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Source: Journal of Wildlife Diseases, 8(3) : 287-295

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-8.3.287

ANESTHESIA OF THE HARP SEAL [1]

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Abstract: The harp seal *(Pagophilus groenlandicus)* was anesthetized with halothane following induction with halothane/nitrous oxide or thiopental sodium. Halothane concentrations of 0.75 - 1.5% were required for surgical anesthesia. The depth of anesthesia was best assessed by heart rate, muscle relaxation and the presence or absence of shivering. Controlled ventilation was required throughout anesthesia and CO_2 homeostasis was maintained by tidal volumes of 22-25 ml/kg at a rate of 10/min. There were two anesthetic deaths: one related to hypothermia, and one to a prolonged induction period.

INTRODUCTION

Anesthesia of pinnipeds presents special problems because of their adaptations for aquatic life[2]. The ability to dive is associated with extensive cardiopulmonary modifications enabling the seal to endure prolonged periods of apnea and great increases in external pressures[1,4,10,11,15]. The conscious seal may respond to noxious stimuli, handling, or strange surroundings with a "dive-like" response which complicates the administration of inhalant or intravenous anesthetics[2].

Pinnipeds have been anesthetized with diethyl ether[9], diethyl ether and nitrous oxide[6], barbiturates[3,7], methoxyflurane[3,8] and halothane[5,14]. The only detailed report describes the use of halothane anesthesia for the California sea lion[14]. Anesthesia for the harp seal has not previously been reported.

MATERIALS AND METHODS

General anesthesia was used 12 times on eight harp seals ranging in age from 3 months to 11 years. The procedures carried out were reduction of a fractured humerous, anesthesia evaluation and electrode placement adjacent to the cochlea. The duration of anesthesia varied from 60 - 250 min. Six additional animals were anesthetized to collect tissue for electron microscopy prior to euthanasia.

Restraint for anesthesia of the harp seal was achieved by strapping the animal to a restraint board fitted with aluminum hoops and car seat-belts. No preanesthetic medication was used.

Induction of anesthesia was achieved via a semi-open system of gas administration in 13 instances. A one-way flow of gas was passed into a loose fitting clear polyethylene bag placed over the head of the seal. The clear bag facilitated observation of the animal and had less tendency to evoke a dive response. To prevent CO_2 accumulation gas flows of 8 - 10 L/min. were maintained allowing the excess gas to escape through the distal end of the bag.

Halothane was gradually added after a brief period during which the seals were allowed to become accustomed to the face-mask and gas flow (O_2 40% and N_2O 60%). Over a 5-10 min. period, the halothane concentration was increased from 0 to 5%. It was only possible to induce anesthesia by this method in young pups. Older animals would either go into a dive response or simply stop breathing as the concentration of halothane reached 1 - 2.5%.

[1] Paper presented at the International Association for Aquatic Anial Medicine Conference, University of Guelph, Guelph, Ontario, Canada, April 29-30, 1971.

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A special vaporizer was used to circumvent this problem. The standard halothane vaporizers will produce maximum concentrations of 5 or 10%. By introducing an ether wick vaporizer[3] into the inflow line, an estimated concentration of 20-30% halothane could be produced. This way, even if the animal took only a few breaths before or between diving responses, enough halothane was inhaled and absorbed to create anesthesia and muscle relaxation. An endotracheal catheter was then rapidly inserted and controlled ventilation was commenced. If the seal was too deeply anesthetized, the excess halothane was quickly removed by ventilation with O_2.

The difficulties encountered with inhalation induction stimulated five trials using the intravertebral extradural vein for the injection of a thiobarbiturate. Thiopental sodium was administered slowly as a 1% solution.

