Drowning is not euthanasia

John W. Ludders, Robert H. Schmidt, F. Joshua Dein,
and Patrice N. Klein

Historically, there has been considerable discussion within the nuisance wildlife control and trapping communities as to whether drowning is a humane method for killing animals. The issue received more attention in 1993, when the American Veterinary Medical Association's Panel on Euthanasia reaffirmed its position that drowning is an unacceptable method (Andrews et al. 1993). For this article, we make a distinction between euthanasia, a "good death" that occurs without pain or distress (Andrews et al. 1993), and death due to killing by other methods. The central issue in this debate is whether drowning animals are rendered unconscious by great levels of carbon dioxide (CO_2, carbon-dioxide-induced narcosis) early in the drowning process and thus are insensitive to the distress and pain associated with drowning.

Proponents of drowning cite an article by Gilbert and Gofton (1982) in which the authors stated that drowning animals die from carbon-dioxide-induced narcosis. However, Gilbert and Gofton (1982) did not report any information on levels of carbon dioxide in blood, which is needed before a determination can be made about the acceptability of drowning as a method of euthanasia. We wish to introduce and clarify information concerning effects of carbon dioxide that have been absent in the debate on drowning.

In their laboratory investigations, Gilbert and Gofton (1982) determined time to death by drowning in mink (*Mustela vison*), muskrat (*Ondatra zibethica*), and beaver (*Castor canadensis*). Readings of the electrical activity of the brain (electroencephalograph, EEG) and of the heart (electrocardiograph, ECG) were recorded from each animal during drowning, and time of death was taken to be the moment when electrical activity of the brain ceased (EEG signal became flat). On average, the EEG signal became flat in mink after 4 minutes, 37 seconds; in muskrats after 4 minutes, 3 seconds; and in beaver after 9 minutes, 11 seconds. However, neither arterial nor venous blood samples were collected before, during, or after the animals drowned, so the partial pressures of carbon dioxide (PCO_2) or oxygen (PO_2) in blood from these animals were not measured. The authors stated that "[d]eath by CO_2 induced narcosis (submersion asphyxia) was evident in beaver, about 50% of muskrats, but 'wet' drowning (defined below) occurred in mink" (Gilbert and Gofton 1982:835). A review article written by Timperman (1972) was referenced to corroborate their conclusion. Timperman's (1972) paper discussed the forensic diagnosis of drowning through identification of diatoms in the lungs of victims. The author mentioned that carbon-dioxide-induced narcosis could be a possible cause of death during drowning, but he also acknowledged that death could be from anoxia. However, he did not provide substantiating data, such as blood gas analyses, to support either factor as the cause of death by drowning.

Proponents of drowning make a distinction between "wet" or "dry" drowning, the former occurring when water enters the lungs and the latter when the lungs remain relatively dry. To some, "dry" drowning implies that because the animal does not inhale water, then death is from CO_2-induced narcosis, although this is most likely incorrect. According to reports of incidents involving human drownings, 2 events may occur following submersion: 1) during the ensuing panic and struggle, water is swallowed and aspiration occurs in

85% of the victims, which leads to "wet" drowning, i.e., the lungs fill with water (Newman and Stewart 1995) and hypoxia and cardiac arrest occur rapidly, the latter probably because the vagal nerve, in response to water contacting the mucous membranes of the larynx or trachea, causes a reflex slowing and arrest of the heart (Suzuki 1996); or 2) during drowning, the act of swallowing water may lead to laryngospasm (an involuntary closure of the glottis or entrance to the airway), thus sealing the airway and preventing water from being aspirated into the lungs (Yagil et al. 1983, Suzuki 1996). Approximately 15% of human drowning victims experience "dry" drowning, in which the lungs remain relatively free of water (Newman and Stewart 1995). Hypoxia and cardiac arrest develop, but often this process is protracted compared to the victims experiencing "wet" drowning. In fact, current research strongly suggests that death occurs more rapidly when water is inhaled because it initiates a reflex vagal inhibition of the heart (Suzuki 1996). Thus, a longer period of consciousness may be associated with "dry" drowning than with "wet" drowning. The accumulated evidence (as discussed below) indicates that the cause of death during drowning is hypoxia and anoxia, not CO_2-induced narcosis.

