Effect of dexmedetomidine hydrochloride on tiletamine hydrochloride–zolazepam hydrochloride anesthesia in alpacas

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OBJECTIVE
To evaluate the effect of IM administration of a tiletamine hydrochloride–zolazepam hydrochloride (TZ) combination with either dexmedetomidine hydrochloride or saline (0.9% NaCl) solution (SS) on the motor response to claw clamping, selected cardiorespiratory variables, and quality of recovery from anesthesia in alpacas.

ANIMALS
5 adult sexually intact male alpacas.

PROCEDURES
Each alpaca was given the TZ combination (2 mg/kg) with dexmedetomidine (5 [D5], 10 [D10], 15 [D15], or 20 [D20] µg/kg) or SS IM at 1-week intervals (5 experiments); motor response to claw clamping was assessed, and characteristics of anesthesia, recovery from anesthesia, and selected cardiorespiratory variables were recorded.

RESULTS
Mean ± SEM duration of lack of motor response to claw clamping was longest when alpacas received treatments D15 (30.9 ± 5.9 minutes) and D20 (40.8 ± 5.9 minutes). Duration of lateral recumbency was significantly longer with dexmedetomidine administration. The longest time (81.3 ± 10.4 minutes) to standing was observed when alpacas received treatment D20. Following treatment SS, 4 alpacas moved in response to claw clamping at the 5-minute time point. Heart rate decreased from pretreatment values in all alpacas when dexmedetomidine was administered. Treatments D10, D15, and D20 decreased Pa_{O_2}, compared with treatment SS, during the first 15 minutes. During recovery, muscle stiffness and multiple efforts to regain a sternal position were observed in 3 SS-treated and 1 D5-treated alpacas; all other recoveries were graded as excellent.

CONCLUSIONS AND CLINICAL RELEVANCE
In TZ-anesthetized alpacas, dexmedetomidine (10, 15, and 20 µg/kg) administered IM increased the duration of lack of motor response to claw clamping, compared with the effect of SS. (*Am J Vet Res* 2016;77:1057–1063)

Among the South American camelids, alpacas (*Lama pacos*) and llamas (*Lama glama*) are both domesticated and popular species in North America. With the increase in the number of camelids as farm and pet animals, the need for veterinary care, including surgical and diagnostic procedures that necessitate anesthesia, has also increased. Acquiring venous access in South American camelids is relatively difficult[1]; thus, drug combinations, such as xylazine hydrochloride and ketamine hydrochloride, administered IM are commonly used as general anesthetic agents, especially for procedures of short duration.[2] Additionally, the use of xylazine, a first-generation α_2-adrenoreceptor agonist, in conjunction with a commercially available 1:1 mixture of tiletamine hydrochloride (a noncompetitive N-methyl-D-aspartate antagonist) and zolazepam hydrochloride (a benzodiazepine) as an anesthetic combination for camelids has been reported.[3] In that latter study,[3] xylazine administered with the TZ combination (2 mg/kg) increased the duration of immobilization in llamas in a dose-dependent manner; however, hypoxemia developed when greater doses of xylazine were used. In comparison with xylazine, dexmedetomidine hydrochloride is an α_2-adrenoreceptor agonist with high selectivity for α_2- versus α_1-adrenergic receptors (1,620:1, compared with 160:1 for xylazine).[4,5] Dexmedetomidine induces sedation and analgesia and provides anesthetic-sparing effects in humans and other animals.[6–8] To the authors' knowledge, there is

ABBREVIATIONS

DAP	Diastolic arterial blood pressure
MAP	Mean arterial blood pressure
SAP	Systolic arterial blood pressure
Sa_{O_2}	Estimated arterial oxygen saturation
SS	Saline (0.9% NaCl) solution
TZ	Tiletamine hydrochloride–zolazepam hydrochloride

no report on the use of dexmedetomidine with a TZ combination in South American camelids.

The aim of the study reported here was to evaluate the effect of IM administration of a TZ combination with either dexmedetomidine or SS on the motor response to claw clamping, selected cardiorespiratory variables, and quality of recovery from anesthesia in alpacas. It was hypothesized that TZ treatment alone would induce only a brief duration of lack of response to claw clamping, and that concurrent IM administration of dexmedetomidine would increase this duration in a dose-dependent manner.

