Local anesthetics are commonly used to relieve pain associated with dental procedures in companion animal practice. The anatomic location of a dental procedure generally dictates the type of dental nerve block performed. Maxillary and infraorbital nerve blocks are performed for procedures involving the upper dental arch or maxilla and provide perioperative analgesia, thereby decreasing intraoperative anesthetic requirements.1,2

The infraorbital nerve is the continuation of the maxillary nerve before it enters the infraorbital canal. It provides the caudal, middle, and rostral superior alveolar nerve branches that supply sensory innervation to the upper dental arch. The caudal branches originate from the infraorbital nerve before it enters the infraorbital canal through the maxillary foramen and supply sensory neurons to the maxillary first molar tooth and MM2 and probably the distal root of the MPM4. The middle branches originate within the infraorbital canal and supply sensory neurons to the 4 maxillary premolar teeth. The rostral branches originate immediately before or after the infraorbital nerve exits the infraorbital canal and supply the MC and incisor teeth on the ipsilateral side. The pterygopalatine nerve originates from the maxillary nerve in the pterygopalatine fossa and gives rise to minor and major palatine nerves that innervate the soft and hard palates, respectively.3

Local anesthetics must be deposited in close proximity to the nerve to elicit an effective conduc-
tion blockade of nerve impulses in the target area. A previous study suggests that at least 3 consecutive nodes of Ranvier need to be desensitized to achieve a successful nerve block. Thus, both the volume and concentration of a local anesthetic are important considerations for achieving an adequate concentration gradient between the site of drug deposition and target nerve to allow for sufficient drug distribution along the nerve to block impulse conduction. Traditionally, veterinarians have used fairly small volumes of local anesthetics to perform various dental nerve blocks. However, results of a 2016 study indicate that the deposition of 1 mL of a bupivacaine-lidocaine mixture into the infraorbital canal of dogs (approx body weight, 20 kg) achieves a reliable and long-lasting blockade of the ipsilateral MC but does not consistently desensitize the MPM4 or MM2. Findings of another study involving canine cadavers suggest that increasing the volume of injectate deposited in the infraorbital canal increases the likelihood of successfully blocking all relevant nerves.

Although dental nerve blocks are performed in veterinary patients on a routine basis, there is a paucity of data regarding their efficacy. The purpose of the study reported here was to assess the desensitization of various oral structures (ipsilateral MC, MPM4, MM2, and hard palate) following the injection of 1, 2, and 3 mL of a lidocaine-bupivacaine mixture (50:50 vol/vol) caudal to and at the caudal aspect of the infraorbital canal of dogs. We hypothesized that a more consistent blockade of the caudal maxillary teeth and hard palate would be achieved with injection of higher volumes of the local anesthetic mixture.

Materials and Methods

Animals

All study procedures were reviewed and approved by the University of California-Davis Institutional Animal Care and Use Committee (IACUC No. 19059). Six 2-year-old sexually intact female mesaticephalic hound-type dogs with a mean ± SD body weight of 23.1 ± 3.2 kg were enrolled in the study. Each dog was considered healthy on the basis of results of a physical examination, CBC, and serum biochemical analysis. The stage of the estrous cycle at the time of study initiation was not determined for any of the dogs. All dogs were purchased from an approved vendor.

Study design

The study had a randomized crossover design. Each dog received 1, 2, and 3 mL of a lidocaine-bupivacaine mixture with at least a 14-day washout period between treatments. The order in which the treatments (1-, 2-, or 3-mL volume) were administered and the side (right or left) used for the first treatment were randomly determined by use of an internet-based randomization program. The opposite side was used for each successive treatment.

Anesthesia and instrumentation

Dogs were anesthetized for each treatment. For each dog prior to each anesthetic episode, food was withheld for at least 12 hours; however, water was available until the dog was transported to the research laboratory for the experiment.

