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Adjunctive Use of Hyperbaric Oxygen Therapy in Veterinary Dentistry and Oral Surgery: A Case Series

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Abstract

Hyperbaric oxygen therapy (HBOT) is utilized as an adjunctive treatment for human and veterinary patients with compromised tissues. Medical records from two veterinary hospitals with HBOT chambers were searched for small animal veterinary dentistry and oral surgery specialty patients. The HBOT records were combined with the medical records from the referring specialty veterinary dentistry and oral surgery services. Clinical indications for HBOT treatments associated with a positive outcome in this case series included resistant bacterial infections, electrical cord injury, bite wound injuries, osteomyelitis, crush/traumatic injuries including mandibular fractures, oral surgery performed at previously irradiated sites, and osteonecrosis, presumably radiation induced. Conditions within this case series that remained unchanged or were associated with partial improvement included preoperative treatment of stomatitis without steroid usage and delayed HBOT treatment for long-term endodontic health of laterally luxated immature permanent mandibular incisors. Eighty-eight percent of the HBOT sessions were tolerated well by the patients in this case series. The most common adverse event was mild anxiety. One patient required oral anxiolytic medications to complete the course of treatment. One patient experienced transient seizure activity and was able to complete that session as well as subsequent sessions at a lower chamber pressure. Future prospective studies are necessary to further evaluate and characterize the potential benefits of HBOT as well as to clarify optimal treatment protocols for specific conditions in veterinary dentistry and oral surgery.

Keywords

hyperbaric oxygen therapy, HBOT, HBO, HBO_2, resistant bacterial infection, compromised tissue, radiation osteonecrosis, osteonecrosis, osteomyelitis, crush injury

Introduction

The Undersea & Hyperbaric Medical Society (UHMS) defines hyperbaric oxygen therapy (HBOT) as near 100% medical grade oxygen delivered at pressures >1.0 atmospheres absolute (ATA).[1] For clinical treatment purposes, a minimum of 1.4 ATA is recommended.[1] The Food and Drug Administration (FDA) recognizes 13 indications for HBOT for human clinical medicine, including carbon monoxide poisoning, idiopathic sudden sensorineural hearing loss, gas gangrene, decompression sickness (diving injury), air and gas bubbles in blood vessels, severe anemia when blood transfusions are not able to be administered, severe and large burns, crush injuries, severe infection of the skin and bone, radiation injury, compromised skin grafts or flaps, sudden vision loss due to blockage of blood flow, and non-healing wounds (i.e., diabetic foot ulcers).[1]

HBOT relies on several laws regarding the behavior of gases. *Boyle's law* describes how a volume of gas is affected by pressure. *Dalton's law* describes how the total pressure of a gas mixture is the summation of the partial pressure of each gas present. *Graham's law* describes the diffusion of gases from higher concentration to lower concentration. *Henry's law* describes the behavior of gases when in contact or within a liquid or tissue. *Fick's law* describes the influence of pressure on the diffusion of gases through a tissue or membrane.[2,3] The properties of these laws allow for HBOT to achieve significantly increased tissue oxygenation independent of hemoglobin.

Increased tissue oxygenation from HBOT has been associated with several physiologic mechanisms of action, including gas bubble reduction, vasoconstriction, reduction of inflammation and edema, angiogenesis, increased leukocyte function,

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Table 1. Soft Tissue Wound Healing Phases, Physiologic Processes, and the Effects of Hyperbaric Oxygen Therapy (HBOT) During the Phases of Wound Healing.[3,4]

Phase of wound healing	Physiologic processes	Effects of HBOT
Inflammatory phase	Hemostasis with fibrin clot formation	Improved tissue oxygenation Direct and indirect antibacterial and antifungal effects Increased leukocyte function
	Influx of inflammatory cells, which in turn are responsible for defense against and elimination of bacteria and diseased tissue[4]	Increased macrophage phagocytosis diminished by hypoxia Reduction of inflammation and edema[3]
Proliferation phase	Significant proliferation of cells, particularly fibroblasts and smooth muscle cells Fibroblasts regulate collagen production, which in turn is responsible for tissue strength Angiogenesis and lymphatic repair occur Re-epithelization occurs[4]	Improved tissue oxygenation Direct and indirect antibacterial and antifungal effects Increased leukocyte function Increased macrophage phagocytosis diminished by hypoxia Fibroblast activation Increased angiogenesis and neovascularization. Increased collagen deposition diminished by tissue hypoxia and hypoperfusion[3]
Maturation/ remodeling phase	Collagenolysis and new collagen production occur[4]	Improved tissue oxygenation Direct and indirect antibacterial and antifungal effects Fibroblast activation Increased collagen deposition diminished by tissue hypoxia and hypoperfusion[3]

increased fibroblast activity, antibacterial and antifungal effects, increased stem cell production, reduction of reperfusion injury, and improved tissue oxygenation.[2,3] The majority of these physiologic effects may be beneficial to the veterinary oral surgery patient with compromised tissues, particularly early in the healing process. Increased leukocyte function, increased fibroblast function, edema reduction, and collagen synthesis are all critical factors during wound healing. The phases of wound healing and the associated potential impacts of HBOT during each phase are summarized in Table 1.

Factors known to impact wound healing include advanced age, poor nutrition, microangiopathy associated with diabetes mellitus, infection, tissue hypoperfusion, and poor tissue oxygenation.[4] HBOT improves tissue oxygenation, promotes angiogenesis, and has antibacterial and antifungal activity.[2,3] These antibacterial and antifungal effects are additionally important to poorly oxygenated and poorly perfused tissues, which have an increased risk for infection.[3,4]

In veterinary medicine, uses for adjunctive HBOT that have been reported include crush injuries, wound management, wound grafts, bone grafts, thermal burns, carbon monoxide poisoning, intervertebral disc disease, head trauma, compartment syndrome, infectious disease (i.e., aerobic bacterial, anaerobic bacterial, and fungal), osteomyelitis, auto-immune disease (i.e., hemolytic anemia), pancreatitis, peritonitis, pyothorax, snake or spider envenomation, post-surgery support, post-cardiovascular arrest support, ischemia/reperfusion injuries, anemia, laryngeal paralysis, non-cardiogenic pulmonary edema, aortic embolisms, and pre-anesthetic oxygenation.[3,5] Specific to equine veterinary medicine, uses for adjunctive HBOT that have been reported in addition to those listed above include laminitis, tendonitis, dummy foal syndrome, exercise-induced pulmonary hemorrhage, and athletic performance recovery.[2,5]

This retrospective case series aims to raise awareness of indications within veterinary dentistry and oral surgery where adjunctive HBOT is currently utilized and may be of clinical benefit. The clinician should be familiar with the benefits and indications, risks and contraindications, and HBOT safety protocols, particularly those specific to veterinary dentistry and oral surgery patients.

