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Authors: Terry J. Kreeger, and Ulysses S. Seal
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Immobilization of Coyotes with Xylazine Hydrochloride–Ketamine Hydrochloride and Antagonism by Yohimbine Hydrochloride

Terry J. Kreeger, Department of Fisheries and Wildlife, University of Minnesota, St. Paul, Minnesota 55108, USA; and Ulysses S. Seal, Research Service, Veteran’s Administration Medical Center, Minneapolis, Minnesota 55417, USA and Departments of Biochemistry and Fisheries and Wildlife, University of Minnesota, St. Paul, Minnesota 55108, USA


One adult male and four juvenile (two female, two male) wild-caught coyotes from South Dakota and one adult wild-caught female from Minnesota were used in this study. The five South Dakota coyotes were housed in indoor-outdoor pens at the Minnesota Zoological Gardens, Apple Valley, Minnesota. The Minnesota coyote was housed in an outdoor kennel approximately 80 km north of the other coyotes. The zoo coyotes were fed a commercial canine formulation once a day (Nebraska Brand, Central Nebraska Packing Inc., North Platte, Nebraska 69101, USA). The adult female was fed dry dog food ad libitum (Purina Brand Dog Chow,Ralston-Purina Co., St. Louis, Missouri 63178, USA). All coyotes were provided water ad libitum.

In October 1985, all coyotes were secured in a hand-held net and immobilized with a single, hand-injected, intramuscular dose of 2.0 mg/kg xylazine HCl (Rompun®, Haver-Lockhart Laboratories, Shawnee, Kansas 66201, USA), 4.0 mg/kg ketamine HCl (Ketaset®, Bristol Labora-
tories, Syracuse, New York 13201, USA), and 0.02 mg/kg atropine sulfate (Atro-
pine Injectable, S.A., Fort Dodge Labo-
ratories, Inc., Fort Dodge, Iowa 50501, 
USA). Upon induction, all coyotes were 
placed in right lateral recumbency with 
heart rate, respiratory rate, rectal tem-
perature, and capillary refill being monitored 
throughout the immobilization period. 
Approximately 20 min was allowed to 
elapse, then all coyotes received 0.2 mg/
kg yohimbine HCl (Sigma Chemical Co., 
St. Louis, Missouri 63178, USA) adminis-
tered via the cephalic vein. Yohimbine 
HCl was constituted at a concentration of 
1.0 mg/ml in sterile 0.9% sodium chloride 
solution. Sterility of this solution was ver-
ified by inoculating on blood and Mac-
Conkey agar and incubating at 37 C for 
24 and 72 hr. Arousal time (AT) and walk 
time (WT) was recorded on all animals. 
AT was the time for yohimbine HCl in-
jection to when the animal opened its eyes 
and raised its head; WT was the time from 
yohimbine HCl injection to when the an-
imal could walk in a directed manner of 
its own accord. One mo later, all the coy-
otes received the same anesthetic doses, 
administered in the same manner, but 
were allowed to recover on their own. 
Thus, each animal served as its own con-
trol. Statistical analysis was by one-way 
ANOVA. Means are reported with stand-
ard deviations.

The mean induction times (time from ini-
tial injection to immobilization) were 
7.3 ± 3.9 min (range = 4–13 min) for the 
first trial and 7.0 ± 3.0 min (range = 3– 
11 min) for the second trial. There was no 
significant difference between these two 
times (P ≥ 0.86). The time from induction 
to injection of yohimbine HCl was 20.2 ± 
11.5 min (range = 11–39 min). The mean 
AT and WT for coyotes reversed with 
yohimbine HCl were 8.2 ± 5.4 min 
(range = 2–18 min) and 13.2 ± 7.7 min 
(range = 7–19 min). The total immobi-
lization time (time from immobilization to 
ambulation) for coyotes receiving yohim-
bine HCl (33.3 ± 9.0 min; range = 21–46 
min) was significantly less than the control 
times (70.5 ± 5.9 min; range = 61–77 min) 
(P < 0.01).

