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SHORT COMMUNICATIONS

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Chemical Immobilization of Red Foxes (*Vulpes vulpes*)

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ABSTRACT: Red foxes (*Vulpes vulpes*) were immobilized with one of the following drug combinations: ketamine/xylazine (n = 22), ketamine/promazine (n = 35), ketamine/midazolam (n = 13), or tiletamine/zolazepam (n = 22). Foxes given ketamine/xylazine had the shortest induction and longest recovery times relative to other drug combinations, whereas foxes given ketamine/midazolam had the longest induction times. Recommended doses for the various combinations are given. Foxes given ketamine/xylazine were given either 0.1, 0.2, 0.4 mg/kg yohimbine, or saline 40 min after anesthetic induction. Administration of yohimbine significantly shortened arousal and recovery times relative to control values (P < 0.001).

Key words: Red fox, *Vulpes vulpes*, chemical immobilization, ketamine HCL, xylazine HCL, midazolam HCL, promazine HCL, tiletamine, zolazepam, yohimbine HCL, experimental study.

Red foxes (*Vulpes vulpes*) have been immobilized with phencyclidine and promazine (Seal et al., 1970) or ketamine and xylazine (Jessup, 1982). Newer immobilizing agents or antagonists have been used successfully on a variety of wild species. We report on the use of some of these newer agents and compare the efficacy of different drug combinations on captive red foxes.

This study was conducted from August 1985 through February 1989. Young (8- to 12-wk-old) fox pups were excavated from their dens in east central North Dakota (47°10′ to 47°20′N, 98°45′ to 99°15′W) and maintained as family units in kennels equipped with den boxes. Foxes were fed frozen commercial canine food (Lang Packing, St. Cloud, Minnesota 56080, USA) and provided water ad libitum. All animals were vaccinated for rabies, canine distemper, canine parvovirus, infectious canine hepatitis, leptospirosis, and parainfluenza (TechAmerica®, Fermenta Animal Health, Omaha, Nebraska 68134, USA). All were periodically treated for ecto- and endoparasites (Ivomec®, Merck, Inc., Rahway, New Jersey 07065, USA). At 4 to 5 mo of age (juvenile), each animal was paired with an unrelated mate and the pair housed separately. At 7 to 10 mo of age (subadult), a pair was selected for surgical implantation of a heart rate/body temperature radio transmitter (Cedar Creek Bioelectronics Laboratory, East Bethel, Minnesota 55011, USA) (Kreeger et al., 1989a). With the exception of Group One below, the anesthetic protocol selected was used for the purpose of implantation of these transmitters. After anesthetic induction, all foxes were weighed and blood samples were taken from the jugular vein.

Induction time (IT), head-up time (HUT), and walk time (WT) were defined. IT was time from injection of anesthetic to loss of consciousness. HUT was time from IT to spontaneous movement of the head. WT was time from IT until the animal could walk in a directed, coordinated manner, but not necessarily judged behaviorally "normal."

Seventy foxes were used in this study. Group One consisted of 22 juveniles foxes (11 males, 11 females) subdivided into four mixed-sex subgroups of five to six animals.

TABLE 1. Recovery times for 4- to 5-mo-old red foxes immobilized with ketamine and xylazine and given either yohimbine or 0.9% saline intravenously 40 min after induction. Means are reported with standard errors.

	Yohimbine dose (mg/kg)			
	0 (control)	0.1	0.2	0.4
n	6	5	5	5[b]
Head-up time (min)	86.2 ± 12.1[a]	6.4 ± 3.6	8.8 ± 2.4	1.6 ± 0.4
Walk time (min)	143.8 ± 28.6[a]	33.2 ± 14.3	38.6 ± 8.8	35.4 ± 10.3

[a] Significantly different from all other doses ($P < 0.05$).
[b] Data do not include one death.

All were physically restrained and injected intramuscularly with 70 to 100 mg ketamine hydrochloride (HCL) (KET; Ketaset®, Bristol Laboratories, Syracuse, New York 13221, USA) and 4 to 5 mg xylazine HCL (XYL; Rompun®, Miles Laboratory Inc., Shawnee, Kansas 66201, USA). Approximately 40 min after induction, the subgroups were given either 0.1, 0.2, 0.4 mg/kg yohimbine HCL (YOH; Sigma Chemical Co., St. Louis, Missouri 63178, USA) or a comparable volume of sterile 0.9% NaCl intravenously (controls). Preparation of YOH was described by Kreeger and Seal (1986a). Statistical analyses for Group One data were based on one-way ANOVA at a significance level of $P < 0.05$. Means are reported with standard errors (SE). Group Two consisted of 35 subadult foxes (18 males, 17 females), 22 of which were from Group One used 3 to 5 mo previously. Foxes were physically restrained and injected intramuscularly with 100 to 150 mg KET and 25 mg promazine HCL (PRO; Sparine®, Wyeth Labs, Philadelphia, Pennsylvania 19101, USA). Group Three consisted of 13 different subadult foxes (7 males, 6 females) which were physically restrained and injected intramuscularly with 125 to 150 mg KET plus 2.5 mg midazolam HCL (MID; Versed®, Roche Laboratories, Nutley, New Jersey 07110, USA). Group Four consisted of 22 other subadult foxes (11 males, 11 females) which were physically restrained and injected intramuscularly with 40 to 55 mg each of tiletamine HCL (TIL) and zolazepam HCL (ZOL) (Telazol®, A. H. Robins, Richmond, Virginia 23220, USA).