Materials and Methods

Patient treatment logs of two veterinary hospitals with HBOT chambers were reviewed for small animal veterinary dentistry and oral surgery patients who received HBOT from September 2017 through December 2021. The HBOT treatment records for each patient were then combined with the medical records from the veterinary dentistry and oral surgery referring institutions. The following information was recorded for each patient in this study: signalment, concurrent medical conditions, concurrent medications, indication for HBOT, oral surgery performed, HBOT chamber type, number of HBOT sessions, ATA of treatment sessions, patient demeanor (when noted) during HBOT sessions, and clinical outcome. Outcomes of the combined surgical and HBOT treatments were ranked on a scale from 0 to 3, with 0 representing no healing, complete dehiscence, or radiographic absence of age-appropriate tooth maturation and 3 representing complete healing, no dehiscence, or radiographic presence of age-appropriate tooth maturation. Outcome scores are detailed in Table 2. Patients were excluded from the study

Table 2. Outcome Scores.

Outcome score	Degree of healing or tooth maturation
0	No healing, complete dehiscence, or radiographic absence of tooth maturation
1	Minimal healing, partial dehiscence, or minimal radiographic evidence of tooth maturation
2	Majority healing, minimal dehiscence, or partial radiographic evidence of tooth maturation including apexogenesis (if applicable)
3	Complete healing, no dehiscence, or radiographic evidence of age-appropriate tooth maturation

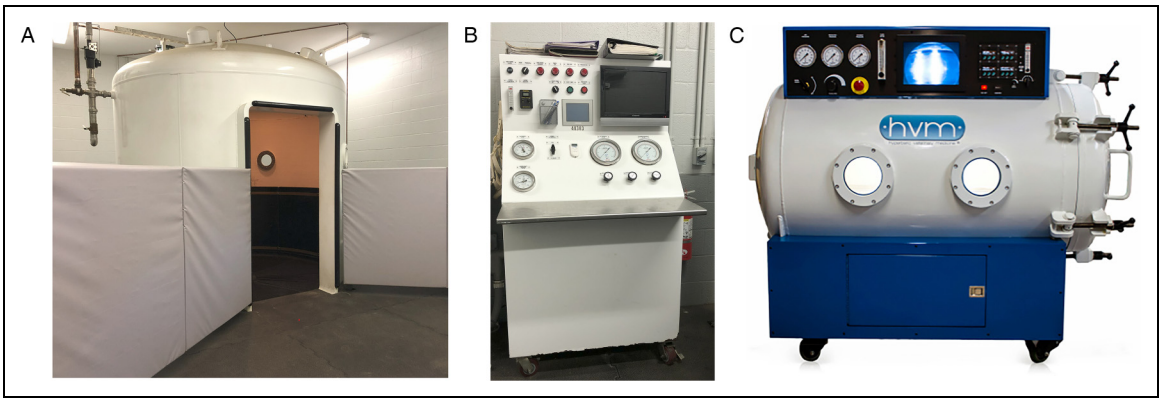

Figure 1. Photographs of (A) equine HBOT chamber, (B) equine HBOT control panel, and (C) small animal HBOT chamber with combined control panel. *Image of hvm chamber model 3200 reproduced with written permission from hyperbaric veterinary medicine (hvm ®).*

if they failed to return for medical progress examinations or follow-up diagnostic imaging at time intervals sufficient for their particular HBOT indication.

The two HBOT facilities in this study utilized different Class C (animal use only) chambers. One chamber was an equine chamber with a free-standing control panel, an Equine Oxygen Therapy chamber, while the other was a small animal chamber with an attached control panel, a hyperbaric veterinary medicine® chamber (Figure 1A–C). Small animal patients referred to the equine facility had received surgical care by a Board-Certified American Veterinary Dental College (AVDC) diplomate. Small animal patients referred to the small animal HBOT facility had received surgical care either by veterinary dentistry and oral surgery residents under the supervision of an AVDC diplomate or by an AVDC diplomate.

At both facilities, patient medical history was reviewed for any contraindications to HBOT prior to initiating therapy, such as pneumothorax, uncontrolled seizures, congestive heart failure, or implanted electronic devices. Patients with a history of trauma or pulmonary disease were required to have thoracic radiographs evaluated for any evidence of pneumothorax, pulmonary bullae, or pulmonary abscesses prior to referral for HBOT.

The standard HBOT treatment at the equine facility was performed with 100% oxygen delivery for 30 min at a compression pressure of 14.7 to 22 pounds of force per square inch area (psi), equivalent to 2.0 to 2.5 ATA inclusive of ambient atmospheric pressure. The standard HBOT treatment at the small animal facility was performed with 100% oxygen delivery for 30 min at compression pressure of 1.5 ATA per session. Small dogs and cats were treated while housed in clear acrylic-only carriers placed within the small animal chamber. At both facilities, treatment sessions were preceded by 15 min of compression and followed by 15 min of decompression. Any variation to the standard session protocols was noted in the HBOT patient session logs at each facility.

Patients were monitored throughout treatment sessions at both locations either directly through a viewing window or indirectly via live feed video. Standard HBOT session safety protocols were followed at both facilities: patient rectal temperature was assessed prior to each HBOT session, patients were visually monitored throughout each session, collars and harnesses were removed, intravenous catheters were covered with 100% cotton materials only, any exposed metal was removed or covered, and electronic devices with or without batteries (ie, diabetic monitoring devices, pacemakers, location tracking dog collars, etc) were not permitted inside the chamber. Patients treated in the small animal chamber were sprayed with a light mist of water prior to entering the HBOT chamber to minimize any possible static.

Results

Dentistry and oral surgery patients receiving HBOT within this case series were cases with clinician concerns for significantly impaired postoperative healing. These cases generally fell into 3 categories: (1) cases with compromised tissues due to infection, (2) cases with compromised tissues due to insufficient healthy tissue to heal (i.e., crushing injuries, devitalization injuries, inflammatory disease), and (3) cases with compromised tissues due to radiation injury. When a case had multiple reasons for HBOT, the primary reason was used for case categorization. These case details are summarized in Table 3. Seven

cases were excluded from the study due to insufficient medical progress evaluation(s) and/or follow-up imaging.