Materials and Methods

Animals

Five sexually intact male alpacas (mean ± SD weight, 54.0 ± 6.5 kg) that were 2 to 4 years old were used in the study. The alpacas were determined to be in good health on the basis of history and results of a physical examination and were acclimated to the housing facility for approximately 14 days prior to the start of the study. The study was approved by the University of Tennessee Institutional Animal Care and Use Committee (protocol No. 2218-1013).

Study design

Each alpaca was used in 5 experiments, according to a Latin square design, with a minimum interval of 7 days between experiments. The treatments consisted of an injectable formulation of a TZ combination[a] (2 mg/kg) with either dexmedetomidine[b] at doses of 5 µg/kg (treatment D5), 10 µg/kg (treatment D10), 15 µg/kg (treatment D15), or 20 µg/kg (treatment D20) or treatment with SS (control treatment) administered as 1 injection IM. The TZ dose was reported as the sum of the tiletamine and zolazepam doses.

Anesthesia

Prior to each experiment, food and water were withheld from the alpacas for approximately 12 hours. The experiment was performed in a quiet room in which the animals were housed for approximately 1 hour prior to the start of each experiment. The doses of the TZ combination and dexmedetomidine were prepared separately and combined in a syringe immediately before administration. A 6-mL injectate volume was achieved by the addition of SS, and the preparation was injected into a semitendinosus or semimembranosus muscle. For the SS experiments, the TZ combination and SS were combined in a syringe to a volume of 6 mL immediately before administration. When the alpaca assumed sternal recumbency with its neck resting on the floor, it was positioned in left lateral recumbency on a padded surface.

Recording and monitoring

Baseline heart rate, respiratory rate, and rectal temperature were determined approximately 30 minutes before the start of each experiment. Heart rate was determined by auscultation, and respiratory rate was determined by observation of thoracic excursions, each over a period of 1 minute. After the animal was positioned in left lateral recumbency, recording of selected cardiorespiratory variables was started and continued, with the alpaca in this position, until recording of these variables was no longer tolerated by the animal. During recumbency, heart rate was monitored continuously with base-apex ECG leads.[c] The SAP, DAP, and MAP were measured with an oscillometric device[d] and recorded at 5-minute intervals. A pressure cuff of appropriate size (cuff width approx 40% of limb circumference [neonatal No. 4 or 5])[9] was placed over the metacarpal artery of the nondependent thoracic limb, and the limb was positioned with the cuff approximately at the level of the base of the heart. Respiratory rate was determined every 5 minutes. Cardiorespiratory data were collected immediately prior to delivery of the noxious stimulus. Rectal temperature was measured with a temperature probe.[c] The values for cardiorespiratory variables and rectal temperature collected during the first 30 minutes after alpacas were rolled into lateral recumbency following induction of anesthesia were analyzed. A sample of arterial blood (1 mL) for blood-gas analysis was collected percutaneously by means of an arterial sample collection kit[e] from a femoral artery at 5, 10, 15, and 20 minutes after attainment of lateral recumbency. Each sample was collected anaerobically and analyzed immediately with a handheld analyzer.[f]

The following characteristics of anesthesia and recovery from anesthesia were recorded: time to lateral recumbency (interval from injection to placement in lateral recumbency [in minutes]), duration of lateral recumbency (interval during which the animal remained in a lateral position [in minutes]), duration of lack of motor response to claw clamping (in minutes), time to standing (interval from injection to standing [in minutes]), and quality of recovery from anesthesia. The response to claw clamping with a 10-inch Vulsellum forceps[g] was assessed every 5 minutes after each alpaca was positioned in lateral recumbency, and the duration of the lack of motor response to claw clamping was recorded. The forceps was closed tightly to the first or second ratchet, depending on claw size, for 60 seconds, but was immediately released if any motor movement was observed. Motor movement was defined as any movement, either reflexive or purposeful, of the limbs or head. The claws were clamped in a rotating order, starting at the lateral claw of the right thoracic limb, and no claw was clamped on more than 2 occasions. The quality of recovery from anesthesia was evaluated on all occasions by the same investigator (RS) with a 3-point scale as follows: 1 = minimal struggling or paddling and standing on the first or second attempt, 2 = mild to moderate struggling and 3 attempts to stand, and 3 = moderate to excessive struggling or paddling during recovery and standing after 4 or more attempts.[10]

