BioOne COMPLETE

CARDIOVASCULAR DEPRESSION AND THERMOREGULATORY DISRUPTION CAUSED BY PENTOTHAL/HALOTHANE ANESTHESIA IN THE HARBOR SEAL, Phoca vitulina 1

Author: SINNETT, EVERETT E.

Source: Journal of Wildlife Diseases, 17(1) : 121-130

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-17.1.121

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.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

CARDIOVASCULAR DEPRESSION AND THERMOREGULATORY DISRUPTION CAUSED BY PENTOTHAL/HALOTHANE ANESTHESIA IN THE HARBOR SEAL, *Phoca vitulina* [1]

EVERETT E. SINNETT, [2] Physiological Research Laboratory, Scripps Institution of Oceanography, La Jolla, California 92093, USA.

ERIC A. WAHRENBROCK, Anesthesia Research Laboratory, School of Medicine, University of California, San Diego, La Jolla, California 92093, USA.

GERALD L. KOOYMAN, Physiological Research Laboratory, Scripps Institution of Oceanography, La Jolla, California 92093, USA.

Abstract: Anesthesia was induced in the harbor seal *(Phoca vitulina)* with an intravenous injection of 10 mg/kg thiopental sodium; this was followed by halothane (1%) anesthesia for up to 9.5 h. Cardiac output was reduced to 30% of the pre-anesthesia value (from an average of 11.5 l/min to 3.5 l/min) while systemic blood pressure fell from an average 150/110 to 80/60. Arterial oxygen partial pressures were somewhat depressed (58-72 Torr) during ventilation with air. Heart rate became stable at 90-100 beats/min. Hypothermia was an occasional problem during the first hour of anesthesia, but this trend reversed and gave way to hyperthermia during prolonged anesthesia.

INTRODUCTION

In recent years, the use of halothane for general anesthesia in marine mammals has become widely accepted. Induction of anesthesia can be accomplished in otariid seals (sea lions and fur seals) by placing a cone over the animal's head and passing a halothane gas mixture through the cone.[12] This type of induction has been found to be troublesome and somewhat dangerous in the harp seal[6] (one of the phocid, or "true" seals — harbor, grey, Weddell, etc.). Induction of anesthesia in harp seals,[6] grey seals,[9] and porpoises[10] can be safely accomplished with an intravenous (i.v.) injection of sodium pentothal; after tracheal intubation, anesthesia can be continued with halothane alone. The animals described appear to tolerate anesthesia well, provided body temperature is maintained, and recovery is prompt, particularly if anesthesia has not been long. However, cardiac output has not been measured in any of these species under anesthesia, and blood pressure during anesthesia has been reported only for the porpoise.[10] This report deals with cardiovascular responses and skin and core temperature changes in harbor seals anesthetized with pentothal/halothane.

MATERIALS AND METHODS

One female and three male harbor seals ranging in age from 12 to 22 months were anesthetized a total of 15 times using pentothal induction followed by 1% halothane anesthesia as part of an experiment on pulmonary blood flow.[14] The female and one of the males had been in

[1] Paper presented at the International Association for Aquatic Animal Medicine Conference and Workshop, San Diego, California, USA, April 23-27, 1978.

[2] Present address: Division of Lung Diseases, N.I.H., Westwood Bldg., Rm. 6A03, 5333 Westbard Avenue, Bethesda, Maryland 20205.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

captivity since shortly after birth. After weaning from a cream, fish, and vitamin formula, they were maintained on frozen herring and vitamins at the Scripps seawater tank facilities. The other two male seals were beached animals kindly supplied by Dr. L. Cornell of Sea World, Inc. They had been cured of respiratory infections and intestinal parasites at least two months before they came under our care and appeared in good health. The four seals ranged in weight from 35 to 52 kg at the time of the experiments, and had steadily gained weight while in our care.

