



# Compendium

# **Hyperbaric Oxygen Therapy**

Cheryl Braswell, DVM
D. Tim Crowe, DVM, DACVS, DACVECC, FCCM

Pet Emergency & Specialty Hospital Thousand Oaks, California

**Abstract:** Hyperbaric oxygen therapy (HBOT) is emerging in veterinary medicine as an effective treatment or adjunct therapy for a variety of disorders in which improving oxygen delivery to tissues is a priority. The primary mechanisms of action of HBOT are (1) immediate hyperoxygenation of plasma and tissues and (2) decreased gas bubble (air embolus) size. With each hyperbaric "dive," secondary physiologic effects are set into motion. This article provides an introduction to HBOT, as well as its benefits, potential indications, contraindications, complications, and future directions in small animal veterinary medicine.

yperbaric oxygen therapy (HBOT) is the therapeutic use of a pressurized, 100% oxygen atmosphere. While HBOT is emerging as an effective treatment or adjunct therapy for a wide variety of medical and surgical conditions in small animal veterinary medicine, it may be underutilized, in part because practitioners may not be sufficiently familiar with how it works and when it may be efficacious. This article gives an overview of HBOT in human and veterinary medicine.

# History of Hyperbaric Oxygen Therapy

The scientific study of the effects of HBOT began as early as 1895, when Scottish physician John Scott Haldane evaluated its effect on carbon monoxide toxicosis in humans. The first clinical application of HBOT in the early 1930s is attributed to Brazilian physician Alvaro Osorio de Almeida. De Almeida's work also addressed the complication of central nervous system toxicity encountered while treating cancer patients with HBOT.

Following De Almeida's work, the US Navy tested HBOT in an effort to find a better therapy for decompression illness in people,<sup>2</sup> which is why an HBOT treatment session is called a "dive."

By the late 1950s, sufficient HBOT research had accumulated for clinicians to study the effects of HBOT on a wide range of human medical and surgical conditions.<sup>2-7</sup> Notable among these studies were the findings of Ita Boerema, a Dutch cardiovascular surgeon who performed various surgical procedures in a hyperbaric oxygen environment,<sup>4</sup> and of I. Churchill Davidson, who launched a clinical trial evaluating HBOT as a potentiator for radiation therapy in cancer patients at St. Thomas Hospital in London.<sup>4</sup>

Veterinarians first used HBOT in the late 1990s to treat traumatic brain injury patients, and the Veterinary Hyperbaric Medicine Society was formed in 2006 to "educate professionals and the public about hyperbaric medicine and its application, collaborate in the discovery of new knowledge, and promote operator and

patient safety." The number of facilities offering HBOT to veterinary patients continues to grow. A list of some of these facilities may be found on the Web site of the Veterinary Hyperbaric Medicine Society (www.vet.utk.edu/vhms/centers.html).

# Gas Laws in the Context of Hyperbaric Oxygen Therapy

Four physical laws pertaining to the properties of gases are important in understanding HBOT (**BOX 1**). In the context of HBOT, because the patient is receiving 100% oxygen, the partial pressure of oxygen ( $Po_2$ ) and, therefore, the density and solubility of oxygen are all maximized.

# Box 1. Gas Laws of Importance in Hyperbaric Oxygen Therapy

- Dalton's law (also called Dalton's law of partial pressures) describes
  the relationship of the pressure of individual gases in a mixture of gases to
  the total pressure of the gas mixture. It states that the total pressure exerted
  by a mixture of gases is equal to the sum of the pressure of each individual
  gas in the mixture.
- Boyle's law relates the volume and density of a gas to the pressure of the
  gas at a constant temperature. It states that the volume of gas is inversely
  proportional to the pressure, while the density of a gas (e.g., oxygen) is
  directly proportional to the pressure.
- Graham's law describes the relationship of pressure (concentration) of a gas to how it moves. It states that oxygen and carbon dioxide (and other gases) move independently, and at different rates, from areas of high pressure to areas of lower pressure.
- Henry's law relates the amount of gas that can be dissolved in a liquid to the
  pressure of the gas above the liquid. It states that the solubility of a gas in a
  liquid is directly proportional to the pressure of the gas in contact with the liquid.