Statistical analysis

Statistical analyses were performed with commercially available statistical software.[h] The effect of treatment on duration of the lack of response to claw clamping was assessed with a generalized linear mixed model, specifying alpaca as the random effect and adjusting for time. Similar models were also used to investigate the effect of treatment on selected cardiorespiratory variables and rectal temperature. The cardiorespiratory variables investigated with this modeling approach were heart rate, respiratory rate, SAP, DAP, and MAP. For each of the cardiorespiratory variables, data collected during the first 30 minutes of each experiment were included in the model as dependent variables, and treatment and time were specified as fixed effects. Model assumption of normality of residuals was assessed with the Shapiro-Wilk test. Least squares means and their SEMs were computed for each dependent variable and compared across treatments. A 2-tailed value of $P < 0.05$ was considered significant for all comparisons. Because the analyses involved multiple comparisons, those were adjusted for by use of the Tukey method.

Results

Following each of the 5 treatments, all alpacas assumed sternal recumbency and tolerated being rolled into lateral recumbency. When the alpacas received treatments D15 and D20, the mean ± SEM duration of lack of motor response to claw clamping was the longest (30.9 ± 5.9 minutes and 40.8 ± 5.9 minutes, respectively). Following SS treatment, only 1 alpaca did not move in response to claw clamping at the 5-minute time point **(Table 1)**. Duration of lateral recumbency was longest for treatments D15 and D20. The time to standing was longest for treatment D20 (81.3 ± 10.4 minutes).

In comparison with baseline values, mean heart rate decreased significantly following all dexmedetomidine treatments; heart rate following each dexmedetomidine treatment was significantly less than that following treatment SS **(Table 2)**. Respiratory rate was less than the baseline value when alpacas received treatment D5. When alpacas received treatment with SS, respiratory rate was greater than values following each dexmedetomidine treatment. Rectal temperature increased significantly from baseline values following all treatments except treatment D5. Mean SAP following treatment D20 and MAP following treatment D15 were greater than for the respective values following treatment SS; mean DAP did not differ among treatments **(Table 3)**.

The Pa_{O_2} and Sa_{O_2} values when alpacas received treatment SS did not differ during the 20-minute measurement period. The Pa_{O_2} values for treatments D10, D15, and D20 were significantly less than those for SS treatment during the first 15 minutes, but there was no significant difference in Pa_{O_2} among treatments at the 20-minute time point (Table 3). At the 5-minute time point, Sa_{O_2} following each dexmedetomidine treatment was significantly less than that following treatment with SS. The Sa_{O_2} values following treatments D10 and D20 remained significantly less than those following treatment with SS for 10 minutes, but there was no significant difference in Sa_{O_2} among treatments at 15 and 20 minutes. Mean Pa_{CO_2} following treatment D20 was significantly greater than that following SS treatment at all time points. In analyzed blood samples, there were no differences in arterial pH, base excess, and bicarbonate concentration among treatments.

Recovery from anesthesia was scored as moderate (score, 2) for 3 alpacas following treatment SS and for 1 alpaca following treatment D5. These animals had moderate muscle stiffness and made ≥ 2 efforts to regain a sternal position. All other alpacas had a recovery score of 1 following each of the 5 experimental treatments. On completion of each experiment, all of the animals were able to walk normally; no immediate or delayed lameness or any residual effect of claw clamping was observed in the week following the experiments.

Table 1—Least squares mean ± SEM times to lateral recumbency, duration of lateral recumbency, time to standing, and duration of lack of motor response to claw clamping in 5 healthy adult alpacas that each received IM administration of a TZ combination (2 mg/kg) with dexmedetomidine at doses of 5, 10, 15, or 20 µg/kg (treatments D5, D10, D15, and D20, respectively) or SS (control) on 5 separate occasions at 1-week intervals.

