FAQs
Analgesia, Sedation, and Anesthesia

Making the Switch from Medetomidine to Dexmedetomidine

A Peer-Reviewed Publication
Dexmedetomidine (Dexdomitor, Pfizer Animal Health) has recently been approved by the FDA Center for Veterinary Medicine as a sedative and analgesic in dogs and cats and as a preanesthetic to general anesthesia in dogs.\(^1\)\(^2\) This drug has been introduced into the United Kingdom and other European countries with similar indications. Within the United States, veterinary practitioners are faced with switching from medetomidine (Domitor, Pfizer Animal Health) to dexmedetomidine (Dexdomitor) because medetomidine has just been phased out and a generic equivalent is not currently available in this country.

In 1997, Ko and colleagues\(^3\) published an article based on questions received from veterinary practitioners regarding the safe and effective use of medetomidine and its specific antagonist, atipamezole (Antisedan, Pfizer Animal Health). Since the launch of Dexdomitor in November 2007, the authors have received numerous questions from veterinarians about this drug and thought that a compilation of the most commonly asked questions and answers would be useful during this transitional period. The answers provided in Part I of this article are based on a review of currently available literature as well as label indications. Part II provides information about the off-label use of dexmedetomidine in combination with other sedative, analgesic, and/or anesthetic agents that has been compiled from published studies and the authors’ collective clinical experience.

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**PART 1: Introducing Dexmedetomidine**

Dexmedetomidine is a synthetic α₂-adrenoreceptor agonist with sedative and analgesic properties. It is the dextrorotary enantiomer of the racemic mixture medetomidine (Figure 1). Dexmedetomidine is commercially available in the United States for use in veterinary patients as Dexdomitor and is indicated for use as a sedative and analgesic in dogs and cats to facilitate clinical examinations and various clinical procedures, including minor surgical and dental procedures. It is also indicated for use as a premedicant prior to general anesthesia in dogs. It is approved for intravenous (IV) and intramuscular (IM) administration in dogs but only IM administration in cats. Dexmedetomidine is also commercially available for use in human patients (Precedex, Hospira) and is indicated for sedation of initially intubated and mechanically ventilated patients during treatment in an intensive care setting.

**What are the similarities and differences between dexmedetomidine and medetomidine?**

Medetomidine is an equal mix of two optical enantiomers, dexmedetomidine and levomedetomidine. Levomedetomidine has been found to have no sedative or analgesic effects. As such, the pharmacologic activity of medetomidine is primarily attributable to dexmedetomidine. Dexmedetomidine is approximately twice as potent as medetomidine in terms of its ability to produce sedation and analgesia. Clinically, this is reflected in the fact that administration of dexmedetomidine requires less drug on a µg/kg or body surface area (BSA) basis. In contrast to Domitor, which is supplied as a 1 mg/ml solution, Dexdomitor is supplied as a 0.5 mg/ml solution, allowing clinicians to use the same injection volume because the dexmedetomidine dilution has the same potency as medetomidine.

A review of current literature revealed a number of studies suggesting that dexmedetomidine differs from medetomidine in several respects. How these differences translate into clinical practice in dogs and cats remains to be fully elucidated. For comparative purposes, a brief overview of these differences is provided.

Dexmedetomidine contains only the active enantiomer without levomedetomidine. As such, only half the amount of the racemic mixture of medetomidine needs to be administered, and therefore metabolized, when compared with medetomidine (which contains both dexmedetomidine and levomedetomidine). Differences in drug metabolism between dexmedetomidine and medetomidine can therefore be expected.

Levomedetomidine has been shown to interfere with the metabolism of other anesthetic drugs in the liver. In human studies, levomedetomidine has been shown to have a greater inhibitory effect than dexmedetomidine on ketamine metabolism by the liver, which may slow recovery after completion of a procedure.

A recent study comparing dexmedetomidine and medetomidine premedication before ketamine anesthesia in 72 cats found that dexmedetomidine-premedicated cats recovered more quickly than cats premedicated with racemic medetomidine. Results of other studies in cats have been equivocal with respect to differences between medetomidine and dexmedetomidine in this species. One small study (six cats) showed no difference between the two drugs in terms of their effects on heart rate, respiratory rate, or body temperature. A more recent study (120 cats) demonstrated that the percentage

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**Figure 1**

Dexmedetomidine contains only the active enantiomer of the racemic mixture medetomidine. Because levomedetomidine has no sedative, analgesic, or cardiorespiratory effects, dexmedetomidine is twice as potent as medetomidine based on a microgram of dexmedetomidine versus a microgram of medetomidine. As currently supplied for veterinary use, however, dexmedetomidine (0.5 mg/ml) has been diluted to be equipotent to medetomidine (1 mg/ml) on a volume-to-volume basis.
of cats with normal heart rate and normal pulse character after drug administration was higher with dexmedetomidine than medetomidine.12

In one study,5 analgesia provided by dexmedetomidine at a dose of 20 µg/kg IV was shown to last longer than that provided by medetomidine at 40 µg/kg IV, suggesting the possibility of greater analgesic duration for dexmedetomidine in dogs.

The analgesic effects of dexmedetomidine (20 µg/kg) last for approximately an hour and can extend into the recovery period if the drug is not reversed with the antagonist, atipamezole.8

How is dexmedetomidine dosed?

The available concentration of dexmedetomidine (0.5 mg/ml; Dexdomitor) provides practitioners with a simple volume-for-volume substitution of dexmedetomidine for medetomidine. Without this formulation, dose determination could be confusing. The package insert of Dexdomitor provides a chart of canine body weight ranges in pounds and kilograms. Practitioners are required to select a dexmedetomidine dosage for a given canine body weight based on the level of sedation/analgesia desired (i.e., premedication/cooperative sedation or full clinical sedation) and the route of administration. The chart provides both an injection volume of Dexdomitor in milliliters as well as a dose in µg/kg. In dogs, there are two FDA-approved Dexdomitor doses for sedation and analgesia (500 µg/m² IM and 375 µg/m² IV) and two approved doses for premedication (125 µg/m² IM and 375 µg/m² IV). The small range in feline body weights resulted in a single FDA–approved Dexdomitor dose (40 µg/kg) for sedation and analgesia in cats.7 This dose is based on the BSA of a 4.5-kg cat, and BSA dosing may still be an important consideration for cats weighing less than 4.5 kg.