# Box 2. Hyperbaric Oxygen Therapy Protocol

#### Prepare the patient

- · Prevent static electricity.
  - -Remove all electronic devices.
  - -Use cotton blankets or bedding.
  - -Remove metal collars.
  - --- Wrap metal objects (e.g., ECG pads) in bandage material
- · Secure catheters.
  - —Cover or wrap catheters to prevent dislodgment.
  - —Place Elizabethan collar, if necessary.
- Assess patient need for anxiolytic.

## Pressurize the chamber

- · Open the oxygen valve to the chamber.
- Initially, keep the effluent valve open to evacuate room air from the chamber.
- To allow pressurization to begin, close the effluent valve.
- During pressurization of the chamber (~10 to 15 minutes), monitor the
  patient for any adverse signs (e.g., aural barotrauma, confinement anxiety).

#### Administer treatment

- Once the desired pressure is reached, begin the prescribed treatment time.
- · Adjust the effluent valve to maintain the desired pressure.

### Decompress the chamber

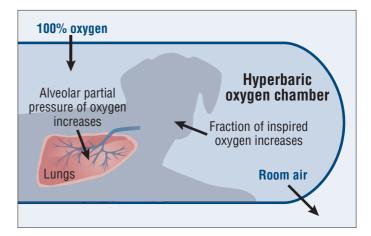
- · Open the effluent valve.
- Decrease the oxygen flow.
- Monitor the pressure within the chamber to make sure decompression takes place over the appropriate time frame (5 to 10 min for low-pressure therapy; 20 min for high-pressure therapy).
- Monitor patient closely for signs of discomfort (e.g., shaking head and ears). If patient discomfort is noted, slow the decompression process.
- When the intrachamber pressure reaches 0 psi, remove and assess the patient.

# **Physiology of Hyperoxygenation**

Oxygen delivery ( $\mathrm{DO_2}$ ) depends on cardiac output, arterial oxygen content ( $\mathrm{Cao_2}$ ), and blood flow. Cao<sub>2</sub> can be calculated using the hemoglobin (Hb) concentration, hemoglobin saturation ( $\mathrm{Sao_2}$ ), and arterial partial pressure of oxygen ( $\mathrm{Pao_2}$ ) as follows:

$$Cao_2 = (1.36 \times Hb \times Sao_2) + (0.003 \times Pao_2)$$

The contribution of dissolved oxygen  $(0.003 \times Pao_2)$  is normally very small; however, under high-pressure hyperbaric conditions (2 atmospheres absolute [ATA]), the content of dissolved oxygen in the plasma can be increased up to 15 times normal. This increase in the oxygen-carrying capacity of the blood is independent of red blood cells (hemoglobin); therefore, in areas where capillary flow is compromised, tissues have a greater opportunity to receive much-needed oxygen.



**Figure 1.** As the room air in the hyperbaric chamber is replaced by 100% oxygen, the partial pressure of oxygen inside the chamber increases (Dalton's law). Simultaneously, as the pressure within the chamber increases, so does the density of oxygen molecules in the chamber (Boyle's law). The resulting increases in the fraction of inspired oxygen and alveolar partial pressure of oxygen make more oxygen available for diffusion into pulmonary capillaries.

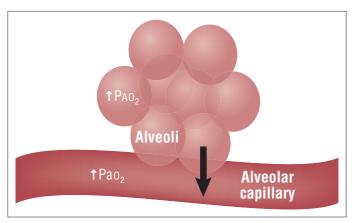
# **Mechanism of Hyperbaric Oxygen Therapy**

Most veterinary high-pressure HBOT sessions use pressures of 2.0 to 3.0 ATA, <sup>12</sup> while low-pressure therapy may be conducted at 1.5 to 2.0 ATA. When lower pressure is used, treatment times may be longer. The average treatment time at 2 ATA is 45 to 60 minutes. <sup>7,13</sup> Small animal patients are typically treated every 8 to 12 hours; however, large animals have been treated up to six times in a 24-hour period, depending on the severity of the condition. <sup>13</sup> A standard HBOT protocol is outlined in **BOX 2**.

