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USE OF THIAFENTANIL–MEDETOMIDINE FOR THE INDUCTION OF ANESTHESIA IN EMUS (*DROMAIUS NOVAEHOLLANDIAE*) WITHIN A WILD ANIMAL PARK

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Abstract: Fifteen adult emu (*Dromaius novaehollandiae*) anesthetic events were successfully undertaken with the use of thiafentanil oxalate (A3080) 0.175 mg/kg i.m. (SD 0.026) and medetomidine 0.092 mg/kg i.m. (SD 0.009) via remote injection. Following induction, the birds were transported to the clinic, where a venous blood gas sample was taken for analysis, which indicated a respiratory acidosis, with a mean arterial pCO_2 of 54.46 mmHg (SD 9.31) and venous pH of 7.135 (SD 0.11), most likely due to moderate bradypnoea. Atipamezole 0.2 mg/kg i.v. (SD 0.02) was administered, immediately followed by orotracheal intubation initiating 2–3% isoflurane with 2 L/min oxygen flow. Parameters evaluated during anesthesia included heart rate, respiratory rate, anesthetic depth, and electrocardiogram readings. Physical exams plus any required procedures were performed in addition to venous blood samples for biochemistry and full blood counts. The birds were then recovered in a crate padded with grass hay with administration of 8.75 mg/kg (SD 1.36) naltrexone (50 mg/mg A3080) administered in equal doses i.v. and i.m. along with 5 mg midazolam i.m. to reduce excitement. Emus were placed in a lateral position and given 4 L/min oxygen via the endotracheal tube, until movement of the head and neck necessitated extubation. Recovery was rapid and smooth in each case with a mean time of 3.1 min from antagonist administration to sternal recovery. On the basis of rapid, smooth, and successful inductions and recoveries, the described dosage of thiafentanil and medetomidine, with administration of midazolam prior to recovery, is recommended for immobilization of adult emus. Due to evidence of respiratory acidosis and bradypnoea, careful monitoring should be instituted throughout and oxygen provision recommended from initial contact.

Key words: anesthesia, *Dromaius novaehollandiae*, emu, medetomidine, midazolam, thiafentanil.

INTRODUCTION

Ratites are notoriously difficult to anesthetize, often providing a challenge to those present. They can kick with power, thrash, or scratch using legs and wings, and thus are dangerous to people working around them.[4,7,18] They also pose a danger to themselves should excess struggling or excitement occur, leading to self-trauma or more serious injuries such as fractured limbs.

Remote injection systems are essential tools to administer drugs to semiwild animals. Smaller darts mean lower weight and faster injection times and so offer an increased percentage of successful drug administration with a reduced possibility of causing severe trauma to the target animal. Repeated remote injections also have an increased risk and can cause extended excitement.[14]

Previously described protocols for anesthesia of emus involve the use of ketamine with xylazine, or intravenous tiletamine–zolazepam either alone or in combination with xylazine and butorphanol.[7,11,12,15] The lack of an ability to fully antagonize these induction drugs, coupled with a requirement for intravenous administration or multiple injections, often makes these impractical in semiwild birds.

The use of thiafentanil oxalate, medetomidine, and ketamine, is a safe and reliable protocol described in a number of species including roan antelope (*Hippotragus equines*), gemsbok (*Oryx gazella*), axis deer (*Axis axis*), and nyala (*Tragelaphus angasi*).[3,4,8,10,25] Thiafentanil is a synthetic opioid with a lower potency and shorter duration of action[4] with faster induction time in elk[16] than carfentanil, another synthetic opioid. Carfentanil and etorphine have been effectively used in ostriches (*Struthio camelus*),[12,19,22] although there exist reports of excitement during induction with some fatalities.[6,21] There are no reported data on the usage of thiafentanil in avian species. Naltrexone is an antagonist against thiafentanil oxalate, with published dosages ranging from 30 mg/mg thiafentanil[7] to 100mg/mg thiafentanil.[26]

Medetomidine is a selective and specific α_2-adrenoreceptor agonist[1,30] that is more potent at both central and peripheral α_2-adrenoreceptors than related drugs such as the commonly used