Each seal underwent two experimental procedures which were separated in time by no less than four weeks. The protocol for each procedure can be outlined as follows. Uninstrumented seals were anesthetized and catheters were placed in the pulmonary artery and descending aorta; measurements of blood pressure and flow were made once the catheters were in place. Following a recovery period of at least six h, measurements of blood pressure and flow were made in awake animals resting quietly on a restraint board. The seals were reanesthetized 24 to 48 h after the end of the first period of anesthesia for the removal of the catheters. This entire procedure (anesthesia for catheter placement, awake measurements, and anesthesia for catheter removal) was accomplished successfully seven times; on one other occasion the catheters could not be positioned properly during the initial period of anesthesia. A more detailed description of the procedures follows. While a venous catheter was available for direct i.v. injections in the instrumented animals, the uninstrumented animals had to be given the pentothal percutaneously. The extradural intravertebral vein[3] was used, and restraint was required to assure intravenous injection. For animals of this size this can be accomplished simply by covering the mouth and eyes of the seal with a large rag; the hind flippers are then held by one assistant while another straddles the seal and restricts body and head movements of the seal with his knees and hands. The pentothal can then be injected with a 9 cm 22 gauge spinal needle by a third person.

Thiopental sodium[2] (25 mg/ml, 10 mg/kg) was rapidly injected intravenously, either through a catheter into the main pulmonary artery or percutaneously into the extradural intravertebral vein. Muscle relaxation was almost immediate, as was the cessation of respiration. The animals were placed on their backs, and tracheal intubation with an appropriate cuffed endotracheal tube was accomplished with the aid of a standard human laryngoscope.

The seals were ventilated mechanically throughout the period of anesthesia with a piston driven pump. A positive end expiratory pressure of one to five cm H_2O was maintained at all times. Gas mixtures ranging from 20-50% O_2 in N_2 with 0.75 to 1% halothane[4] were used to maintain anesthesia. End tidal CO_2 and halothane concentrations were monitored[5] and adjustments in tidal volume, frequency, and inspired halothane were made to maintain concentrations of CO_2 and halothane close to 5.3 and 1%, respectively.

The initial period of anesthesia, for catheter placement and cardiac output measurement, was always the longest. Heart rate[6][7] and body (rectal)

[2] Pentothal®, Abbott Laboratories, North Chicago, Illinois 60064, USA.

[4] Fluothane®, Ayerst Laboratories, Inc., New York, New York 10017, USA.

[5] Model LB-2 medical gas analyzer, Beckman Instruments Inc., Fullerton, California 92634, USA.

[6] Brush EKG preamplifier for Model 200 Multichannel recorder, 3631 Perkins Ave., Cleveland, Ohio 44114, USA.

[7] Model 7788A eight channel recorder, Hewlett-Packard, Palo Alto, California 94304, USA.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

temperature [8] measurements were obtained within 5 min of the induction of anesthesia and were monitored throughout this period of anesthesia. An arterial catheter was inserted into either the right or left fore flipper artery and was advanced until its tip was located in the descending aorta, as determined fluoroscopically.[9] When it had been advanced to this position it was possible to obtain blood pressure [10] and blood samples for gas analysis;[11] when the catheter tip was located more peripherally, blood pressure was either damped or unreadable, and blood samples were unobtainable. A Swan-Ganz thermal dilution catheter [12] was inserted into the external jugular vein and was advanced until its tip had been positioned in the main pulmonary artery. Fluoroscopy [9] was required for this procedure also, and, along with blood pressure measurements, [7] [10] was used to confirm proper positioning. The catheter was used for determining cardiac output by the thermal dilution technique.[5]

Once the catheters had been properly positioned, a set of measurements were made while the seals were still under anesthesia. These measurements included cardiac output, systemic blood pressure, body temperature, heart rate, and blood gases. The halothane was then turned off, lidocaine [13] was infiltrated around the incisions, and the seals were allowed to recover. Shortly after extubation they were placed on a foam rubber-lined metal restraint board in the shape of a truncated V. Two canvas flaps fastened to the sides of the board were laced over the seal's back and completed the restraint. The lacing was kept loose enough to allow easy breathing, but tight enough to prevent undue movement and to protect the catheter positioning. The seals accepted the restraint quite well and did not appear stressed; in fact, they quite often appeared to fall asleep on the board.