In dogs, body weights and mass-specific metabolic rates vary across breeds.18 Within-breed differences also exist because individual animals have different body conformations and may be under- or overweight relative to the breed standard. As such, dexmedetomidine doses are based on an animal’s BSA rather than body weight to minimize variations in sedative and analgesic effects.

<table>
<thead>
<tr>
<th>Weight (kg)</th>
<th>Dexmedetomidine Dose for Cooperative Sedation/Preanesthesia in Dogs (125 µg/m² IM)</th>
<th>Dexmedetomidine Dose for Moderate Sedation/Preanesthesia in Dogs (375 µg/m² IM or IV)</th>
<th>Dexmedetomidine Dose for Profound Sedation in Dogs (500 µg/m² IM)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total dose in ml†</td>
<td>Dose in ml/kg†.</td>
<td>Dose in µg/kg</td>
</tr>
<tr>
<td>2</td>
<td>0.04</td>
<td>0.02</td>
<td>10.02</td>
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<tr>
<td>2.5</td>
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<tr>
<td>5</td>
<td>0.08</td>
<td>0.015</td>
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<tr>
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</tr>
<tr>
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</tr>
<tr>
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<td>0.011</td>
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<td>0.010</td>
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<tr>
<td>40</td>
<td>0.30</td>
<td>0.007</td>
<td>3.69</td>
</tr>
</tbody>
</table>

*The preanesthetic dose (125 µg/m² IM, cooperative sedation/preanesthesia) can be used IM with an opioid (morphine, 0.25 mg/kg; hydromorphone, 0.05 mg/kg; or butorphanol, 0.2 mg/kg) to produce a moderate degree of sedation similar to that induced by 375 µg/m² IM of dexmedetomidine.
†The total doses in ml and doses in ml/kg in this table have been derived from the µg/kg BSA dose and have been rounded up or down, as appropriate, to accommodate practical dosing. As a result, a perfect match in values based on calculation conversions between columns will not necessarily occur.
BSA is defined as the measured or calculated surface of an animal’s body and is a better indicator of metabolic mass than body weight because it is less affected by abnormal adipose (fat) tissue mass.\textsuperscript{19,20} There is a disproportionate relationship between basal metabolic rate (BMR) and surface area that was encapsulated in Brody’s famous mouse-to-elephant curve, which demonstrates that the “specific” BMR (per unit of body weight) decreases with increasing body size.\textsuperscript{21} In other words, the BMR of a mouse is higher per unit weight than that of an elephant based on mass proportionality.\textsuperscript{20,22} Clinically, this is of particular importance in small animals because the specific (mass-related) metabolic rate of animals decreases with increasing body size.

The clinical significance of using BSA dosing for dexmedetomidine in dogs and cats is elaborated on below and, ideally, dogs and cats should be weighed to allow for accurate dose calculation. Suggested dosing guidelines (for different weight ranges) are included in the package insert; however, individual patient evaluations can influence the final dosage administered. Whether body weights are estimated or actual weights determined, the BSA conversion charts provided in Tables 1 and 2 should be used as a guide to determine dose requirements in ml/kg and µg/kg, thereby ensuring that the dosage of dexmedetomidine is not skewed drastically, leading to gross under- or overdosage.

**What are the cardiorespiratory and other physiologic effects of dexmedetomidine?**

Desirable effects associated with the administration of dexmedetomidine are sedation, analgesia, and muscle relaxation. Other physiologic responses include peripheral vasoconstriction, hypertension, bradycardia, reduced respiratory rate, decreased body temperature, and increased urine production.\textsuperscript{1,2,4,5,8}

A persistent bradycardia is characteristic of both dexmedetomidine and medetomidine. The reduction in heart rate is produced by two different time-dependent mechanisms; which mechanism predominates at a particular time is a function of the interval since drug administration. During the initial phase after dexmedetomidine administration, an increase in systemic vascular resistance produces reflex bradycardia, which is a normal physiologic response to the increased blood pressure that follows peripheral vasoconstriction. In the second phase, approximately 15 to 20 minutes after administration, the initial vasoconstriction wanes and a reduction in sympathetic tone becomes the predominant mechanism underlying bradycardia.\textsuperscript{3} The effect of dexmedetomidine on heart rate can outlast its sedative and analgesic properties, and heart rate should be monitored in animals recovering from dexmedetomidine sedation.\textsuperscript{5} The reduction in cardiac output induced by dexmedetomidine or medetomidine is similar and is primarily attributed to the increase in afterload produced by increased systemic vascular resistance.\textsuperscript{5}

Dexmedetomidine shows little or no effect on respiration in spontaneously breathing anesthetized animals.\textsuperscript{4} Because of its sedative effect, respiratory rate decreases within 5 minutes of IV administration and 15 minutes of IM administration and returns to baseline values by 180 minutes after administration.\textsuperscript{1,2} Body temperature slowly decreases (on average, by 2.1°F to 2.5°F over 180 minutes) to below pretreatment levels in sedated animals.\textsuperscript{1,2} In a recent study,\textsuperscript{7} investigators found that although a decrease in rectal temperature was observed in dogs treated with dexmedetomidine (or medetomidine), their temperatures generally remained within clinically acceptable limits. As part of a best practice protocol, animals should be kept at a warm and consistent temperature by using an exogenous heat source during any procedure and recovery period.

**What would be an unacceptably low heart rate in a dexmedetomidine-sedated patient, and should profound bradycardia be managed?**

An appropriate heart rate is one that is sufficient to maintain cardiac output and blood pressure. Normal heart rates should be based on an animal’s body size instead of a predetermined range. Bradycardia can be considered dangerous when cardiac output is adversely affected and when mean arterial blood pressure falls below 60 to 70 mm Hg.

Dexmedetomidine-induced bradycardia is dose- and route-dependent, with heart rates as low as 28 to 32 bpm having been recorded.\textsuperscript{1} Before making a decision to treat bradycardia in a dexmedetomidine-sedated animal, the patient’s blood pressure and heart rate should be evaluated and interpreted in conjunction with each other. The use of pulse quality in animals sedated with dexmedetomidine or medetomidine may not be as practical given the vasoconstrictive nature of these drugs.

Modern blood pressure monitors allow blood pressure to be accurately measured in dexmedetomidine-sedated patients. Bradycardic animals usually do not require intervention as long as mean arterial blood pressure can be maintained above 70 mm Hg.

