Henry Ford Hospital Medical Journal

Volume 38 | Number 1 Article 5

3-1990

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Management of Chronic Staphylococcal Osteomyelitis of the Temporal Bone: The Use of Hyperbaric Oxygen

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Hyperbaric oxygen (HBO) is an effective adjunct in the management of selected otolaryngologic problems including radiation-induced necrosis of the temporal bone, malignant external otitis, mandibular osteoradionecrosis and refractory osteomyelitis, soft tissue head and neck necrotizing fasciitis, compromised skin flaps and grafts, acute air or gas embolism, and otologic barotrauma. We describe the management of a patient with insidious Staphylococcus aureus *osteomyelitis of the temporal bone by the use of HBO preoperatively and postoperatively in conjunction with surgical debridement. The possible application of angiogenic agents and tetracycline bone-labeling in combination with HBO therapy in the management of refractory neurotologic disease is discussed. (Henry Ford Hosp Med J 1990;38:16-20)*

In the past 15 years many studies have reported the application of hyperbaric oxygen (HBO) as an adjunct to the treatment of radiation-induced necrosis of the temporal bone, malignant external otitis, mandibular osteoradionecrosis and refractory osteomyelitis, soft tissue head and neck necrotizing fasciitis, compromised skin flaps and grafts, and acute air or gas embolism. The use of HBO has also been investigated in the treatment of sudden sensorineural hearing loss, tinnitus, and vertigo secondary to acute otologic hypoxia and barotrauma (1-13). Information regarding the indications for HBO therapy, its mechanisms of action, and details of treatment protocol have been documented under the auspices of the Hyperbaric Oxygen Committee of the Undersea Medical Society (14,15).

Conditions for which HBO is accepted as indicated therapy are listed in Table 1. Mandatory rules for HBO administration include: 1) abstinence of smoking during the entire course of therapy; 2) avoidance of flammable synthetic clothing (ie, nylon); 3) avoidance of flammable topical materials, such as petroleum jelly, glycerin, lanolin products, hair spray, oils, tonic, wigs, mustache wax, cosmetics, cologne, or perfume; 4) patients must avoid gum or candy because of the risk of seizures; and 5) flammable reading material may not be brought into the chamber. Certain drugs and medical conditions which may hasten or delay the onset of HBO toxicity should be considered in the selection of patients (Tables 2 and 3).

Side effects of HBO therapy are the result of hyperoxygenation and barotrauma. These include transient reversible myopia, maturation of existing cataracts, seizures, nitrogen narcosis, and the adult respiratory distress syndrome or pulmonary fibrosis which are caused by hyperoxygenation. Pressure-related side effects include eustachian tube dysfunction, otologic barotrauma, sinus "squeeze," pneumothorax and/or pneumomediastinum, gastrointestinal gas expansion, arterial gas emboli, and decompression sickness. With appropriate patient screening and education, the incidence of these complications is minimal (15).

The beneficial effects of HBO therapy are the result of: 1) hyperoxygenation of tissues, hemoglobin, and plasma; 2) vasoconstriction without hypoxia which reduces tissue edema, compartment pressure, and intracranial pressure; 3) pressure effects which reduce gas bubble size; and 4) antimicrobial activity from enhanced leukocytic bactericidal activity and reduced production of toxins in soft tissue. A secondary effect of HBO therapy is enhanced wound healing because of fibroblast proliferation, improved osteoclastic function, and increased red blood cell deformability.

Pretherapy evaluation should include a chest x-ray and spirometry to rule out chronic obstructive pulmonary disease, blebs, and bullae, as well as a careful search for optic neuritis and cataracts. Claustrophobia and morbid obesity may preclude treatment depending on the type of chamber and method of oxygen administration.

The following report illustrates the management of an unusual case of *Staphylococcus aureus* osteomyelitis of the temporal bone which was secondary to cholesteatoma and chronic mastoiditis.