Category 1. Cases With Compromised Tissues due to Infection

Cases 1 and 2 had existing infections and were treated with a combination of surgical debridement, antibiotic therapy, and HBOT. The infections resolved. Both cases were treated with empirical postoperative antibiotics. Case 1 was treated with clindamycin[a] (5.5 mg/kg) per os (PO) every 12 h for 8 days. Case 2 was treated with cefpodoxime proxetil[e] (9 mg/kg) PO every 24 h for 14 days. These antibiotics, based on culture and sensitivity results, were ultimately found to be ineffective for the identified organism, methicillin-resistant *Staphylococcus pseudintermedius* (MRSP). Case 1 had surgical debridement and repair including an intraoral splint for an iatrogenic mandibular fracture during mandibular canine tooth extraction at the primary care veterinarian office. Oral surgery was followed by 5 sessions of HBOT at 1.5 ATA in a small animal chamber on days 1, 5, 8, 10, and 12. Culture and sensitivity results became available after the third HBOT session, and the patient had already improved. Antibiotic therapy was modified to an antibiotic effective against the MRSP cultured, chloramphenicol[d] (55.7 mg/kg) PO every 8 h for 14 days. Case 2 had surgical debridement and repair of a full-thickness incisional dehiscence associated with MRSP infection at the vertical center of a previous lip melanoma excision, followed by 5 sessions of HBOT at 2.0 ATA in an equine chamber on postoperative days 3, 4, 5, 6, and 7. Since case 2 responded to oral surgery followed by HBOT and the discharge had fully resolved before the culture results were available, organism-specific antibiotic therapy was not pursued. Patient progress is depicted in Figure 2A–D.

Category 2: Cases With Compromised Tissues due to Insufficient Healthy Tissue to Heal

Cases 3 through 8 were cases with various underlying pathology, each resulting in clinician concern for insufficient healthy tissue to heal. Case 3 was the only patient in the case series with a pre-existing medical history of seizures, which were well controlled with medical management. Case 3 had osteomyelitis, an unfavorable mandibular fracture, and bilateral oronasal fistulae. The mandibular fracture occurred during meal consumption 2 days after extraction of the left mandibular first molar tooth (309) at the primary care clinic, which had been performed with both lingual and buccal bone removal. The first oral surgery included extraction of retained tooth roots, fracture repair with an intraosseous suture to align the fragment sections, and placement of a demineralized bone matrix with cancellous bone chips[u] graft at the fracture site. In addition, debridement and local closure of the oronasal fistulae was performed without significant flap creation. Two HBOT sessions at 1.5 ATA in the small animal chamber were performed on days 0 and 1. Sixteen days after the first surgery, a second surgery to modify the repair was performed due to shifting of the mandibular repair, which caused a mucosal defect on the lingual aspect of the mandible. The bone margins in this area were smoothed, and the mucosa was sutured closed. Crown height reduction with vital pulp therapy of the right mandibular canine tooth (404) was performed due to ongoing palatal soft tissue trauma. Two additional sessions of HBOT were performed at 1.5 ATA in the small animal chamber on day 0. The patient healed well other than the bilateral oronasal fistulae, and the vital pulp therapy was deemed successful at the last radiographic medical progress evaluation performed 2 years postoperatively. At that time, persistent oronasal fistulae were addressed with large mucogingival flaps.

Case 4 was a pediatric feline patient that experienced a crush-type oral injury due to canine bite wounds. Bilateral caudal mandibular fractures were treated with surgical debridement, laceration repair, nonrigid maxillomandibular fixation (labial button technique) followed by 4 sessions of HBOT at 1.5 ATA in the small animal chamber on postoperative days 4 and 7. Two sessions were performed on each day with 4 h between treatments on day 4 and 7 h between treatments on day 7. The patient had healed well at follow-up imaging 14 weeks later.

Case 5 was the only case in the series where HBOT was utilized preoperatively to reduce oral inflammation. Case 5 was an FIV + adult cat with stomatitis. To avoid steroid treatment due to the potential immune compromise of the patient, 5 daily preoperative sessions were performed at 1.5 ATA in the small animal chamber on days −5, −4, −3, −2, and −1. The gingiva and mucosa were noted to be equally as hemorrhagic and friable after HBOT as they were prior to the sessions. After the HBOT sessions were completed, partial mouth extractions were performed, and ongoing medical management after surgery was required.

Case 6 utilized HBOT for endodontic health in addition to soft tissue and bony healing. Case 6 was a juvenile dog with bilateral rostral mandibular fractures, including lateral luxation of the immature permanent mandibular incisors, sustained during a fence related injury. Oral surgery, including focal areas of debridement, laceration repair, repositioning of the laterally luxated dentition in the fracture alveoli, and intraoral splint placement from the left mandibular canine tooth, extending along the labial aspect of the mandibular incisors, to the right mandibular canine tooth (304-404) was performed ~37 h after the patient was presented at an emergency service. Two HBOT sessions were performed ~6 h apart at 1.5 ATA in a small animal chamber on postoperative day 4. Successful soft tissue and bony healing occurred. The intraoral splint was removed 10 weeks later. Radiographic evidence of dentin deposition and apexogenesis was present on progress evaluation at 2 years, demonstrating additional tooth development since the time of injury. However, the radiographic appearance was not consistent with the normal age-related development of the rest of the dentition, indicating an eventual loss of tooth vitality.

Table 3. Summary of Case Details.