	Treatment				
Variable	**SS**	**D5**	**D10**	**D15**	**D20**
Time to lateral recumbency (min)	5.6 ± 1.2[a,b]	8.3 ± 1.2[a]	6.8 ± 1.2[a,b]	4.8 ± 1.2[b]	4.6 ± 1.2[b]
Duration of lateral recumbency (min)	17.1 ± 6.1[a]	31.1 ± 6.1[b]	41.3 ± 6.2[b,c]	45.1 ± 6.1[c,d]	55.3 ± 6.1[d]
Time to standing (min)	33.4 ± 10.4[a]	53.2 ± 10.4[a,b]	63.8 ± 10.4[b]	62.8 ± 10.4[b]	81.3 ± 10.4[c]
Duration of lack of motor response to claw clamping (min)	0.0 ± 5.9[a]	11.9 ± 5.9[a]	26.4 ± 6.0[b]	30.9 ± 5.9[b,c]	40.8 ± 5.9[c]

Treatments consisted of an injectable formulation of a TZ combination mixed with a dose of dexmedetomidine or SS administered as 1 injection IM. The TZ dose is reported as the sum of the tiletamine hydrochloride and zolazepam hydrochloride doses. A 6-mL injectate volume was achieved by the addition of SS, and the preparation was injected into a semitendinosus or semimembranosus muscle. Time to lateral recumbency (interval from injection to placement in lateral recumbency [in minutes]), duration of lateral recumbency (interval during which the animal remained in a lateral position [in minutes]), duration of lack of motor response to claw clamping (in minutes), time to standing (interval from injection to standing [in minutes]), and quality of recovery from anesthesia were recorded. Motor movement was defined as any movement, either reflexive or purposeful, of the limbs or head.

[a–d]For a given variable, values with different superscript letters are significantly ($P < 0.05$) different.

Table 2—Least squares mean ± SEM heart rate, respiratory rate, and rectal temperature in the 5 healthy adult alpacas in Table 1 that each received IM administration of a TZ combination (2 mg/kg) with dexmedetomidine at doses of 5, 10, 15, or 20 µg/kg (treatments D5, D10, D15, and D20, respectively) or SS (control) on 5 separate occasions at 1-week intervals.

	Treatment				
Variable	**SS**	**D5**	**D10**	**D15**	**D20**
Heart rate (beats/min)					
Baseline	46 ± 3[a,*]	41 ± 3[a,*]	41 ± 3[a,*]	43 ± 3[a,*]	45 ± 3[a,*]
Treatment	46 ± 4[a,†]	31 ± 3[b,*]	31 ± 3[b,*]	30 ± 3[b,*]	35 ± 3[b,*]
Respiratory rate (breaths/min)					
Baseline	22 ± 2[a,*†]	23 ± 2[a,*†]	25 ± 2[a,*]	16 ± 2[a,†]	18 ± 2[a,*†]
Treatment	32 ± 3[b,†]	18 ± 3[b,*]	20 ± 3[a,*]	16 ± 3[a,*]	19 ± 3[a,*]
Rectal temperature (°C)					
Baseline	37.4 ± 0.3[a,*]	38.2 ± 0.3[a,†]	38.1 ± 0.3[a,*†]	37.9 ± 0.3[a,*†]	37.8 ± 0.3[a,†]
Treatment	38 ± 0.3[b,†]	38.2 ± 0.3[a,*†]	38.6 ± 0.3[b,‡]	38.2 ± 0.3[b,†]	38.3 ± 0.3[b,*]

Baseline heart rate, respiratory rate, and rectal temperature were determined approximately 30 minutes before the start of each experiment. Heart rate, respiratory rate, and rectal temperature values collected over the first 30 minutes after alpacas were rolled into lateral recumbency after induction of anesthesia were analyzed and are reported here.

[*†‡]Within a row, values with different superscript symbols are significantly ($P < 0.05$) different.

[a,b]For a given treatment, values of a given variable with different superscript letters are significantly ($P < 0.05$) different.

Table 3—Least squares mean ± SEM cardiorespiratory variables in the 5 healthy adult alpacas in Tables 1 and 2 that each received IM administration of a TZ combination (2 mg/kg) with dexmedetomidine at doses of 5, 10, 15, or 20 µg/kg (treatments D5, D10, D15, and D20, respectively) or SS (control) on 5 separate occasions at 1-week intervals.