The HUT and WT for foxes given YOH was significantly shorter than for control foxes ($P = 0.0001$ and 0.001, respectively; Table 1). There was no difference in HUT or WT among YOH doses ($P = 0.94$). Cataleptoid muscle rigidity and tremors usually followed intravenous YOH administration. One male fox who received 0.2 mg/kg YOH and one female who received 0.4 mg/kg YOH had rectal temperatures of at least 43.3 C during this period. Both foxes were immersed in water after body temperature was measured, but only the male survived. Ambient temperature during this episode was about 33.0 C. In general, recoveries in foxes given YOH were characterized by increased activity as the animal attempted to become sternal and stand. The foxes would often raise their heads only to drop them with some force for a period of time prior to becoming sternal or standing.

Statistical analyses were not performed on data among groups since foxes were not randomly assigned and because two age groups were used. Induction times appeared to differ among all four groups with Group One foxes recording relatively short IT and Group Three relatively long IT (Table 2). Group One tended to have a longer HUT compared to the other groups. Inductions for all drug combinations were smooth, as were recoveries with the exception of Group One foxes given YOH. Recoveries without YOH were character-

TABLE 2. Induction and arousal times for red foxes immobilized with either ketamine/xylazine, ketamine/promazine, ketamine/midazolam, or tiletamine/zolazepam. Means are reported with standard errors.

Group	*n*	Drug combination	Amount (mg/kg)	Induction time (min)	Head-up time (min)
1	22[a]	Ketamine	22.7 ± 0.6		
		Xylazine	1.2 ± 0.03	1.4 ± 0.1	86.2 ± 1.21[c]
2	35[b]	Ketamine	27.9 ± 0.1		
		Promazine	5.2 ± 0.1	2.7 ± 0.2	27.3 ± 0.3
3	13[b]	Ketamine	30.8 ± 1.2		
		Midazolam	0.6 ± 0.02	4.9 ± 0.7	18.6 ± 4.0
4	22[b]	Tiletamine	5.3 ± 0.2		
		Zolazepam	5.3 ± 0.2	3.6 ± 0.5	25.1 ± 2.5

[a] Juvenile (4 to 5 mo) foxes.
[b] Subadult (7 to 10 mo) foxes.
[c] Head-up time for Group One foxes receiving saline only (n = 6).

ized by tremors, but not the increased activity seen after YOH administration.

The relationship between YOH dose and HUT and WT in red foxes was similar to that reported for gray wolves (*Canis lupus*) (Kreeger et al., 1987); increasing the YOH dose did not shorten recovery times. Higher doses of YOH are probably contraindicated due to the extreme tachycardia seen in canids immobilized with ketamine and xylazine then given doses of YOH ≥ 0.2 mg/kg (Kreeger et al., 1987). The positive chronotropic effects of both KET (Folts et al., 1975) and YOH (Gomes et al., 1980) apparently act synergistically to markedly increase heart rate, since this tachycardia is not seen when YOH is given to animals anesthetized with drugs other than KET (Kreeger et al., 1989b).

Another caution in the use of YOH in animals anesthetized with KET and XYL is the time of YOH administration after the last KET injection. Yohimbine is an alpha$_2$-adrenergic antagonist (Goldberg and Robertson, 1983) which acts on presynaptic adrenoceptors to antagonize the effects of XYL (Langer, 1981). Yohimbine does not antagonize KET anesthesia (Kreeger and Seal, 1986b). For a smooth recovery, sufficient time must be allowed for the animal to metabolize KET prior to YOH administration. If the effects of XYL were antagonized through the use of YOH before sufficient KET had been metabolized, the animal would recover from what is effectively pure KET anesthesia. Rough induction and recovery effects of KET alone have been well documented (Wright, 1982). We feel that the two cases of hyperthermia were caused by this phenomenon, coupled with warm ambient temperatures that resulted in a rapid increase in endogenous heat production.