Case #	Signalment	Indication for HBOT	Concurrent medical conditions	Concurrent medications	Oral surgery	HBOT chamber type	ATA	No. of sessions	Session days	Outcome score	Time of last progress evaluation
Case 1	10-year-old FS dachshund	MRSP osteomyelitis Iatrogenic, transverse, displaced, rostral mandibular fracture at extraction site of 304	Poor bone quality 304-305; facial edema after induction; periodontal disease	Clindamycin[a], tramadol[b], carprofen[c] Antibiotic modified to chloramphenicol[d]	Debridement, intra-oral splint placement	Small animal	1.5	5	1, 5, 8, 10, 12	3	30 months
Case 2	8.5-year-old M Newfoundland	MRSP incisional infection; dehiscence of excision of lip malignant melanoma	Hypoalbuminemia, hypoproteinemia	Cefpodoxime proxetil[e], carprofen[c]	Debridement, surgical repair	Equine	2.0	5	3, 4, 5, 6, 7	2	9 months
Case 3	14-year-old M dachshund	Osteomyelitis, unfavorable, oblique, caudal mandibular fracture at 309	Well-controlled seizures; allergies	Oclacitinib[f], zonisamide[g], tramadol[b], grapiprant[h], cefovecin sodium[i], antibiotic modified to amoxicillin/clavulanic acid[j], gabapentin[k], buprenorphine[l]	(1) Fracture repair, intraosseous wire, local closure of ONF	Small animal	1.5	2	0, 1	1	24 months
		Sublingual mucosal dehiscence presumed secondary to contact with shifted portion of left mandible, VPT		Amoxicillin[m], gabapentin[k], tramadol[b]	(2) Debridement of shifted left mandible at fracture site lingual aspect, surgical closure, VPT 404	Small animal	1.5	2	0 (twice)	Mucosa, bone 3 Tooth vitality 3	24 months
Case 4	0.3-year-old M DSH	Crush injury/bite wound; bilateral caudal mandibular fractures	None	Robenacoxib[n], buprenorphine[l], clindamycin[a]	Debridement, laceration repair, labial button technique MMF	Small animal	1.5	4	2 (twice), 7 (twice)	3	3.5 months
Case 5	3-year-old MN DSH	Preoperative treatment as anti-inflammatory for mucosa and gingiva prior to partial mouth extractions; without steroid administration	FIV positive; ventricular premature contractions	Buprenorphine[l]	Partial mouth extractions, gingival biopsy	Small animal	1.5	6	−30, −5, −4, −3, −2, −1	0	1 month
Case 6	0.5-year-old M Goldendoodle	Mandibular fracture with mucosal	None	Clindamycin[a],	Focal debridement, laceration repair,	Small animal	1.5	2	4 (twice)	Mucosa, bone 3	18 months

(continued)

Table 3. (continued)

Case #	Signalment	Indication for HBOT	Concurrent medical conditions	Concurrent medications	Oral surgery	HBOT chamber type	ATA	No. of sessions	Session days	Outcome score	Time of last progress evaluation
		laceration, lateral luxation of mandibular incisors with alveolar fracture and moderate displacement of fracture segment		carprofen[c], gabapentin[k]	intraoral splinting of laterally luxated dentition					Tooth vitality 2	
Case 7	0.3-year-old F Miniature Poodle	Electrical cord injury, devitalization of soft tissue and bone, loss of soft tissue with denuded bone	None	Amoxicillin/clavulanic acid[j], meloxicam[o]	Debridement of soft tissue and bone	Small animal	1.5	6	0, 1 (twice), 2, 3, 4, 5	2	20 months
Case 8	8-year-old M dachshund	Advanced periodontal disease with absent surrounding soft tissue (lips, vestibule, mucosa), bilateral ONF	Systolic heart murmur, heartworm antigen positive; kerato-conjunctivitis sicca OD, anemia, hookworms	Doxycycline[p], carprofen[c], ophthalmic cyclosporine[q]	Extractions, bilateral ONF repair with flexible sheet of demineralized cortical bone[u]	Small animal	1.5	7	0, 1, 2, 3, 4, 6, 7	2	15 months
Case 9	11-year-old FS beagle	Presumptive radiation osteonecrosis 2 years after radiation therapy	Lingual soft tissue sarcoma, previous SRT 2 years prior, stable renal azotemia	Clindamycin[a], metronidazole[r], carprofen[c]	(1) Bone and soft tissue debridement	None	N/A	0	N/A	0	9 months
			Urinary tract infection, systolic heart murmur	Antibiotics switched to amoxicillin/clavulanic acid[j]	(2) Left rostral mandibulectomy	Equine	2.0, 2.5, 2.0	5	1, 2, 3, 5, 8	2	7 months
				Gabapentin[k], carprofen[c]	(3) Right rostral mandibulectomy	Equine	2.0	3	7, 8, 9	2	5 months
Case 10	13-year-old MN Goldendoodle	Extractions at previously irradiated site 4 years prior	Laryngeal paralysis, previous CAA, previous SRT 4 years prior, osteoarthritis, increased liver enzymes, rostral mandibular mass	Grapiprant[h], amoxicillin/clavulanic acid[j], acetaminophen/codeine[s]	Extractions	Equine	2.0	3	1, 4, 5	3	18 months
Case 11	2-year-old FS Labrador Retriever	Maxillectomy at previously irradiated site 3 months prior; osteonecrosis noted on histopathology	Previous SRT 3 months prior, undifferentiated soft tissue sarcoma	Amoxicillin/clavulanic acid[j], carprofen[c], gabapentin[k], trazodone[t]	Caudal maxillectomy	Equine	2.0	6	1, 4, 5, 6, 7, 8	3	11 months

Abbreviations: ATA, atmospheres absolute; HBOT, hyperbaric oxygen therapy.
[a]Multiple treatments on the same day.