When a patient is placed into the hyperbaric chamber and pressurization begins, the action of replacing the room air in the hyperbaric chamber with 100% oxygen gradually increases the fraction of inspired oxygen (Fio<sub>2</sub>) in conjunction with the atmospheric pressure (**FIGURE 1**). The increased concentration of oxygen molecules increases the partial pressure of oxygen (Dalton's law), so as the patient breathes, the concentration of oxygen molecules delivered to each alveolus and, therefore, the alveolar partial pressure of oxygen (PAO<sub>2</sub>) are increased. This increase in partial pressure leads to a corresponding increase in the density of the oxygen molecules in the alveoli (Boyle's law), resulting in more oxygen being available for diffusion into pulmonary capillaries.

Because gases move from areas of higher pressure to areas of lower pressure (Graham's law), the increased PAO<sub>2</sub> results in an increase in oxygen molecules diffusing across the alveolar epithelium into the blood. The high density and pressure of oxygen in the alveoli also increase the solubility of oxygen in the blood (Henry's law), resulting in a significant increase in the arterial partial pressure of oxygen (PaO<sub>2</sub>; FIGURE 2). In turn, this high PaO<sub>2</sub> creates a pressure gradient that determines the movement (amount and rate of diffusion) of oxygen from the blood through the capillary endothelium, the interstitial space, and the intracellular compartment (Graham's law; FIGURE 3). Therefore, a significantly





**Figure 2.** A high  $P_{AO_2}$  increases the solubility of oxygen in the adjacent blood (Henry's law), resulting in a significant increase in  $Pao_2$ .

increased Pao<sub>2</sub> ultimately results in an increased rate of oxygen delivery into the interstitial space and then into the cells (**FIGURE 3**).

# **Hyperbaric Chambers**

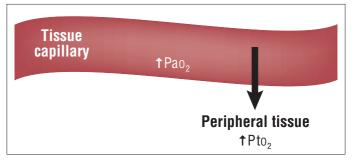
Human HBOT chambers are either monoplace (accommodating one person) or multiplace (accommodating several people at one time). Veterinary practices can acquire previously owned monoplace equipment from human facilities to treat their patients. Veterinary-specific hyperbaric chambers are also available, with some being large enough for ambulatory equine patients. Access to hyperbaric oxygen as a treatment modality is currently limited due to the modest number of facilities with the appropriate equipment.

# **Benefits of Hyperbaric Oxygen Therapy**

The primary effects of HBOT are hyperoxygenation and decreased gas bubble size. As described above, hyperoxygenation increases the oxygen-carrying capacity of the blood and, subsequently, the diffusion of oxygen into tissues (Graham's law).  $^{14}$  Tissue Po $_{2}$  may remain elevated by more than 10% above normal for up to 3 hours after a single hyperbaric dive.  $^{15}$ 

Decreased gas bubble size is paramount in the treatment of mechanical vascular obstruction from decompression sickness<sup>16</sup> or air embolism<sup>17</sup>; smaller bubbles pass through the vasculature more easily, thereby clearing the obstruction. Additionally, the smaller surface area of smaller bubbles reduces the activation of platelets, complement, and Hageman factor (factor XII).<sup>16</sup>

Other beneficial effects of HBOT include increases in leukocyte oxidative killing capacity,  $^{18,19}$  modulation of nitric oxide production,  $^{20-22}$  and modification of growth factors and cytokine effects through regulation of their levels and/or receptors.  $^{23-25}$  HBOT also decreases production of clostridial  $\alpha$  toxins  $^{26}$  and is synergistic with some antibiotics such as fluoroquinolones, amphotericin B, and aminoglycosides, all of which use oxygen to cross the cell membrane.  $^{27-30}$  An additional benefit is decreased vasogenic edema. Although the mechanism has not been conclusively elucidated, it is proposed that hyperoxic vasoconstriction to normal tissues



**Figure 3.** A high  $Pao_2$  creates a pressure gradient with adjacent tissue ( $Pto_2$ ) that increases the amount and rate of diffusion of oxygen from the blood through the capillary endothelium, the interstitial space, and, ultimately, the intracellular compartment (Graham's law).

may be involved while blood supply to is chemic tissues remains unaffected.  $^{\rm 14,31}$ 