Case Report

Since childhood this 48-year-old woman had chronic tympanic membrane perforation with suppurative otitis media of the right ear. Fifteen years earlier she had been seen for granulomatous mastoiditis, otorrhea, and middle ear cholesteatoma. Cultures of middle ear drainage grew *Staphylococcus aureus* and *Pseudomonas aeruginosa*. Her morbid obesity and endomorphic habitus limited access to her veins

Submitted for publication: October 17, 1989.
Accepted for publication: April 24, 1990.
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Table 1
Accepted Indications for Use of Hyperbaric Oxygen Therapy

1. Radiation necrosis: Osteoradionecrosis: soft tissue radionecrosis
2. Refractory osteomyelitis
3. Refractory mycoses
4. Necrotizing and mixed soft tissue infections
5. Gas gangrene (Clostridial)
6. Compromised skin grafts or flaps
7. Healing enhancement in selective problem wounds
8. Acute traumatic ischemia (crush injuries)
9. Decompression sickness
10. CO_2 and cyanide poisoning; smoke inhalation
11. Arterial gas (air) embolism
12. Acute cerebral edema
13. Special consideration: thermal burns; acute anemia

Table 2
Factors Hastening Hyperbaric Oxygen Toxicity

Adrenocortical steroids	Dextroamphetamine
CO_2 inhalation	Epinephrine
Vitamin E deficiency	Norepinephrine
Hyperthyroidism	Insulin
Hyperthermia	Diamox
	Narcotic agents

Table 3
Factors Delaying Onset of Hyperbaric Oxygen Toxicity

Adrenergic blocking drugs	Intermittent O_2 exposure
Antioxidants	Hypothyroidism
Chlorpromazine	Starvation
Anticonvulsants	Hypothermia
Ganglionic blocking drugs	Acclimation to hypoxia
Glutathione	Reserpine
Gamma-aminobutyric acid	Succinate
Disulfiram	Trisaminomethane

and made either computed tomography (CT) or magnetic resonance imaging (MRI) very difficult. She had a 30+ pack/year smoking history and multiple topical and systemic drug allergies and idiosyncratic sensitivities.

The patient's condition worsened progressively over this 15-year period. Foul otorrhea was associated with spiking fevers (40° C [104° F), cellulitis and diffuse swelling over the temporal, parietal, and infraorbital areas, with woody induration of the auricle, and obliterative mastoid granulation. Deep pain persisted in the temporal, postauricular, and retro-orbital regions. Intermittent right facial twitching correlated with surgically documented intralabyrinthine and vertical facial nerve dehiscence (Figs 1A and 1B). Despite multiple surgical procedures and long-term antibiotic therapy, mastoid biopsies consistently demonstrated necrotic and hypertrophic mucosa, cholesteatoma, and fibroblastic proliferation. Granuloma formation was secondary to powdered chloramphenicol crystals. Cultures demonstrated methicillin-resistant *Staphylococcus aureus, Pseudomonas aeruginosa,* and *Bacteroides melaninogenicus.* Antibiotic treatment, all of which failed, included imipenem and vancomycin, imipenem and gentamicin, cefazolin sodium, and ciprofloxacin.

Systemic candidiasis, *Clostridia difficile* gastroenteritis, catheter-induced *Staphylococcus aureus* septicemia, cephalic vein thrombophlebitis, vancomycin-induced "red neck syndrome," and gentamycin-induced oscillopsia and ataxia all occurred, as complications of the long-term medical therapy.

Over a ten-year period the patient had ten surgical procedures, including tympanoplasty, extended radical mastoidectomy, temporal craniectomy with intradural pericranial graft, partial petrous apicectomy, facial nerve decompression, and mastoid cavity fat obliteration. CT of the right temporal bone demonstrated marked irregularity of the anterior wall of the right external auditory canal, highly suggestive of chronic osteomyelitis. CT also demonstrated dehiscence of the right tegmen tympani and lateral aspect of the petrous ridge, opacification of the mastoid, and early inflammatory changes in the right cerebellum suggesting an evolving intracranial abscess (Figs 2A and 2B).

Because conventional medical and surgical management had failed, the patient was referred for adjunct HBO therapy. After preoperative bone-labeling with tetracycline, the patient underwent extended radical revision mastoidectomy and split-thickness skin graft of the cavity. "Leathery" granulomatous tissue and osteitic bone were removed from the middle ear, eustachian tubal orifice, anterior bony canal wall, and facial ridge. Postoperatively, the patient received 20 outpatient HBO treatments (90 minutes daily at 2.4 atmospheres of absolute pressure [ATA]). After only ten HBO treatments, high-resolution CT demonstrated marked improvement in the irregularity of the external auditory canal wall and better definition of the petrous bone (Fig 2C). The deep pain, granulation tissue, and cellulitis resolved (Fig 3). The Westergren sedimentation rate returned to near normal (29 mm/hr from 83 mm/hr).