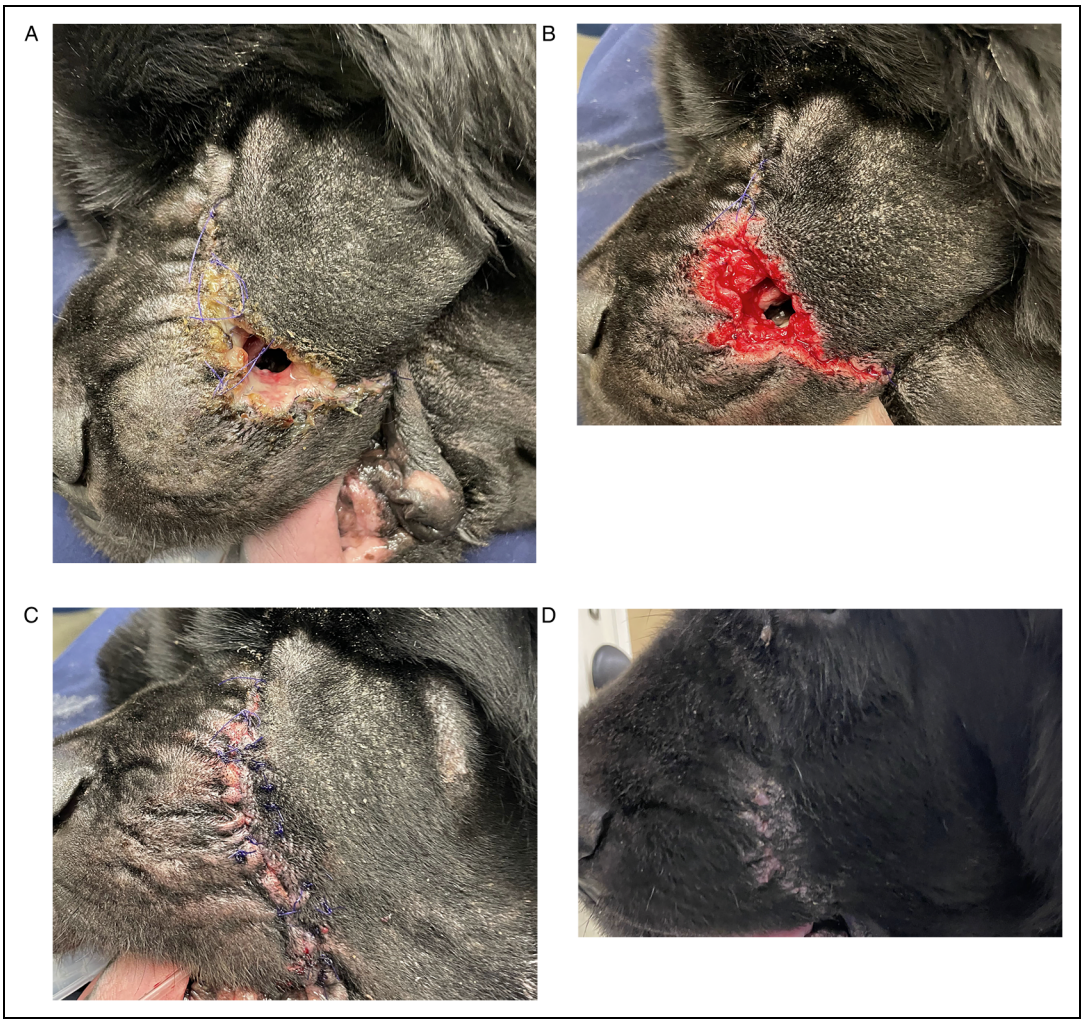

Figure 2. Case #2. Surgical repair followed by HBOT sessions for repair of a full-thickness dehiscence associated with MRSP infection at the vertical center of a previous melanoma excision. (A) Preoperative. (B) Intraoperative. (C) Immediately postoperative. (D) Four-week postoperative.

Cases 7 and 8 had significant loss of surrounding tissue available for oral surgery. Case 7 was a juvenile miniature poodle with an electrical cord bite injury sustained 10 days prior that resulted in loss of palatal mucosa, maxillary and mandibular gingiva and mucosa, lip margins, and a focal area of the vestibule on the side of the injury. Oral surgery included lavage of the wounds and debridement of diseased bone and soft tissue. Seven HBOT sessions at 1.5 ATA in the small animal chamber were performed on days 0, 1 (2 sessions on day 1, 4 h apart), 2, 3, 4, and 5 to achieve maximal soft tissue healing before pursuing further oral surgery. At 2 weeks, the lips were observed to have healed completely, and at 4 weeks the palatal mucosa had healed and the denuded area of mandibular bone was noted to be markedly reduced in size. Three to four months later, additional surgery was required to extract teeth with delayed or absent eruption and to close soft tissue over the remaining small areas of denuded mandibular bone in the region of those unerupted teeth.

Case 8 was an adult male dachshund with advanced periodontal disease at the time of rescue with notable absence of the maxillary buccal mucosa, vestibule, and lips in the regions of the right maxillary second premolar to the first molar teeth (106-109) and the left maxillary second premolar to the second molar teeth (206-210) as well as oronasal/oroantral fistulae bilaterally. Haired skin was present in lieu of mucosa in these regions. The first oral surgery included extraction of the mandibular dentition affected by periodontal disease and an anesthetized oral examination with cone beam computed tomography (CBCT) to plan for definitive oronasal fistula repair. HBOT was not pursued after the first oral surgery as the mandibular gingiva and mucosa were deemed healthy enough for routine healing.

One week later, bilateral oronasal fistula repair utilizing flexible sheets of a demineralized cortical bone membrane[v] was performed. Following the second oral surgery, 7 sessions of HBOT at 1.5 ATA in the small animal chamber were performed on days 0, 1, 2, 3, 4, 5, and 6. At the 2 week medical progress examination, small, focal dehiscence at the most caudal aspect of both of the oronasal/oroantral fistulas was

