury, it is possible that spinal cord ischemia could be reduced and secondary injury may be attenuated.

The present study was limited by its retrospective nature. Although this study included only patients for which the medical record specifically stated that nociception was absent, errors in recording information were possible because a variety of individuals were responsible for examining patients and recording information in the medical record. The anesthesia records were also an area with opportunity for error because during some periods, such as when the patient was moved from imaging to surgery, there were times when blood pressure was not recorded every 5 minutes. This made it difficult to precisely record all instances of hypotension and accurately determine the duration of hypotension. Finally, because of the worse prognosis associated with absent nociception in dogs with acute IVDH, the number of dogs in which surgical intervention was elected by owners was low. It is possible that small effects associated with hypotension during anesthesia could have been detected with a larger sample size.

**Acknowledgments**

Presented in poster form at the International Veterinary Emergency and Critical Care Symposium, Indianapolis, September 2014.

The authors thank Dr. George Moore for assistance with statistical analyses.

**Footnotes**

a. Stata SE, version 12.1, StataCorp, College Station, Tex.

**References**

From this month’s AJVR

Effect of the duration of food withholding prior to anesthesia on gastroesophageal reflux and regurgitation in healthy dogs undergoing elective orthopedic surgery

Sivert Viskjer and Lennart Sjöström

OBJECTIVE
To compare the incidence of and risk factors for gastroesophageal reflux (GER) and regurgitation associated with preanesthetic food withholding for periods of 18 hours (overnight) and 3 hours in healthy dogs undergoing elective orthopedic surgery.

ANIMALS
82 healthy (American Society of Anesthesiologists physical status classification I or II) client-owned dogs.

PROCEDURES
Food was withheld for 18 hours (18-hour group [n = 41]) or each dog was allowed to consume half its daily ration of canned food approximately 3 hours (3-hour group [n = 41]) prior to induction of anesthesia. In each anesthetized dog, a pH catheter was introduced through the oropharynx into the distal portion of the esophagus; the pH was continuously recorded throughout the period of anesthesia. Gastroesophageal reflux was defined as pH < 4.0.

RESULTS
Gastroesophageal reflux was significantly associated with age, dorsal recumbency, and duration of preanesthetic food withholding. Regurgitation was significantly associated with duration of GER and duration of preanesthetic food withholding. During anesthesia, 25 (61%) dogs in the 3-hour group had GER and 12 (48%) of those dogs regurgitated gastric content; 18 (43.9%) dogs in the 18-hour group had GER and 2 (11.1%) of those dogs regurgitated gastric content. The mean lowest pH measured in the refluxate in the 3-hour group (2.3) was significantly greater than that in the 18-hour group (1.3).

CONCLUSIONS AND CLINICAL RELEVANCE
Among the study dogs undergoing orthopedic surgery, consumption of a light meal 3 hours prior to anesthesia was associated with significantly greater odds of reflux and regurgitation, compared with overnight food withholding. (Am J Vet Res 2017;78:762–769)

See the midmonth issues of JAVMA for the expanded table of contents for the AJVR or log on to avmajournals.avma.org for access to all the abstracts.