Stedman's Medical Dictionary (1995:1176) defines narcosis as a "[g]eneral and nonspecific reversible depression of neuronal excitability, produced by a number of physical and chemical agents, usually resulting in stupor rather than in anesthesia." Hypercarbia, or an excess of carbon dioxide (CO_2) in blood, can cause narcosis. In animals, CO_2 is a normal byproduct of oxygen (O_2) metabolism, and it is eliminated from the body through the lungs and the process of pulmonary ventilation (Guyton 1991). The relationship of CO_2 production to O_2 utilization is expressed as the respiratory exchange ratio, generally accepted to be around 0.8; it indicates that in general, less CO_2 is produced for a given amount of metabolized O_2 (Guyton 1991).

Several studies, involving numerous animal species in which blood gases were measured, indicate that carbon-dioxide narcosis does not occur until the partial pressure of carbon dioxide in arterial blood ($PaCO_2$) exceeds 95 millimeters of mercury (mm Hg) and true anesthesia occurs only when $PaCO_2$ exceeds 200 mm Hg. For example, laboratory rats exposed to 100% CO_2 at various chamber fill rates started to show evidence of CO_2

narcosis (they became uncoordinated) after $PaCO_2$ exceeded 123 mm Hg (Hewett et al. 1993). The same rats became immobile only after $PaCO_2$ exceeded 212 mm Hg, and they finally lost the pedal reflex to painful stimulation (toe pinch) after $PaCO_2$ exceeded 332 mm Hg (Hewett et al. 1993).

A study of the narcotic properties of carbon dioxide in dogs sheds more light on the issue of CO_2-induced narcosis (Eisele et al. 1967). In this study, the narcotic and anesthetic properties of CO_2 were determined in 2 ways: 1) by determining the MAC (the minimum alveolar concentration of an inhalant anesthetic that prevents purposeful movement by an animal exposed to a painful stimulus) for the inhalant anesthetic halothane (2-bromo-2-chloro-1,1,1-trifluoroethane), and then, in a step-wise manner, replacing the halothane with CO_2 while maintaining a constant plane of anesthesia; and 2) by administering only CO_2 to dogs and recording the $PaCO_2$ when each dog was anesthetized and unresponsive to a painful stimulus. The results indicated that increasing levels of $PaCO_2$ above 95 mm Hg were increasingly narcotic. At a $PaCO_2$ of 95 mm Hg the narcotic effect of CO_2 was minimal as it reduced the MAC of halothane by only 0.08%. In this study, anesthesia was produced at an average $PaCO_2$ of 222 mm Hg.

Drowning animals, of course, are not breathing 100% CO_2, let alone air; in fact, they are not breathing at all. Because the drowning animal cannot breathe, it uses all of the O_2 available in its blood, and CO_2 accumulates because of oxygen metabolism. As previously noted, the respiratory exchange ratio indicates that the rate of O_2 utilization is greater than the rate of CO_2 production (Guyton 1991), and this fact is demonstrated by numerous animal studies. In dogs that were drowned with either cold salt water (CSW) or cold fresh water (CFW), $PaCO_2$ increased significantly, but after 10 minutes of immersion it never exceeded 64.8±4.9 mm Hg in either group (Conn et al. 1995). However, PaO_2 significantly decreased in both groups; after 4 minutes of immersion, PaO_2 was 16.4±1.5 mm Hg in the CFW group and 18.8±21.6 mm Hg in the CSW group, and after 10 minutes of immersion it was 9.6±3.8 and 8.8±1.9 in the CFW and CSW groups, respectively. Similar results were found in another study involving anesthetized, intubated dogs that inhaled a fixed quantity (20 ml/kg) of fresh water (Rai et al. 1980). Prior to inhaling water, the PaO_2 and $PaCO_2$ were 100 mm Hg and 35 mm Hg, respectively. Five minutes after inhaling

water, the PaO_2 and $PaCO_2$ were 35 mm Hg and 52 mm Hg, respectively. During 40 minutes of observation, $PaCO_2$ never exceeded 60±0.5 mm Hg (mean ± SEM) and the PaO_2 did not exceed 47±5.5 mm Hg. The results from these 2 studies show that $PaCO_2$ levels were well below those necessary to induce CO_2 narcosis and that the dogs were hypoxemic (inadequate oxygen in blood).

In a study that measured cerebral blood flow and arterial blood gases in ducks (*Anas platyrhynchos*) held under water for more than 4 minutes, the average PaO_2 was 52 mm Hg (minimum recorded was 37 mm Hg) at 4.61 minutes, while the average $PaCO_2$ was 51 mm Hg (Stephenson et al. 1994). These numbers indicate that the ducks were hypoxemic and hypercarbic and that $PaCO_2$ was not at levels known to produce narcosis. However, PaO_2 had decreased to hypoxemic levels, and had the ducks not been killed by decapitation, the PaO_2 would have continued to decrease to levels incompatible with life, i.e., the ducks would have died from anoxic asphyxiation.