The seals were allowed several hours to recover from the anesthesia before more measurements of cardiac output, blood pressure, blood gases, and heart rate were made. Several sets of such awake, quiet measurements were recorded over a 24 to 48 hour period, and these serve as our reference measurements for the anesthesia study.

When the experimental protocol on the awake seals had been completed, the seals were reanesthetized, as already described, for catheter removal. This procedure was generally performed as rapidly as possible with minimal monitoring of the animal (heart rate and rectal temperature); however, time was taken during two of these procedures to measure skin (dorsal midline approximately midbody) and pulmonary arterial temperature (via the Swan-Ganz thermistor); on one of these occasions systemic blood pressure was also measured until the catheter was removed.

After the removal of the catheters, antiseptic analgesic powder [14] was applied and the wounds were closed. The seals were returned to the water within 3 to 12 h and ate normally shortly thereafter. They were given tetracycline [15] prophylactically for a week, and recovery was rapid and uneventful.

---

[8] Model 421 Thermistor Probe and Model 43TD Tele-thermometer, Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio 45387, USA.

[9] Philips Medio 50, 2785 Kurtz Street, San Diego, California 92120, USA.

[10] Statham P23 Db transducer, Gould-Statham Instruments, Inc., Hato Rey, Puerto Rico 00919, USA.

[11] Radiometer model BMS-3 and PHM-72 blood gas analyzer and meter, London Co., Westlake, Ohio 44145, USA.

[12] Edwards Laboratories, Santa Ana, California 92705, USA.

[13] Xylocaine® hydrochloride, 1%, Astra Pharmaceutical Products Inc., Worcester, Massachusetts 01606, USA.

[14] Eye and Wound Powder, Fort Dodge Laboratories, Inc., Fort Dodge, Iowa 50501, USA.

[15] Tetrachel®, Rachelle Laboratories, Inc., Long Beach, California 90801, USA.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

RESULTS

The results of the studies on awake, quietly resting seals will be presented first, for the effects of anesthesia to be appreciated more fully.

The heart rate of the quietly resting harbor seal can vary considerably, depending on the respiratory cycle; during periods of spontaneous apnea, sometimes lasting up to one minute, the heart rate fell as low as 24 beats/min. During periods of tidal ventilation the heart rate was 60-70 beats/min; after the longer apneic periods or during struggling, the heart rate rose as high as 120-130 beats/min. Cardiac output was also affected by the respiratory cycle. The mean of 134 determinations was 11.5 l/min, but during apneustic periods it was observed to drop as low as 2.9 l/min. Despite the variations in heart rate and cardiac output, systemic blood pressure was maintained within a narrow range. This range varied from seal to seal, being as high as 160/120 Torr and as low as 140/100 Torr. Blood gas values were apparently affected by respiration also; samples drawn at random had arterial oxygen partial pressures (PaO_2) averaging 72 Torr with a range of 38-101 Torr, while samples drawn at the end of a period of rapid ventilation had PaO_2's averaging 96 Torr, with a range of 87-107 Torr.

Figures 1 and 2 show the effects of the pentothal injection and up to an hour of halothane anesthesia on heart rate, systemic blood pressure, and pulmonary arterial and skin temperature. These measurements were made in one seal during the catheter removal stage at the end of two separate experiments. In both cases they show that the heart rate rose sharply with the injection, remained elevated for about 10 min, and then stabilized at 95-100 beats/min. The systemic blood pressure (measured in one experiment) showed a narrowing of the pulse pressure within seconds of the injection, and a falloff of the mean pressure starting 10 to 15 min after the injection. Figure 2 shows skin and pulmonary artery temperatures for the two experiments. Skin temperature at the position measured rose to within 2.5 degrees C of core body temperature within one minute, and was within 1 C of the pulmonary artery temperature less than 10 min after induction. This rapid cutaneous warming served to drop the core body temperature 1.5 to 2.5 C in 10 min or less. Although skin temperature was measured at only one site, our subjective observations were that skin warming was patchy both temporally and spatially; some areas of the back did not warm, while flippers warmed and then cooled.