# **Potential Indications for Hyperbaric Oxygen Therapy**

In veterinary medicine, scientific evaluation of HBOT is scarce; therefore, HBOT applications and protocols are largely adopted from human studies. Literature searches on HBOT in veterinary medicine yield primarily anecdotal case reports and conference proceedings.<sup>8,12,13,32-35</sup> In 2000, I (D. T. C.) discussed more than 1400 hyperbaric oxygen treatments given to dogs and cats over a 2-year period.33 This report covered HBOT for a wide variety of conditions, such as sepsis, peritonitis, osteomyelitis, encephalitis, spinal cord injury, traumatic shearing and crush wound injury, snake envenomation with resultant swelling and necrosis, and aortic thromboembolism, with the results assessed as "extremely favorable" in most cases. Failure to respond to HBOT was noted as a prognosticator of an unfavorable outcome. I have also reported retrospective studies on the use of HBOT that demonstrated benefits in veterinary patients with spinal cord injuries/compressions, head injuries, or stroke-like conditions<sup>34</sup> as well as in wound healing.<sup>35</sup>

Hyperoxygenation is potentially indicated in conditions characterized by ischemic insult, 36,37 such as aortic thromboembolism, gastric dilatation-volvulus, traumatic brain injury, and stroke, and after cardiopulmonary-cerebral resuscitation. It has been documented that an increased oxygen gradient between a wound and its surrounding environment will stimulate angiogenesis and, subsequently, increased proliferation of fibroblasts<sup>12</sup>; therefore, HBOT may also be useful for nonhealing wounds, skin flaps or grafts, 38,39 radiation injuries, 40,41 and crush injuries. 42,43 Patients with sepsis, osteomyelitis, diskospondylitis, gangrene, fungal infections, and necrotizing fasciitis may benefit from HBOT's effect on leukocyte killing capacity, antibiotic synergy, and clostridial toxin production.44,45 Additionally, the modification of growth factors and cytokine effects induced by hyperoxygenation is helpful in inflammatory conditions such as pancreatitis,46 heatstroke,47 and envenomation.12

A series of neurologic cases treated with HBOT, presented at the 2007 American College of Veterinary Internal Medicine Forum and the 2008 International Veterinary Emergency & Critical



Care Society Symposium, included patients with global ischemia, head trauma, fibrocartilaginous emboli, intervertebral disk extrusion, and inflammatory central nervous system disease. <sup>12,31</sup> The presenter felt that the adjunct use of HBOT was instrumental in the improvement and recovery of these patients.

At our facility (Pet Emergency & Specialty Hospital, Thousand Oaks, CA), more than 1700 HBOT sessions have been administered to treat a wide variety of conditions, including wounds, envenomation, central vestibular disease, osteomyelitis, traumatic brain injury, and pancreatitis. We retrospectively evaluated HBOT as an adjunct therapy in the management of emergent central nervous system conditions. The favorable results obtained prompted an ongoing international, multicenter, prospective study. In our experience, veterinary small animal practitioners often do not recognize situations in which their patients could benefit from this treatment modality.

#### **Contraindications**

Awareness of contraindications to HBOT is important to providing safe, optimal care. In veterinary medicine, HBOT contraindications are often inferred from human studies.

Absolute contraindications to HBOT include the following<sup>33</sup>:

- Untreated pneumothorax or pneumomediastinum
- High risk of aspiration, as in comatose, unconscious, or semiconscious patients
- Respiratory failure requiring mechanical ventilatory assistance (if using a monoplace chamber)
- Implantation of older types of pacemakers
- Uncontrolled seizure activity
- Nonmammalian species, due to differences in respiratory physiology and/or anatomy

Relative contraindications to HBOT include the following:

- Upper respiratory infections
- Asthma
- Pregnancy
- · History of spontaneous pneumothorax