The concurrent administration of an experimental α₂-adrenoceptor antagonist (L-659,066) with
dexmedetomidine has been shown to alleviate this bradycardic response without affecting the sedative quality of dexmedetomidine, and further work is required to assess whether L-659,066 could prove clinically useful in attenuating the peripheral α₂-adrenoceptor effects of dexmedetomidine without compromising its sedative or analgesic effects.

Is it appropriate to use an anticholinergic in conjunction with dexmedetomidine?

Several options are available if treatment of bradycardia becomes necessary. Both anticholinergics and atipamezole (Figure 2) have been used to alleviate dexmedetomidine-induced bradycardia. The choice of drug depends on the nature of the procedure, time to completion, and the need to manage pain. Atipamezole reverses not only the dexmedetomidine-induced bradycardia but also the drug’s sedative and analgesic effects, which may interrupt or complicate the procedure being performed.

The recommended route of administration for atipamezole is IM; however, to rapidly reverse bradycardia in an emergency situation, it can be given as a single IV bolus (off label) or at a rate of 5 to 20 µg/kg IV over the course of several minutes. As stated, it is important to remember that atipamezole will antagonize dexmedetomidine sedation and analgesia, thereby reducing the depth of anesthesia. There are no clinical studies supporting the intraoperative use of IV atipamezole, and this technique should be used with caution in emergency situations.

The administration of an anticholinergic before the onset of α₂-agonist–induced bradycardia is more effective than when administered with (or following) an α₂-agonist; however, because of the potential for arrhythmias associated with anticholinergic administration in the presence of an α₂-agonist, routine administration of an anticholinergic before, with, or after medetomidine and dexmedetomidine is not recommended. In a recent study evaluating the sedative and cardiorespiratory effects of acepromazine or atropine administered before dexmedetomidine, atropine prevented dexmedetomidine-induced bradycardia but was associated with a marked hypertensive response.

How is dexmedetomidine used alone in dogs?

Dexmedetomidine can be used in any case in which medetomidine was previously considered a drug of choice. It can be used alone as a preanesthetic before the induction of IV or inhalation anesthesia (mild sedation, 125 µg/m² IM; moderate sedation, 375 µg/m² IM) or as a sedative–analgesic agent for diagnostic or invasive procedures (profound sedation and analgesia, 375 µg/m² IV or 500 µg/m² IM).

As discussed, when using dexmedetomidine alone, it is important to use BSA dosing instead of body weight–based dosing. This will help minimize any variation in the degree of sedation and analgesia attributable to body conformation. A dose chart complementing that provided with the package insert of Dexdomitor is given in Table 1.

Table 1 illustrates the relationship between the dexmedetomidine dose (µg/kg) and an animal’s body weight (kg). It is important to note that the µg/kg dose increases as an animal’s body weight decreases. For dogs, a body weight of 15 kg represents a “pivot point” below which dose requirements for dexmedetomidine may be relatively greater by virtue of greater BSA:body weight ratios. Clinically, this is important because it means that smaller dogs (<15 kg) will need relatively more dexmedetomidine per unit body weight than larger dogs (>15 kg). For example: For an IM dose of 500 µg/m², a dog weighing 2 kg is dosed at 40.08 µg/kg (0.16 ml total dose volume), whereas a dog weighing 40 kg is dosed at 14.77 µg/kg (1.18 or ~1.2 ml total dose volume).

One of the values of dexmedetomidine when used before anesthesia induction is its dose-sparing effect on
induction and maintenance drugs commonly used for general anesthesia. Premedication with 125 µg/m² of dexmedetomidine has been shown to markedly reduce anesthetic requirements. Induction drug requirements for intubation were shown to be reduced by between 30% to 61% on average, depending on the preanesthetic dose and the induction drug used. Mean isoflurane concentration during major procedures was lowered by 40% to 60% for dexmedetomidine-treated dogs compared with control dogs not receiving dexmedetomidine. The extent of this drug-sparing effect is proportional to the dose of dexmedetomidine administered. In our experience, combining the 125 µg/m² dose of dexmedetomidine (as listed in Table 1) with an opioid (see Table 3 for opioid dosages) provides an effective preanesthetic combination in dogs. However, if more profound sedation is desired, higher doses of dexmedetomidine alone (as listed in Table 1) can be used, or dexmedetomidine can be used in combination with an opioid (as listed in Table 3). The dexmedetomidine–opioid combination provides effective sedation and analgesia and also exerts a dose-sparing effect on induction and maintenance anesthetics (as discussed above).

In a similar fashion to medetomidine microdoses, dexmedetomidine microdoses can be used to facilitate endotracheal intubation and smooth recovery from general anesthesia. In addition, microdoses of dexmedetomidine have been used as a constant-rate infusion adjunct during propofol or isoflurane anesthesia and for intra- and postoperative pain management. However, none of these uses are approved by the FDA.

**How is dexmedetomidine used alone in cats?**

Dexmedetomidine is approved for use in cats as a sedative and analgesic to facilitate clinical examinations, minor surgical procedures, and minor dental procedures. As is true in dogs, it can be used in cats as a substitute for medetomidine.

At the label dose of 40 µg/kg IM, dexmedetomidine induces a moderate to deep level of sedation and provides chemical restraint and analgesia sufficient for clinical examinations and procedures, including radiography, ultrasonography, ear examinations and treatment of otitis, drainage of abscesses, grooming and bathing, suture removal, collection of blood samples, and oral examination and dentistry.

Onset of sedation occurs within 5 minutes after IM administration, and the window of profound sedation is 15 to 60 minutes after IM administration. Most cats return to their presedated state within 180 minutes after initial IM administration without atipamezole reversal. In a study comparing the effects of dexmedetomidine with xylazine, approximately 57% of 122 cats that received dexmedetomidine (40 µg/kg IM) and 68% of 120 cats that received xylazine (2.2 mg/kg IM) vomited during the first 5 minutes after administration, regardless of whether they were fasted.

We have used lower dose rates (5 to 30 µg/kg IM) to induce mild to moderate sedation. The use of dexmedetomidine via the IV route has also been reported. Lower doses appear to be more frequently associated with vomiting than the label dose, particularly when administered subcutaneously or IM.