To achieve endotracheal intubation the seal was positioned in sternal recumbency and the jaws were held apart by an assistant. The tongue was pulled forward and the epiglottis was depressed with a laryngescope (Figure 1), allow-

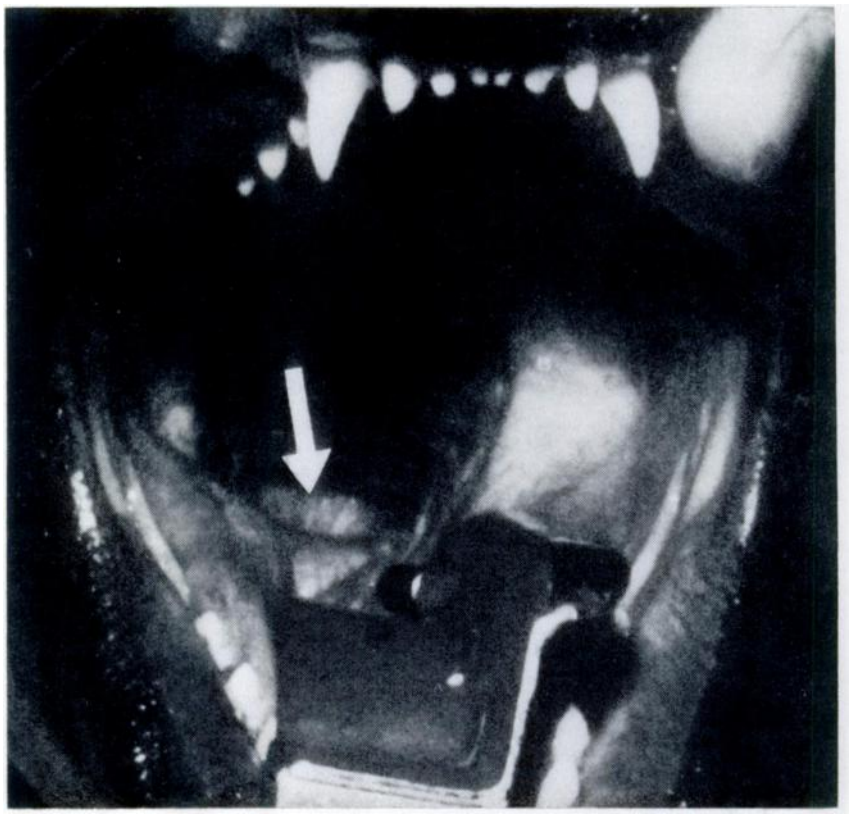

FIGURE 1. View of the larynx of a seal positioned in sternal recumbency. The arrow points to the closed vocal cords.

[3] No. 8 Ether vaporizer, Ohio Chemical (Canada), Montreal.

ing insertion of a cuffed endotracheal catheter (26-38 French) under direct vision. Visualization of the larynx was difficult because of the narrow pharynx and folds of mucous membrane lateral to the vocal cords. Larygospasm was frequently present and topical desensitization with a lidocaine aerosol did not seem to be of much benefit. The lubricated endotracheal catheter was gently, but firmly, inserted into the larynx. Occasionally, it was necessary to use a small catheter initially, replacing it with a larger one when the depth of anesthesia was stabilized.

Following induction of anesthesia the nitrous oxide flow was discontinued and anesthesia was maintained with halothane using a semi-closed circle system with O_2 flows of 4-5 L/min. The seals were kept in as light a level of anesthesia as was compatable with the surgery. In most instances, a venous access was established into the hind flipper or extradural vein and 10 ml/kg of a balanced electrolyte solution[4] was administered during the period of surgery.

Heart rate and rhythm were determined by electrocardiographic monitoring[5]. Three electrodes were used with one placed under each fore flipper and the third one just ventral and posterior to the heart. A vane type respirometer[6] was used to measure ventilation frequency and volume. Ventilation was controlled throughout and, in most cases, the frequency was arbitrarily set at 10/min. Periodically blood samples were obtained for assessment of acid-base equilibrium. Body temperature (rectal) was monitored continuously.

RESULTS

Figure 2 depicts a typical anesthetic record. Despite the avoidance of premedication with an anticholingeric agent, tracheo-bronchial secretions and salivation were minimal. Laryngeal stimulation during endotracheal catheterization did not result in reflex alteration of the heart rate.

Induction with halothane/nitrous oxide usually took 15-20 min. and it was successful in 12 of 13 instances. The one exception was an 11 year old female that had not adapted well to captivity. The animal remained anorectic and resisted handling for the 3 months after capture. During induction this seal went into a dive response that lasted 40 min. Eventually, respirations commenced, induction was completed, and the animal was stabilized on halothane. However, 30 min. after induction, cardiac asystole occurred. Surgery was discontinued and a heart beat was temporarily restored with external cardiac massage. Ventricular fibrillation followed and this did not respond to resuscitative procedures. It was not possible to obtain acid-base data from this particular seal.