Long term measurements of body temperature and heart rate were made during the periods of anesthesia in which the catheters were first inserted. The data on body temperature are presented in Figure 3. Core body temperature reached a minimum within 30 to 120 min in each experiment and then rose steadily for the duration of anesthesia. Heating pads were used only when the body temperature fell to 34 C and then only until the temperature had begun to climb. Ice packs were applied when the body temperature rose to 38.5. The rise in body temperature after two h averaged about 0.67 C per hour.

The heart rate became stable after 0.5 h and averaged 90 to 100 beats/min throughout the remainder of the period of anesthesia. Both cardiac output and mean systemic blood pressure were considerably depressed during long term halothane anesthesia. Figure 4 shows that the average cardiac output was only 30% of the awake value, despite a higher mean heart rate. Systemic blood pressure was always much lower than in the awake animals, with values ranging from 90/70 to 65/50. The blood pressure rose to 110/90 in one seal when the halothane was reduced to 0.5%, but this level of halothane is inadequate to provide surgical anesthesia. Blood PaO_2's were also depressed even in normocapnia (see Table 1). For instance, one (hypercapnic) seal breathing a gas mix-

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

ture having an inspired PO_2 of 534 Torr had a PaO_2 of 120 Torr, while another (normocapnic) seal breathing gas with a PO_2 of 147 Torr had a PaO_2 of 62 Torr.

Cardiac arrhythmias occurred twice during long term anesthesia. Bradycardia was seen in one seal after 9.5 h of anesthesia at a body temperature of 39.4 C. Atropine and sodium bicarbonate were administered and the halothane was turned off. Recovery of normal rhythm took about five min. One other seal with a left ventricular catheter developed a cardiac arrhythmia at a