A study in which blood gases were measured in beaver during submersion sheds more light on the drowning issue, especially as it relates to furbearers. After venous and arterial catheterization to sample blood, European beaver (*Castor fiber*) were forcefully submerged in water for up to 10 minutes (Clausen and Ersland 1970). From the authors' figures, the following conclusions can be drawn. Throughout the period of submersion, $PaCO_2$ increased but never exceeded 100 mm Hg; it took 7.5 minutes of submersion before $PaCO_2$ exceeded 95 mm Hg. The PaO_2 rapidly decreased during the first 7 minutes of submersion, but both PaO_2 and arterial hemoglobin saturation with oxygen were at hypoxemic levels ($PaO_2 < 50$ mm Hg and saturation<50%) within 5 minutes from the start of submersion. Thus the beavers were hypoxemic 2-3 minutes before $PaCO_2$ reached 95 mm Hg.

The method by which great CO_2 concentrations kill animals is anesthesia-induced respiratory arrest and the ensuing tissue hypoxia–anoxia (Mullenax and Dougherty 1963, Andrews et al. 1993). In fact, the time to death is prolonged when oxygen is used with CO_2. When a gas mixture consisting of approximately 70% CO_2, 24% N_2, and 6% O_2 was used to kill mink, for example, the 5 test animals survived for at least 15 minutes in the gas mixture (Hansen et al. 1991). One animal died 6 minutes after being removed from the gas mixture, but the 4 other animals fully recovered.

The preceding evidence demonstrates that in drowning animals, hypercarbia lags behind hypoxia and anoxia and that drowning animals die from hypoxia and anoxia. All of this suggests that drowning animals experience hypoxemia-induced discomfort and distress before CO_2 narcosis occurs, if narcosis occurs at all. This raises the question: do animals experience distress during drowning? For the following reasons, we believe that the answer is yes. The classic stress response consists of changes in heart rate and increases in blood pressures and circulating blood levels of epinephrine and norepinephrine and other stress-related hormones (Moberg 1985). In rats breathing 100% CO_2 (CO_2 anoxia), plasma norepinephrine increased significantly and was released from the sympathetic nervous system and not the adrenal medulla (Borovsky et al. 1998). The authors concluded that the response was mainly from hypoxia, not from CO_2 in and of itself (Borovsky et al. 1998).

In a model of asphyxia in which rats were strangled (anoxic asphyxia), mean serum norepinephrine and epinephrine concentrations were significantly greater in the strangled group compared to the non-strangled group (norepinephrine=5.4±2.6 ng/mL vs. 2.8±0.1 ng/mL, $P<0.001$ and epinephrine=6.0±3.4 ng/mL vs. 3.8±3.0 ng/mL, $P<0.05$; Hirvonen et al. 1997). The author concluded that the data supported the idea that catecholamine concentrations increased in blood upon suffocation and could be used as indicators of hypoxia (Hirvonen et al. 1997).

In dogs that were drowned with either cold salt water (CSW) or cold fresh water (CFW), epinephrine and norepinephrine concentrations (pg/mL) increased significantly after immersion and continued to rise throughout the experimental period (Conn et al. 1995). Prior to immersion, epinephrine was 206±25 in the CFW group and 133±67 in the CSW group. After 10 minutes of immersion, it had risen to 174,650±1,750 in the CFW group and 153,250±4,585 in the CSF group. Prior to immersion, norepinephrine was 224±46 in the CFW group and 374±182 in the CSW group, and by 10 minutes it had reached 63,025±4,946 in the CFW group and 50,400±1,796 in the CSF group. The authors noted that though the greater values reported in their study could be partly attributed to sudden cold stress that has been described after cold-water immersion, a more important etiological factor is likely to be anoxic–ischemic stress producing a catecholamine surge (Conn et al. 1995).

Thus, the accumulated data indicate that hypoxia-anoxia readily elicit the stress response in a variety of animal species.

To summarize, data from several studies and a variety of animal species indicate that CO_2 can produce narcosis, but only at partial pressures in arterial blood exceeding 95 mm Hg. Furthermore, data from rats and dogs suggest that a level of CO_2-induced narcosis sufficient to render an animal insensible to the discomfort, anxiety, and stress associated with hypoxemia is probably above 123 mm Hg; true CO_2-induced anesthesia, and thus insensibility, does not occur until $PaCO_2$ exceeds 200 mm Hg.

