

USE OF TELAZOL® TO IMMOBILIZE FEMALE NORTHERN SEA LIONS (EUMETOPIAS JUBATUS) IN ALASKA

Authors: Loughlin, Thomas R., and Spraker, Terry

Source: Journal of Wildlife Diseases, 25(3) : 353-358

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-25.3.353>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

USE OF TELAZOL® TO IMMOBILIZE FEMALE NORTHERN SEA LIONS (*EUMETOPIAS JUBATUS*) IN ALASKA

Thomas R. Loughlin¹ and Terry Spraker²

¹ National Marine Mammal Laboratory, National Marine Fisheries Service, 7600 Sand Point Way, NE, Seattle, Washington 98115, USA

² Veterinary Diagnostic Laboratory, Department of Pathology, College of Veterinary Medicine, Colorado State University, Fort Collins, Colorado 80523, USA

ABSTRACT: Twenty-nine female northern sea lions (*Eumetopias jubatus*) were immobilized using Telazol® in dosages ranging from 1.8 to 8.1 mg/kg. Best results were achieved with Telazol® dosages ranging between 1.8 and 2.5 mg/kg which resulted in smooth induction and recovery. Optimal injection location was in the muscle mass of the lower back and hip. Dosages >3.5 mg/kg resulted in a tendency toward hypothermia. Six mortalities occurred which were partially caused by the location of drug injection and perhaps the high dosage.

Key words: Telazol®, otariids, northern sea lions, *Eumetopias jubatus*, chemical immobilization, anesthetic, field study.

INTRODUCTION

There have been few successful reports of chemical immobilization of otariid pinnipeds. Galápagos fur seals (*Arctocephalus galapagoensis*) and Galápagos sea lions (*Zalophus californianus wolfebaeki*) were restrained using 2 to 4 mg/kg ketamine hydrochloride mixed with 0.3 to 0.4 mg/kg xylazine hydrochloride delivered by blowpipe (Trillmich, 1983). Somewhat higher dosages of ketamine/xylazine were required for Antarctic fur seals (*A. gasella*) with a mortality rate of 20% for affected animals (Bester, 1988). However, use of ketamine hydrochloride alone on California sea lions (*Zalophus californianus*) resulted in high mortality and irregular behavior at sea after recovery (R. DeLong, pers. commun.). Alternatively, ketamine hydrochloride has proven to be very useful for immobilization of phocid pinnipeds, particularly northern and southern elephant seals (*Mirounga angustirostris* and *M. leonina*) and gray seals (*Halichoerus grypus*) (Parry et al., 1981; Antonelis et al., 1987; Bester, 1988).

Telazol®, a 1:1 mixture of tiletamine hydrochloride and zolazepam hydrochloride, has been used successfully to immobilize a variety of wild and domestic animals and is currently approved for domestic dogs and cats (Gray et al., 1974; Schobert, 1987). It is characterized by rapid, smoothly in-

duced, profound analgesia, cataleptoid anesthesia, and normal pharyngeal-laryngeal reflexes (Schobert, 1987). It has been used successfully to immobilize marine mammals, such as polar bears (*Ursus maritimus*), elephant seals and male Atlantic walrus (*Odobenus rosmarus*) (Gray et al., 1974; Stirling et al., 1985; Stirling and Sjare, 1988); however its use on otariid pinnipeds has been equivocal. Based on information in Gray et al. (1974), Schobert (1987) concluded that California sea lions are not good candidates for Telazol® because of a high mortality rate. In the review by Gray et al. (1974) five California sea lions (sex and age not provided) were given dosages of Telazol® ranging from 0.8 to 1.5 mg/kg. Two of the five animals died, although the drug's role as a cause of mortality was unknown. The drug had the desired effect on the three remaining California sea lions. This report presents the results of our studies using Telazol® to immobilize female northern (Steller) sea lions (*Eumetopias jubatus*).

The northern sea lion is the largest member of the Otariidae; the mass of males is more than 800 kg and females average 250 kg (Calkins and Pitcher, 1982). The species' range extends around the North Pacific Ocean rim from Hokkaido, Japan, through the Kurile Islands, Aleutian Islands, and central Bering Sea, and south

TABLE 1. Telazol® dosage summary and weight for individual female northern sea lions studied during 1987 and 1988. Estimation of mass was based on subjective appraisal of body length.

