PHYSIOLOGIC AND ELECTROCARDIOGRAPHIC RESPONSES OF AMERICAN RIVER OTTERS (LUTRA CANADENSIS) DURING CHEMICAL IMMOBILIZATION AND INHALATION ANESTHESIA

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PHYSIOLOGIC AND ELECTROCARDIOGRAPHIC RESPONSES OF AMERICAN RIVER OTTERS (*LUTRA CANADENSIS*) DURING CHEMICAL IMMOBILIZATION AND INHALATION ANESTHESIA

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ABSTRACT: Rectal temperatures and heart rates of American river otters (*Lutra canadensis*) decreased significantly ($P < 0.05$) during chemical immobilization with i.m. ketamine hydrochloride in combination with xylazine hydrochloride and acepromazine and during inhalation anesthesia with isoflurane. Anesthetized otters showed a tendency for apnea during induction and while dorsally recumbent, which was reflected by a respiratory acidosis on arterial blood gases. Declines in rectal temperatures and heart rates were not found to be a function of dosage (mg/kg) of the ketamine combination used except for rectal temperatures of otters in relatively poor body condition (inanition). The electrocardiograms of isoflurane-anesthetized otters were similar to those recorded on immobilized otters with the exception of an r′ deflection in the ventricular depolarization complex (RSr′). Electrocardiographic criteria were not found which predicted the degree of right ventricular or generalized cardiac enlargement seen radiographically.

INTRODUCTION

American river otters were reintroduced into Oklahoma waterways in 1984 and 1985 pilot studies by the Oklahoma Department of Wildlife Conservation to determine habitat suitability. Otters acquired from southern Louisiana were evaluated clinically as to health status (Hoover et al., 1984, 1985) and surgically implanted with an intra-abdominal radiotelemetry device (Hoover, 1984) prior to their release. In this report, we present and compare the combined physiologic data gained during chemical immobilization and inhalation anesthesia of these two groups of river otters. Portions of this information have appeared previously in separate reports (Hoover, 1984, 1985; Hoover et al., 1984, 1985). Electrocardiographic and blood gas data for anesthetized river otters are included in this report.

MATERIALS AND METHODS

In 1984, 10 otters were immobilized and subsequently anesthetized. In 1985, 10 additional otters were immobilized initially on day 3; eight otters (two died, on days 5 and 7 respectively) were immobilized and subsequently anesthetized on day 15; and these eight otters were immobilized again on day 24.

Otters were restrained (immobilized) for clinical evaluations with a combination of ketamine hydrochloride (HCl) (Ketaset, Bristol Laboratories, Syracuse, New York 13221, USA), xylazine HCl (Rompun, Haver-Lockhart Bayvet Division, Shawnee, Kansas 66203, USA), and acepromazine (PromAce, Fort Dodge Laboratories, Inc., Fort Dodge, Iowa 50501, USA), and they were anesthetized for surgery with isoflurane (Forane, Ohio Medical Anesthetics, Madison, Wisconsin 53707, USA). Ten ml (1,000 mg) of ketamine HCl was mixed with 0.5 ml (10 mg) of xylazine HCl and 0.5 ml (5 mg) of acepromazine to arrive at the concentrations in the ketamine combination. This combination was given by intramuscular injection at a mean dosage of 16.8 mg/kg (range 13.1–21.9 mg/kg) of the ketamine HCl based on known body weights. Inhalation anesthesia was maintained by precision vaporizers via an endotracheal tube. The midrange isoflurane concentration was 1.25% (range 0.25–2.50%) with a midrange oxygen flow of 1.75 liters/min (range 1–3 liters/min). Heart and respiratory rates and rectal temperatures were monitored during immobilization and anesthesia.