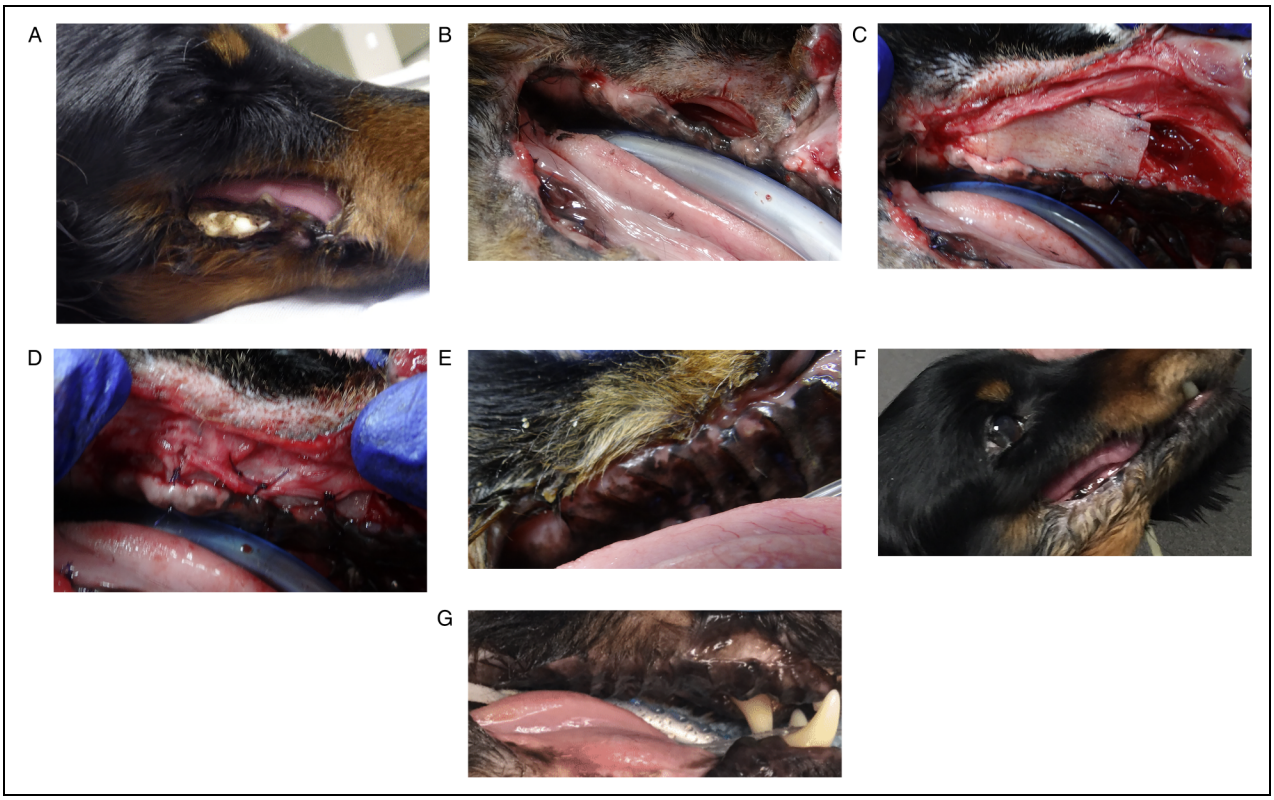

Figure 3. Case #8. Right side oronasal fistula repair in the absence of surrounding mucosa, vestibule, and lips. (A) Presentation. (B) Preoperative right side. (C) Intraoperative. (D) Surgical closure. (E) One-month postoperative demonstrating full fistula closure. (F) Conscious appearance 1 month postoperative. (G) Fourteen-month anesthetized examination.

observed. At the 6 week medical progress examination, the right oronasal/oroantral fistula had healed completely. The persistent left oronasal/oroantral fistula was repaired utilizing a flexible sheet of demineralized cortical bone membrane[u] and a palatal mucosal flap. HBOT was not pursued after the left oronasal fistula revision surgery due to financial constraints. A persistent 1 mm fistula remained. The patient was asymptomatic, so additional surgery was not pursued. Patient progress is depicted in Figures 3A–G and 4A–H.

Category 3: Cases With Compromised Tissues due to Radiation Injury

Cases 9, 10, and 11 were cases with oral surgery performed within a field that had been previously irradiated. Case 9 had stereotactic radiation therapy (SRT) with a total of 33 Gray (Gy) performed 2 years prior to oral surgery. The patient was presented for treatment of exposed, infected mandibular bone. The first surgery included aggressive local bone and soft tissue debridement and flap closure. Postoperative HBOT was declined by the owner at the time of the first surgery. The mucopurulent discharge resolved; however, complete dehiscence of the surgical site occurred, resulting in a recurrence of mandibular bone exposure. No additional healing occurred, and 2 months later, a second surgery to excise bone affected by the presumptive radiation osteonecrosis was performed. Attempts to contact the radiation oncologist to confirm the radiation field before the second surgery were unsuccessful.

The second surgery included a left rostral mandibulectomy and extraction of the right mandibular first incisor tooth (401) due to periodontal disease. Five HBOT sessions were performed on postoperative days 1, 2, 3, 5, and 8. The first HBOT session at 2.0 ATA in the equine chamber was well tolerated. Transient seizure activity (whole-body tremors) occurred at chamber pressure 2.5 ATA during the second session. Chamber pressure was decreased to 2.0 ATA, the seizure activity resolved, and the session was completed at the lower pressure. Additional HBOT sessions were completed at chamber pressure 2.0 ATA, and no further seizure activity occurred. Healing was achieved other than a focal area at the symphysis. Over the next several months, no further healing was achieved with medical management.

Prior to the third oral surgery, the radiation field was confirmed to include a portion of the right mandible, including the site of the previously extracted tooth 401. A right rostral mandibulectomy was performed, followed by 3 HBOT sessions at 2.0 ATA on postoperative days 7, 8, and 9. No seizure activity occurred. Healing was achieved other than a small, focal area at the rostro-dorsal aspect of the right mandible. The patient was asymptomatic during this exposure and remained unchanged over the next 5 months, at which time the patient was euthanized due to lingual sarcoma recurrence.

Figure 4. Case #8. Left side oronasal fistula repair in the absence of surrounding mucosa, vestibule, and lips. (A) Presentation. (B) Preoperative. (C) Intraoperative. (D) Immediate postoperative. (e) One-month postoperative. (F) One-month postoperative conscious appearance. (G) Immediate postoperative palatal flap to close the persistent oroantral fistula. (H) Fourteen-month postoperative persistent, reduced, asymptomatic oroantral fistula.

Case 10 had SRT with a total of 27 Gy 4 years prior to oral surgery which consisted of extractions and bony debridement due to an odontogenic abscess within the radiation field. Histopathology was not performed. Three sessions of HBOT at 2.0 ATA in an equine chamber on postoperative days 4, 5, and 6 were performed, and the surgical site healed fully without complications.

Case 11 had SRT with a total of 36 Gy performed ~3 months prior to oral surgery. A caudal maxillectomy was performed within the radiation field and histopathology of the excised section revealed osteonecrosis, presumably radiation induced. Six sessions of HBOT were performed at 2.0 ATA in an equine chamber on days 1, 4, 5, 6, 7, and 8, and the surgical site healed completely without complications (Figure 5). After oral surgery followed by HBOT sessions, both cases 10 and 11 were observed to have healed completely at medical progress examinations.

Discussion

This case series includes dentistry and oral surgery patients who may have benefited from adjunctive HBOT. This modality can provide the oral surgeon with an additional therapeutic tool to treat patients with compromised tissues. In this case series, the oral diseases treated with HBOT and associated with a positive outcome included the following: resistant bacterial infections, oral trauma/crush injury, mandibular fractures, electrical cord injury, osteomyelitis, oral surgery within a previous radiation therapy field, and presumptive radiation osteonecrosis. Conditions within this case series that remained unchanged or were associated with partial improvement included preoperative HBOT for stomatitis without steroid treatment in an FIV + cat and delayed surgery and postoperative HBOT for long-term endodontic health of laterally luxated immature permanent mandibular incisors.

If electing to pursue HBOT for patients with compromised tissues, in addition to the potential benefits previously described, the oral surgeon should be aware of contraindications to HBOT, potential risks associated with therapy, and the treatment plan and protocols to be utilized by the HBOT facility.

The absolute contraindication to HBOT is an unvented pneumothorax due to trapped gas undergoing pressure changes exacerbating pneumothorax symptoms. Patients with pneumothorax venting via a Heimlich valve are permitted within the chamber.