# **Complications**

As with contraindications, complications of HBOT are mostly inferred from human medicine. Oxygen toxicity from the formation of reactive oxygen species (ROS) is a potentially serious complication of HBOT.<sup>33</sup> ROS originate from the addition of an electron to molecular oxygen (i.e., the reduction of the molecule). Because this electron is unpaired in its valence shell, the resultant molecule is highly reactive and can cause significant cellular damage.<sup>48</sup> Cellular oxidant stress (and subsequent damage) is determined by the relationship between ROS formation and ROS elimination by antioxidant defenses.<sup>48</sup> Natural antioxidant defenses within the central nervous system can become overwhelmed if they are overexposed to oxygen; when this occurs, oxygen-induced seizures may result.<sup>48,49</sup> Careful decompression for oxygen-induced seizures is recommended; anticonvulsant therapy is not required.<sup>48,49</sup> Seizures

typically do not recur if future hyperbaric treatments are conducted at a lower pressure or shorter length of exposure.<sup>48</sup> While ROS are implicated in pulmonary toxicity in human studies,<sup>48</sup> no published veterinary reports citing this relationship presently exist.

Confinement anxiety and aural barotrauma (ear discomfort) are less serious complications of HBOT.<sup>7</sup> If aural barotrauma is noted during compression or decompression, the rate of the process is slowed. For containment anxiety, anxiolytic medication is recommended. However, patients receiving anxiolytics must be assessed before HBOT to ensure that they are not profoundly sedate and therefore at risk for aspiration.

# **Future Directions**

Further scientific research is necessary to maximize the potential of HBOT in small animal veterinary medicine. Treatment protocols need to be standardized. Studies are needed to compare the effectiveness of low-pressure versus high-pressure HBOT and to determine the optimal length and frequency of individual treatments for specific conditions. No published reports to date delineate long-term outcomes from HBOT, present a cost-benefit analysis, or identify potential differences in treatment sensitivity or complications by species and breed.

#### **Conclusion**

HBOT is an underutilized treatment modality that is potentially beneficial for a variety of disorders in which improving oxygen delivery to tissues is a priority. It also may have positive effects in infectious and inflammatory conditions. Knowing the physiology, mechanisms of action, indications, contraindications, and possible complications of HBOT can help small animal veterinary practitioners be better prepared to identify patients that may benefit from this important emerging therapy.

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## References

- 1. Haldane J. The action of carbonic oxide on man. J Physiol 1895;18(5-6):430-462.
- **2.** Clarke D. History of hyperbaric therapy. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:3-24.
- **3.** De Almeida AO. Recherches sur l'action toxique des hautes pressions d'oxygene. *Proc Societe de Biologie de Paris* 1934;66:1225.
- **4.** Kindwall EP. A history of hyperbaric medicine. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing, 2008:3-22.
- **5.** Boerema I, Kroll JA, Meijne NG, et al. High atmospheric pressure as an aid to cardiac surgery. *Arch Chir Neerl* 1956;8:193-211.
- **6.** Gray LH, Conger AD, Ebert M, et al. Concentration of oxygen dissolved in tissues at time of irradiation as a factor in radiation therapy. *Br J Radiol* 1953;26:638.
- 7. Clarke D. Effects of pressure. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:513-526.
- **8.** Crowe DT, Hittenmiller DJ. Hyperbaric oxygen therapy for traumatic brain injury. *Proc Int Vet Emerg Crit Care Symp* 1998:809.
- Veterinary Hyperbaric Medicine Society. Accessed June 2010 at http://www.vet.utk.edu/vhms/.
- **10.** Ganong WF. Gas transport between lungs and the tissues. In: Ganong WF, ed. *Review of Medical Physiology*. 22nd ed. New York, NY: McGraw-Hill Companies; 2005:666-670.