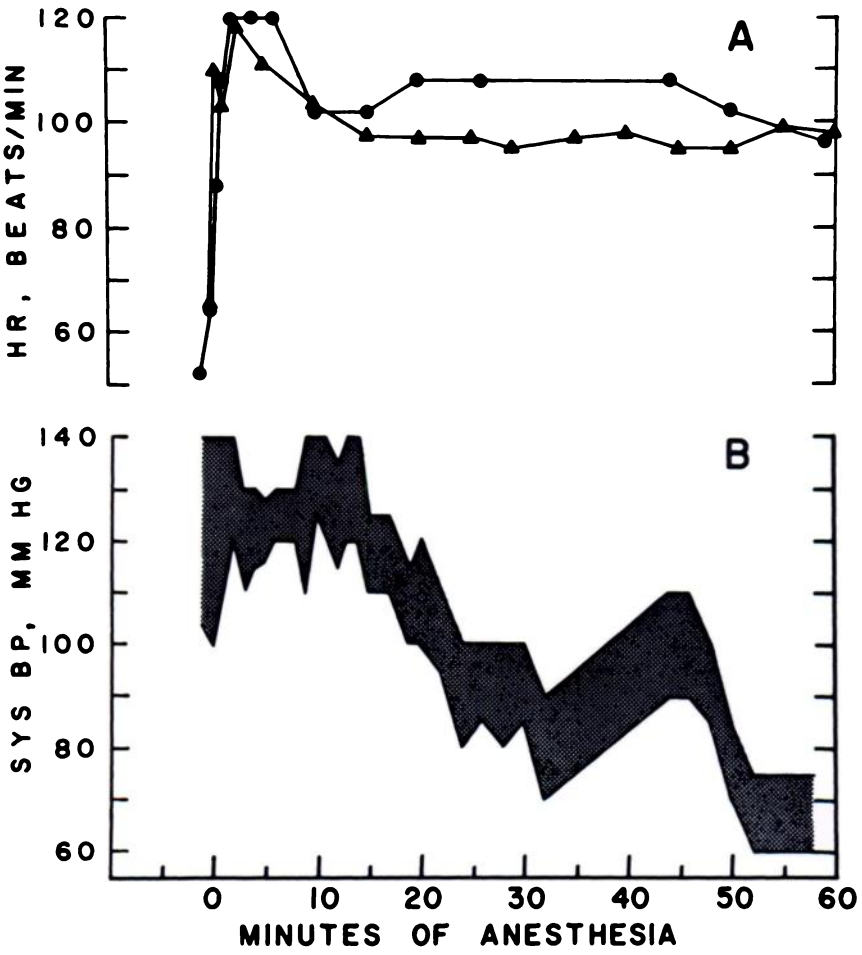


FIGURE 1. Short term effects of pentothal/halothane anesthesia in the seal on heart rate and blood pressure. A. Changes in heart rate are shown for two experiments on a single animal. B. Systemic systolic and diastolic pressures are indicated by the extremes of the shaded area for a single experiment. Note the rapid narrowing of the pulse pressure and the later fall in mean pressure. The corresponding heart rate data are marked by triangles in A.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

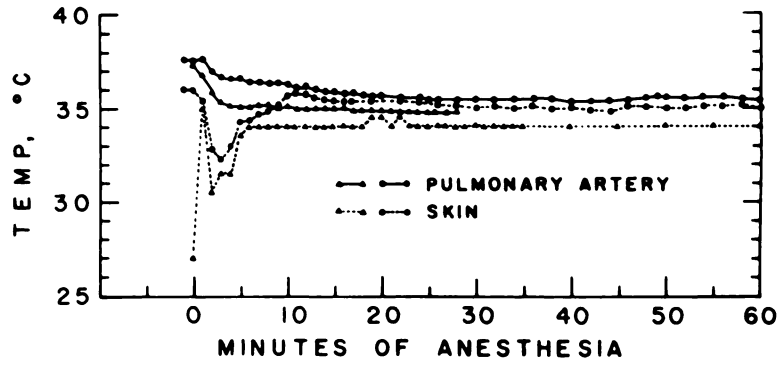


FIGURE 2. Short term effects of pentothal/halothane anesthesia in the seal on skin and core body temperature. Included are data from two separate experiments, marked by circles and triangles. The fall in skin temperature at 2-4 minutes occurred after placing the animal on its back on a metal table. Note how closely skin temperature approaches core body temperature.

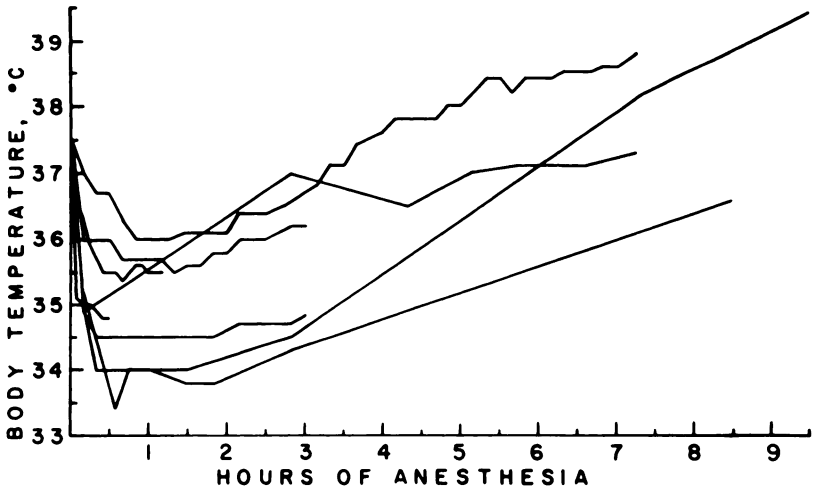


FIGURE 3. Core body temperature during prolonged anesthesia in the seal. The data are from eight periods of anesthesia on four animals, and include data on either rectal or pulmonary arterial blood temperature. An initial temperature of 37.5 C was measured in two seals, and this temperature was assumed to be the initial value for the other experiments as well.

body temperature of 37.5 C; this seal was not given any drugs, but recovered normal cardiac rhythm when the halothane was shut off and the catheter was withdrawn to the aorta.