At the research laboratory, a 20-gauge, 4.8-cm catheter was aseptically placed in a cephalic vein. Anesthesia was induced by IV administration of propofol (mean ± SD dose delivered, 4.2 ± 0.6 mg/kg) titrated to effect. Following orotracheal intubation with an appropriately sized cuffed endotracheal tube, anesthesia was maintained with isoflurane in oxygen delivered via a partial rebreathing anesthetic circuit. An infrared gas analyzer was used to measure 

\( \text{ETCO}_2 \) and \( \text{ETiso} \). Intermittent positive pressure ventilation was used to maintain the \( \text{ETCO}_2 \) between 30 and 40 mm Hg (actual mean ± SD \( \text{ETCO}_2 \) maintained, 36 ± 3 mm Hg). A forced-air warming system was used to maintain esophageal temperature at approximately 38°C (actual mean ± SD esophageal temperature maintained, 37.9 ± 0.2°C). The dog was positioned in dorsal recumbency on a warm water blanket and instrumented with a multiparameter monitor that recorded the lead II ECG and used oscillometry to monitor heart rate (actual mean ± SD heart rate, 109 ± 18 beats/min) and MAP (actual mean ± SD MAP, 67 ± 9 mm Hg). Each dog received an IV infusion of lactated Ringer solution (5 mL/kg/h) by use of a fluid administration pump while anesthetized.

Treatment administration

The 3 study treatments consisted of the injection of 1, 2, or 3 mL of a mixture (50:50 vol/vol) of 2% lidocaine solution and 0.5% bupivacaine solution approximately 2 cm caudal to and at the caudal aspect of the infraorbital canal on the designated side (half of the total volume was injected at each site). For each treatment, the infraorbital foramen was palpatated through the oral mucosa by moving the upper lip dorsally on the side designated for injection. A 22-gauge, 4.5-cm-long IV catheter (including stylet) was inserted at the rostral aspect of the infraorbital canal. Then, the stylet was retracted 2 to 3 mm, and the catheter was advanced further into the infraorbital canal. For the initial 4 experiments (treatment-dog combinations), the catheter was advanced to its full length through the infraorbital canal parallel to the upper dental arch, and the stylet was removed. This positioned the end of the catheter at a point parallel to the lateral canthus of the eye. A syringe containing the assigned treatment was attached to the free end of the catheter. Negative pressure was applied to the catheter to ensure that it was not in a blood vessel before the entire treatment volume was injected. For all the remaining experiments, the catheter was inserted through the infraorbital canal as previously described, but only half of the treatment volume was injected with the catheter fully inserted through the canal. The catheter was then withdrawn 2 cm, and
the remaining stimulation volume was injected after ensuring that the end of the catheter was not within a blood vessel. The treatment volume was injected over 30 seconds at each injection site. After the entire treatment volume was injected, the catheter was removed, and pressure was applied at the catheter site for approximately 20 to 30 seconds.

**REMP**

At each stimulation site, 2 shielded unipolar stimulating needle electrodes were inserted approximately 5 mm apart and as close as possible to the dental-gingival margin. Two shielded unipolar stimulating needle electrodes were also inserted SC over the digastricus muscle on the side ipsilateral to the injected treatment to record the REMP from each stimulation site. Another electrode was inserted SC over the dorsal cervical region to serve as a ground. The REMP was measured with an evoked potential measurement system that used a proprietary software program.

To determine the maximal stimulus at each site for each measurement time, an electrical stimulus was applied to the site, and the intensity of the current was varied to obtain maximal amplitude of the first REMP waveform with minimal stimulus artifact. A 0.5-millisecond pulse width at a frequency of 1 Hz for 20 seconds was used, and the machine determined the average of the resulting waveforms. The intensity of current varied between 15 and 100 mA. Before each REMP measurement cycle, the impedance of each recording electrode was checked to ensure it was < 30 kΩ, and physiologic variables (heart rate, MAP, esophageal temperature, ET<sub>CO<sub>2</sub></sub>, and ET<sub>iso</sub>) were recorded.