Intravenous administration of thiopental sodium via the extradural vein resulted in a smooth, rapid induction even in large aggressive seals. All five seals showed apnea without bradycardia following administration of 2-4 mg/kg. Palpebral, pupillary and oral reflexes were present and the seals were capable of incoordinated movement. Under these circumstances cyanosis could be detected within 90-120 sec. and it was necessary to rapidly administer more anesthetic so that endotracheal intubation could be achieved and controlled ventilation commenced. This required a total dose of 5-8 mg/kg thiopental sodium.

Typically, the concentration of halothane required for maintenance of surgical anesthesia varied between 0.75 and 1.5%. It was often difficult to assess anesthetic depth as the classical reflexes were of little value. Table 1 lists the changes in signs, or reflexes, observed with varying anesthetic depths.

The preanesthetic heart rate varied from 80 - 140/min. with the seal resting

[4] Normosol, R., Abbot, Montreal, Canada.
[5] Birtcher Model 335 electrocardiograph and model 425 oscilloscope, J. R. Stevens Co., Toronto, Canada.
[6] Wright Respirometer, Canox Ltd., Toronto, Canada.

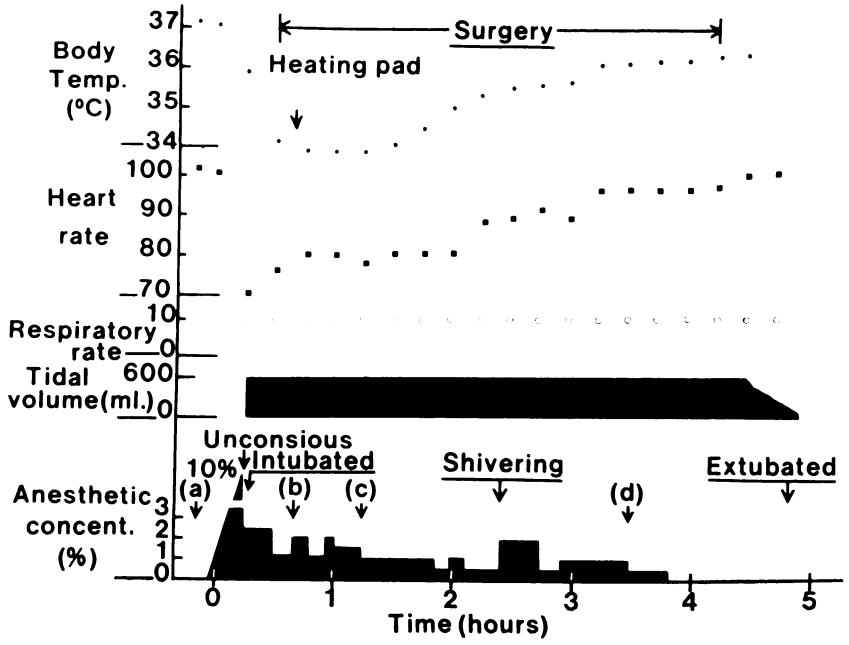

FIGURE 2. Various parameters during halothane anesthesia in a 3 month old harp seal weighing 30 kg. Halothane concentrations in excess of 10% were used for induction of anesthesia. Venous blood (flipper vein) was periodically collected for acid-base evaluation (a,b,c,d). Moderate hypercapnia (P_vCO_2 60 - 65 mm Hg) and acidosis (pH 7.21 - 7.30) was present throughout anesthesia.

TABLE 1. Monitoring Anesthetic Level (Halothane) in the Harp Seal.

	Increasing Depth of Anesthesia		
Sign or Reflex	Light	Surgical	Deep
Heart Rate	Stable or increased	Stable	Decreased
Respiration	Apnea	Apnea	Apnea
Shivering and/or movement	Yes	No	No
Response to painful stimuli	Yes	No	No
Palpebral and corneal reflexes	Absent	Absent	Absent
Muscle relaxation (jaw tone)	Poor	Moderate	Complete
Capillary refilling time	Rapid	Moderate	Slow

on the restraining board. Post-induction heart rate was 72 - 110/min. and the rate during maintenance halothane anesthesia was 80 - 110/min., with the lower rates usually occurring during deeper anesthesia. The heart rate was usually quite stable for any particular seal under a constant level of anesthesia.