Induction times for immobilization were measured from the injection of otters with the ketamine combination until all righting reflexes (coordinated ability to assume and maintain

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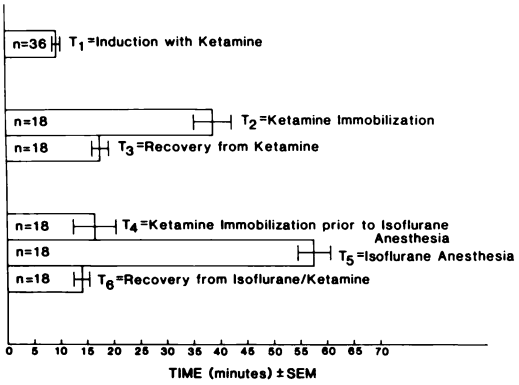

FIGURE 1. Duration of induction, immobilization, and recovery with ketamine combination, and the maintenance of isoflurane anesthesia and recovery in American river otters. T_1 = from ketamine combination injection to loss of righting reflexes; T_2 = from T_1 to return of righting reflexes; T_3 = from T_2 to being ambulatory and responsive to external stimuli; T_4 = from T_1 to initiation of isoflurane anesthesia; T_5 = from T_4 to termination of isoflurane anesthesia; and T_6 = from T_5 to return of righting reflexes.

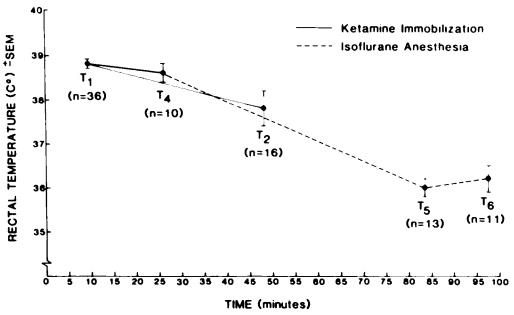

FIGURE 2. Rectal temperatures of American river otters taken during immobilization by ketamine combination and isoflurane anesthesia.

sternal recumbency) were lost. Immobilization time was measured from the end of induction until righting reflexes returned. Recovery times from immobilization were measured from the return of righting reflexes until the otters were ambulatory and had coordinated responses to stimuli from touch, sound, and sight. Anesthesia time was measured from the start of isoflurane with oxygen inhalation by immobilized otters until the termination of isoflurane administration. Recovery time from isoflurane/ketamine combination anesthesia was measured from the termination of isoflurane administration to otters until righting reflexes returned.

In 1985, arterial blood gas values of eight anesthetized otters were determined on a Corning 175 Automatic pH/Blood Gas System (Corning Medical and Scientific, Houston, Texas 77060, USA), and corresponding venous total CO_2 and anion gap values were determined on an RA1000 System (Technicon Corp., Tarrytown, New York 10591, USA). Values were calculated for venous bicarbonate using HCO_3^- = total CO_2 × 0.95 and for venous anion gap using AG = [Na^+ and K^+] − [Cl^- and HCO_3^-] (Schaer, 1982).

Thoracic radiographs were taken prior to obtaining electrocardiograms (ECG's) on the otters (Hoover et al., 1984, 1985). The ECG's were recorded as described previously (Hoover et al., 1984; Hoover, 1985) on the 10 otters while immobilized in 1984 and on the eight otters anesthetized with isoflurane in 1985 using an FD-31P ECG/Phono System (Fukuda Denshi Corp., Tokyo, Japan) as in 1984. Two additional chest leads were obtained in 1985 which approximate CV_6LU (C_3)—left sixth intercostal space at the costochondral junction—and V_{10} (C_4)—over the dorsal spinous process of the seventh thoracic vertebra (Detweiler and Patterson, 1965). Electrocardiographic amplitudes were measured to the nearest 0.05 millivolts and durations to the nearest 5 milliseconds.

The means, standard errors of the means (±SEM), standard deviations from the means (±SD), and ranges were determined for the data. Means were compared where appropriate by *t*-tests for paired dependent observations or for independent observations with equal or unequal variances as determined by *F*-test. The physiologic response data for immobilized and anesthetized river otters were analyzed by linear regression and two-way analysis of variance (ANOVA). The level of significance for all tests was $P < 0.05$.