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xylazine or detomidine,[24] and has previous reported usage in ratites.[13,31] Atipamezole is a highly selective α_2-adrenoreceptor antagonist against medetomidine, shown in a variety of species.[1,23] Dosages of atipamezole at 2.5 and 5 times medetomidine for antagonism of effects have been shown to be effective in two avian species, namely, domestic pigeons (*Columba livia*) and yellow-crowned Amazon parrots (*Amazona ochrocephala ochrocephala*).[23] Midazolam is a benzodiazepine that can reduce anxiety during either anesthetic induction or recovery,[6,20] and thus was used in this study in an attempt to reduce thrashing during recovery, a problem encountered in alternative ratite anesthesia protocols.[3,8,27] Both medetomidine and thiafentanil are known to cause both respiratory and cardiac depression,[17,23] but it was hypothesized that the high potency and rapid onset of action may result in a smoother induction with minimal excitatory phase and the ability to use low-volume remote injection systems, and be fully antagonized.

MATERIALS AND METHODS

The study involved three female and five male emus, residents of a mixed-species drive-through exhibit, weighing from 33 to 45 kg and aged from 5 to 16 yr old. Fifteen anesthetic events were used for the study, undertaken for physical examination, required procedures, or euthanasia in one case of severe chronic lameness. No birds with clinical signs of systemic illness, or with any indication that they may be an increased anesthetic risk, were used. Because of the drive-through management of the animals, starvation prior to anesthesia was not done.

Each animal was estimated at 45 kg prior to anesthesia, and 13 birds were administered with 7 mg thiafentanil oxalate (A3080, 10 mg/ml, ZooPharm Inc., Laramie, Wyoming 82070, USA) and 3.6 mg medetomodine hydrochloride (medetomidine, 20 mg/ml, ZooPharm), an estimated dose of 0.156 mg/kg thiafentanil and 0.08 mg/kg medetomidine. Two of these emus required 200 mg ketamine hydrochloride (Ketaset®, 100 mg/ml, Fort Dodge Animal Health, Iowa, 50501, USA), which was hand injected into the thigh musculature. The remaining two birds were given 5.5 mg thiafentanil with 3.6 mg medetomidine and one of these required an additional 100 mg ketamine, again hand injected. Ratings, on a scale of 0–5 with 5 being extremely strenuous and 0 being minimal movement, were given for the initial pursuit of the animal and for activity of the bird postdarting. This score took any activity into account, including thrashing, running, or other excitatory phase reactions. Times of events during the procedure were kept throughout, with darting time (T_d), initial signs, sternal recumbency, and lateral recumbency recorded. Initial contact, or T_0, then became the starting point for recording the following events: blood gas sampling, isoflurane commencement, atipamezole administration, isoflurane discontinuation, naltrexone administration, extubation time, sternal recovery, and standing recovery. An induction event was considered successful when the bird achieved lateral recumbency, with the head down, and could be handled and transported safely.

The induction drugs were given in the lateral thigh muscles with the use of a 1 ml, gel collar, three-quarter-in.-needle Type C Pneudart (Pneu-Dart Inc., Williamsport, Pennsylvania 17701, USA) utilizing a CO_2 pistol and barrel (Daninject, North America, Fort Collins, Colorado 80527, USA). Once the emus achieved lateral recumbency, leg ropes were applied, heart rate and respiratory rate were recorded, and plane of anesthesia assessed. A net stretcher was used to load the birds and transport them to the clinic, where a right jugular venous blood sample was taken for immediate IStat analysis (HESKA® Corporation, Loveland, Colorado 80538, USA), using the matching CG4+ (HESKA) cartridge, to assess blood gas and related values. In addition to this, a venous blood gas sample was taken from a fully conscious semitame emu for comparison to the samples from anesthetized birds, because of a lack of published data on emu venous blood gas parameters.

Following this a dose of 0.175 mg/kg atipamezole (Antisedan®, 5 mg/ml, Pfizer Animal Health, New York, New York 10017, USA) based on the estimated weight of 45 kg, was administered intravenously and an 8–9 mm endotracheal tube placed. This procedure was done in an effort to lower the combined cardiorespiratory depressive effects of isoflurane, medetomidine, and thiafentanil. Oxygen at 2 L/min and 2–3% isoflurane (Isoflurane, Vet One, Meridian, Idaho 83680, USA) were then supplied via a parallel breathing system. Respiratory and heart rate were monitored throughout the procedures, with the use of a three-lead electrocardiogram (Passport XG, Datascope® Corporation, Fairfield, New Jersey 07004, USA), auscultation, and visually watching movements of both breathing and cardiac cycles. The electrocardiogram leads were attached to the bird via crocodile clips attached in each axilla and immediately caudal to the keel with 70% isopropyl alcohol (isopropyl alcohol, 70%, Vet