Significant arrhythmias were not observed in any of the successful anesthesias, even during induction. In the two unsuccessful anesthesias, electrocardiographic monitoring using standard Lead II did provide advance warning of circulatory failure with ventricular extrasystoles, atrioventricular block, asystole and ventricular fibrillation being observed. The responses of seals to emergency cardiac drugs (epinephrene, isoproterenol, calcium gluconate, sodium bicarbonate) and cardiac defibrillation appeared to be similar to that of terrestrial mammals.

Table 2 lists the results of blood acid-base analyses obtained from venous blood samples collected at varying periods throughout anesthesia. At a frequency of 10/min., tidal volumes (V_t) of 22 to 25 ml/kg were required to maintain CO_2 homeostasis. There was no appreciable metabolic acidosis during the period of anesthesia and any acidemia was due to CO_2 retention.

Typically body temperature dropped suddenly from a preinduction value of 36 - 37C, to a post-inductive value of 34C. The temperature could be kept at 34 - 35C by application of a thermostatically controlled heating pad at 40C. Simultaneous measurement of rectal, esophageal and skin temperature in one seal showed no appreciable difference between rectal and esophageal temperature throughout the period of anesthesia (Figure 3). Skin temperature (lateral thorax) was initially far less than rectal temperature but increased steadily following induction until a plateau was reached 120 min. after induction.

In one of the earlier anesthesias, no attempt was made to support body temperature and the result was a progressive

TABLE 2. Acid-Base Equilibrium with Controlled Ventilation During Anesthesia*.

Seal	Time after Induction (min.)	Body Temp. (C)	V_t f	V_t (ml/kg)	pHv	Pv_{CO2} mm Hg)	HC03 (mEq/L)
A_2 (two years)	Pre-induction	35.2			7.35	55	28.5
	+30	34.0	10	10	7.09	96	28.0
	+42	33.5	10	20	7.22	68	27.0
	+48**	34.0	25	10-20	7.24	52	22.6
B_5 (one year)	+20	33.8	10	20	7.20	68	26.0
	+35	33.5	10	20	7.21	70	27.5
	+80	35.0	10	22	7.22	64	25.6
	+155	35.0	12	22	7.34	56	30.0
C_6 (3 mos.)	Pre-induction	37.1			7.39	48	28.5
	+20	34.1	10	18	7.21	65	25.5
	+60	33.4	10	18	7.29	62	29.0
	+195	36.0	10	18	7.30	60	28.8
C_8 (8 mos.)	+90	35.0	10	25	7.41	40	25.1

* The acid-base values were determined at 37C and the reported values are not corrected to body temperature. Ventilation volumes are at BTPS.