RESULTS

The duration of effects for ketamine combination and isoflurane in river otters is presented in Figure 1. The ketamine combination at these dosages induced a smooth and rapid immobilization (T_1) of sufficient duration (T_2) for clinical evaluations. Otter recovery period from being able to maintain sternal recumbency to being ambulatory (T_3) was not considered excessive. The ketamine combination was also efficacious in tracheal intubation and induction of isoflurane anesthesia for ap-

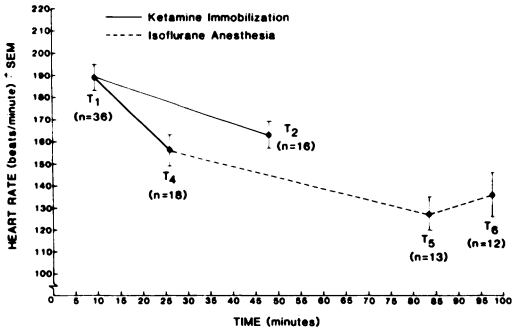

FIGURE 3. Heart rates of American river otters taken during immobilization by ketamine combination and isoflurane anesthesia.

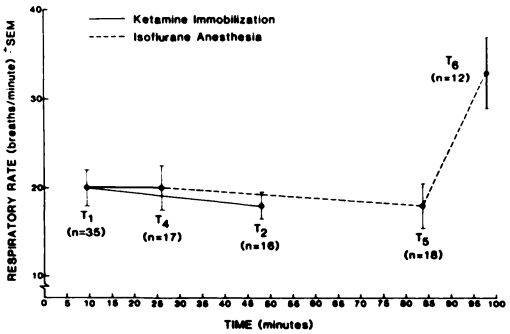

FIGURE 4. Respiratory rates of American river otters taken during immobilization by ketamine combination and isoflurane anesthesia.

proximately 15 min (T_4) following initial immobilization. Isoflurane produced good surgical anesthesia, the depth of which was controlled easily by precision vaporizers. Otter recovery from termination of isoflurane anesthesia (T_5) to being able to maintain sternal recumbency (T_6) was rapid and smooth.

The means (±SEM) for rectal temperatures are presented in Figure 2, and heart rates are presented in Figure 3. The decreases in rectal temperatures and heart rates during immobilization (from T_1 to T_2) and anesthesia (from T_1 or T_4 to T_5) were significant.

Respiratory rates of river otters are presented in Figure 4 and appeared to be unaffected during immobilization or anesthesia as long as otters were in sternal or lateral recumbency. The increases in respiratory rates during anesthesic recovery (T_5 to T_6) were significant.

Eight otters immobilized on day 3, 1985, had a significantly smaller decrease in heart rates (mean ± SEM = 9 ± 15 beats/min) than when immobilized on day 24 (mean ± SEM = 41 ± 18 beats/min) by t-test with equal variances (F' = 1.46, with 7 and 7 df). However, there were no significant differences by t-tests between days 3 and 24, 1985, in the respective decreases in rectal temperatures, respiratory rates, or the duration of immobilization in these eight otters. In a two-way ANOVA, the decreases observed in otter rectal temperatures and heart rates during immobilization (Figs. 2 and 3 from T_1 to T_2) and the duration of immobilization (Fig. 1, T_2) were not found to be a function of dosage (mg/kg) of ketamine combination administered or of the days (3 and 24) otters were immobilized. However, there was a significant linear regression model ($P < 0.01$, $R^2 = 0.68$) for the decreases in rectal temperature observed ($n = 8$) at T_2 versus dosage of ketamine combination administered on day 3, 1985:

$$\text{decrease rectal temperature (C)} = 8.91 - (0.69 \times \text{mg/kg ketamine})$$

The eight otters anesthetized for surgery on day 15, 1985, and the 10 otters anesthetized for surgery in 1984 were similar in rectal temperature and heart rate decreases, respiratory rates at termination of anesthesia (T_5) and recovery (T_6), and lengths of recovery from anesthesia by t-tests with equal or unequal variances as determined by F-test. In a two-way ANOVA, decreases in rectal temperatures and heart rates observed during anesthesia (Figs. 2 and 3 at T_5) and the duration of anesthetic recovery (Fig. 1, T_6) were not found to be a function of dosage (mg/kg) of ketamine combination administered for immobilization (anesthetic in-

TABLE 1. Blood gas determinations for eight American river otters under isoflurane and oxygen anesthesia.[a]