The relationship between different drug combinations confirmed our empirical observations with canid immobilizations over the years. That is, the combination of KET and XYL results in rapid induction coupled with extended recovery times if YOH is not administered. Likewise, it has been our experience with wolves (T. J. Kreeger, unpubl. data) that KET combined with MID resulted in extended induction times while arousal times were short, often requiring boosters of KET to maintain anesthesia. The combinations of KET and PRO or TIL and ZOL achieved quick inductions and acceptable recovery times in red foxes and appeared to be good drug combinations to use on this species. These two drug combinations also provide good cardiovascular and respiratory support compared to the potential bradycardic, hypotensive and respiratory depressant effects of XYL (Clark et al., 1982). While KET can induce convulsions in canids (Wright,

1982), only one convulsion in 70 trials was noted in this study. This occurred in a female fox anesthetized with KET and PRO. If convulsions are unacceptable, we would recommend the use of either KET/MID or TIL/ZOL as both of these combinations benefit from the anticonvulsant effects of benzodiazepine tranquilizers (Richter, 1981).

Based on these results and our experience with wild canid anesthesia, we recommend the following dosages for immobilization of red foxes: (1) 30 mg/kg ketamine plus 5 mg/kg promazine; (2) 20 mg/kg ketamine plus 1 mg/kg xylazine; 0.1 mg/kg yohimbine given 45 to 60 min after the last ketamine administration; (3) 30 mg/kg ketamine plus 1 mg/kg midazolam; or (4) 5 mg/kg tiletamine plus 5 mg/kg zolazepam (i.e., 10 mg/kg of Telazol®).

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LITERATURE CITED

CLARK, D. M., R. A. MARTIN, AND C. A. SHORT. 1982. Cardiopulmonary responses to xylazine/ketamine anesthesia in the dog. Journal of the American Animal Hospital Association 18: 815–821.

FOLTS, J. D., S. AFONSO, AND G. G. ROWE. 1975. Systemic and coronary haemodynamic effects of ketamine in intact anesthetized and unanaesthetized dogs. British Journal of Anaesthesiology 47: 686–693.

GOLDBERG, M. R., AND D. ROBERTSON. 1983. Yohimbine: A pharmacological probe for study of the alpha$_2$-adrenoreceptor. Pharmacological Review 35: 143–180.

GOMES, C., G. TROLIN, M. HENNING, AND B. PERRSON. 1980. Pre- and postsynaptic alpha-adrenoceptor antagonists: Differentiated cardiovascular effects in the rat. Clinical and Experimental Hypertension 2: 273–296.

JESSUP, D. A. 1982. Restraint and chemical immobilization of carnivores and furbearers. *In* Chemical immobilization of North American wildlife, L. Nielsen, J. C. Haigh, and M. E. Fowler (eds.). Wisconsin Humane Society, Inc., Milwaukee, Wisconsin, pp. 227–244.

KREEGER, T. J., AND U. S. SEAL. 1986a. Immobilization of coyotes with xylazine hydrochloride-ketamine hydrochloride and antagonism by yohimbine hydrochloride. Journal of Wildlife Diseases 22: 604–606.

———, AND ———. 1986b. Failure of yohimbine hydrochloride to antagonize ketamine hydrochloride immobilization of gray wolves. Journal of Wildlife Diseases 22: 600–603.

———, A. M. FAGELLA, U. S. SEAL, L. D. MECH, M. CALLAHAN, AND B. HALL. 1987. Cardiovascular and behavioral responses of gray wolves to ketamine-xylazine immobilization and antagonism by yohimbine. Journal of Wildlife Diseases 23: 463–470.

———, D. MONSON, V. B. KUECHLE, U. S. SEAL, AND J. R. TESTER. 1989a. Monitoring heart rate and body temperature in red foxes (*Vulpes vulpes*). Canadian Journal of Zoology 67: In press.

———, R. E. MANDSAGER, U. S. SEAL, M. CALLAHAN, AND M. BECKEL. 1989b. Physiological response of gray wolves to butorphanol-xylazine immobilization and antagonism by naloxone and yohimbine. Journal of Wildlife Diseases 25: 89–94.

LANGER, Z. 1981. Presynaptic regulation of the release of catecholamines. Pharmacological Review 32: 337–362.

RICHTER, J. J. 1981. Current theories about the mechanisms of benzodiazepines and neuroleptic drugs. Anesthesiology 54: 66–72.

SEAL, U. S., A. W. ERICKSON, AND J. G. MAYO. 1970. Drug immobilisation of the Carnivora. International Zoo Yearbook 10: 157–170.

WRIGHT, M. 1982. Pharmacological effects of ketamine and its use in veterinary medicine. Journal of the American Veterinary Medical Association 180: 1462–1471.

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