Because of the small range in feline body weights, a dose of 40 µg/kg (based on a 4.5-kg cat) was approved for dexmedetomidine. BSA dosing, however, may be an important consideration for cats weighing less than 4.5 kg. A dose chart incorporating the BSA principles is presented in Table 2. It is important to note that, as is the case in dogs, the µg/kg dose increases as a cat’s body weight decreases. Clinically, this implies that smaller cats (<4.5 kg) will need relatively more dexmedetomidine per unit body weight than larger cats (>4.5 kg).
Medetomidine has been successfully used in a variety of combinations for more than a decade, and dexmedetomidine can replace medetomidine in all such combinations. For example, dexmedetomidine can be combined with opioids (e.g., butorphanol, buprenorphine, morphine, hydromorphone), dissociative agents (e.g., ketamine), benzodiazepines (e.g., midazolam, diazepam), and tiletamine–zolazepam (Telazol, Fort Dodge Animal Health) to produce a wide range of dose-related responses in dogs and cats ranging from mild sedation to a surgical plane of anesthesia. Various combinations are listed in Tables 3, 4, and 5. The underlying dosing strategy for these combinations is to start with a low dose in a given patient and supplement additional amounts as required. The protocols in Tables 3, 4, and 5 are based on our experience with dexmedetomidine and represent modifications of published medetomidine combinations. These protocols, however, have not been validated in published studies nor are they approved by the FDA.

What are the similarities and differences between dexmedetomidine–ketamine protocols and dexmedetomidine–opioid protocols?

Pharmacologically, the combination of an α2-agonist (xylazine, medetomidine, or dexmedetomidine) with ketamine is considered to be a true anesthetic combination, unlike the combination of an α2-agonist with an opioid (xylazine–butorphanol or dexmedetomidine–butorphanol), which is thought to provide only sedation and analgesia. Despite this pharmacologic distinction, similar degrees of sedation and analgesia have been documented with medetomidine–ketamine and medetomidine–butorphanol combinations in dogs. Our clinical observations suggest that a similar relationship exists between dexmedetomidine–ketamine and dexmedetomidine–butorphanol combinations.

One advantage of using dexmedetomidine–ketamine is that animals so treated tend to have higher heart rates than those receiving dexmedetomidine–opioid combinations. This is likely to be related to the fact that ketamine stimulates sympathetic tone, whereas opioids enhance parasympathetic tone synergistically with dexmedetomidine. Profound sinus bradycardia, first- and second-degree atrioventricular block, and sinus arrest are more likely in patients receiving dexmedetomidine–opioid combinations than dexmedetomidine in combination with a dissociative agent (e.g., ketamine, tiletamine–zolazepam). Blood pressure in dexmedetomidine–ketamine–treated patients may be higher than when dexmedetomidine–opioid combinations are used.

Clinically, we think that dexmedetomidine used in combination with either ketamine or an opioid provides a more consistent level of sedation than when the same dose of dexmedetomidine is used alone. This is in agreement with findings from a study in which medetomidine was incorporated into ketamine and opioid protocols.

Atipamezole reversal of the dexmedetomidine component of a dexmedetomidine–ketamine combination is characterized by a different response when compared with reversing the dexmedetomidine in a dexmedetomidine–opioid combination. If the dexmedetomidine component in the dexmedetomidine–ketamine combination is antagonized too early (less than 40 minutes after ketamine administration), the effect of the ketamine will predominate, leading to a dissociative recovery evidenced by headshaking, salivation, tongue flicking, and significant muscle tremors. This is particularly marked in dogs. In contrast, when used in combination with an opioid, dexmedetomidine can be reversed at any time with minimal impact on the quality of the animal’s recovery or time to recovery after atipamezole administration. After dexmedetomidine is reversed, the residual effect of opioids is usually limited to mild sedation and analgesia (with the intensity of the analgesia being dependent on the dose, nature of the opioid used, and time since administration).

Can buprenorphine be combined with dexmedetomidine in dogs and cats?

Yes, although it is important to understand that the onset of action and depth of sedation associated with this combination will not be as profound as that seen with some other dexmedetomidine–opioid combinations in dogs and cats. Buprenorphine is a partial µ-agonist and, as such, does not produce the same degree of analgesia as morphine or hydromorphone. Buprenorphine also has a ceiling effect. A key advantage of buprenorphine is that it has a longer duration of analgesic action (6 to 8 hours) than the other opioids.

We recommend that when using this combination, buprenorphine (20 µg/kg IM) be given 30 minutes before dexmedetomidine instead of administering the two drugs together. Buprenorphine has a very slow onset of action (30 to 40 minutes); giving it...
A 25-kg, 4-year-old Labrador retriever in good health was presented for dental cleaning and tooth extractions. Basic blood work was normal. The dog was premedicated with dexmedetomidine (0.23 ml IM) in combination with hydromorphone (0.05 mg/kg IM). The dexmedetomidine dose used was based on the values provided in Table 1 (preanesthetic/cooperative sedation, 125 µg/m² or 0.22 ml for a body weight of 25 kg = 4.31 µg/kg). The dexmedetomidine and hydromorphone were drawn up separately, mixed in the same syringe, and administered as a single IM injection. The dog appeared to be moderately sedated within 8 minutes after drug administration. When prompted, the dog was able to walk to the induction table 10 minutes after receiving the dexmedetomidine–hydromorphone preanesthetic combination.

Placement of an IV catheter was easily achieved after premedication with dexmedetomidine–hydromorphone. The dog was induced with propofol at 3 mg/kg IV given to effect, intubated, and then maintained on isoflurane. Notice that the dog was connected to an electrocardiograph to monitor cardiac rhythm and that the technician’s fingers were palpating the pedal artery pulse while the dog was being induced. Bradycardia (heart rate, 48 bpm) was noted while blood pressure remained within normal limits during the early stage of anesthesia maintenance. Bradycardia subsided as the dental procedure was started. Blood pressure was well maintained throughout the entire procedure.