	Mean ± SD	Range
Arterial blood[b]		
pH	7.25 ± 0.06	7.16–7.33
pCO_2 (torr)	50.4 ± 7.2	41–62
pO_2 (torr)	352.8 ± 46.0	285–449
HCO_3^- (meq/liter calculated)	22.1 ± 2.3	18–26
BE (meq/liter calculated)	(−5.3) ± 3.0	(−11)–(−1)
Venous blood[c]		
Total CO_2 (meq/liter)	26.9 ± 2.4	22–30
Anion gap (meq/liter)	13.0 ± 4.0	8–21
Calculated[d] HCO_3^- (meq/liter)	25.5 ± 2.3	21–29
Calculated[d] anion gap (meq/liter)	14.4 ± 3.8	9.5–21.9

[a] Isoflurane (Forane, Ohio Medical Anesthetics, Madison, Wisconsin 53707, USA) and oxygen delivered by precision vaporizers via endotracheal tubes.

[b] Corning 175 Automatic pH/Blood Gas System (Corning Medical and Scientific, Houston, Texas 77060, USA).

[c] RA1000 System (Technicon Corp., Tarrytown, New York 10591, USA).

[d] Venous HCO_3^- = total CO_2 × 0.95 and anion gap = (Na^+ and K^+) − (HCO_3^- and Cl^-).

duction) or of the years otters were anesthetized.

The arterial blood gas and corresponding venous values for otters anesthetized with isoflurane and oxygen are presented in Table 1. The low value for pO_2 (285 torr) occurred in an otter with clinical bronchopneumonia. The high value for pCO_2 (62 torr) and low value for pH (7.16) occurred in an otter with apnea during the initiation of inhalation anesthesia.

The mean, standard deviation, and range for lead II ECG's and the % occurrence of the sum of positive and negative QRS deflections for all leads recorded on anesthetized river otters are presented in Table 2. The mean lead II ECG for anesthetized otters is illustrated in Figure 5, and the observed ranges are included. Comparisons were made between the ECG's of immobilized otters in 1984 and those of anesthetized otters in 1985. Anesthetized otters had significantly ($P \leq 0.05$) faster heart rates (beats/min) with correspondingly shorter PR and QT intervals (msec) by t-test for independent samples and equal variances ($F = 2.84$, 1.04, and 3.16, respectively, with 9 and 7 df). The ECG's of immobilized or anesthetized river otters did not have a Q wave (first negative deflection) in the QRS complexes (ventricular depolorization). However, seven of eight anesthetized river otters did have an r′ (second positive deflection) in the QRS complex (Fig. 5)—RSr′ complex—that was not seen in immobilized otters—RS complex.

DISCUSSION

In 1985, otters were held in captivity for 15 days prior to surgery and for 10 days after. During this holding period, there was considerable reduction in the number and severity of clinical abnormalities seen, a significant ($P < 0.05$) increase in mean (±SEM) body weight of 1.13 ± 0.31 kg, and a commensurate increase in body fat (Hoover et al., 1985). Based on thorough clinical evaluations, the eight otters anesthetized in 1985 (day 15) were considered to be in better health than the 10 otters anesthetized in 1984 (Hoover et al., 1985).

Immobilization with the ketamine combination was satisfactory for clinical evaluations and procedures involving minimal pain (Hoover, 1984; Hoover et al., 1985). Hyperthermia has been reported with the use of ketamine HCl in European otters (*Lutra lutra*) by Von Reuther (1983) and Von Reuther and Brandes (1984) and in one American river otter by Kane (1979). In these American river otters, there was a significant decrease ($P < 0.05$) in rectal temperatures by t-test for paired observations with the ketamine combination alone in 1985 (Hoover et al., 1985) and in

TABLE 2. Electrocardiograms[a] of eight American river otters during isoflurane anesthesia.[b]