- **11.** Hammarlund CE. The physiologic effects of hyperbaric oxygenation. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:39–70.
- **12.** Lyman R. Hyperbaric oxygen therapy in the emergent and critical care patient: mechanisms of action and case examples. *Proc Int Vet Emerg Crit Care Symp* 2008.
- **13.** Geiser DR. Putting the pressure on disease: overview of large animal hyperbaric therapy. *Proc Int Vet Emerg Crit Care Symp* 2008:213-218.
- **14.** Piantadosi CA. Pulmonary gas exchange, oxygen transport, and tissue oxygenation. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy.* Philadelphia, PA: Saunders; 2008:133-158.
- **15.** Wells CH, Goodpasture JE, Horrigan DJ, et al. Tissue gas measurements during hyperbaric oxygen exposure. *Proc Sixth Int Cong Hyperb Med* 1977:118-124.
- **16.** Moon RE. Gorman DF. Decompression sickness. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:283-319
- **17.** Kindwall EP. Gas embolism. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:517-534.
- **18.** Allen DB, Maguire JJ, Mahdavian M, et al. Wound hypoxia and acidosis limit neutrophil bacterial killing mechanisms. *Arch Surg* 1997;132:991-996.
- **19.** Babior BM. Oxygen-dependent microbial killing by phagocytes. *N Engl J Med* 1978; 198:659-668.
- **20.** Fadini GP, Miorin M, Facco M, et al. Circulating endothelial progenitor cells are reduced in peripheral vascular complications of type 2 diabetes mellitus. *J Am Coll Cardiol* 2005;45:1449-1457.
- **21.** Tepper OM, Galiano RD, Capla JM, et al. Human endothelial progenitor cells from type II diabetics exhibit impaired proliferation, adhesion, and incorporation into vascular structures. *Circulation* 2002;106:2781–2786.
- 22. Thom SR. Effects of hyperoxia on neutrophil adhesion. *Undersea Hyperb Med* 2004; 31:123-131.
- **23.** Sheikh AY, Gibson JJ, Rollins MD, et al. Effect of hyperoxia on vascular endothelial growth factor levels in a wound model. *Arch Surg* 2000;135:1293-1297.
- **24.** Feng J, Gibson J, Constant J, et al. Hyperoxia stimulates macrophage vascular endothelial growth factor (VEGF) production. *Wound Repair Regen* 1998;6:A252.
- **25.** Bonomo SR, Davidson JD, Yu Y, et al. Hyperbaric oxygen as a signal transducer: upregulation of platelet derived growth factor-beta receptor in the presence of HBO2 and PDGH. *Undersea Hyperb Med* 1998;25:211-216.
- **26.** Korhonen K, Klossner J, Hirn M, et al. Management of clostridial gas gangrene and the role of hyperbaric oxygen. *Ann Chir Gynaecol* 1999;88:139-142.
- **27.** Hopf HW, Kelly M, Shapshak D. Oxygen and the basic mechanisms of wound healing. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy.* Philadelphia, PA: Saunders; 2008:203-228.
- **28.** Adams K, Mader J. Aminoglycoside potentiation with adjunctive hyperbaric oxygen therapy in experimental *Pseudomonas aeruginosa* osteomyelitis [abstract 69]. Undersea and Hyperbaric Medical Society Annual Scientific Meeting; Baltimore, MD;1987.
- **29.** Mader JT, Shirtliff ME, Bergquist SC, et al. Antimicrobial treatment of chronic osteo-myelitis. *Clin Orthop Relat Res* 1999:47-65.
- **30.** Verklin RM Jr, Mandell GL. Alteration of effectiveness of antibiotics by anaerobiosis.