Intubation was easily achieved, and the dog was maintained on isoflurane (1.25% to 1.75%) throughout the dental procedure. The dog also received a dose of carprofen (Rimadyl, Pfizer Animal Health) at 4.4 mg/kg SC prior to the dental cleaning and extraction. Balanced electrolyte fluid was administered IV at 10 ml/kg/hr for the duration of the procedure. As part of the patient’s pain management, a dental block using a lidocaine-bupivacaine combination was also administered before any teeth were extracted. A second dose of hydromorphone (0.05 mg/kg IM) was administered at the conclusion of anesthesia for additional postoperative pain relief.
before administering dexmedetomidine allows its peak sedative and analgesic effects to match those of dexmedetomidine, thereby providing optimal sedation and muscle relaxation. A recent study compared the restraint and sedation achieved using dexmedetomidine in combination with butorphanol (0.2 mg/kg IM), buprenorphine (0.015 mg/kg IM), or diazepam (0.4 mg/kg IV) in dogs undergoing hip radiographic examination and requiring hindlimb manipulation (hip-extended or stress radiographic views) for pelvic radiography. The investigators concluded that the overall quality of sedation in the dexmedetomidine–buprenorphine–sedated dogs was poor, and additional buprenorphine was required to complete the procedure. In contrast, dexmedetomidine–butorphanol was seen to induce excellent sedation with sufficient muscle relaxation to allow for the completion of the diagnostic procedure.

Can dexmedetomidine be substituted for medetomidine in the medetomidine–ketamine–butorphanol combination (“kitty magic”) in cats?

Dexmedetomidine is a scientifically based substitute for medetomidine. Although not FDA approved, dexmedetomidine can be administered by IM injection in combination with ketamine and butorphanol. This combination can be used to achieve varying degrees of sedation and analgesia as well as a surgical plane of anesthesia. This combination is also less likely to induce vomiting because of the rapid onset of anesthesia and depression of the vomiting center.

Table 6 reflects doses we have used for various procedures in the average cat (4.5 kg [10 lb]). The combination can be administered at one of three doses to achieve sedation/anesthesia and analgesia suitable for mild, moderate, and extremely painful or invasive procedures. The injection volumes of each of the three drugs in the combination range from 0.1 to 0.3 ml for a 4.5-kg cat.

Sedation/anesthesia occurs within 3 to 5 minutes when all three drugs are mixed together in the same syringe and administered as a single IM injection. Cats can be intubated after administration of the highest dose, and this dose allows for 40 to 45 minutes of surgical plane general anesthesia. IV administration of these IM doses will produce a deeper plane of anesthesia; however, the general recommendation is to halve the IM dose if the IV route is used.

A volume of atipamezole equal to that of the dexmedetomidine can be used for reversal after the procedure has been completed if indicated by patient management. Although atipamezole is frequently used to reverse sedation in cats, it is important to remember that atipamezole is not FDA approved for use in cats.

Atipamezole administration antagonizes both the sedative and analgesic effects of dexmedetomidine; additional analgesia must be provided for cats undergoing painful procedures. In our experience, postoperative pain management for the type of procedures associated with this drug combination can include an additional dose of butorphanol (0.2 mg/kg IM) or buprenorphine (15 to 20 µg/kg IM). The use of buprenorphine takes advantage of its longer duration of analgesic action relative to butorphanol and other commonly used opioids. Alternative analgesics include other types of opioids such as hydromorphone (0.05 to 0.1 mg/kg IM) or morphine (0.25 to 0.5 mg/kg IM) depending on preference and availability. Administration of these drugs is unlikely to be antagonized by previously administered butorphanol, which will most likely have been metabolized by the end of the procedure. NSAIDs such as carprofen or

<table>
<thead>
<tr>
<th>Species</th>
<th>Drug Dose</th>
<th>Drug Dose</th>
<th>Drug Dose</th>
<th>Drug Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dexmedetomidine (µg/kg)</td>
<td>Butorphanol (mg/kg)</td>
<td>Hydromorphone (mg/kg)</td>
<td>Morphine (mg/kg)</td>
</tr>
<tr>
<td>Dogs</td>
<td>5–10 IV</td>
<td>0.1–0.2 IV</td>
<td>0.03–0.05 IV</td>
<td>0.25–0.5 IV</td>
</tr>
<tr>
<td></td>
<td>15–20 IM</td>
<td>0.3–0.4 IM</td>
<td>0.05–0.1 IM</td>
<td>0.5–1 IM</td>
</tr>
<tr>
<td>Cats</td>
<td>15–25 IV</td>
<td>0.2–0.3 IV</td>
<td>0.03–0.05 IV</td>
<td>–</td>
</tr>
<tr>
<td></td>
<td>20–40 IM</td>
<td>0.3–0.4 IM</td>
<td>0.05–0.1 IM</td>
<td>–</td>
</tr>
</tbody>
</table>

*Note that dexmedetomidine dosages in this table are higher than the 125 µg/m² IM doses listed in Table 1.

†Note that buprenorphine has a slower onset than other opioids.
Meloxicam can be administered perioperatively for additional pain control.

Reported side effects associated with this combination are respiratory changes characterized by slow respiratory rate, an apneustic (intermittent) breathing pattern, hypoventilation, and apnea. If hypoventilation or apnea occurs, endotracheal intubation accompanied by assisted or controlled ventilation providing 100% oxygen using an anesthetic breathing circuit or Ambu bag is necessary to alleviate the respiratory depression. Respiratory depressive effects are typically noted within the first 5 to 10 minutes after drug administration. Hypothermia will occur, and body temperature should be properly maintained by using an exogenous heat source during both the procedure and the recovery period.

Can dexmedetomidine replace medetomidine in a tiletamine-zolazepam-medetomidine-butorphanol (TTD) protocol?

Yes, given that dexmedetomidine is the active component in medetomidine. The TKX (tiletamine-zolazepam [Telazol], ketamine, and xylazine) protocol was introduced in 1993 and has been widely used in cats, particularly in trap-and-neuter and shelter operations. Two drawbacks associated with this protocol are the limited analgesia it provides as a result of the absence of opioids and the fact that it is principally suitable only for use in cats because of the prolonged and erratic recovery associated with dissociative agents in dogs. These limitations led to a modification of the protocol by replacing xylazine with medetomidine and ketamine with butorphanol, creating the TTD protocol (tiletamine-zolazepam [Telazol], butorphanol [Torbugesic, Fort Dodge Animal Health], and medetomidine [Domitor]). The TTD combination has proved suitable for use in dogs and cats and has largely replaced the TKX combination. Similar to other medetomidine or dexmedetomidine anesthetic combinations, the TTD (or TTDex, see below) protocol is not FDA approved in either cats or dogs.