DISCUSSION

Our data indicate that the longer term (greater than two hours) effects of halothane anesthesia in the harbor seal are more severe than might have been

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

supposed based on experiments in man and in other animals.[1,2,13,15] We found an unexpectedly large depression of cardiac output and systemic blood pressure, hyperthermia during long term anesthesia, and depression of arterial oxygen saturation. The initial drop in body temperature and the heart rate

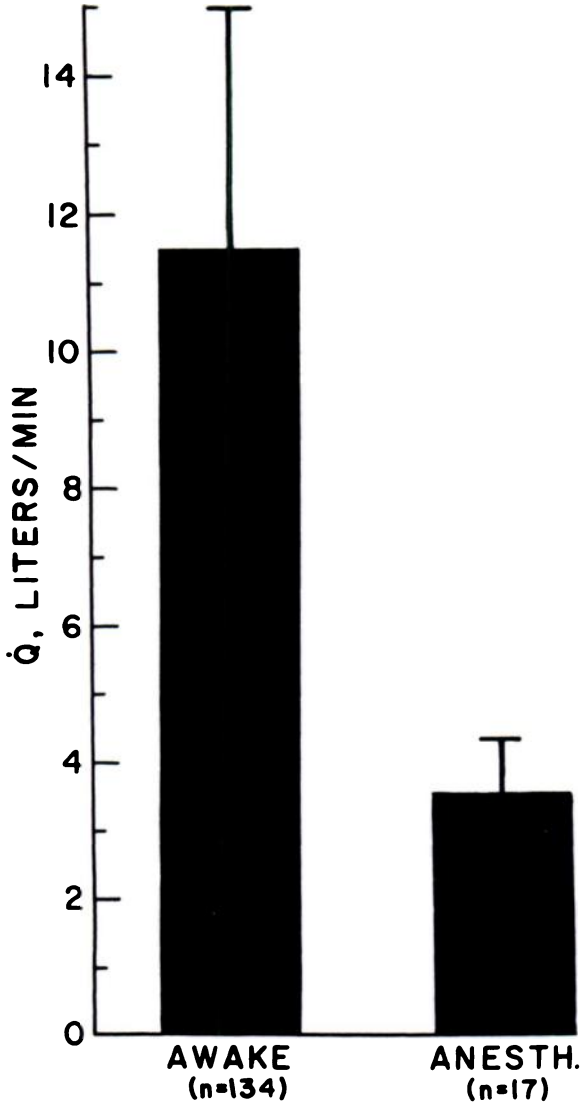


FIGURE 4. Cardiac output in awake and anesthetized seals. The means of n cardiac output estimates (n for each condition is given in parentheses) are indicated by the solid bars, while the standard deviations are indicated by the thin vertical lines.

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

response upon induction with pentothal were not unexpected.

The cardiac output, which had not been measured previously in anesthetized marine mammals, was depressed to a much greater degree than might be expected with 1% halothane. In dogs[1] given 1.5% halothane the cardiac output dropped at 30 min to 57% of the awake value, but had recovered to 83% of the awake value at 120 min. In man,[2] 1% halothane caused a drop to 78% in the first hour of anesthesia, with a recovery to 107% of the awake value in the fifth hour. Our data were not taken longitudinally during the period of anesthesia, but were all made at least two hours after induction. The drop to 30% of the awake value represents a much more profound depression of cardiac output than has been reported in other intact animal experiments. Halothane has been shown[13] to depress cardiac contractility directly, and the recovery of cardiac output in intact animals has been ascribed to reflex compensation in the circulatory system. Perhaps these reflexes are inhibited by halothane more severely in harbor seals than they are in humans or dogs.

Our data on the heart rate effects of pentothal/halothane anesthesia in the seal are similar to those found for the porpoise[11] and for dogs.[15] Since heart rate was elevated during anesthesia, the marked fall in cardiac output is thus due to an even greater drop in stroke volume, reflecting in part the direct effect of halothane on cardiac contractility.[13]

It is also known[7,13] that an elevated right atrial pressure can inhibit venous return and thus depress cardiac output. This poses a dilemma for those working with anesthetized phocid seals, since they have been shown[4] to have compliant chest walls; positive end expiratory pressure (PEEP) is required to prevent lung collapse, but this same pressure acts to inhibit venous return. A systematic study on the effects of varying amounts of PEEP and apneustic plateau respiration on cardiac output in seals seems warranted.