Month examined	Approximate mass (kg)	Dosage (mg)	mg/kg
June 1987	226	1,750	7.7
June 1987	215	1,750	8.1
June 1987	247	1,500	6.1
June 1987	204	1,000	4.9*
June 1987	215	1,000	4.7
June 1987	227	1,000	4.4
October 1987	250	1,000	4.0
October 1987	250	800	3.2*
October 1987	272	800	2.9
October 1987	272	800	2.9*
October 1987	215	800	3.7
October 1987	204	750	3.7
October 1987	272	750	2.8*
June 1988	225	400	1.8
June 1988	225	500	2.2
June 1988	225	500	2.2
June 1988	225	400	1.8
June 1988	225	400	1.8
June 1988	225	400	1.8
June 1988	225	500	2.2
June 1988	225	500	2.2
June 1988	225	500	2.2
June 1988	225	1,100	4.9*
June 1988	225	500	2.2
June 1988	225	500	2.2
June 1988	196	500	2.6
June 1988	225	500	2.2
June 1988	225	800	3.6
June 1988	225	800	3.6*

* Died.

to the Channel Islands off California (Loughlin et al., 1984). The centers of abundance and distribution for this species are the Gulf of Alaska and Aleutian Islands, respectively. Within this range, the population currently numbers about 70,000 adult animals but it is declining at a rate of at least 2.7%/yr (Merrick et al., 1987). In trying to determine the cause(s) of the decline, we marked the animals by attaching telemetry devices in a study of the life history and population dynamics of the northern sea lion. To attach these devices we used Telazol® to immobilize the animals.

MATERIALS AND METHODS

During June and October 1987 and June 1988, 29 inactive, lactating, adult, female northern sea lions on Marmot Island, Alaska (58°N, 152°W) were immobilized with Telazol®. Lyophilized Telazol® (A. H. Robins, supplied by Wildlife Laboratories, Inc., Fort Collins, Colorado 80526, USA) was hydrated with sterile water in the field to the desired concentration, usually 200 to 250 mg/ml. The concentration was higher than manufacturer recommended levels to facilitate use of small syringes. Estimates of body mass were based on information (curvilinear and standard lengths and mass) in Calkins and Pitcher (1982) and on unpublished data collected by them during 1985 and 1986. Animals were remotely injected using a Telinject® vario air-pistol (Telinject USA Inc., 23655 San Fernando Road, Newhall, California 91321, USA) with pistol barrel and 5 or 10 ml syringe, depending on dosage; the syringe used 2 × 60 mm collared needles. We tried, but were not always successful, to place shots in the muscle masses of the shoulder, back, and hip.

We recorded the minutes from needle entry to when ataxia was first noticed (AT); we then recorded the minutes until the animal was down and unable to move away upon our approach (AD). When possible, we also recorded cataleptoid anesthesia [from AD until the animal lifted its head (HL)] and was able to ambulate (recovery). For some animals we also monitored rectal body temperature with a digital thermometer at 10 min intervals, and we measured respiratory rate by visual inspection. Environmental temperature and weather also were recorded. A bland ophthalmic ointment (Ilotycin®, Dista Products Co., Eli Lilly and Company, Indianapolis, Indiana 48285, USA) was applied to the eyes to protect from desiccation and approximately 4.4 mg/kg oxytetracycline (Liquamycin LA-200®, Agricultural Division, Pfizer Inc., New York, New York 10017, USA) was injected intramuscularly. Each animal was individually marked with plastic, numbered tags in the rear of each front flipper, and a radio transmitter was glued to the nape with quick-drying epoxy resin to facilitate resighting of drugged animals and to monitor behavior while they were on land.

Statistical analysis was by one-way analysis of variance (ANOVA) and χ^2 contingency tables (Snedecor and Cochran, 1980). A general linear interactive model (GLIM), which includes ANOVA, analysis of covariance, and χ^2 , was used to compare the site of injection to dosage and month (Numerical Algorithms Group, 1987). A value of $P \leq 0.05$ was considered significant.