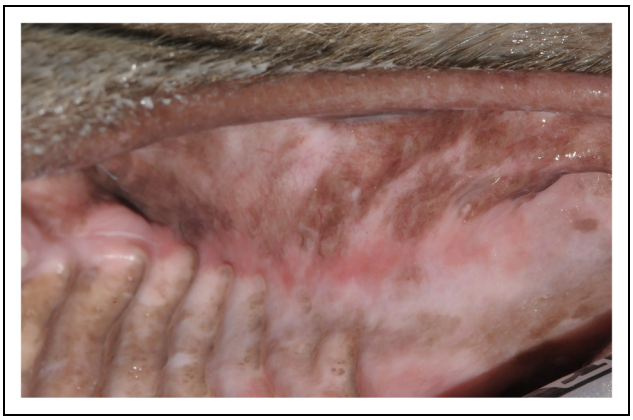

Figure 5. Case #11. Follow-up anesthetized, intraoral photograph of left caudal maxillectomy site completely healed in previous radiation field with osteonecrosis present on histopathology.

Patients with a history of trauma should have thoracic radiographs obtained prior to receiving HBOT. Due to differences in respiratory physiology, HBOT should not be pursued in non-mammalian species.[6]

Relative contraindications are related to trapped gas, oxygen toxicity, or pressure changes. Relative risks associated with trapped gas include intraocular gas, bronchospasm/chronic obstructive pulmonary disease, recent thoracic surgery or trauma, and dental disease such as incomplete fillings, non-vital teeth, or periapical disease. Relative risks related to oxygen toxicity potentially include the concurrent use of bleomycin or doxorubicin, optic neuritis, or retinopathy. Relative risks related to pressure changes include respiratory infections that could potentially interfere with the ability to equalize ear pressure, otitis media, and implanted devices (i.e., pacemakers which may not be designed to function or withstand increased pressure). Patients with congestive heart failure or uncontrolled seizures should not undergo HBOT treatments.[6] Pre-existing, well-controlled seizures are not a contraindication to HBOT. Any metal that cannot be removed should be covered within the chamber due to the risk of spark with the highly combustible nature of oxygen. The metallic component of the intraoral splints in this case series was covered by a bis-acrylic composite[w] and therefore deemed safe for HBOT treatments. Veterinary dentistry patients with metal crowns, particularly those with multiple crowns that could contact in occlusion, should not be permitted within the HBOT chamber. Catastrophic events can occur when safety protocols are not followed. Explosive chamber fires have occurred secondary to sources of potential sparks being inappropriately brought into HBOT chambers.[7]

Risks of therapy include otic barotrauma, pulmonary barotrauma, barodontalgia, reversible myopia, irreversible cataract progression, and oxygen toxicity seizures. The most frequent side effect reported in human clinical medicine is otic barotrauma.[6,10] Oxygen toxicity seizures are more likely at higher chamber pressures and when the patient is febrile.[9] As a standard safety precaution, rectal temperature was obtained in all pets prior to HBOT sessions in this case series. To avoid pretreatment hyperthermia, clinicians should consider avoiding medications associated with a possible increase in body temperature (i.e., opioids), for several hours before scheduled sessions. Patient rectal temperature has been shown to decrease slightly during HBOT sessions, including during sessions when the patient is panting.[9] Oxygen toxicity-induced seizures have been reported in 0.02% to 0.03% of human patients and informally observed in <0.35% at 2 ATA in veterinary patients.[9] In 2 retrospective studies evaluating a combined 515 HBOT sessions in veterinary patients at 2.0 ATA, no oxygen toxicity seizures were observed.[8,9] In this retrospective case series, 1 patient experienced transient seizure activity at 2.5 ATA, which resolved as the chamber pressure was lowered to 2.0 ATA.

Claustrophobia and anxiety are also a risk of HBOT. In this case series, 3 patients were noted to display anxious behaviors, such as head shaking, panting, pawing, and/or chewing. Of these 3 patients, 1 required oral anxiolytic medication prior to future HBOT sessions. Two patients were noted to be restless during decompression of their first few sessions, but not during subsequent sessions. Anxious behavior did not prevent any of the patients in this series from completing the recommended course of HBOT. Interestingly, all the patients within this case series that had been observed to have anxiety had been treated at the equine facility. The pets treated within the equine chamber had substantially more room to ambulate which could account for their restless behaviors. In addition, the HBOT treatment logs at the equine facility included a comment section for the behavior of the patient during every session, thereby potentially increasing observer sensitivity. A large retrospective study reviewed 230 HBOT sessions at 2.0 ATA for 78 dogs and 12 cats and found 76 mild clinical adverse events reported, with the most frequent being head shaking, panting, and swallowing. None of these events required intervention.[8] It has been informally observed that most adverse events typically occur during compression and decompression, presumably due to otic pressure changes.[8] In this retrospective case series, 88% of the HBOT sessions were well tolerated.

While the HBOT facility typically determines the HBOT treatment protocol, the veterinary oral surgeon can influence initiation of therapy and therefore should be aware that the time interval to treatment can be a potential factor influencing the clinical outcome of HBOT treatments. For the treatment of central retinal artery obstruction in people, HBOT is recommended within the first 24 h of injury, along with supplemental oxygen between HBOT treatments.[1] Avoiding a time delay to treatment is especially important for patients with vascular supply disruption, such as acute blindness after an anesthetized oral procedure or luxated dentition.

When evaluating the frequency of pulp necrosis for luxated permanent dentition in a prospective human study, the most important prognostic factors were the type of injury and the stage of root development.[11] Laterally luxated teeth with an open apex (i.e., immature permanent dentition) had a much higher rate of pulp survival (>90%) at 1 year when compared with laterally luxated teeth with a closed apex (>20%), presumably due to an increased area for repair and neovascularization to occur.[11] While neovascularization starts in the first few days,

vasculature can be identified by microradiography with contrast at 10 days in the apical half of the pulp and at 30 days in the entirety of the pulp of replanted, immature canine dentition.[12] The risk of pulp necrosis at 1 year of a laterally luxated immature permanent tooth increases >10-fold if a concurrent crown fracture is present.[13] Endodontic survival of immature permanent dentition for an additional year would allow for apexogenesis to occur and thus the potential for endodontic therapy to be pursued if indicated. At 2 years post injury, case 6 was noted to have radiographic evidence of apexogenesis, tooth root resorption, and a lack of continued age-appropriate development. Case 6 had a time delay of 37 h from injury/lateral luxation of the immature permanent mandibular incisors to oral surgery, followed by a delay of 4 days prior to HBOT. HBOT may have been more impactful for long-term endodontic health if performed immediately after injury and during the period prior to potential pulp revascularization.