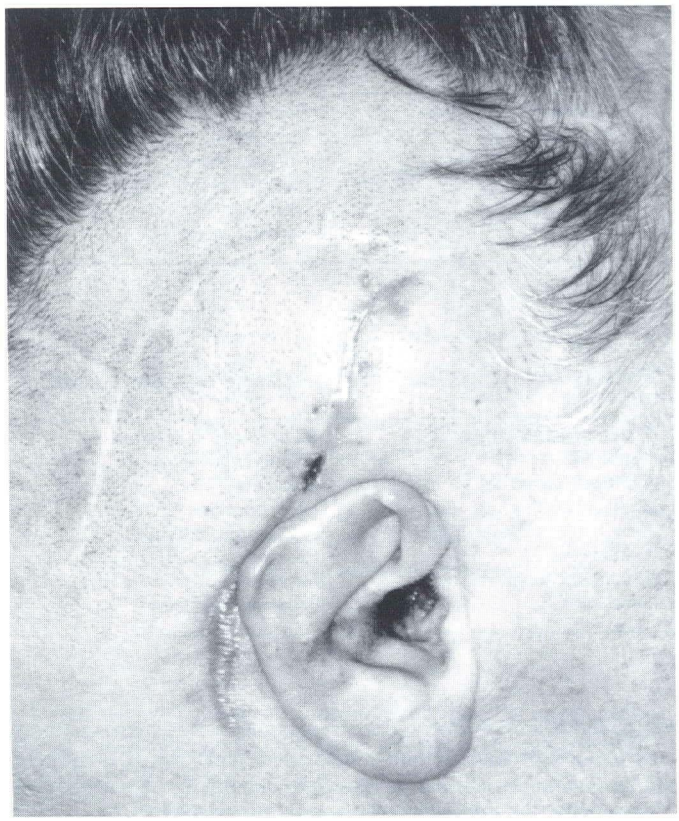
No evidence of temporal bone osteomyelitis persists one and a half years later. The patient wears a right bone conduction hearing aid. Her ataxia and oscillopsia have markedly improved, and she is ambulatory and capable of light work.

Discussion

Previous otolaryngologic reports have stressed the importance of combined antibiotic treatment and adjunct HBO therapy for treatment of radiation-induced osteomyelitis, osteoradionecrosis, and *Pseudomonas* "malignant" or necrotizing external otitis of the temporal bone (16-24). The present case suggests that HBO therapy is also effective in the adjunct management of chronic osteomyelitis of the temporal bone, which occurred as a complication of cholesteatoma and otomastoiditis.

Assessment of chronic osteomyelitis of the temporal bone must include detection of host compromising factors. Such evaluation requires sequential radiologic imaging with CT and radionuclide bone and soft tissue scintigraphy. Antibiotic therapy must be culture-directed and prolonged.

Warning signals suggesting development of chronic osteitis/osteomyelitis of the temporal bone include deep pain (temporal, parietal, postauricular, retro-orbital), intermittent foul otorrhea with spiking fevers, periauricular cellulitis, woody induration of the pinna, chronic mastoid-cutaneous fistula, fibrotic mastoid granulation tissue, intermittent facial twitching suggestive of facial canal dehiscence, and persistent leukocytosis with elevated sedimentation rate. Impending (neur)otologic complication may be signaled by the following: 1) acute exacerbation of purulent, malodorous drainage often preceded by headache or otalgia; 2) low-grade or spiking fever; 3) systemic symptoms, such as lethargy, emesis, blurred vision, altered mental status, apha-