	Treatment				
Variable	**SS**	**D5**	**D10**	**D15**	**D20**
SAP (mm Hg)	141 ± 8[a]	134 ± 8[a,b]	148 ± 8[a,b,c]	152 ± 7[a,b,c]	165 ± 8[c]
DAP (mm Hg)	99 ± 11[a]	97 ± 11[a]	117 ± 11[a]	127 ± 9[a]	112 ± 11[a]
MAP (mm Hg)	114 ± 8[a]	113 ± 8[a,b]	131 ± 8[a,b]	139 ± 7[b]	132 ± 8[a,b]
Sa_{O_2} (mm Hg)					
5 min	93.2 ± 9.3[a,*]	86.3 ± 10.3[b,*]	63.4 ± 10.7[b,*]	66.2 ± 9.3[b,*]	59.6 ± 9.3[b,*]
10 min	96.7 ± 9.5[a,*]	87.5 ± 10.3[a,b,*]	68.3 ± 10.3[b,*]	79.0 ± 9.3[a,b,†]	69.0 ± 9.3[b,†]
15 min	97.3 ± 10.1[a,*]	92.0 ± 10.3[a,*]	86.3 ± 10.3[a,†]	89.4 ± 9.3[a,‡]	74.0 ± 9.5[a,†]
20 min	96.7 ± 11.3[a,*]	92.3 ± 10.3[a,*]	86.8 ± 10.3[a,†]	92.2 ± 9.3[a,‡]	75.1 ± 9.7[a,†]
Pa_{O_2} (mm Hg)					
5 min	77.6 ± 9.2[a,*]	57.4 ± 10.2[a,b,*]	36.5 ± 10.7[b,*]	46.8 ± 9.2[b,*]	39.4 ± 9.2[b,*]
10 min	86.6 ± 9.2[a,*]	64.4 ± 10.2[a,b,*]	41.6 ± 10.2[b,*]	60.6 ± 9.2[b,†]	54.6 ± 9.2[b,†]
15 min	88.8 ± 10.0[a,*]	72.4 ± 10.2[a,b,*]	59.9 ± 10.2[b,†]	67.4 ± 9.2[b,†]	65.2 ± 9.5[b,†‡]
20 min	86.3 ± 11.5[a,*]	72.6 ± 10.2[a,*]	59.6 ± 10.2[a,†]	73.4 ± 9.2[a,†]	72.6 ± 9.7[a,‡]
Pa_{CO_2} (mm Hg)					
5 min	44.5 ± 1.9[a,*]	45.4 ± 2.1[a,b*]	49.6 ± 2.3[a,b,*]	50.2 ± 1.9[a,b,*]	50.4 ± 1.9[b,*]
10 min	43.0 ± 1.9[a,*]	44.0 ± 2.1[a,b,*]	49.8 ± 2.1[b,*]	48.0 ± 1.9[a,b,*]	49.1 ± 1.9[b,*]
15 min	41.3 ± 2.3[a,*]	44.6 ± 2.1[a,b,*]	46.0 ± 2.1[a,b,*]	47.7 ± 1.9[b,*]	47.6 ± 2.0[b,*]
20 min	38.7 ± 2.8[a,*]	42.6 ± 2.1[a,b,*]	45.8 ± 2.1[b,*]	47.0 ± 1.9[a,b,*]	45.6 ± 2.1[b,*]

The SAP, DAP, and MAP were measured with an oscillometric device and recorded at 5-minute intervals. Blood pressure values collected over the first 30 minutes after alpacas were rolled into lateral recumbency after induction of anesthesia were analyzed and are reported here. Cardiorespiratory data were collected immediately prior to delivery of the noxious stimulus. A sample of arterial blood (1 mL) for blood-gas analysis was collected percutaneously by means of an arterial sample collection kit from a femoral artery at 5, 10, 15, and 20 minutes after attainment of lateral recumbency. Each sample was collected anaerobically and analyzed immediately with a handheld analyzer.

[*†‡]For a given treatment, values of a given variable with different superscript symbols are significantly ($P < 0.05$) different.

[a–c]Within a row, values with different superscript letters are significantly ($P < 0.05$) different.

See Table 1 for key.

Discussion

In the present study, the administration of a TZ combination (2 mg/kg) IM without the addition of dexmedetomidine allowed all 5 alpacas to be placed in lateral recumbency, but only 1 alpaca had a lack

of motor response to claw clamping. A lack of motor response to claw clamping was considered by the authors to represent a surgical plane of anesthesia. Concurrent IM administration of dexmedetomidine at a dose of 10, 15, or 20 µg/kg significantly prolonged the duration of the TZ-induced lack of motor response to claw clamping.