One) on the skin in order to aid conduction. Respiration and heart rate were recorded every 5 min, from initial contact to extubation. In addition to this corneal reflex, muscle relaxation and jaw tone were monitored. Pulse oximetry and indirect blood pressure monitoring with cuff placement proximal to the tarsus were largely unsuccessful. In the event of any manual assistance with respiration being required, a dressing was placed around the caudal cervical area to prevent expansion of the trachea where the cartilaginous support is absent.[6]

Once physical examination and any required procedures were completed, isoflurane was discontinued, the patients were moved to a straw-bedded recovery crate and administered naltrexone hydrochloride (naltrexone, 50 mg/ml, ZooPharm) at 50 times the milligram dosage of thiafentanil, half of which was given intravenously and half intramuscularly. A 5-mg dose of midazolam hydrochloride (midazolam, 5 mg/ml, Baxter Healthcare Corporation, Deerfield, Illinois 60015, USA) i.m. was also given to the birds at this stage. Oxygen at 4 L/min was supplied via the orotracheal tube, and maintained until resistance to the tube was displayed by the birds by swallowing or excess head movement. At this point, the emus were extubated and left to recover inside the crate, while monitoring their response and keeping outside disturbances to a minimum. Times were recorded for sternal and standing recovery inside the crate. Release into the exhibit was done within 1–3 hr, and regular behavioral observations performed over the subsequent 72 hr.

A two-tailed *t*-test assuming unequal variance (Microsoft® Office Excel® 2007, Microsoft Corporation, Redmond, Washington 98052, USA) was used to compare differences in pH, pCO_2, heart rate, respiratory rate, and recovery times between birds requiring ketamine and those without, as well as a one-tailed variations in heart rate and respiratory rate following medetomidine antagonism. For each variable, $P < 0.05$ was considered statistically significant. Pearson's product-moment correlation coefficient was used to determine the degree of correlation between induction dosages and arterial pH, pCO_2, mean heart rate and mean respiratory rate. This statistical method was also utilized in the evaluation of procedure length and recovery times.

RESULTS

Pursuit of the animals was scored at a low level, with pursuit graded at a mean of 0.3 (SD 0.5). The emus also displayed a low level of activity after T_d, with a mean of 1.3 (SD 0.9). They generally progressed from mild ataxia, to walking backwards slowly, falling into sternal recumbency and rolling into lateral.

Table 1. Times of events after remote injection of adult emus (*Dromaius novaehollandiae*) with thiafentanil/medetomidine.

Event	*n*	Mean (minutes after T_d)	Range	SD
Initial signs	15	2.6	1–5	1.2
Sternal recumbency	15	4.7	2–9	2.3
Lateral recumbency	14[a]	7.0	2–15	3.9
Initial contact (T_0)	15	9.9	5–16	3.6

[a] $n=14$ because one subject was held up by an object in the field and was physically placed into lateral recumbency.

A light–medium plane of anesthesia was achieved with the initial induction dose in each of the 15 animals, with three exceptions. These animals required a hand injection of 100–200 mg ketamine i.m. after achieving lateral recumbency, because of excess limb and head movements.

The mean times to sternal and lateral recumbency post darting were 4.7 min (SD 2.3) and 7.0 minutes (SD 3.9), respectively, with initial contact, or T_0 at 9.9 min (SD 3.6) (Table 1). Venous blood samples taken for blood gas analysis, shown in Table 2, were taken prior to administration of atipamezole, after the birds were transported to the clinic, at a mean of 9.8 min (SD 2.7) post T_0, These, as shown in Table 2, displayed a significant respiratory acidosis, with a mean arterial pCO_2 of 54.46 mmHg (SD 9.31) and a venous pH of 7.135 (SD 0.11). Correlation coefficient values for arterial pH and arterial pCO_2 at -0.043 and 0.126, respectively, suggest minimal correlation between the parameters and dosage of induction drugs. No significant difference, at $P < 0.05$, was found between the pH and pCO_2 of groups administered with ketamine and those without ketamine.