TABLE 2. Mean (\bar{x}) time to first signs of ataxia (AT), time to when animal was considered down (AD), first signs of recovery (HL), and complete recovery for northern sea lions injected with Telazol® during 1987 and 1988.

	All cases	June 1987	October 1987	June 1988
AT (min)				
\bar{x} (SD) ^a	4.9 (3.2)	6.4 (4.9)	4.2 (1.7)	3.9 (2.1)
Range (n) ^b	1.2–16.5 (25)	2–16.5 (6)	2–7 (6)	1.3–8.4 (13)
AD (min)				
\bar{x} (SD)	11.3 (6.6)	14.0 (6.8)	7.2 (6.8)	11.9 (7.2)
Range (n)	4.1–25 (25)	6–20.5 (6)	5–13 (6)	4.1–25 (13)
HL (min)				
\bar{x} (SD)	68.3 (46.5)	^c	121.6 (53.5)	49.1 (32.6)
Range (n)	5–180 (16)	60–172 (2)	75–180 (3)	5–92 (11)
Recovery (min)				
\bar{x} (SD)	131.8 (30.1)	^c	130.0 (45.8)	138 (23.3)
Range (n)	90–180+ (9)	106 (1)	90–180+ (3)	110–168 (5)

^a SD, standard deviation.

^b n, sample size.

^c Sample size too small to calculate mean.

RESULTS

Mean Telazol® dosage for female northern sea lions in June 1987 was 5.98 mg/kg ($n = 6$), for October 1987 it was 3.31 mg/kg ($n = 7$), and for June 1988 it was 2.44 ($n = 16$); for all cases mean dosage ranged from 1.8 to 8.1 mg/kg ($\bar{x} = 3.4$ mg/kg; SD = 1.7; Table 1). Mean AT time for all cases was 4.9 min (SD = 3.2); mean AD was 11.3 min (SD = 6.6), mean HL was 1 hr 8 min (SD = 46.5), and mean recovery was 2 hr 11 min (SD = 30.1) (Table 2). The AT times for animals studied in all three study periods were not significantly different from each other (ANOVA, $F = 1.52$, $P = 0.76$), nor were AD times significantly different during these study periods (ANOVA, $F = 1.91$, $P = 0.83$). Interestingly, AD in June of both years was much higher (twice in June 1987) than in October 1987, but the low sample size and high variability probably masked any statistical significance. The sample sizes were too small to compare HL and recovery times for these periods.

Mean rectal temperature for all cases was 36.4 C ($n = 45$; SD = 1.6; range 33.8 to 37.7 C) and mean respiration rate was

13.3 breaths/min ($n = 102$; SD = 5.4; range 6 to 26). There was no apparent correlation between respiratory rate and dosage ($r^2 = 0.044$).

High dosage of Telazol® (>3.5 mg/kg) resulted in a tendency toward hypothermia; however, at recommended (1.8 to 2.5 mg/kg) or low (<1.8 mg/kg) dosages there was no change in vital parameters. At low and recommended dosages the palpebral and pedal reflexes remained functional, although they were absent at high dosages.

Of the 29 animals tested, three were injected in the muscle of the cheek or neck, six in the shoulder, three in the back, six in the chest, three in the abdomen, and eight in the hip area. Three mortalities occurred when injected in the shoulder, one in the neck, one in the cheek, and one in the abdomen. In all but one mortality there was no indication of an impending problem to the injection of Telazol®. Five sea lions suddenly developed respiratory arrest and could not be revived. The sixth was a female injected in the neck and began convulsions 11 min after the injection which progressed into excessive spasmodic locomotion for nearly 5 min. Death oc-

curred 3 hr after the injection. The six mortalities occurred at dosages of 2.8, 2.9, 3.2, 3.6, 4.9, and 4.9 mg/kg (Table 1).