The dose of the TZ combination was chosen on the basis of findings of previous studies[3,10] in llamas in which TZ (2 mg/kg) induced recumbency but only a short period of immobilization in response to noxious stimulation. In the present study, the TZ combination alone prevented movement in response to stimulation in only 1 alpaca, and that occurred at 5 minutes after administration. This finding was consistent with the effect of a TZ combination, at the same dose, in llamas.[3] This finding confirmed that a dose of 2 mg of TZ/kg is unsuitable as a sole anesthetic agent for surgical procedures in South American camelids.

Concurrent administration of α_2-adrenoreceptor agonists has been reported to potentiate the anesthetic effects of TZ in llamas,[3] sheep,[11] rats,[12] and pigs.[13] The doses of dexmedetomidine used in the present study were based on findings of a pilot study in alpacas in which doses of 30 and 40 µg/kg were associated with severe respiratory depression and bradycardia when administered with a TZ combination (2 mg/kg). Thus, the maximum dose of dexmedetomidine evaluated in the present study was 20 µg/kg. A range of dexmedetomidine doses was assessed to determine whether a dose-response effect exists for the interaction of dexmedetomidine and TZ in alpacas, as has been established for the interaction of xylazine and TZ in llamas.[3] In the present study, coadministration of dexmedetomidine at a dose of 5 µg/kg with the TZ combination (2 mg/kg) did not significantly increase the duration of the lack of motor response to claw clamping, compared with the effect of SS treatment (control treatment). On the basis of this finding, the authors conclude that dexmedetomidine administered IM at a dose of 5 µg/kg with a TZ combination (2 mg/kg) does not provide a surgical plane of anesthesia in alpacas; however, that dexmedetomidine-TZ treatment did induce recumbency and may be suitable for brief periods of restraint for non-noxious procedures. Concurrent administration of dexmedetomidine at 10, 15, or 20 µg/kg increased the duration of lack of response to claw clamping, but the statistical comparison of the results did not indicate a dose-dependent effect, which may be a reflection of the small sample size (type II error). A previous study[3] revealed that xylazine had a dose-dependent effect on the duration of immobilization when used in conjunction with a TZ combination in llamas.

In the alpacas in the present study, heart rate decreased following all dexmedetomidine treatments, compared with the respective baseline values and findings following SS treatment. This decrease in heart rate was consistent with the reported effects of α_2-adrenoreceptor agonists and is caused by centrally mediated sympatholysis or transient peripheral vasoconstriction.[14] Bradycardia has also been associated with the administration of the less selective α_2-adrenoreceptor agonist xylazine in TZ-treated llamas.[3]

Overall, there was little effect of dexmedetomidine on arterial blood pressure in the study alpacas, which was consistent with the effects of xylazine when used in conjunction with a TZ combination (2 mg/kg) in llamas.[3] Dexmedetomidine, when administered IV, has a biphasic effect on blood pressure involving a brief increase, which is attributed to stimulation of peripheral $\alpha_{2\text{-B}}$ adrenergic receptors on vascular smooth muscle, followed by a dose-dependent decrease.[4] In the previous studies and present study, the lack of significant difference in blood pressure in llamas and alpacas when they receive treatment with SS or dexmedetomidine may be attributable to the fact that the α_2-adrenoreceptor agonists were administered IM.

In the present study, respiratory rate increased from baseline when alpacas were given the TZ combination with SS treatment, which may be the reflection of a lighter plane of anesthesia, compared with that achieved with the dexmedetomidine treatments. The addition of dexmedetomidine was associated with a small but significant decrease in respiratory rate only when alpacas received treatment D5. Although $Paco_2$ following treatment D20 was greater than that following treatment with SS, the $Paco_2$ values following each treatment remained < 60 mm Hg, which did not indicate that clinically important ventilatory compromise had been induced by any of the study treatments. The observed effects of dexmedetomidine on $Paco_2$ were consistent with the reported effects of xylazine when administered with a TZ combination in llamas.[3]