The REMP was measured at the vestibular aspect of MC on the side contralateral to the injected treatment and the vestibular aspect of MC on the side contralateral to the injected treatment. For each experiment, 3 baseline REMP recordings were obtained at 10-minute intervals for each stimulation site. The end of the injection of the assigned treatment was designated time 0, and the REMP was measured before (baseline); at 5, 10, 15, 30, 45, and 60 minutes after treatment administration; and every 20 minutes thereafter until the stimulation sites were no longer desensitized or for up to 6 hours. Each REMP measurement cycle (ie, measurement at all 5 locations) took approximately 2 to 3 minutes to complete.

For each dog, carprofen (2 mg/kg, IV) was administered immediately following completion of all REMP measurements. Then, isoflurane administration was discontinued, and the dog was allowed to recover from anesthesia.

**Statistical analysis**

For each experiment and data acquisition time, the REMP for the noninjected contralateral MC was used as a control to normalize the REMP values obtained for the 4 ipsilateral stimulation sites. For each treatment and stimulation site, the primary outcome of interest was the normalized area under the first REMP waveform, although the REMP amplitude (height of the waveform) and time to onset (time from application of the stimulus to initiation of the waveform) and duration (time elapsed between initiation of the stimulus and return to the same voltage) of desensitization were also assessed.

The following equation was used to normalize the area under the first REMP waveform for all ipsilateral measurements:

\[
\text{normalized value} = \left( \frac{C}{TC} \right) \times \left( \frac{TT/TRC}{} \right) \times 100
\]

where \(C\) is the mean baseline area under the first REMP waveform for the noninjected contralateral MC, \(TC\) is the subsequent area under the first REMP waveform for the noninjected contralateral MC at time \(t\), \(TT\) is the subsequent area under the first REMP waveform for a particular injected ipsilateral stimulation site (MC, MPM4, MM2, or hard palate) at time \(t\), and \(TRC\) is the mean baseline area under the first REMP waveform for that injected ipsilateral stimulation site. A stimulation site (oral structure) was considered desensitized or blocked if a flat line was achieved during REMP measurement or the normalized area under the first REMP waveform was < 15% after treatment injection and was considered recovered (ie, no longer desensitized) when the area under the first REMP waveform was > 15% of the control value.

Descriptive statistics were generated. Data for onset and duration of desensitization were summarized as the median and range. The effects of treatment (1, 2, or 3 mL of the lidocaine-bupivacaine mixture; injectate volume) and stimulation site (MC, MPM4, MM2, and hard palate) on the probability that a block occurred were evaluated by use of mixed multilevel logistic regression with QR decomposition. Mixed linear regression was used to evaluate the effects of treatment and stimulation site on onset and duration of the nerve block. All models included an identity covariance matrix structure to control for individual dog as a random effect. Wald tests with the Bonferroni method were used when post hoc multiple comparisons were performed to examine the effects of treatment volume and injection site on the dependent variables. Values of \(P < 0.05\) were considered significant. Dogs with missing data were retained during the construction of all models. All analyses were performed with commercial statistical software.

**Results**

All dogs completed each of the 3 treatments. However, because of an oversight, desensitization of the hard palate was assessed for only 3 of the 6 dogs following administration of the 1-mL treatment and for only 5 dogs following administration of the 2- and 3-mL treatments. No adverse effects associated with the study treatments were observed in any of the dogs.
A mean ± SD ET_{ISO} of 1.8 ± 1.2% was necessary to maintain an anesthetic plane sufficient for prevention of movement in response to electrical stimulation of the gingival tissue or hard palate mucosa. That ET_{ISO} generally did not suppress autonomic responses (an increase in heart rate or MAP) to the noxious (electric) stimulus; however, those responses were transient, and the heart rate and MAP quickly returned to prestimulation levels after application of the electric stimulus was discontinued.