Heart-rate mean was 63.9 (SD 9.6), which remained relatively constant throughout each procedure and a mean respiratory rate of 7.5 (SD 2.7). Two of the birds had respiratory rates that fell to 1–2 breaths per minute and were provided with positive pressure ventilation at 6–8 breaths per minute. Pearson product-moment correlation coefficients, with values of 0.123 and 0.073, were calculated for induction drug dosage

Table 2. Venous blood gas results taken from 14[a] anesthetized emus (*Dromaius novaehollandiae*) with the use of a CG4+ IStat cartridge, with reference values from other species[2] and one fully conscious emu.

Parameter	Mean	Range	SD	Fully conscious	Adult ostrich values[2]
Lactate (mmol/L)	8.35	3.81–16.26	3.60	0.96	
tCO_2 (mmol/L)	29.8	21–50	7.27	31	23.5–27.9
pH	7.135	6.931–7.329	0.11	7.436	7.408–7.420
pCO_2 (mmHg)	81.1	57–119.2	16.28	43.8	34.5–47.3
pO_2 (mmHg)	29.6	18–43	7.37	54	
HCO_3 (mmol/L)	27.6	18.7–48.6	7.47	29.5	22.5–26.7
Base Excess (mmol/L)	−1.5	−14 to 23	8.90	5	2.2
sO_2%	36.7	21–60	11.02	89	
Art pH (Calculated)[b]	6.885	6.700–7.082	0.11	7.185	
Art pCO_2 (Calculated)[c]	54.46	40.3–75.9	9.31	32.79	
Art HCO_3 (Calculated)[d]	23.49	16.3–41.6	6.31	25.47	

[a] $n=14$, as one emu was euthanized.
[b] Arterial pH calculated from venous result using 0.961 (venous pH) + 0.039.
[c] Arterial pCO_2 calculated from venous result using 0.572 (venous pCO_2) + 7.735.
[d] Arterial HCO_3 calculated from venous result using 0.845 (venous HCO_3) + 0.538.

against heart rate and respiratory rate, respectively. There was no significant difference, at $P < 0.05$, in heart rate or respiratory rate between birds administered with ketamine and those without ketamine. Statistical evaluation of heart rate immediately prior and post atipamezole administration indicated a significant difference at $P < 0.05$. This difference is shown in Figure 1, with a clear increase in heart rate between 15 and 20 min, with values of 62.9 (SD 9.5) and 74.2 (SD 19.8), coinciding with the mean atipamezole administration time of 14.9 min (SD 5.8). Respiratory rate evaluation at the same time points was not significantly different at $P < 0.05$, although it did increase from 6.9 (SD 2.85) to 8.2 (SD 3.17). With regards to the duration of anesthesia provided by the induction drugs alone, the longest time period from T_0 to beginning isoflurane administration was 19 min, or 30 min post T_d, and the plane of anesthesia was suitable for handling and monitoring at this point.

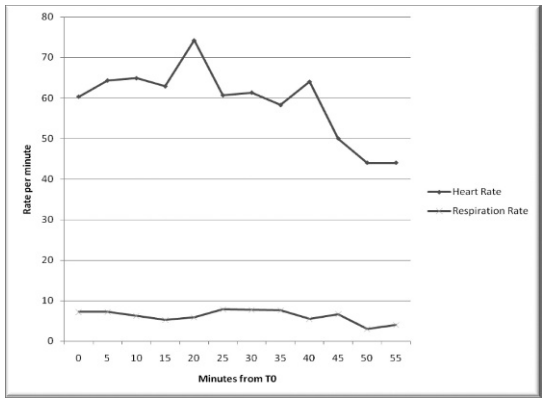

Figure 1. Mean heart rate and respiratory rate versus time during anesthesia in 15 emus. Atipamezole administered at a mean of 14.9 min (SD 5.8) after initial contact (T_0).

Recovery times are summarized in Table 3, which shows the timings for each stage of recovery. The mean time from administration of the naltrexone to sternal recumbency was 3.1 min (SD 2.14), and to standing was 7.8 min (SD 5.28). The birds that required ketamine to aid induction showed increased recovery times, with a mean of 4.7 min (SD 2.52) to sternal with ketamine compared to 2.7 min (SD 1.95) without. This difference was, however, not significant at $P < 0.05$.