To incorporate dexmedetomidine into this protocol, practitioners can replace the medetomidine component with an equal volume of dexmedetomidine. Table 7 provides appropriate dosages for the different drug combinations comprising the Telazol-Torbugesic-Dexdomitor (TTDex) combination. Two approaches can be used to formulate this combination. The first involves drawing up each component separately based on the dosages listed in Table 7 and then mixing them in the same syringe immediately before drug administration. The second approach is to reconstitute the Telazol powder using 2.5 ml of butorphanol (10 mg/ml) and 2.5 ml of dexmedetomidine (Dexdomitor, 0.5 mg/ml) as diluents (in a similar fashion to TKX). Each milliliter

### Table 4

<table>
<thead>
<tr>
<th>Dexmedetomidine-Ketamine Combinations* in Dogs and Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug Dose</strong></td>
</tr>
<tr>
<td><strong>Species</strong></td>
</tr>
<tr>
<td><strong>Dexmedetomidine (µg/kg)</strong></td>
</tr>
<tr>
<td><strong>Ketamine (mg/kg)</strong></td>
</tr>
<tr>
<td><strong>Dogs</strong></td>
</tr>
<tr>
<td>5–10 IV</td>
</tr>
<tr>
<td>15–20 IM</td>
</tr>
<tr>
<td><strong>Cats</strong></td>
</tr>
<tr>
<td>15–25 IV</td>
</tr>
<tr>
<td>20–40 IM</td>
</tr>
</tbody>
</table>

*The combinations induce a surgical plane of anesthesia that lasts 20 minutes (low doses) to 40 minutes (high doses).

### Table 5

<table>
<thead>
<tr>
<th>Dexmedetomidine–Butorphanol–Midazolam and Dexmedetomidine–Butorphanol–Diazepam Combinations for Profound Sedation and Immobilization of Healthy Dogs and Cats*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug Dose</strong></td>
</tr>
<tr>
<td><strong>Species</strong></td>
</tr>
<tr>
<td><strong>Dexmedetomidine (µg/kg)</strong></td>
</tr>
<tr>
<td><strong>Butorphanol (mg/kg)</strong></td>
</tr>
<tr>
<td><strong>Midazolam or Diazepam (mg/kg)</strong></td>
</tr>
<tr>
<td><strong>Dogs</strong></td>
</tr>
<tr>
<td>2.5–5 IV</td>
</tr>
<tr>
<td>5–10 IM</td>
</tr>
<tr>
<td><strong>Cats</strong></td>
</tr>
<tr>
<td>10–15 IV</td>
</tr>
<tr>
<td>20–25 IM</td>
</tr>
</tbody>
</table>

*Butorphanol can be replaced with another opioid at the doses listed in Table 3.
of the reconstituted TTDex solution contains 100 mg of tiletamine–zolazepam, 5 mg of butorphanol, and 250 µg of dexmedetomidine. It is important to note that while these approaches to reconstituting and using Telazol in combination with other sedatives and analgesics have been used frequently,39–41 they represent an off-label, non–FDA-approved use of these drugs.

The TTDex combination, when administered at a dose of 0.01 ml/kg IM, can be used to achieve mild to moderate sedation in cats and dogs; this dose can also serve as a premedicant before IV propofol induction and isoflurane or sevoflurane maintenance anesthesia. For more profound sedation (e.g., such as that required to manipulate a dog’s limbs during an orthopedic examination), TTDex can be dosed at 0.02 ml/kg IM. To use TTDex for injectable anesthesia, a dose of 0.03 ml/kg IM is recommended; this dose provides a surgical plane of anesthesia for 35 to 45 minutes. For IV administration, the recommended IM doses should be halved in both dogs and cats; however, if a deeper plane of anesthesia is desired, the full IM dose can be given IV. With a dose of 0.03 ml/kg IM, dogs and cats will become laterally recumbent 5 to 8 minutes after injection and can be intubated and maintained on 100% oxygen only. If anesthesia needs to be extended, isoflurane or sevoflurane may be used.

Reversal of TTDex with atipamezole can be achieved once a procedure has been completed. The reversal with atipamezole in dogs and cats anesthetized with the TTDex combination should, however, be reserved for emergency situations only. Reversal of the dexmedetomidine component in this combination may be associated with a rough recovery characterized by dissociative-induced muscle rigidity and convulsion in dogs (atipamezole reverses only the dexmedetomidine-induced sedation and muscle relaxation, thereby allowing the tiletamine component of Telazol to become the dominant agent). Atipamezole reversal may not be as effective in cats if given within 30 to 40 minutes of TTDex administration, as the Telazol and butorphanol components will still be active. The volume of atipamezole used for reversal is half of the TTDex injection volume and should be given IM.

What precautions need to be taken when using dexmedetomidine alone or in drug combinations?

Appropriate patient selection is of paramount importance for the safe and effective use of dexmedetomidine as a sole agent or in combination. Dexmedetomidine should not be used in dogs or cats with severe cardiovascular disease, respiratory disorders, liver or kidney disease, diabetes, or in conditions of shock, severe debilitation, or stress due to extreme heat, cold, or fatigue.1,2

Cardiac and respiratory function should be monitored at 3- to 5-minute intervals in patients sedated with dexmedetomidine whether used alone or in combination protocols. As with all anesthetics, equipment for endotracheal intubation and ventilatory support should be available in the event of severe respiratory depression or apnea. Manual ventilation can easily be achieved using an anesthetic machine and breathing circuit or an Ambu bag. Enriched oxygen (100%) is advisable should hypoxemia occur.