- J Lab Clin Med 1977;89:65-71.
- **31.** Lyman R. Hyperbaric oxygen therapy in small animal neurology. *Proc Am Coll Vet Intern Med* 2007.
- **32.** Crowe DT. On the front lines: Life-saving emergency medicine and critical care procedures. *Proc Calif Vet Med Annu Conf* 2001:103-160.
- **33**. Crowe DT. Hyperbaric oxygen therapy in veterinary medicine: a case series at Carson-Tahoe Veterinary Hospital. *Hyperbaric Medicine Today* 2000;1(2):13-15.
- **34.** Crowe DT. Physiotherapy and hyperbaric oxygen therapy in the critical patient. *Proc* 11th Annu Sci Meet 2002;324-35.
- **35.** Crowe DT. Bovine small intestinal submucosal and hyperbaric oxygen used in severe wounds. *Proc Am Coll Vet Surg* 2000.
- **36**. Baynosa RC, Zamboni WA. Microcirculation and ischemia-reperfusion: basic mechanisms of hyperbaric oxygen. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:735-754.
- **37**. Buras JA. Garcia-Covarrubias L. Ischemia-reperfusion injury and hyperbaric oxygen therapy: Basic mechanisms and clinical studies. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:159-186.
- **38**. Nemiroff PM. Hyperbaric oxygen in skin grafts and flaps. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:835-850.
- **39.** Kagan SH. Management of chronic wounds. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:349-372.
- **40.** Marx RE. Radiation injury to tissue. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:851-904.
- **41.** Feldmeier JJ. Hyperbaric oxygen therapy for delayed radiation injuries. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:231–256.
- **42**. Strauss MB, Miller SS. The role of hyperbaric oxygen in crush injury, skeletal muscle-compartment syndrome, and other acute traumatic ischemias. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:755-790.
- **43.** Strauss MB, Garcia-Covarrubias L. Crush injuries: justification of and indications for hyperbaric oxygen therapy. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy.* Philadelphia, PA: Saunders; 2008:427-450.
- **44.** Park MK, Falzon CC, Whelan HT. Effects of hyperbaric oxygen in infectious diseases: basic mechanisms. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed. Flagstaff, AZ: Best Publishing; 2008:535-576.
- **45.** Niinikoski J. Hyperbaric oxygen therapy in chronic osteomyelitis. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:419-427.
- **46.** Cuthbertson CM, Christophi C. Potential effects of hyperbaric oxygen therapy in acute pancreatitis. *ANZ J Surg* 2006;76(7):625-630.
- **47**. Tsai HM, Gao CJ, Li WX, et al. Resuscitation from experimental heatstroke by hyperbaric oxygen therapy. *Crit Care Med* 2005;33(4):813-818.
- **48.** Clark JM. Oxygen toxicity. In: Kindwall EP, Whelan HT, eds. *Hyperbaric Medicine Practice*. 3rd ed, Flagstaff, AZ: Best Publishing; 2008:73-85.
- **49.** Clark JM. Oxygen toxicity. In: Neuman TS, Thom SR, eds. *Physiology and Medicine of Hyperbaric Oxygen Therapy*. Philadelphia, PA: Saunders; 2008:527-563.





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1.	Oxygen delivery depends on  a. cardiac output.  b. arterial oxygen content.  c. blood flow.  d. all of the above	6.	law relates the amount of gas that can be dissolved in a liquid to the pressure of the gas above the liquid.  a. Boyle's  b. Dalton's
2	The primary effects of HBOT are hyperoxygenation and		c. Graham's
۷.	<ul> <li>a. decreased edema.</li> <li>b. enhanced antimicrobial effectiveness.</li> <li>c. increased fibroblast proliferation.</li> <li>d. decreased gas bubble size.</li> </ul>	7.	<ul> <li>d. Henry's</li> <li>Which of the following is not a contraindication for HBOT?</li> <li>a. untreated pneumothorax</li> <li>b. nonmammalian species</li> <li>c. unconscious patient</li> </ul>
3.	After a single HBOT dive, tissue $P_{0_2}$ can remain elevated by more than above normal for up to 3 hours.		d. controlled seizures
4.	<ul> <li>a. 5%</li> <li>b. 10%</li> <li>c. 20%</li> <li>d. 50%</li> </ul> The secondary effects of HBOT do not include <ul> <li>a. increased leukocyte killing ability.</li> <li>b. increased flow of fluid into tissues.</li> <li>c. increased fibroblast proliferation.</li> </ul>		<ul> <li>What is the most significant potential complication of HBOT?</li> <li>a. containment anxiety</li> <li>b. aural barotrauma</li> <li>c. decreased proprioception</li> <li>d. oxygen toxicity</li> <li>If signs of aural barotrauma are observed during decompression,</li> <li>a. the rate of decompression should be slowed.</li> <li>b. the rate of decompression should be increased.</li> </ul>
5.	<ul> <li>d. decreased clostridial α toxin production.</li> <li>law relates the volume and density of a gas to the pressure at a constant temperature.</li> <li>a. Boyle's</li> <li>b. Dalton's</li> <li>c. Graham's</li> <li>d. Henry's</li> </ul>	10.	<ul> <li>c. decompression should be discontinued.</li> <li>d. the chamber should be repressurized.</li> <li>Which of the following is a potential indication for HBOT in veterinary medicine?</li> <li>a. nonhealing wounds</li> <li>b. osteomyelitis</li> <li>c. head trauma</li> </ul>
			d. all of the above