The data suggested a lack of a linear relationship between length of procedure and recovery time, and this was further indicated by a Pearson's product-moment correlation coefficient of −0.205. Release back to the drive-through setting exhibit was performed within 1–3 hr of standing time, with no ataxia evident at this time.

DISCUSSION

The immobilization and subsequent recovery of semiwild animals is greatly facilitated by smooth and short induction and recovery periods, which was seen in this study with the use of the described protocols. In addition to this, the dosage can be administered in a single low-volume dart, thus reducing the possibility of problems associated with repeated dartings.

The mean score of 1.3 for activity postdarting is indicative and supportive of the observations of a smooth induction, with the absence of a severe excitatory phase. This lowers the risk of exer-

Table 3. Stages of recovery and times in 14[a] anesthetized emus (*Dromaius novaehollandiae*) with the use of a combination of thiafentanil and medetomidine with isoflurane for maintenance.

Event	*n*	Mean (minutes after T_d)	Range (minutes after T_d)	SD
Isoflurane off	14	23.7	21–37	12.6
Naltrexone administration	14	25.9	24–39	12.6
Extubated	14	27.5	25–40	12.3
Sternal recovery	14	29.0	26–44	12.3
Standing recovery	14	33.7	29–54	12.6

[a] $n=14$ for recovery timings, as one subject was euthanized.

tional myopathy or death as previously seen with other opioids in rheas, emus, and ostriches.[5,21,25,29]

The three birds requiring ketamine in addition to the thiafentanil–medetomidine induction dose were all from the initial four animals immobilized. At this stage of the study, the remote injection site was below the ventral border of feathering overlying the lateral hindlimb, which, in retrospect, had a much lower muscle mass than proximal to this muscle mass. Following this, the remaining 11 birds had the target site for remote injection moved proximally, to aim for a larger mass of muscle but sometimes requiring a blind shot through the feathers, shown in Figure 2. None of the birds injected at this more proximal site required any additional drugs. One of the three subjects requiring ketamine was given 0.123 mg/kg thiafentanil and 0.081 mg/kg medetomidine, one of the lowest calculated dosages, which may have also played a role in the requirement for additional induction drugs.

The results from the venous blood gas analysis, compared to the normal values obtained from a fully conscious bird and in comparison to mammalian values, indicate a significant respiratory acidosis, with lowered pH and hypercapnia. This is most likely due to hypoventilation.[5] Possible additional reasons for the low venous pO_2 of 29.6 mmHg (SD 7.37) include the fact that avian erythrocytes consume oxygen up to 10 times faster than mammalian erythrocytes.[9] This would result in alteration of the values with a delay in running the sample, or from causing jugular stasis for longer time period because of problems sampling. Steps were taken to minimize these effects, by running samples immediately after rapid jugular sampling. Precooled syringes could also have been utilized, as they may have reduced this effect further by slowing the metabolism of the avian blood cells.[9]

Normal respiratory rates for ostriches under anesthesia have been reported to be either 25–40 or above 8 breaths per minute.[6,15] An overall mean respiratory rate of 7.5 breaths per minute (SD 2.7) is indicative of some respiratory depression, a known side effect of opioid, alpha-2, and isoflurane administration[17] with two birds requiring positive pressure ventilation to assist with breathing. The birds requiring assistance with breathing did not receive any ketamine, and were actually dosed in the lower third of the dose range due to their weight. Results from the blood gas analysis suggest that simply using respiratory rate as is not an accurate indicator of the requirement for intervention to assist with breathing.

The lack of a constant pulse oximetry reading makes full evaluation of the whole-body effects of this respiratory depression difficult to assess fully.

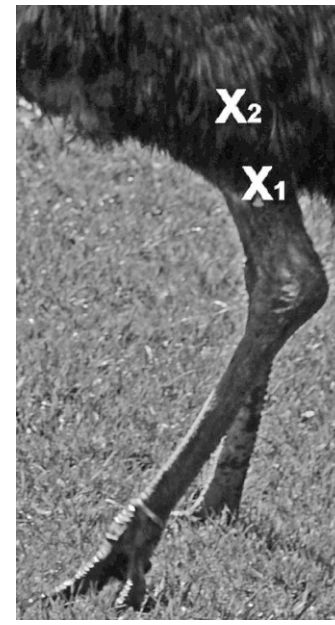

Figure 2. Original and preferred darting anatomy in an emu (*Dromaius novaehollandiae*). X1: original darting site, just proximal to the hock. X2: preferred darting site, into the lateral thigh musculature, sometimes requiring a shot through the feathers.