### Table 6

Dexmedetomidine–Butorphanol–Ketamine (“Kitty Magic”) Combination in Cats*

<table>
<thead>
<tr>
<th>Drug Dose</th>
<th>Dexmedetomidine (0.5 mg/ml)</th>
<th>Butorphanol (10 mg/ml)</th>
<th>Ketamine (100 mg/ml)</th>
<th>Atipamezole† (5 mg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Profound sedation–analgesia</td>
<td>0.1 ml (11.1 µg/kg)</td>
<td>0.1 ml (0.22 mg/kg)</td>
<td>0.1 ml (2.2 mg/kg)</td>
<td>0.1 ml (111.1 µg/kg)</td>
</tr>
<tr>
<td>Castration or laceration repair</td>
<td>0.2 ml (22.2 µg/kg)</td>
<td>0.2 ml (0.44 mg/kg)</td>
<td>0.2 ml (4.4 mg/kg)</td>
<td>0.2 ml (222.2 µg/kg)</td>
</tr>
<tr>
<td>Ovariohysterectomy, onychectomy, abdominal procedures</td>
<td>0.3 ml (33.3 µg/kg)</td>
<td>0.3 ml (0.66 mg/kg)</td>
<td>0.3 ml (6.6 mg/kg)</td>
<td>0.3 ml (333.3 µg/kg)</td>
</tr>
</tbody>
</table>

*Based on a 4.5-kg (10-lb) cat. All drugs (except atipamezole) can be mixed in one syringe and administered as a single IM injection. If given IV, the drug doses in this combination should be halved; however, if a deeper plane of anesthesia is desired, the full IM dose can be given IV.
†If reversal is necessary for safe recovery.
Corneal dryness may occur during dexmedetomidine sedation. The patient’s eyes should be lubricated and protected with eye ointment. Body temperature may decrease after sedation or anesthesia with dexmedetomidine; therefore, sedated animals should be kept warm with an exogenous heat source during the procedure and the recovery period.12 Muscle tremors or twitching may occur during dexmedetomidine sedation and should be carefully differentiated from a light plane of sedation or extrapyramidal or seizure activity.

In older dogs and cats, it may be appropriate to decrease the dexmedetomidine dose. In very excited or agitated patients or any patient with elevated noradrenaline levels, dexmedetomidine, like medetomidine, may have variable efficacy. When dealing with these patients, it is recommended to allow them to calm down before administering dexmedetomidine or to consider using another drug technique as described above.

**Can dexmedetomidine be used in microdoses?**

Although not FDA approved for such use, microdoses of medetomidine have been used to facilitate induction of and recovery from anesthesia.30,31 A study investigating the effects of IV diazepam or microdose medetomidine on propofol-induced sedation in dogs found that the IV administration of 1 µg/kg of medetomidine 45 seconds before propofol induction in dogs resulted in a 38% reduction in the total propofol induction dose when compared with 0.4 mg/kg diazepam (36%) and propofol alone.30 The administration of medetomidine at 5 µg/kg IV in combination as part of a typical diazepam–ketamine (one-to-one volume-to-volume) induction protocol significantly improved muscle relaxation and extended the duration of sedation in dogs.31 In dogs and cats, medetomidine can be used at 1 to 2 µg/kg IV to rapidly smooth rough anesthetic recoveries; we are of the opinion that microdoses of dexmedetomidine at 0.5 to 1 µg/kg IV exert a similar dose-sparing effect on propofol induction and improve muscle relaxation. At 2 µg/kg IV, dexmedetomidine can be used to extend the duration of sedation associated with a diazepam–ketamine induction and to smooth rough anesthetic recoveries in these patients. Clinically, IM microdoses (3 to 4 µg/kg) of dexmedetomidine can also be used in dogs or cats to improve the effect of other IM sedative and anesthetic drugs when there is no IV access.

Microdoses of dexmedetomidine can also be used as an adjunct to isoflurane or sevoflurane to enhance the anesthetic effects, especially for procedures that are particularly noxious but not necessarily painful (e.g., ear flushing or retroflexive rhinoscopy). Animals undergoing these procedures frequently wake prematurely from inhalant anesthesia and require higher concentrations of inhalants to remain appropriately anesthetized. The administration of a microdose of dexmedetomidine (1 µg/kg IV) to these patients as an adjunct to the isoflurane or sevoflurane maintenance frequently leads to a more stable plane of anesthesia.

### Table 7

**Tiletamine–Zolazepam–Butorphanol–Dexmedetomidine (TTDex) Combination in Dogs and Cats**

<table>
<thead>
<tr>
<th>Desired Purpose</th>
<th>IM Dosages of Individual Components*</th>
<th>Volume (ml/kg IM) of Reconstituted TTDex†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premedication (mild to moderate sedation)</td>
<td>Tiletamine–zolazepam, 1 mg/kg Butorphanol, 0.05 mg/kg Dexmedetomidine, 2.5 µg/kg</td>
<td>0.01</td>
</tr>
<tr>
<td>Chemical restraint (profound sedation)</td>
<td>Tiletamine–zolazepam, 2 mg/kg Butorphanol, 0.1 mg/kg Dexmedetomidine, 5 µg/kg</td>
<td>0.02</td>
</tr>
<tr>
<td>Surgical plane of anesthesia</td>
<td>Tiletamine–zolazepam, 3 mg/kg Butorphanol, 0.15 mg/kg Dexmedetomidine, 7.5 µg/kg</td>
<td>0.03</td>
</tr>
</tbody>
</table>

*Each agent (tiletamine–zolazepam [Telazol, Fort Dodge Animal Health], butorphanol, and dexmedetomidine) may be drawn up separately according to the dosage listed in this table. If given IV, drug doses in this combination should be halved; however, if a deeper plane of anesthesia is desired, the full IM dose can be given IV.

†Alternately, Telazol powder can be reconstituted using 2.5 ml of butorphanol (10 mg/ml) and 2.5 ml of dexmedetomidine (Dexdomitor, 0.5 mg/ml) as diluents. If given IV, drug doses in this combination should be halved; however, if a deeper plane of anesthesia is desired, the full IM dose can be given IV.
Microdoses of dexmedetomidine appropriate for clinical use in dogs and cats can be made by diluting dexmedetomidine 10-fold (from the original concentration of 500 µg/ml) using sterile injection water or saline to yield a final concentration of 50 µg/ml.

How should dexmedetomidine-sedated animals be monitored?

As is the case with other sedative agents, patients receiving dexmedetomidine, either alone or in combination with other agents, should be properly monitored. Heart rate, respiratory rate, oxygen saturation of hemoglobin using pulse oximetry (Spo2), pulse quality, noninvasive blood pressure, electrocardiography, and body temperature are the main vital signs used to monitor cardiorespiratory performance of animals. Monitoring end-tidal carbon dioxide can also be useful. However, monitoring of Spo2 is often a challenge because of the dexmedetomidine-induced vasoconstriction, and the value calculated by pulse oximetry may not be accurate.