We recognize that drowning has been a traditional wildlife management technique, especially for trapping aquatic mammals such as beaver, muskrat, nutria (*Myocastor coypus*), mink, and river otters (*Lontra canadensis*). In some states, trappers have been encouraged to drown non-aquatic mammals captured in cage traps, including raccoons (*Procyon lotor*), striped skunks (*Mephitis mephitis*), and opossums (*Didelphis virginiana*). Drowning is a method of killing animals that is convenient for humans. However, the concept of euthanasia is independent of traditions and convenience, and drowning can not be considered euthanasia. As we noted at the beginning of this article, euthanasia is a "good death" that occurs without pain or distress. Time is an important element in euthanasia, and any technique that requires minutes rather than seconds to produce death can not be considered euthanasia. We encourage wildlife administrators, researchers, animal care and use committees, managers, and trappers to consider these findings as they develop wildlife euthanasia technique guidelines and Best Management Practices for Trapping (Proulx and Barrett 1989, Friend et al. 1994, Hamilton et al. 1998).

Literature cited

ANDREWS, E. J., B. T. BENNETT, J. D. CLARK, K. A. HOUPT, P. J. PASCOE, G. W. ROBINSON, AND J. R. BOYCE. 1993. Report of the AVMA panel on euthanasia. Journal of the American Veterinary Medical Association 202: 229-249.

BOROVSKY, V., M. HERMAN, G. DUNPHY, A. CAPLEA, AND D. ELY. 1998. CO2 asphyxia increases plasma norepinephrine in rats via sympathetic nerves. American Journal of Physiology 274: R19-22.

CLAUSEN, G., AND A. ERSLAND. 1970. Blood O2 and acid-base changes in the beaver during submersion. Respiration Physiology 11: 104-112.

CONN, A. W., K. MIYASAKA, M. KATAYAMA, M. FUJITA, H. ORIMA, G. BARKER, AND D. BOHN. 1995. A canine study of cold water drowning in fresh versus salt water. Critical Care Medicine 23: 2029-2037.

EISELE, J. H., E. I. EGER, AND M. MUALLEM. 1967. Narcotic properties of carbon dioxide in the dog. Anesthesiology 28: 856-865.

FRIEND, M., D. E. TOWEILL, R. L. BROWNELL, V. F. NETTLES, D. S. DAVIS, AND W. J. FOREYT. 1994. Guidelines for proper care and use of wildlife in field research. Pages 96-105 in T. A. Bookhout, editor. Research and Management Techniques for Wildlife and Habitats. The Wildlife Society, Bethesda, Maryland.

GILBERT, F. F., AND N. GOFTON. 1982. Terminal dives in mink, muskrat, and beaver. Physiology and Behavior 28: 835-840.

GUYTON, A. C. 1991. Textbook of medical physiology. W.B. Saunders, Philadelphia, Pennsylvania.

HAMILTON, D. A., B. ROBERTS, G. LINSCOMBE, N. R. JOTHAM, H. NOSEWORTHY, AND J. L. STONE. 1998. The European Union's wild fur regulation: A battle of politics, cultures, animal rights, international trade and North America's wildlife policy. Transactions of the North American Wildlife and Natural Resources Conference 63: 572-588.

HANSEN, N. E., A. CREUTZBERG, AND H. B. SIMONSEN. 1991. Euthanasia of mink (Mustela vison) by means of carbon dioxide (CO2), carbon monoxide (CO) and nitrogen (N2). British Veterinary Journal 147: 140-146.

HEWETT, T. A., M. S. KOVACS, J. E. ARTWOHL, AND B. T. BENNETT. 1993. A comparison of euthanasia methods in rats, using carbon dioxide in prefilled and fixed flow rate filled chambers. Laboratory Animal Science 43: 579-582.

HIRVONEN, J., M. L. KORTELAINEN, AND P. HUTTUNEN. 1997. Pulmonary and serum surfactant phospholipids and serum catecholamines in strangulation. An experimental study on rats. Forensic Science International 90: 17-24.

MOBERG, G. P. 1985. Biological response to stress: Key to assessment of animal well-being? Pages 27-49 in G. P. Moberg, editor. Animal Stress. American Physiological Society, Bethesda, Maryland.