Significant changes in Pao_2 were detected in the alpacas of the present study, but owing to the experimental design, comparison of posttreatment blood gas data with baseline values was not possible. However, there is no reason to assume that baseline blood gas values were abnormal because these animals were healthy, and the goal was to compare the effect of treatments on blood gas values. Low Pao_2 values were evident at 5 minutes after administration of all treatments, and this decrease was more pronounced when alpacas received dexmedetomidine (Pao_2 values were ≤ 60 mm Hg after 5 minutes of recumbency). Low Pao_2 values were also recorded when a TZ combination was administered in conjunction with acepromazine or butorphanol[10] or xylazine[3] in llamas. Mechanisms responsible for decreased Pao_2 during anesthesia and recumbency include hypoventilation, ventilation-perfusion mismatching, and intrapulmonary shunting from perfusion of nonventilated areas of the lungs.[15,16] Despite low Pao_2 values when alpacas received dexmedetomidine in the present study, clinical signs of hypoxemia (eg, cyanosis and tachypnea) were not noted during any experiment. Although the lack of tachypnea in these animals may have been a

result of the respiratory depressant effect of anesthetics, it was more likely that the absence of the clinical signs of hypoxemia was due to the tolerance of New World camelids for low oxygen partial pressures as an evolutionary adaptation to high-altitude living.[17–20] A potential limitation in the interpretation of the blood gas results in the present study was that the handheld analyzer used for blood gas analysis was not validated for use in New World camelids and the reported values for Sa_{O_2} were, therefore, considered an estimation. Nevertheless, the use of this handheld analyzer in other camelid studies[3,10] has been reported.

Recovery from anesthesia was considered smooth and uncomplicated when the study alpacas received dexmedetomidine at the doses > 5 µg/kg. Although the investigators were not blinded to the treatments, only 1 investigator (RS) evaluated and scored the quality of the recoveries to reduce any potential interobserver variability. In a previous study,[i] administration of a TZ combination at a dose of 4.4 mg/kg resulted in muscle tremors, rigidity, and hyperthermia during recovery from anesthesia in 1 of 4 llamas. No muscle rigidity and tremor were observed during the recovery phase in the alpacas of the present study, which may be a result of administration of a comparatively lower dose of the TZ combination.

In the alpacas of the present study, rectal temperature increased from baseline during the 30-minute period after drug administration for all treatments except D5, despite the fact that the ambient temperature remained constant at approximately 21°C. The increase in rectal temperature was not clinically important; however, an increase in body temperature of 1.8°C (3.2°F) over a 25-minute period was reported when a TZ combination (4 mg/kg) was used as the sole anesthetic agent in llamas.[i] In contrast, no significant increase in body temperature was reported when a TZ combination (2 mg/kg) was administered with xylazine[3] or butorphanol,[10] under the same conditions as those in the present study. The increase in body temperature in TZ-treated llamas seems to be primarily related to an increase in muscle activity and rigidity, especially when the drug combination is used alone or at higher doses[i]; however, no obvious signs of increased muscle activity were noted during the 30-minute period of recumbency in which rectal temperature was recorded for alpacas in the present study. Nevertheless, despite the lack of observable signs, it was likely that the change in rectal temperature was the result of an increase in basal metabolism because of an increase in muscle activity.

The present study revealed the clinical efficacy of the TZ combination (2 mg/kg) with dexmedetomidine at doses of 10, 15, or 20 µg/kg in providing a lack of motor response to claw clamping, indicative of a surgical plane of anesthesia, for a period of 20 to 40 minutes (depending on the dose) in alpacas. Intramuscular administration of the TZ combination (2 mg/kg) with dexmedetomidine at a dose of 5 µg/kg induced only a brief period of lack of response to claw clamping. Although transient hypoxemia was associated with dexmedetomidine administration, the authors do not consider this to be clinically important in healthy alpacas; however, oxygen supplementation is recommended for sick or debilitated animals.

Acknowledgments

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Footnotes

a. Telazol, Zoetis, Florham Park, NJ.
b. Dexdomitor, Zoetis, Florham Park, NJ.
c. 1100 Patient Monitor, Criticare Systems, Waukesha, Wis.
d. Dinamap Veterinary Blood Pressure Monitor 8300, Critikon, Tampa, Fla.
e. Pro-Vent arterial blood sampling kit, Smiths Medical, Keene, NH.
f. i-STAT portable clinical analyzer, Heska Corp, Loveland, Colo.
g. Miltex, Lake Success, NY.
h. SAS, version 9.3 TS Level 1M2, SAS Institute Inc, Cary, NC.
i. Klein L, Tomasic M, Olson K. Evaluation of telazol in llamas (abstr), in *Proceedings*. Annu Meet Am Coll Vet Anesth 1989;23.

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