Improvements in the technology of cardiorespiratory monitors has allowed for improved and easier monitoring of α2-agonist-sedated animals. Older monitors frequently fail to provide accurate or reliable readings in medetomidine-sedated animals. At present, several monitors stand out as being relatively reliable in providing measurements of Spo2, electrocardiography, and blood pressure in dexmedetomidine-sedated animals. These monitors include Innomed-InnoCare-vet (Lumic International, Baltimore, MD), PC-VetGard+ (Vmed Technology, Mill Creek, WA), and Cardell (Sharn Veterinary, Tampa, FL).

How can a dexmedetomidine constant-rate infusion be used in dogs and cats?

Constant-rate infusion (CRI) is the approved use for dexmedetomidine in humans for sedation. Several studies have evaluated dexmedetomidine CRI in dogs. It has been demonstrated that dexmedetomidine CRIs at 0.5 and 3 µg/kg/hr (with a loading dose of 0.5 and 3 µg/kg IV, respectively) reduced isoflurane requirement (minimum alveolar concentrations) by 18% and 59%, respectively.29,45 In another study, dexmedetomidine CRI was used in dogs undergoing soft tissue or orthopedic surgeries. These dogs were given IV doses of 5 µg/kg of dexmedetomidine and 10 µg/kg of buprenorphine followed by propofol induction and isoflurane maintenance supplemented with dexmedetomidine CRI at 1 to 3 µg/kg/hr. These authors also concluded that a 5 µg/kg IV loading dose of dexmedetomidine followed by 1 µg/kg/hr CRI produced the most favorable results.

In a separate study, dogs were anesthetized and maintained on isoflurane or propofol CRI for a 2-hour period. After anesthetic induction, dexmedetomidine was initiated with a 1 µg/kg loading dose followed by dexmedetomidine CRI at 1 µg/kg/hr. Dexmedetomidine was then continued for 22 hours after recovery from general anesthesia. The authors concluded that dexmedetomidine CRI resulted in typical α2 hemodynamic changes (hypertension and reduction of heart rate) with minimal respiratory effects and appeared to be an efficacious adjunct during and after propofol or isoflurane anesthesia in healthy dogs.

Although not FDA approved for CRI, these studies demonstrate that dexmedetomidine CRI is a reliable and valuable adjunct to isoflurane anesthesia in maintaining surgical anesthesia in healthy dogs. Dexmedetomidine CRI can also be used in dogs anesthetized with isoflurane alone, propofol alone, or with opioid premedication followed by propofol induction and maintained on isoflurane. The advantages of using dexmedetomidine CRI as an anesthetic adjunct include (1) an inhalant anesthetic–sparing effect, (2) the provision of a stable plane of anesthesia during the intraoperative period, thereby minimizing “alpine anesthesia” response (i.e., peaks and valleys of deep to light to deep planes of anesthesia) to surgical stimulation, (3) the provision of additional analgesia, and (4) sparing of other intraoperative CRI analgesics (morphine–lidocaine–ketamine or fentanyl). The long-term effects of prolonged dexmedetomidine CRI have yet to be determined clinically.

Some researchers also describe the use of dexmedetomidine CRI in soft tissue and orthopedic cases. Healthy dogs and cats (American Society of Anesthesiologists’ class I and II) can be premedicated with one of the following:

- Dexmedetomidine alone (dogs, 5 to 8 µg/kg IM; cats, 10 to 20 µg/kg IM)
- Acepromazine (0.02 mg/kg IM) with hydromorphone (0.05 to 0.1 mg/kg IM)
- A combination of dexmedetomidine (5 µg/kg IM) with acepromazine (0.02 mg/kg IM) and hydromorphone (0.1 mg/kg IM)

All animals can then be induced with propofol (2 to 3 mg/kg to effect) and maintained on isoflurane for surgery. Immediately after induction, an IV bolus of dexmedetomidine (0.5 to 1 µg/kg) can be given as a loading dose, followed by 0.5 µg/kg/hr CRI for the entire procedure. A recent study demonstrated that
the intraoperative heart rate, blood pressure, Spo2, and end-tidal carbon dioxide were well maintained in these cases. The study involved a range of surgical procedures with varying intensity of nociceptive stimulation.

When using dexmedetomidine CRI as an anesthetic adjunct to inhalant anesthesia for surgical procedures, it is important to understand that while dexmedetomidine CRI does act to markedly spare inhalant anesthetics (isoflurane or sevoflurane) and other analgesic agents, it is only an adjunct to anesthesia. If one allows the inhalant anesthetic concentration to be turned down too low (<1% isoflurane or <2.5% sevoflurane), there is an increased probability that the surgical patient may awaken prematurely and without warning if subjected to an extremely painful stimulus. To avoid this situation, the inhalant anesthetic concentration should be appropriately adjusted and the animal’s plane of anesthesia closely monitored while it is receiving the dexmedetomidine CRI.

Further studies are required to objectively evaluate the concurrent administration of dexmedetomidine CRI with fentanyl or morphine–lidocaine–ketamine to better understand the dose sparing provided by “nano concentrations” (0.5 µg/kg/hr) of dexmedetomidine on these intra- and postoperative analgesics.

Can dexmedetomidine be administered to puppies and kittens?

The safety of dexmedetomidine has not been adequately investigated in dogs younger than 16 weeks or cats younger than 12 weeks.12 We do not recommend its use in very young puppies and kittens because these animals do not have a fully developed sympathetic or parasympathetic nervous system, nor do they have the myocardial mass of an adult. As such, these animals are less able to maintain cardiac output in the presence of bradycardia and may become hypotensive despite the peripheral vasoconstriction. That being said, there is no best way to anesthetize very young patients if inhalant anesthesia is not an option. Situations may present themselves in which dexmedetomidine or a dexmedetomidine combination becomes a consideration (e.g., rescue or spay–neuter clinics with feral or unowned dogs and cats, ease of injection, economics, lack of inhalant anesthesia). Reversal of dexmedetomidine with atipamezole may become advisable when surgical procedures are completed in these cases.

Conclusion

Dexmedetomidine is the active component of medetomidine. As such, at equal pharmacologic doses, the same indications for the clinical use of medetomidine apply to the use of dexmedetomidine in dogs and cats. The continued use of dexmedetomidine alone and in combination with other agents in daily practice will allow the full advantages and disadvantages of this drug and the various combinations discussed to become more fully elucidated.

References