The proportion of dogs for which desensitization was successfully achieved varied by treatment and oral structure (Table 1). Test site was significantly (P = 0.004) associated with the probability of desensitization (successful nerve block) after controlling for subject (dog) and treatment (injectate volume). The MC was more likely to be successfully desensitized than the MM2 (P = 0.029) and hard palate (P = 0.004). Likewise, MPM4 was more likely to be successfully desensitized than the MM2 (P = 0.023) and hard palate (P = 0.004). The likelihood of a successful block did not differ significantly between MC and MPM4 (P = 0.953) or between MM2 and the hard palate (P = 0.189).

When subject and oral structure were controlled, treatment was not significantly (P = 0.064) associated with the likelihood of a successful block. The likelihood of successful block did not differ significantly between the 1- and 2-mL treatments (P = 1.00), 1- and 3-mL treatments (P = 0.540), or the 2- and 3-mL treatments (P = 0.080).

When a nerve block was successfully achieved, the time to onset of desensitization was not significantly associated with test site (P = 0.272) or treatment (P = 0.449) after the subject was controlled. However, the duration of desensitization differed significantly (P = 0.001) among oral structures. The duration of desensitization for MC was significantly longer than that for the hard palate (P = 0.002) but did not differ significantly from that for MPM4 (P = 0.152) or MM2 (P = 0.202). The duration of desensitization for MPM4 was significantly longer than that for MM2 (P = 0.035) and the hard palate (P < 0.001). The duration of desensitization did not differ significantly (P = 0.116) between the MM2 and hard palate. Treatment was not significantly (P = 0.437) associated with the duration of desensitization.

### Discussion

Results of the present study indicated that the volume of local anesthetic injected caudal to and at the caudal aspect of the infraorbital canal of dorsally recumbent dogs was not significantly associated with the efficacy or duration of desensitization (nerve block). For this study, we chose to position dogs in dorsal recumbency for injection of the local anesthetic because many dental procedures are routinely performed with dogs positioned in that recumbency. Additionally, dorsal recumbency provided better access to the stimulating electrodes used for REMP measurements. We chose to use a 50:50 (vol/vol) mixture of lidocaine and bupivacaine on the basis of findings of another study, which indicate that such a mixture provides a rapid onset of desensitization and has a longer duration of effect than injection of lidocaine alone. A long duration of effect is desirable for postoperative analgesia, especially when invasive dental procedures are performed.

The volumes of local anesthetics used to perform infraorbital nerve blocks in dogs in general practice are typically smaller than volumes of the lidocaine-

### Table 1

<table>
<thead>
<tr>
<th>Injectate volume (mL)</th>
<th>Oral structure*</th>
<th>No. of dogs in which the treatment resulted in desensitization of the oral structure</th>
<th>Time to onset of desensitization (min)</th>
<th>Duration of desensitization (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>MC</td>
<td>4</td>
<td>12.5 (5–80)</td>
<td>145.5 (75–230)</td>
</tr>
<tr>
<td></td>
<td>MPM4</td>
<td>6</td>
<td>12.5 (5–120)</td>
<td>247.5 (15–315)</td>
</tr>
<tr>
<td></td>
<td>MM2</td>
<td>2</td>
<td>5 (5–5)</td>
<td>130 (5–255)</td>
</tr>
<tr>
<td></td>
<td>Hard palate‡</td>
<td>1</td>
<td>10</td>
<td>35</td>
</tr>
<tr>
<td>2</td>
<td>MC</td>
<td>6</td>
<td>22.5 (5–45)</td>
<td>197.5 (50–375)</td>
</tr>
<tr>
<td></td>
<td>MPM4</td>
<td>6</td>
<td>7.5 (5–15)</td>
<td>312.5 (115–395)</td>
</tr>
<tr>
<td></td>
<td>MM2</td>
<td>5</td>
<td>15 (5–45)</td>
<td>75 (50–335)</td>
</tr>
<tr>
<td></td>
<td>Hard palate‡</td>
<td>2</td>
<td>27.5 (10–45)</td>
<td>62.5 (15–100)</td>
</tr>
<tr>
<td>3</td>
<td>MC</td>
<td>5</td>
<td>30 (5–100)</td>
<td>230 (120–355)</td>
</tr>
<tr>
<td></td>
<td>MPM4</td>
<td>5</td>
<td>15 (10–15)</td>
<td>245 (15–345)</td>
</tr>
<tr>
<td></td>
<td>MM2</td>
<td>2</td>
<td>7.5 (5–10)</td>
<td>162.5 (70–255)</td>
</tr>
<tr>
<td></td>
<td>Hard palate‡</td>
<td>1</td>
<td>45</td>
<td>15</td>
</tr>
</tbody>
</table>