Leads	Σ QRS complex deflections (%)					
	Pos.	Isoelectric	Neg.		Mean ± SD	Range
Standard				Heart rate	174 ± 9/min	160–180/min
I	100	—	—	Rhythm	NSR[c]	
II	100	—	—			
III	87.5	12.5	—	Mean electrical axis		
Augmented				Frontal plane	54 ± 13°	35–68°
AVR	—	—	100			
AVL	50	12.5	37.5	Lead II		
AVF	100	—	—	P (msec)[d]	51 ± 6	40–60
Chest				P (mV)[e]	0.24 ± 0.6	0.15–0.30
C_1	87.5	—	12.5	PR (msec)	86 ± 8	75–95
C_2	—	25	75	QT (msec)	184 ± 11	170–200
C_3	100	—	—	ST segment (mV)	(−0.01) ± 0.06	(−0.10)–(−0.05)
C_4	50	25	25	T (mV)	0.45 ± 0.11	0.30–0.60
				QRS (msec)	39 ± 5	30–45
				S (mV)	(−0.34) ± 0.25	(−0.70)–(−0.05)
				Q (mV)	0 ± 0	—
				R (mV)	1.82 ± 0.48	1.30–2.60
				r′	0.07 ± 0.04	0.00–0.10

[a] FD-31P ECG/Phono System (Fukuda Denshi Corp., Tokyo, Japan).
[b] Paper speed was 50 mm/sec, and sensitivity of 1 millivolt = 10 mm.
[c] NSR = normal sinus rhythm.
[d] msec = milliseconds.
[e] mV = millivolts.

conjunction with isoflurane in 1984 and 1985 (Hoover, 1984; Hoover et al., 1985). These decreases were found despite moderate ambient temperatures (15–22 C) during immobilization and efforts made to minimize body heat loss during surgery (Hoover, 1984; Hoover et al., 1985). The increased loss of body heat (decreased rectal temperatures) with increased dosage of ketamine combination used to immobilize otters on day 3, 1985 (which was not found on day 24, 1985) may have been associated with the relative lack of body fat stores (inanition) observed on clinical evaluations (physical examinations and radiographically) on day 3 (Hoover et al., 1985).

Increases in heart rate (25–50%) with the use of ketamine HCl have been reported in European otters by Jenkins and Gorman (1981). In these American river otters, there was a significant decrease ($P < 0.05$) in heart rates by t-test for paired observations with the ketamine combination alone in 1985 (Hoover et al., 1985) and in conjunction with isoflurane in 1984 and 1985 (Hoover, 1984; Hoover et al., 1985). During three of the 18 anesthesias, 0.05 mg/kg atropine sulfate (Eli Lilly and Co., Indianapolis, Indiana 46285, USA) given IV was efficacious in treating otters with moderate (<100 beats/min) but progressive bradycardia.

Anesthetized river otters tended to become apneic during periods of dorsal recumbency for surgery (Hoover, 1984), and ventilation assistance was given as needed to assure adequate exchange of oxygen and carbon dioxode. Arterial blood gases of anesthetized otters indicated the presence of a respiratory acidosis (low blood pH [<7.30–7.45] and elevated pCO_2 [>29–42

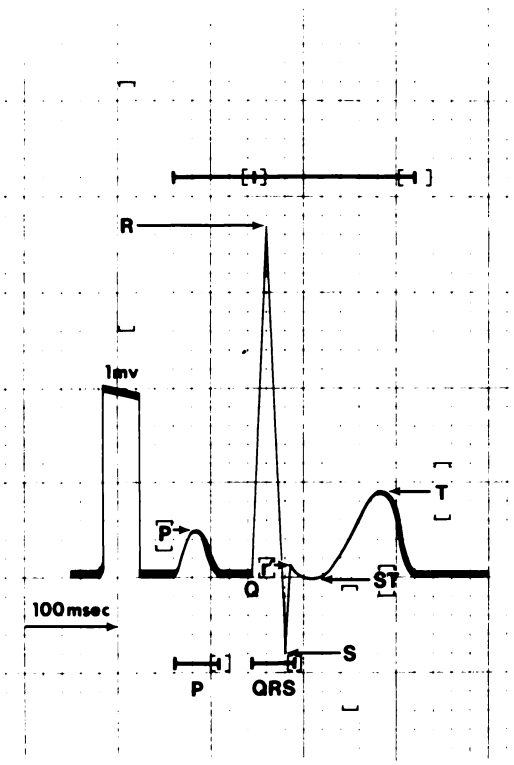

FIGURE 5. Scale representation of the mean lead II electrocardiogram for American river otters anesthetized with isoflurane at paper speed 50 mm/sec. Small box equals 0.1 mV on a vertical axis and 20 msec on a horizontal axis. The observed ranges are contained within the brackets.

torr] with normal serum bicarbonate [17–24 meq/liter] [Schaer, 1982]). This demonstrates the importance of monitoring river otters for apnea during anesthetic induction and anesthesia, and providing adequate oxygen flow (≥2 liters/min) and ventilation assistance when needed.