Our measurements of systemic blood pressure in anesthetized seals also appear to be the first reported, although data are available on systemic blood pressure in two species of porpoise.[10] Both of these species appear to tolerate pentothal/halothane anesthesia much better than do harbor seals, since the former had mean blood pressures during anesthesia of 115 Torr *(Tursiops truncatus)* and 130 Torr *(Lagenorhynchus obliquidens)* while the harbor seals had mean blood pressures as low as 60 Torr. The blood pressure drop caused by anesthesia was 5 to 15 Torr in the porpoises, while in the harbor seals it was on the order of 60 Torr. These findings in the seal probably reflect a reduced stroke volume and vascular dilatation, while the stroke volume and vascular tone in the porpoise may be preserved. Data on

TABLE 1. Inspired PO_2 (PIO_2) and blood gases in anesthetized seals.

Seal	PIO_2	PaO_2	$PaCO_2$	pH
3	534	120	55	7.23
1	320	185	43	7.32
4	225	57	55	7.25
2	185	96	33	7.36
1	154	72	43	7.33
1	147	62	42	7.32
2	147	58	32	7.33
3	134	57	48	7.25
1	97	34	41	7.34

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

cardiac output and stroke volume in anesthetized porpoises are required to confirm this speculation.

The effects of halothane on the blood pressure of men[2] and dogs[1] is less severe than it was in our seals. In men the blood pressure fell roughly 25% during the first hour of 1% halothane anesthesia and showed no recovery after 5 hours, while in dogs given 1.5% halothane it fell to 62% of the awake value at 30 minutes but then recovered to 86% of the awake value at 120 minutes. Again it would seem that the seal's reflex control of hemodynamics are more adversely affected than other species studied.

The problem of hypothermia in marine mammals undergoing barbiturate and/or halothane anesthesia has been recognized in California sea lions,[12] in harp seals,[6] and in porpoises.[8] We have shown that this drop in core body temperature is due to an extremely rapid rise in skin temperature with resultant loss of heat to the environment. Skin temperature rose to within 1 degree C of core temperature within ten minutes. Smaller rises in skin temperature have been observed in man[2] during induction with halothane with a resultant drop in core body temperature of about one-half degree C. An unexpected effect of long term anesthesia in the harbor seal, however, was that body temperature began to rise steadily after about an hour and a half of anesthesia; all of the other researchers have only stressed the need to prevent hypothermia. We suspect that hyperthermia may have contributed to at least one of the two episodes of cardiac arrhythmia that we observed, and would recommend *against* warming a moderately hypothermic animal if a long period of anesthesia is anticipated. More severe hypothermia (below about 34 C) should be treated, since hypothermia has contributed to anesthetic death in harp seals.[6]

Reports on blood gas data in anesthetized marine mammals are scarce. Arterial blood gases in the porpoise[11] were maintained within physiologically normal limits (PaO_2 95-120 Torr, $PaCO_2$ 30-45 Torr, pH 7.2-7.4), but the gas mixture used to attain this condition was not explicitly stated. Venous PCO_2 and bicarbonate values were used as indicators for maintaining CO_2 homeostasis in the harp seal,[6] but no indication of arterial oxygenation was given. Our data indicate that respiratory acidosis or alkalosis can be avoided by closely monitoring end tidal PCO_2, but that the use of compressed air is inadequate to maintain arterial oxygen saturation. In our opinion, pure oxygen should be avoided for long term anesthesia due to the danger of atelectasis, but 50% O_2 in N_2 can be safely administered for long periods and should offset the deficiency in arterial oxygenation. We were able to maintain alveolar ventilation in our animals at an adequate level using the 50% O_2 mixture and a standard piston driven ventilator instead of using the previously recommended apneustic plateau,[11] but perhaps the latter technique would provide good arterial oxygenation at a lower inspired PO_2. However, the possible adverse effects of the apneustic plateau on venous return should also be considered.