Each dog received each injectate volume (treatment) in a randomized order with at least a 14-day washout period between treatments. An oral structure was considered desensitized if a flat line was achieved during REMP measurement or the normalized area under the first REMP waveform was < 15% after treatment injection and was considered recovered (ie, no longer desensitized) when the area under the first REMP waveform was approximately 15% of the control value. The contralateral MC was used as the control. A range is not provided when an oral structure was desensitized in only 1 dog.

*Ipsilateral to the side in which the treatment was injected. †Only assessed in 3 of the 6 dogs. ‡Only assessed in 5 of the 6 dogs.
bupivacaine mixture evaluated in the present study. Previous studies\(^5\) of infraorbital nerve blocks in dogs involved the injection of local anesthetics at volumes smaller than those administered in the present study at various depths within the infraorbital canal. In 1 study,\(^7\) injection of 0.5 mL of a 2% lidocaine solution at a depth of 0.5 cm into the infraorbital canal of dogs similar in size to those of the present study failed to provide adequate desensitization for posterior rhinoscopy. It is possible the injection failed to block nerve impulses from the major, minor, and accessory branches of the palatine nerve and caudal nasal nerve, all of which originate from the ptgypalatine nerve, a branch of the maxillary nerve. Additionally, the caudal aspect of the nasal cavity is also innervated by the ethmoidal nerve, which is a branch of the ophthalmic nerve. Therefore, an infraorbital nerve block may not be the most appropriate approach for desensitizing the nasal cavity. In another study,\(^8\) injection of 1 mL of a 2% chloroprocaine solution at an unspecified depth within the infraorbital canal of dogs successfully desensitized (as determined by measurement of REMP) all ipsilateral teeth evaluated including the MC, MPM4, and maxillary first molar tooth. In other studies, injection of 0.3 mL of a 2% mepivacaine solution\(^1\) or 0.3 mL of a 0.5% bupivacaine solution\(^2\) at a depth of approximately 0.5 cm in the infraorbital canal of isoflurane-anesthetized Beagles (body weight, approx 9 kg) resulted in an approximately 20% decrease in the MAC of isoflurane when an electrical stimulus was applied to the ipsilateral MC; however, that stimulus induced an increase in heart rate and blood pressure. This suggests that the nerve blocks performed in those studies\(^1,2\) were only partially effective because complete blockade of the noxious stimulus to the CNS should have prevented any change in heart rate and blood pressure and increased the magnitude of the reduction in MAC for isoflurane. Theoretically, complete blockade of a noxious stimulus should cause a 100% reduction in the MAC for isoflurane, but that is not pharmacologically possible because animals begin to regain consciousness when the MAC of isoflurane approaches 0.5%. In another study,\(^10\) epidural administration of morphine to halothane-anesthetized dogs caused an approximately 40% decrease in the MAC for halothane, but the end point was difficult to determine because the dogs were spontaneously moving (regaining consciousness) when the MAC was decreased by that amount. If the injection of 0.3 mL of local anesthetic 0.5 cm into the infraorbital canal as previously described\(^1,2\) achieved a complete block of the infraorbital nerve, the MAC for isoflurane should have been decreased by approximately 40%; thus, that approach does not appear to yield a complete block of the infraorbital nerve. Results of another study\(^5\) conducted by our research group suggest that injection of 1 mL of a local anesthetic at approximately two-thirds the depth of the infraorbital canal in approximately 20-kg dogs results in consistent desensitization of the ipsilateral MC but inconsistent desensitization of MPM4 and MM2.