We compared the mortalities by study period and location of injection and found no significant difference based on study period ($\chi^2 = 2.81$; 2 df) although there was 17% mortality in June 1987, 43% in October 1987, and 13% in June 1988. There was a significant difference when comparing the location of injection. There were significantly more mortalities when injected anterior to or in the shoulder than posterior to the shoulder ($\chi^2 = 9.67$; 1 df). We also compared site of injection with dosage and month and found that site of injection was the only significant factor ($G^2 = 9.260$; $P < 0.995$); neither dose ($G^2 = 0.280$; $P < 0.403$) nor month ($G^2 = 2.480$; $P < 0.885$) was significant by itself or conditionally after the site of injection was included. The relative risk of mortality with an anterior injection to that of a posterior injection was approximately 24:1.

In three cases during June 1987, more than one injection was required, and in two of those the animal died. In one mortality the animal was given injections of 500 mg (in the right abdomen), and two more injections of 300 mg and 300 mg each (in the left chest muscle) totaling 1,100 mg or 4.9 mg/kg. In another mortality the animal was given 500 mg in the left shoulder then 300 mg (position not recorded) totaling 800 mg or 3.6 mg/kg. In the third case the animal was given 500 mg in the flipper then 300 mg in the hip totaling 800 mg or 3.6 mg/kg; this animal survived. No other animals were given repeat injections.

Activity patterns and general behavior of drugged and normal animals were monitored from observation posts for at least 2 wk after the study while the animals were on land, or through August in 1988 by remote telemetry stations. We were unable to distinguish any behavioral abnormality in drugged animals 2 days after injection. Most animals behaved in a normal fashion within 1 day after the injection; their attendance patterns were sim-

ilar to non-drugged sea lions at Marmot Island and to sea lions at Ugamak Island in the eastern Aleutian Islands (Merrick et al., 1988).

Mean air temperature at noon during June and July in both years was 10.7 C with overcast skies or light fog and variable winds to about 15 knots. During October 1987 mean air temperature was 3.2 C with rain and clouds and variable winds to 28 knots.

DISCUSSION

Our observations of Telazol® induced immobilization were similar to those of Stirling and Sjare (1988) for male Atlantic walruses ($\bar{x} = 1.51$ mg/kg, $n = 10$; best results at 2.0–2.25 mg/kg). The rear flipper and pelvic regions were the first to exhibit reduced mobility, and the front flippers and head were last; the reverse occurred during recovery. This sequence occurred with all our drugged animals regardless of the placement of injection.

One of our greatest sources of error in dosage level was our estimation of body mass. The amount of drug given to an animal was based on our subjective appraisal of its weight, based on its assumed length and robustness. However, even with these inaccuracies we feel confident that our appraisals were within ± 20 kg of the actual weight. Also, A. H. Robins Co. states that dogs and cats [and experience by one of us (TS) on brown and black bears] have tolerance for higher than recommended doses of Telazol® and recommends high initial dosages to reduce the likelihood of supplemental injections. Therefore, we gave high initial doses to our first study animals then reduced the dosage during later studies for optimal results.

One mortality occurred during June 1987 (17%), three in October 1987 (43%), and two in June 1987 (13%). The drug used in June 1987 was manufactured in France and during the other two study periods it was manufactured in the United States. We did not record any noticeable effect caused by this difference, except our sub-

jective opinion that the drug seemed to be more potent during October than June 1987 but not during June 1988. However, no statistically significant difference was found in the parameters that we measured. The high mortality during October may have been the result of physiological differences between the study periods. During June, northern sea lions are reproductively active whereas during October they are not (however, some animals are still nursing pups then) and have completed or are near the end of the annual molt.

Animals injected posterior to the shoulders in the muscle masses of the back or hip region exhibited the best response to the drug. They had smooth induction and recovery with minimal to no muscle tremors. Our subjective appraisal was that the reaction of animals injected posterior to the shoulder was more consistent, and the animals had fewer visible behavioral side effects than animals injected more anteriorly. We do not recommend that northern sea lions be darted anterior to the shoulder or in the shoulder with Telazol®. The reason for this difference in response to injection site was not determined, but could have been due to the concentration of the drug, rate of drug absorption, character of circulation, and perhaps blubber thickness in this area. Also, most of the mortalities occurred during the first two study periods when we were using high dosages, and the combination of high dosage, high concentration, and location probably accounted for the mortalities.

During our research in June 1988, we observed two female sea lions that had been immobilized with Telazol® during 1987. Each behaved normally and exhibited no apparent long-term behavioral effects from the drug and one of the females had a newborn pup.