Case 7 had a delay of 10 days after electrical cord injury prior to referral to a veterinary dentistry and oral surgery specialist. Future studies would be needed to evaluate if immediate HBOT after an electrical cord injury would help minimize the extent of the loss of gingiva and mucosa. Time for necrotic tissue to become apparent prior to debridement surgery would still be required. HBOT could then additionally be pursued after debridement surgery.

Treatment protocols for resistant bacterial infections following surgical debridement should also begin without delay whenever possible. For both cases 1 and 2, surgical debridement followed by HBOT resulted in resolution of visible symptoms of the clinical infections before the patients were treated with appropriate antibiotics based on culture results. It is particularly noteworthy that the MRSP infection associated with case 2 resolved with surgical debridement followed by HBOT without ever being treated with an organism-specific antibiotic. The oral surgeon may want to consider adjunctive HBOT when treating multidrug-resistant infections.

While conflicting reports are present in the human literature regarding the efficacy of HBOT in treating oral surgery patients who previously received radiation therapy, the UHMS describes and reviews the Marx treatment protocol. This protocol includes treating radiation osteonecrosis with 30 sessions prior to surgery, to excise all affected bone, and then to follow surgery with 10 additional sessions and includes treating extractions in a previously irradiated field with 20 sessions prior to extractions followed by 10 sessions post extractions.[1] In a retrospective study of 37 human patients having extractions after radiation therapy, 4% of patients treated with HBOT (20 sessions prior to extractions and 10 sessions postoperatively at 2.4 ATA for 90 min) developed osteonecrosis compared with 15% of the patients who had extractions without HBOT.[13] This extended protocol may not be financially feasible for the average veterinary client. The patients in this case series with radiation injury concerns treated at the equine facility were recommended to have a minimum of 5 daily postoperative HBOT treatments and to start HBOT treatments on postoperative day 1. It is important to note that HBOT alone without excising bony osteonecrosis is not expected to resolve radiation osteonecrosis even with a prolonged course of HBOT treatment.[1]

Although the oral surgeon will influence initiation of HBOT treatments via timely referral, ultimately the treatment plan will be determined by the HBOT facility. Small animal patients are typically treated with once to twice daily treatments for 50 to 90 min at an ATA of 1.4 to 2.8.[3] While veterinary treatment protocols are extrapolated from human clinical use, the number of treatments per day and the total number of treatments are often influenced by chamber availability, HBOT technical support, owner time and financial constraints, and the availability of medical boarding for pets at the facility. The small animal HBOT facility in this case series was able to provide medical boarding for patients to support twice daily HBOT sessions. The equine facility in this case series had HBOT technical support constraints in addition to not providing medical boarding for small animal patients; thus they performed HBOT sessions once daily.

Proper surgical techniques, such as large, tension-free mucogingival flaps to repair oronasal fistulae, are still required when utilizing HBOT. In case 3, an attempt to debride and close the bilateral oronasal fistulae with only local surgical management was made due to time limitations associated with the mandibular fracture repair procedure. While the bilateral oronasal fistulae were not the focus of that surgical procedure, they did not heal with adjunctive HBOT without proper surgical techniques. These fistulae were later repaired with appropriate, large, tension-free, mucogingival flaps.

An inherent limitation of this study is the nature of a retrospective case series. These cases represent a heterogenous population of canine and feline patients with various pre-existing medical conditions, indications for HBOT, and postoperative care. As this case series is not a prospective, randomized, controlled study, one cannot comment if these patients would have healed equally well with or without HBOT. An additional limitation of this study is that the patients receiving treatment at the equine facility all had oral surgery performed by an AVDC diplomate. Patients receiving HBOT at the small animal facility either had surgery performed by an AVDC diplomate or a dentistry and oral surgery resident under the supervision of an AVDC diplomate. Thus, varying degrees of surgical experience were present in this case series.

Further studies are needed to confirm and further characterize the potential benefits of HBOT for compromised tissues and identify the ideal treatment protocols for each disease condition. In the interim, since HBOT may provide clinical benefits, it should be considered as an adjunctive treatment modality for oral surgery for patients with compromised tissues.

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Materials

(a) Clindamycin, Chronus Pharmaceuticals, Inc., Setauket, NY, USA.
(b) Tramadol, Sun Pharmaceuticals Industries Limited, Cranbury, NJ, USA.
(c) Carprofen, Dechra Veterinary Products, Overland Park, KS, USA.
(d) Chloramphenicol, GPS Compounding Pharmacy, NC, USA.
(e) Cefpodoxime proxetil, Zoetis, Inc., Piscataway, NJ, USA.
(f) Oclacitinib maleate, Zoetis, Inc., Piscataway, NJ, USA.
(g) Zonisamide, Eisai Co. Ltd, Nutley, NJ, USA.
(h) Grapiprant, Aratana Therapeutics Inc., Leawood, KS, USA.
(i) Cefovecin sodium, Zoetis, Inc., Piscataway, NJ, USA.
(j) Amoxicillin/clavulanic acid, Zoetis, Inc., Piscataway, NJ, USA.
(k) Gabapentin, Epicur Pharma, Mount Laurel, NJ, USA.
(l) Buprenorphine, Epicur Pharma, Mount Laurel, NJ, USA.
(m) Amoxicillin, Zoetis, Inc., Piscataway, NJ, USA.
(n) Robenacoxib, Elanco Animal Health, Greenfield, IN, USA.
(o) Meloxicam, Boehringer Ingelheim, Iselin, NJ, USA.
(p) Doxycycline hyclate, West-Ward Pharmaceuticals Corp., Eatontown, NJ, USA.
(q) Ophthalmic cyclosporine, Merck Animal Health, Madison, NJ, USA.
(r) Metronidazole, Unichem Pharmaceuticals, East Brunswick, NJ, USA.
(s) Acetaminophen/codeine, Mallinckrodt, SpecGx LLC, Webster Groves, MO, USA.
(t) Trazodone, Teva Pharmaceuticals, Parsippany, NJ, USA.
(u) PerioMix®, Veterinary Transplant Services®, Inc., Kent, WA, USA.
(v) Ossiflex™, Veterinary Transplant Services®, Inc., Kent, WA, USA.
(w) ProTemp™, 3M™ ESPE, St. Paul, MN, USA.

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