In a study\(^9\) involving canine cadaver heads, better staining of the maxillary and ptgypalatine nerves was achieved when 0.5 mL of methylene blue stain was injected within the caudal portion of the infraorbital canal with the tip of the needle or catheter positioned where a line parallel to the infraorbital canal intersects a line perpendicular to the infraorbital canal from the lateral canthus of the eye, compared with the staining of those 2 nerves when 0.5 mL of methylene blue stain was injected by a traditional percutaneous approach to the maxillary nerve. In an in vivo study,\(^5\) injection of 1 mL of local anesthetic within the infraorbital canal of dogs at a point approximately two-thirds of its length resulted in inconsistent desensitization of MPM4 and MM2.

For a nerve block to be effective, it is imperative that the local anesthetic be deposited in close proximity to the nerve so that the targeted portion of the nerve is exposed to the drug. A successful peripheral nerve block requires blockade of at least 70% of the transmembrane sodium channels over 3 consecutive nodes of Ranvier.\(^11\) In mammalian nerve fibers, the internodal distance is determined principally by the diameter of the fiber and can vary from 0.2 to 2 mm.\(^12\) Therefore, the volume of local anesthetic injected should be sufficient to cover 6 mm of the targeted portion of the nerve to ensure blockade of at least 70% of the transmembrane sodium channels over 3 consecutive nodes of Ranvier. Results of the present study and other studies\(^5,9\) suggested that injection of 0.5 to 1.0 mL of local anesthetic at various depths within the infraorbital canal of dogs can effectively desensitize MC and MPM4. From an anatomic perspective, it is not surprising that deposition of a local anesthetic within the infraorbital canal exposes a sufficient length of the nerve to the drug to effectively desensitize the ipsilateral MC.

In the previously described canine cadaver study,\(^9\) injection of 0.5 mL of methylene blue stain within the infraorbital canal resulted in some staining of the maxillary nerve in 34 of 37 (92%) heads and staining of > 6 mm of the maxillary nerve in 24 (65%) of those heads; additionally, the ptgypalatine nerve was stained in 26 of 37 (70%) heads. In an experiment by Fizzano et al\(^6\) that involved the cadavers of mesaticephalic dogs with body weights similar to the dogs of the present study, 1, 1.5, or 3 mL of a 1% methylene blue solution was injected through a 20-gauge, 5-cm-long over-the-needle catheter, which was inserted through the infraorbital canal so that the catheter tip was positioned near the ptgypalatine fossa. Results of that study\(^6\) indicate that 5 mL of injectate was required to stain all branches of the maxillary nerve including the palatine and caudal superior alveolar nerves. Collectively, findings from studies\(^6,9,13\) of maxillary nerve block approaches in canine cadavers indicate that injection of a fairly large volume (3 mL) of stain in the caudal portion of the infra-
orbital canal consistently results in better staining of the maxillary nerve and its branches than other approaches and injectate volumes, which is desirable for rhinoscopy and various dental procedures involving the caudal portion of the upper dental arch. In an in vivo crossover experiment, pain and stress responses were compared for dogs that underwent rhinoscopy and nasal biopsy specimen collection following infraorbital injection of 3 mL of a 0.5% bupivacaine solution or physiologic saline (0.9% NaCl) solution. The mean blood pressure and serum cortisol concentration in response to the rhinoscopy and biopsy procedures were higher when saline solution was administered than when bupivacaine was administered; however, the mean intraoperative heart rate, serum norepinephrine concentration, incidence of purposeful movement, and postoperative pain score did not differ significantly between the 2 treatments.6