In summary, we have found that the induction of anesthesia in the harbor seal can be accomplished easily with an i.v. injection of sodium pentothal (10 mg/kg) which is followed immediately by tracheal intubation and maintenance of anesthesia with 1% halothane. Our data indicate that cardiovascular depression is severe, however, and further, that both hypo- and hyperthermia may need to be dealt with during long term anesthesia.

These findings should not be taken as a condemnation of the use of halothane anesthesia in marine mammals, since in relative terms it appears to be the safest agent in general use. We hope, though, that those using less sophisticated monitoring equipment will find our data

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use

useful in anticipating difficulties and understanding halothane anesthesia in marine mammals. We also expect that our work will stimulate research to better define the cardiovascular state of marine mammals subjected to different types of mechanical ventilation during anesthesia.

Acknowledgements

This research was supported by NHLBI grants 16157, 17731, 12373, and 14169.

LITERATURE CITED

1. AHLGREN, I., K.F. ARONSEN, I. BJÖRKMAN and S. WETTERLIN. 1978. The hemodynamic effect of halothane in the normovolemic dog. Acta anaesth. scand. 22: 83-92.
2. EGER, E.I., II, N.T. SMITH, R.K. STOELTING, D.J. CULLEN, L.B. KADIS and C.E. WHITCHER. 1970. Cardiovascular effects of halothane in man. Anesthesiology 32: 396-409.
3. HARRISON, R.J. and J.D.W. TOMLINSON. 1956. Observations on the venous system in certain Pinnipedia and Cetacea. Zool. Soc. Lond., Proc. 126: 205-333.
4. LEITH, D.E. 1976. Comparative mammalian respiratory mechanics. Physiologist 19: 485-510.
5. MARUSCHAK, G.F., E.A. MEATHE, J.F. SCHAUBLE and A. FRONEK. 1974. A simplified equation for thermal dilution cardiac output. J. Appl. Physiol. 37: 414-416.
6. McDONELL, W. 1972. Anesthesia of the harp seal. J. Wildl. Dis. 8: 287-295.
7. PERMUTT, S. and P. CALDINI. 1978. Regulation of cardiac output by the circuit: venous return. In: *International Conference on Cardiovascular System Dynamics*. Ed. by Baan et al., M.I.T. Press, Cambridge, pp. 465-479.
8. RIDGWAY, S.H. 1972. Homeostasis in the aquatic environment. In: *Mammals of the Sea: Biology and Medicine*. Ed. by S.H. Ridgway. Charles C. Thomas, Springfield, Illinois, pp. 704-726.
9. —— and P.L. JOYCE. 1975. Studies on seal brain by radiotelemetry. Rapp. P.-v. Reun. Cons. int. Explor. Mer. 169: 81-91.
10. —— and J.G. McCORMICK. 1971. Anesthesia of the porpoise. In: *Textbook of Veterinary Anesthesia*. Ed. by L.R. Soma. Williams and Wilkins Co., Baltimore, Md., pp. 394-403.
11. ——, —— and E.G. WEVER. 1974. Surgical approach to the dolphin's ear. J. Exp. Zool. 188: 265-276.
12. —— and J.G. SIMPSON. 1969. Anesthesia and restraint for the California sea lion, *Zalophus californianus*. J. Am. vet. med. Ass. 155: 1059-1063.
13. STEPHEN, C.R. and D.M. LITTLE, Jr. 1961. *Halothane*. Williams and Wilkins, Baltimore, pp. 31-44.
14. SINNETT, E.E., G.L. KOOYMAN, and E.A. WAHRENBROCK, 1978. Pulmonary circulation of the harbor seal. J. Appl. Physiol. 45: 718-727.
15. VATNER, S.F. and N.T. SMITH. 1974. Effects of halothane on left ventricular function and distribution of regional blood flow in dogs and primates. Circ. Res. 34: 155-167.

Received for publication 23 June 1978

Downloaded From: https://complete.bioone.org/journals/Journal-of-Wildlife-Diseases on 28 Mar 2024
Terms of Use: https://complete.bioone.org/terms-of-use