However, the calculation of arterial blood gas values from venous samples does correlate well with actual arterial values, especially those for pCO_2, pH, and HCO_3^-, with error ranges within those considered acceptable for laboratory equipment, and well within limits of error acceptable in clinical practice.[28] The method used in this case was therefore suitable for assessment of blood gas status, and offered the advantage, compared to arterial sampling, of being more rapid because of the ease of right jugular visualization and subsequent phlebotomy.

The bradypnoea seen, along with the respiratory acidosis, were the main problems associated with the protocol. The financial constraints of the project meant that repeated blood gas analysis, after antagonism of the medetomidine and with oxygen supplementation, could not be performed. These values would have given some indication as to the respiratory depressive effects of medetomidine. Oxygen supplementation at initial contact with the animal would be a suggested tool. Alternatively, the possibility of a protocol with slightly lower drug dosages may be possible, with darting at position X2 (see Fig. 2).

Cardiovascular effects appeared to be minimal, despite both opioids and medetomidine being known to cause cardiovascular depression[17,23] with heart rate relatively constant throughout procedures, at a mean of 63.9 bpm (SD 9.6), shown in Figure 1. This is within the range of 45–80 that is considered to be the normal range in anesthetized ratites.[15] A significant increase was found with heart rate following administration of atipamezole. The rate then declined and stayed relatively steady with a gradual decrease until recovery was initiated. Most of the procedures were short, with only four anesthetic events contributing to heart and respiratory values after 40 min, and only one anesthesia accounting for the values at 50 and 55 minutes. The rise at 15–20 min was almost certainly due to antagonism of the cardiodepressive medetomidine, with the decline after this most likely due to the effects of isoflurane. Indirect blood pressure monitoring was largely unsuccessful with the equipment available with the cuff placed over the cranial tibial artery immediately proximal to the hock. An alternative position for placement of the cuff could have been overlying the metatarsal artery, as used in ostriches.[13] From a practical standpoint, medial metatarsal vein catheterization was successful in every case, with good visualization of the vein. There were no abnormalities noted in any of the electrocardiogram readings, although no published data currently exist for normal complex amplitudes or durations in emus.

Recovery in the birds was smooth in each case, with zero to minimal thrashing or excitement and no evidence of self-trauma during recovery. This was probably influenced by the administration of midazolam, in addition to recovery within a dark and quiet contained space. Recovery was also rapid, with a mean of 3.1 min from naltrexone administration to achieving a sternal position, increased, although not to a statistically significant level, by addition of ketamine to the induction dose. The length of procedures had a very low negative correlation with the recovery times, possibly linked to increasing time under anesthesia with metabolism of thiafentanil, and ultimately leading to reduced recovery times. Additional, longer duration anesthetic events would need to be performed to assess this theory fully. Minimal correlation was also found between dosage of induction drugs and heart rate or respiratory rate. Addition of ketamine to the induction dose appeared to have no adverse safety effects, with no significant difference between groups when comparing pH, pCO_2, recovery times, heart, or respiratory rates.

There were no deleterious effects seen after anesthesia with the birds. Release into the drive-through setting was done within 1–3 hr depending on staffing and timing factors, and no birds showed any remaining drug effects on release, ambulating normally with no evidence of ataxia or renarcotization following regular observations for 72 hr postanesthesia. The initial thiafentanil–medetomidine dose was found to be sufficient in most of the cases, and, without ketamine, retained the capacity to use single, low-volume remote injection systems with complete antagonism availability.

CONCLUSION

The dosages used in this study, coupled with remote injection in the thigh musculature, provided an excellent and reliable combination for induction of anesthesia in adult emus. Caution should be taken regarding the noted bradypnoea and significant respiratory acidosis. Oxygen supplementation at initial contact may improve the hypercapnia, or adjustments to the induction protocol, as discussed, may be warranted, whereas ketamine offers an additional induction drug to use if required. The dosages used with the concentrations available allowed the use of a single low-volume dart, enhancing practicality and safety. The ability to antagonize the thiafen-

tanil and medetomidine fully allowed for rapid and smooth recoveries, with no lasting effects seen.

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