In the present study, we injected fairly large volumes (1, 2, and 3 mL) of local anesthetic deep beyond the infraorbital canal of dogs, with the expectation that such an approach would provide consistent desensitization of the caudal maxillary teeth and hard palate. Desensitization of the ipsilateral MC was not achieved in the first dog following injection of the 3-mL treatment. Therefore, we decided to modify the protocol and inject half the assigned volume with the 4.5-cm-long catheter fully inserted into the infraorbital canal and the other half after the catheter was withdrawn 2 cm from the canal, with the expectation that the modified protocol would lead to the desensitization of the ipsilateral MC as well as the caudal maxillary teeth and hard palate. The catheters used in the Fizzano et al9 experiments were 5 cm long, whereas the catheters used in the present study were only 4.5 cm long, despite the fact that dogs in both studies were mesaticephalic with similar body weights. Thus, it is likely that, in the present study, the first half of the injectate was deposited near the pterygopalatine fossa. It was expected that injection of local anesthetic at that location would result in consistent desensitization of the caudal superior alveolar and palatine nerves and the loss of REMP from MM2 and the hard palate. Findings of the present study indicated that increasing the local anesthetic injectate volume from 1 to 3 mL did not improve the efficacy of the block in desensitizing MM2 and the hard palate. Results of the Fizzano et al9 study and the present study suggested that extrapolation of data from cadavers to live animals in clinical situations must be done carefully because the likelihood of a successful nerve block is dependent on a combination of anatomic, physiologic, procedural, and pharmacological factors. It has been proposed that the distribution of dye in cadavers may not accurately reflect the distribution of local anesthetic in live animals.13 Consequently, various approaches and injectate volumes must be evaluated in live animals to assess the efficacy of nerve blocks. Lidocaine concentrations of 10 mg/mL can cause vasodilation of skeletal muscle arterioles.15

It is possible that administration of lidocaine to the dogs of the present study caused local vasodilation and increased blood flow to the surrounding medial and lateral pterygoid muscles, thereby leading to an increase in lidocaine uptake into the systemic circulation. Nevertheless, the duration of desensitization induced by the injectate volumes assessed in the present study was adequate for most dental procedures.

For most experiments (dog-treatment combinations) of the present study, the treatment was divided and deposited at approximately 2 cm caudal to and at the end of the infraorbital canal. The injectate deposited caudal to the infraorbital canal (ie, with the catheter fully inserted) had a larger physical space (the pterygopalatine fossa) to diffuse into than the injectate deposited at the caudal aspect of the infraorbital canal. Therefore, it is possible that a large portion of the injectate deposited with the catheter fully inserted diffused away from the nerve. The caudal superior alveolar nerves and pterygopalatine nerves arise from the ventral surface of the infraorbital and maxillary nerves, respectively.3,9 Because the treatments of the present study were administered with the dogs positioned in dorsal recumbency, it is also possible that a portion of the drug deposited with the catheter fully inserted settled away from the desired site of action owing to the effect of gravity. It has been demonstrated that gravity can significantly affect the distribution of local anesthetics in peripheral nerve blocks and epidurals.37 In the previously described study involving canine cadaver heads, when staining of the maxillary and pterygopalatine nerves was not achieved, the dye was always found medial to the maxillary nerve close to the palatine bone. The investigators of that study proposed that lateromedial insertion of the cannula into the infraorbital canal could have resulted in a lack of proximity between the tip of the cannula and the nerve. Although the angle of catheter insertion was not monitored in the present study, it could have affected the location of local anesthetic deposition when the treatment was injected with the catheter fully inserted. It is also possible that the local anesthetic was injected in the interstitium of the medial pterygoid muscle, and its spread was limited by muscle fascia. However, it is unlikely that the muscle body would have been penetrated given that IV catheters were used for administration of the study treatments.

The success of a nerve block is dependent not only on the correct anatomic deposition of the local anesthetic but on the absolute drug mass used as well. The mass of a drug is influenced by its volume and concentration. In a study involving rats, when equipotent doses of lidocaine were used at different volumes to perform sciatic nerve blocks, the intraneural drug concentration achieved was greater when higher volumes were used, but the functional characteristics of the nerve blocks did not differ significantly among the volumes evaluated. The investigators of that study theorized that the administra-
tion of larger volumes of the drug (albeit at lower concentrations) led to an increase in the uptake of the drug into the superficial epineural compartment. It is possible that a similar phenomenon occurred in the present study.