** At recovery with the seal conscious and breathing spontaneously.

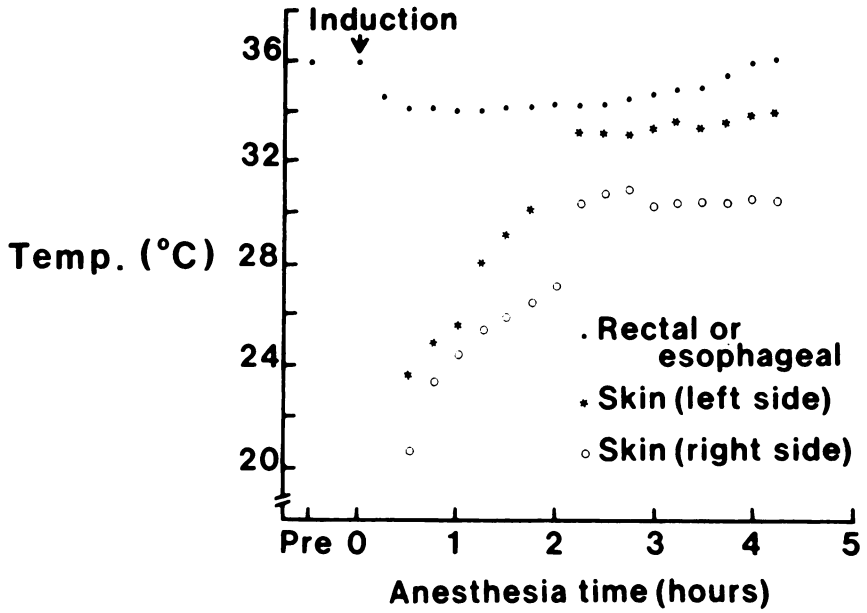

FIGURE 3. Temperature changes during halothane anesthesia in a 1 year old harp seal. Immediately after induction of anesthesia the seal was placed on a heating pad maintained at 40C.

and fatal hypothermia. An atrioventricular block appeared 45 minutes after induction at a body temperature of 27C (Figure 4). The block was progressive and unresponsive to atropine or isoproternol. The arrhythmia eventually progressed to asystole and, although resuscitation procedures were temporarily successful, ventricular fibrillation followed. It was possible to electrically defibrillate the heart but after a brief period, it would once more fibrillate so resuscitative efforts were terminated.

The recovery time following termination of halothane administration varied with the length of anesthesia, but was never over 1 hour. Usually, halothane administration was discontinued prior to the end of surgery allowing recovery soon after completion of the operation.

When the seal started to shiver, ventilation frequency and volume were decreased to permit CO_2 accumulation. Return of consciousness was evidenced by flipper and chewing movements, and a return of palpebral and eye preservation reflexes. At this point, the endotracheal catheter was removed and spontaneous ventilations normally resumed. Two animals, although fully conscious, appeared to have some residual respiratory depression with failure to resume regular ventilation rhythm. Bradycardia was not evident. Intravenous administration of 0.25 mg/kg doxapram hydrochloride[7] illicited a dramatic response with full resumption of respiratory rhythm and amplitude as well as increased reflex responsiveness.

All recovered seals were fully ambulatory in 2 or 3 hours after termination of anesthesia, although they were kept in shallow water (4 - 8 cm) for the first 24 hours. None of the animals showed any complications in the post-anesthetic period that were related to anesthesia.

[7] Dopram, A. H. Robins Co., Montreal.

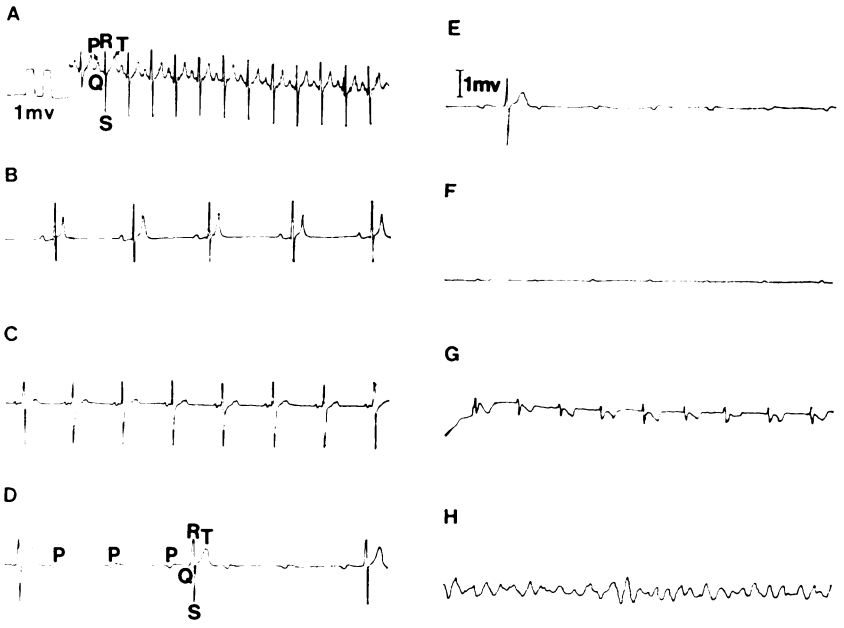

FIGURE 4. Electrocardiographic changes recorded during halothane anesthesia in a 1 year old harp seal. Lead II, paper speed 25 mm/sec. (A) Preanesthetic control, (B) Post-induction 5 min, (C) 30 min, (D) 45 min. — Atrioventricular block. Body temperature of the seal was 27C, (E) Artiroventricular block is more pronounced, (F) Cardiac asystole with atrial contractions, (G) Post-resuscitation recording, (H) Terminal ventricular fibrillation.