Telazol® in dosages ranging from 1.8 to 2.5 mg/kg had the best results to immobilize northern sea lions because at these levels induction and recovery were smooth, depression of the respiratory rate was minimal, and hypothermia was unlikely. At

those levels any errors associated with determination of the animal's size were reduced allowing for the possibility of a second injection if needed. Also, best results were achieved when injection was in the muscle mass of the lower back or hip.

ACKNOWLEDGMENTS

We are grateful to W. Lance for supplying the Telazol® and to J. Baker, D. Calkins, P. Gearin, D. McAllister, R. Merrick, D. DeGhetto, K. Shevljagin and V. Vladimirov for assistance during field operations. The manuscript was improved by comments from J. Davis, G. Duker, D. Gorenberg, R. Henth, R. Merrick and two anonymous reviewers. A. York provided statistical advice. Reference to trade names does not imply endorsement by the National Marine Fisheries Service, NOAA.

LITERATURE CITED

- ANTONELIS, G. A., JR., M. S. LOWRY, D. P. DEMASTER, AND C. H. FISCUS. 1987. Assessing northern elephant seal feeding habits by stomach lavage. *Marine Mammal Science* 3: 308-322.
- BESTER, M. N. 1988. Chemical restraint of Antarctic fur seals and southern elephant seals. *South African Journal of Wildlife Research* 18: 57-60.
- CALKINS, D. G., AND K. W. PITCHER. 1982. Population assessment, ecology and trophic relationships of Steller sea lions in the Gulf of Alaska. U.S. Department of Commerce, NOAA, OC-SEAP, Final Reports 19, Anchorage, Alaska, pp. 445-546.
- GRAY, C. W., M. BUSH, AND C. C. BECK. 1974. Clinical experience using CI-744 in chemical restraint and anesthesia of exotic specimens. *Journal of Zoo Animal Medicine* 5: 12-21.
- LOUGHLIN, T. R., D. J. RUGH, AND C. H. FISCUS. 1984. Northern sea lion distribution and abundance: 1956-1980. *The Journal of Wildlife Management* 48: 729-740.
- Merrick, R. L., P. GEARIN, S. OSMEK, AND D. WITTHROW. 1988. Field studies of northern sea lions at Ugamak Island, Alaska during the 1985 and 1986 breeding seasons. NOAA Tech. Memo. NMFS F/NWC-143, U.S. Department of Commerce, Seattle, Washington, 60 pp.
- , T. R. LOUGHLIN, AND D. G. CALKINS. 1987. Decline in abundance of the northern sea lion, *Eumetopias jubatus*, in Alaska, 1956-86. *Fishery Bulletin*, U.S. 85: 351-365.
- NUMERICAL ALGORITHMS GROUP. 1987. The GLIM system release, 3.77 manual, 2nd ed. Royal Statistical Society, Oxford, England, 303 pp.
- PARRY, K. S., S. ANDERSON, AND M. A. FEDAK. 1981. Chemical immobilization of gray seals. *The Journal of Wildlife Management* 45: 986-990.

- SCHOBERT, E. 1987. Telazol use in wild and exotic animals. *Veterinary Medicine* October 1987: 1080-1088.
- SNEDECOR, G. W., AND W. G. COCHRAN. 1980. *Statistical methods*, 7th ed. Iowa State University Press, Ames, Iowa, 507 pp.
- STIRLING, I., E. BROUGHTON, L. O. KNUTSEN, M. A. RAMSAY, AND D. S. ANDRIASHEK. 1985. Immobilization of polar bears with Telazol on the western coast of Hudson Bay during summer 1984. *Canadian Wildlife Service Progress Notes* No. 157, Canadian Wildlife Service, Edmonton, Alberta, Canada, 7 pp.
- , AND B. SJARE. 1988. Preliminary observations on the immobilization of male Atlantic walruses (*Odobenus rosmarus rosmarus*) with Telazol. *Marine Mammal Science* 4: 163-168.
- TRILLMICH, F. 1983. Ketamine xylazine combination for the immobilisation of Galapagos sea lions and fur seals. *Veterinary Record* 112: 279-280.

Received for publication 15 November 1988.