In the present study, the mean durations of desensitization for MC and MPM4 were significantly longer than those for MM2 and the hard palate, regardless of treatment. For peripheral nerve blocks to be effective, it is critical that the local anesthetic remain in contact with nerve tissue, which is easily achieved when the drug is deposited within the infraorbital canal. Moreover, the pressure generated by deposition of the local anesthetic within the noncompliant infraorbital canal might have impaired blood perfusion within the canal and delayed absorption of the local anesthetic into the systemic circulation, thereby resulting in longer exposure of the nerve to the drug. That may explain the apparently more consistent and longer duration of desensitization of the ipsilateral MC and MPM4 observed in the present study.

Nerve damage and intravascular or intraneural injection are potential complications that can result from the use of an IV catheter to perform an infraorbital nerve block, but none of those complications were observed in the present study. Threading a catheter or cannula through the infraorbital canal can damage and cause prolonged desensitization of the nerve. To prevent that from happening in the present study, the stylet was retracted into the catheter before the catheter was advanced through the infraorbital canal. Also, the catheters used in the present study were composed of fluorinated ethylene propylene polyester, a flexible material with a low coefficient of friction, which was unlikely to cause damage to structures within the infraorbital canal. The catheter was aspirated prior to administration of any injections, which ensured that the local anesthetic was not injected into a blood vessel. The risk of intraneural injection was negligible because the stylet was removed from the catheter prior to treatment injection and no excessive pressure was noted during any injection. On the basis of our clinical experience and data from the present study and other studies, in which an infraorbital approach was used for nerve blocks in dogs, adverse effects associated with that approach appear to be minimal. However, complications associated with infraorbital nerve blocks will be better elucidated as more literature becomes available.

Six dogs each received three treatments in a randomized crossover manner in the present study. The enrollment of more dogs in the study might have increased the power and allowed us to identify additional significant differences among treatments in regard to the efficacy of desensitization of the assessed oral structures. Also, for some experiments, the area under the first REMP waveform for some oral structures remained < 15% of the control value 6 hours after treatment injection, which indicated that those structures were still desensitized. It is likely that the mean duration of desensitization for some of the oral structures would have increased had we continued monitoring the REMP until all oral structures were no longer desensitized. However, this would have prolonged the duration that the dogs were anesthetized.

Findings of the present study indicated that increasing the volume of local anesthetic used for an infraorbital nerve block in dogs provided minimal benefits in terms of block efficacy and duration. Our experience with the dogs of this study suggested that a modified approach by which half of the local anesthetic volume was deposited just past the caudal aspect of the infraorbital canal and the other half was deposited close to the middle and rostral superior alveolar nerves at the caudal aspect of the infraorbital canal might improve desensitization of all teeth in the ipsilateral maxillary arch, compared with deposition of the entire injectate volume at the caudal aspect of the infraorbital canal. However, with use of the modified approach, in some instances, the ipsilateral MC remained sensitive even when MPM4 was desensitized, which suggested that the concentrations of lidocaine and bupivacaine used in this study were insufficient to fully penetrate and block the conduction of nerve impulses in large nerves such as the maxillary nerve. Further research is necessary to evaluate the effect of local anesthetic concentration on the efficacy of nerve blocks commonly used for oral procedures in veterinary patients.

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e. Carescape B650, GE Healthcare, Waukesha, Wis.

f. Mark 4, Bird Corp, Palm Springs, Calif.

g. Bair Hugger, Arizant Healthcare Inc, Eden Prairie, Minn.

h. Baxter Healthcare Corp, Deerfield, Ill.

i. Hospira Inc, Lake Forest, Ill.

j. B. Braun Melsungen AG, Melsungen, Germany.

k. Grass Products, Warwick, RI.

l. Viking IVD, Nihon Kohden America Inc, Irvine, Calif.

m. Stata Corp LP, College Station, Tex.

Footnotes