DISCUSSION

In harp seal pups and young adults halothane/nitrous oxide induction was safe and controllable, although relatively slow. The more favourable reports on inhalation induction have either been in the eared seals[14] or young animals[6,9]. Older animals were able to sustain prolonged periods of apnea seriously interfering with the uptake of the inhalation agents. The anesthetic death of the 11 year old female was undoubtedly due, in part, to the prolonged dive response during induction resulting in metabolic and respiratory acidosis. Upon termination of the dive response, induction was likely completed before oxygen depletion and the acidosis were corrected. Subsequent controlled ventilation was probably insufficient to eliminate the CO_2 accumulation, although it is only possible to speculate since blood samples were not obtained.

Thiopental sodium administration via the extradural vein provided a rapid and dependable means of induction, and this was particularly advantageous in the larger more aggressive seals. Rapid intubation and commencement of controlled ventilation was essential. The thiobarbiturate did not appear to pool in the peripheral circulation and there was good correlation between administered dose and central nervous system depression. There were insufficient recovery cases to know if thiopental sodium induction prolongs recovery or results in residual respiratory depression. Similar techniques have been used with success in the harbour seal[6,7] and California sea lion[5].

The concentration of halothane required for surgical anesthesia varied between 0.75 and 1.5% and this is similar to requirements for the California sea lion[11]. The degree of anesthetic depression was best assessed by heart rate, muscle relaxation and the presence or absence of shivering.

All narcotics and anesthetics depress the ventilatory response to CO_2 in therapeutic and anesthetic dosages and clinical usage is usually associated with a degree of alveolar hypoventilation and respiratory acidosis. The respiratory center of the seal appears to be particularly susceptible to drug depression, perhaps because of a reduced ventilatory response to CO_2[9,10].

Intravenous or inhalation general anesthesia of the harp seal always resulted in respiratory arrest, often occurring prior to the onset of unconsciousness and muscle relaxation. The Weddell seal apparently responds similarly, whereas the harbour seal may or may not become apneic and the California sea lion usually does not become apneic[13]. Nevertheless, to prevent hypoventilation, assisted or controlled ventilation is recommended even with the latter species[11]. The alveolar or effective ventilation should be such as to maintain CO_2 homeostasis, particularly if the duration of anesthesia is long. At a frequency of 10/min., tidal volumes of 22-25 ml/kg were sufficient to prevent hypercapnia.

General anesthesia would seem to modify the regulatory mechanisms associated with the cardiovascular response to a dive. The sudden onset of bradycardia typical of a dive response was never seen in an anesthetized harp seal subjected to surgical stimulation, even when under light enough anesthesia to permit shivering. Perhaps because of this, anesthetized seals cannot tolerate respiratory arrest with cessation of oxygen intake much longer than non-diving mammals. It was not unusual to see cyanosis of the oral mucous membranes following thiobarbiturate induction of anesthesia if intubation took longer than 90 - 120 seconds. In contrast, periods of apnea of up to 10 minutes are tolerated following succinylcholine chloride immobilization[12].

Agents which product a marked hypothalamic depression, ganglionic block, or peripheral vasodilation will alter thermoregulatory control in Pinnipedia[14]. Phenothiazine tranquilizers, barbiturates, methoxyflurane and halothane are all capable of inducing hypothermia in pinnipeds. With halothane the hypothermia is likely associated with peripheral vasodilation, since skin temperature approached core body temperatures following induction of anesthesia. Hypothermia potentiates anesthesia and interferes with cardiac conduction. Body temperature must be monitored carefully during seal anesthesia and some means of safely administering external heat must be available.

Acknowledgement

Dr. K. Ronald, Dean, College of Biological Science, University of Guelph, generously provided the opportunity for the author to work with the harp seals and to utilize them for anesthesia studies. A number of graduate students and support staff in the Zoology Department, University of Guelph provided technical assistance.

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Received for publication May 28, 1971