MULLENAX, C. H., AND R. W. DOUGHERTY. 1963. Physiologic responses of swine to high concentrations of inhaled carbon dioxide. American Journal of Veterinary Research 24: 329-332.

NEWMAN, A. B., AND R. D. STEWART. 1995. Submersion Incidents. Pages 1209-1233 in P. S. Auerbach, editor. Wilderness Medicine: Management of Wilderness and Environmental Emergencies, Mosby, St. Louis, Missouri.

PROULX, G., AND M. W. BARRETT. 1989. Animal welfare concerns and wildlife trapping: Ethics, standards and commitments. Transactions of the Western Section of the Wildlife Society 25: 1-6.

RAI, U. C., P. K. BHARDWAJ, AND M. MOHAN. 1980. Effects of aspirated and swallowed water in mongrel dogs subjected to freshwater drowning. Indian Journal of Physiology and Pharmacology 24: 197-204.

STEDMAN'S MEDICAL DICTIONARY. 1995. Twenty-sixth Edition. Williams and Wilkins, Baltimore, Maryland.

STEPHENSON, R., D. R. JONES, AND R. M. BRYAN, JR. 1994. Regional cerebral blood flow during submergence asphyxia in Pekin duck. American Journal of Physiology 266: R1162-1168.

SUZUKI, T. 1996. Suffocation and related problems. Forensic Science International 80: 71-78.

TIMPERMAN, J. 1972. The diagnosis of drowning. A review. Forensic Science 1: 397-409.

YAGIL, R., Z. ETZION, AND A. OREN. 1983. The physiology of drowning. Comparative Biochemistry and Physiology 74A: 189-193.

John W. Ludders (top photo) is an associate professor and chief of section in anesthesiology in the Department of Clinical Sciences at the College of Veterinary Medicine, Cornell University. He received his B.S. (zoology) and his D.V.M. from Washington State University and did his residency in veterinary anesthesiology at the University of California, Davis. His research interests are in analgesia and anesthesia for birds.
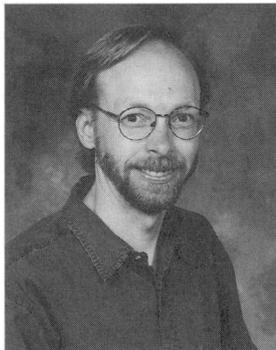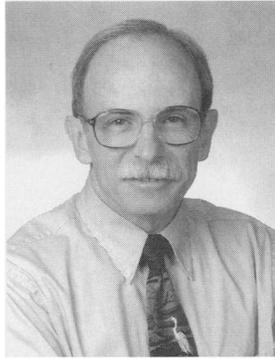
Robert H. Schmidt (bottom photo) is an associate professor in the Department of Fisheries and Wildlife at Utah State University. He received his B.S. in natural resources from Ohio State University; an M.S. in forestry, fisheries, and wildlife from the University of Nebraska, Lincoln; and an M.S. and Ph.D. in biological ecology from the University of California, Davis. His interests cover the spectrum of wildlife policy, ecology, and management. Robert was president of the Western Section of The Wildlife Society in 1989 and currently serves as president of the National Animal Damage Control Association and as an Executive Board member of the Wildlife Damage Management Working Group of TWS.

F. Joshua Dein is animal welfare officer at the United States Geological Service-Biological Research Division, National Wildlife Health Center. Trained as a biologist and a veterinarian, he is responsible for providing technical assistance to managers and researchers in areas such as captive animal management, capture and immobilization of wildlife, biological sample collection, telemetry implantation, euthanasia, and disease monitoring. He also has interests in electronic information resources, moderating the Wildlife Health mailing list and the Wildlife Health information Partnership. **Patrice M. Klein** is the wildlife veterinarian for the Humane Society of the United States (HSUS) and the veterinary director of the HSUS Wildlife Rehabilitation Training Center, Cape Cod, Massachusetts. She received her B.A. in biology from Hofstra University in 1976, an M.S. in pharmacology-toxicology from St. John's University, New York in 1983, and her V.M.D. from the University of Pennsylvania School of Veterinary Medicine in 1988. She has extensive training and experience in pathology and is a diplomate in the American College of Poultry Veterinarians. From 1990 to 1995, Pat was the center veterinarian at the United States Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland, where she was responsible for the health management of endangered species of birds such as whooping cranes and Mississippi sandhill cranes and evaluated the effects of environmental pollutants on avian species. She is currently working with Humane Society International on international wildlife rehabilitation programs in Central and South America.