Within 5 min of receiving yohimbine 
HCl, the adult male coyote developed a 
fixed behavior pattern characterized by 
mouth gaping and face scratching with 
both forepaws. This behavior lasted ap-
proximately 10 min. One juvenile female 
also demonstrated unusual post-reversal 
behavior consisting of rhythmic, side-to-
side head twitching and hyperreflexia. 
This behavior lasted for almost 35 min. 
Neither of these behavior patterns were 
observed when the animals recovered 
without yohimbine HCl.

Xylazine HCl is an alpha2-adrenergic 
agonist that produces sedation and anal-
gesia by stimulating central presynaptic 
adrenoceptors. This stimulation inhibits 
the influx of calcium during an action poten-
tial to prevent norepinephrine release 
at the nerve terminal (Starke, 1977, Rev. 
362). Yohimbine HCl is an alpha2-adren-
ergic antagonist (Goldberg and Robertson, 
1983, Pharmacol. Rev. 35: 143–180) that 
has been shown to reverse xylazine HCl-
induced blockade of neural transmission 
in several species (Hsu and Shulaw, 1984, 
Hatch et al., 1985, Am. J. Vet. Res. 46: 
371–375).

The definitive site of action for keta-
nine HCl remains to be determined. Cho-
linergic (Leeuwin et al., 1984, Br. J. 
Pharmacol. 82: 339–347), serotonergic, 
gamma-aminobutyric acid, dopaminergic 
180: 1462–1470), sigma opioid (Smith et 
al., 1980, Anesthesiology 53: 35) and 
N-methylaspartate (Thomson et al., 1985, 
Nature 313: 479–481) receptors have all 
been implicated as sites of action. Besides 
adrenergic activity, yohimbine HCl may
be capable of influencing cholinergic (Zettler and Thorner, 1973, Pharmacology 10: 238–251), serotonergic (Sanghvi and Gershon, 1970, Eur. J. Pharmacol. 11: 125–129), and dopaminergic (Scaotton et al., 1980, J. Pharmacol. Exp. Ther. 215: 494–499) receptors. Thus, there may be some interaction between ketamine HCl and yohimbine HCl at common receptor sites.

The ability of yohimbine HCl to antagonize ketamine HCl anesthesia is equivocal. Domestic cats anesthetized with ketamine HCl (20.0 mg/kg), then given yohimbine HCl (0.25 mg/kg), had significantly shorter arousal times than did controls, but walk times were unchanged, or even lengthened (Hatch et al., 1983, Am. J. Vet. Res. 44: 417–423). Yohimbine HCl (0.5 mg/kg) failed to reverse ketamine HCl (10.0 mg/kg) anesthesia in rhesus monkeys (Macaca mulatta) (Lynch and Line, 1985, Lab. Anim. Sci. 35: 417–418) as well as in wolves (Canis lupus L.) (Kreeger and Seal, 1986, J. Wildl. Dis. 22: 600–603). Yohimbine HCl could be acting as a general stimulant to either reverse or override at least some of the effects of ketamine HCl (Hsu and Lu, 1984, J. Am. Vet. Med. Assoc. 185: 886–888). Thus, reversal of xylazine HCl–ketamine HCl anesthesia is probably due primarily to the antagonism of xylazine HCl by yohimbine HCl. The fixed behavior pattern observed in the two coyotes could be due to a residual ketamine HCl effect unmasked after antagonism of xylazine HCl.


Atropine sulfate was included in this drug protocol to preclude any xylazine HCl-induced bradycardia (Klide et al., 1975, Am. J. Vet. Res. 36: 931–935).

Even though inductions were fairly quick and smooth, it should be remembered that these animals were caged and not overly stimulated prior to immobilization. Coyotes chased, or otherwise greatly excited, would be expected to require higher immobilizing doses (Nielsen et al., 1982, op. cit.).

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