On the basis of thoracic radiographs taken at the time of electrocardiograms, three river otters had generalized cardiomegaly; one had right ventricular enlargement which was attributed to cor pulmonale; and one had mild idiopathic right ventricular enlargement (Hoover et al., 1984, 1985). When ECG's of these otters were compared with those of the 13 otters having normal radiographic cardiac silhouettes, no accepted criteria (Ettinger and Suter, 1970; Tilley and Gompf, 1977; Tilley, 1985) were found that predicted these radiographic findings. The severity of radiographic cardiac changes seen in these otters does not appear to be sufficient to result in significant alteration of the ECG's. The RSr′ complex (Tilley, 1985) may suggest some alteration in the relative rates of right and left ventricular depolarizations (myocardial depression) of river otters under isoflurane anesthesia.

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BOOK REVIEW . . .

Handbook of Diseases of Saskatchewan Wildlife. Gary A. Wobeser. Saskatchewan Parks and Renewable Resources, 3211 Albert Street, Regina, Saskatchewan S4S 5W6, Canada. 1985. vii + 65 pp. $10 Canadian.

This publication is aimed at hunters and naturalists, as well as biologists and wildlife technicians in Saskatchewan, who may encounter abnormalities in tissue, or sick or dead wildlife. Its objective is to explain the cause and significance of diseases or conspicuous lesions, and the significance of some parasites. Despite the narrow geographic scope, and the unsophisticated technical background of the intended audience, this publication would be a useful first reference for wildlife biologists, veterinarians, laboratory diagnosticians and students of wildlife disease throughout northern North America. Anecdotal and observational information is presented that is not readily available elsewhere. It joins similar publications describing wildlife diseases in Ontario, Colorado, Alaska and Wyoming, and is among the best of this genre.

Presented in a small (15 × 23 cm) staple-bound format readily carried in the field, this booklet is written in simple language, and the text is supplemented by a Glossary of Technical Terms. Fifteen conditions affecting birds, and over 40 affecting mammals, are considered in the 50 pages of text, under the etiologic groupings of Bacterial, Viral, Fungal, Parasitic, Toxic and Non-infectious Diseases. Each condition is considered succinctly under the headings: Cause; Species Affected; Occurrence in Saskatchewan; Ecology; Clinical Disease; Pathology; Specimens for Diagnosis; and Significance. There is a clear Table of Contents, but no index. The text is illustrated by 60 color figures, and is supplemented by a section on Specimen Collection and Handling and a brief Bibliography. There is an Appendix consisting of four tables listing conditions encountered in hunter-killed game; zoonoses; diseases of the skin or external surface; and causes of abnormal behavior in wild mammals. While not all conditions described in detail are listed in relevant tables, these do provide a useful point of entry to the text, provided the reader consults the Table of Contents. A section addressing the issue of rehabilitation of injured birds is included.

The conditions selected for discussion are appropriate, and the content of each section is clearly written, generally in sufficient detail to permit understanding, while avoiding irrelevant facts. Significant errors of omission are not evident, spelling mistakes are rare and the information presented is, with only arguable exceptions, accurate. The author seems to have relied upon authorities with which some would quibble when describing the host ranges of some cestodes. Reference to yersiniosis as a zoonosis might have been tempered by an observation that wildlife have not (at least to this reviewer's knowledge) been implicated directly as a source of human infection in North America. Giardiasis, which is not referred to in this booklet, may be much more significant in this context. The illustrations all complement the text, and their quality is good, though some might benefit from the use of arrows to guide the lay reader to the lesion. Diagrams might have helped clarify the life-cycles of some parasites.

The author is to be commended for making his extensive knowledge and experience so clearly available to his "public." I am sure that they, in turn, will reward him with further opportunities to gather more "bits and pieces" to contribute to our understanding of wildlife diseases, in Saskatchewan, and more generally.

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