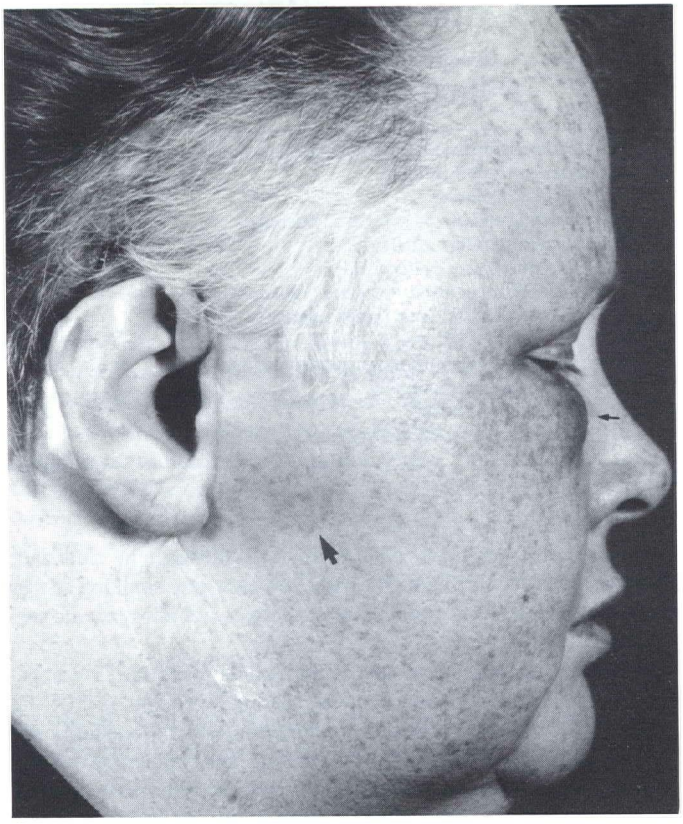


Fig 1 (A and B)—Staphylococcus aureus *osteomyelitis of the right temporal bone secondary to chronic suppurative otitis media with cholesteatoma. Cellulitis and swelling over the temporal and infraorbital regions. Rubbery, odiferous mastoid granulation tissue. Woody edema of the auricle. Spiking fevers (40 °C) associated with sedimentation rate four times normal.*

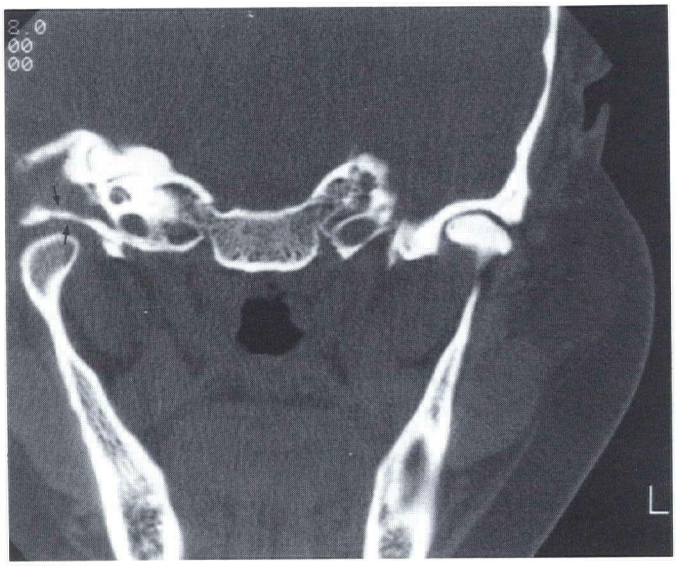


Fig 2A—Pre HBO therapy: high-resolution CT of the right temporal bone (coronal view). Erosion of anterior bony wall of external auditory canal. Opacification of the mastoid cavity.

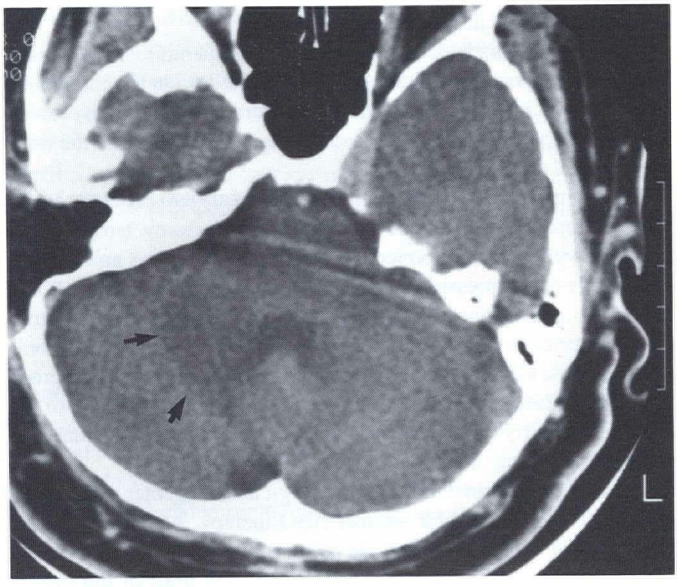


Fig 2B—Pre HBO therapy: high-resolution CT (axial view). Evolving abscess of the right cerebellum.

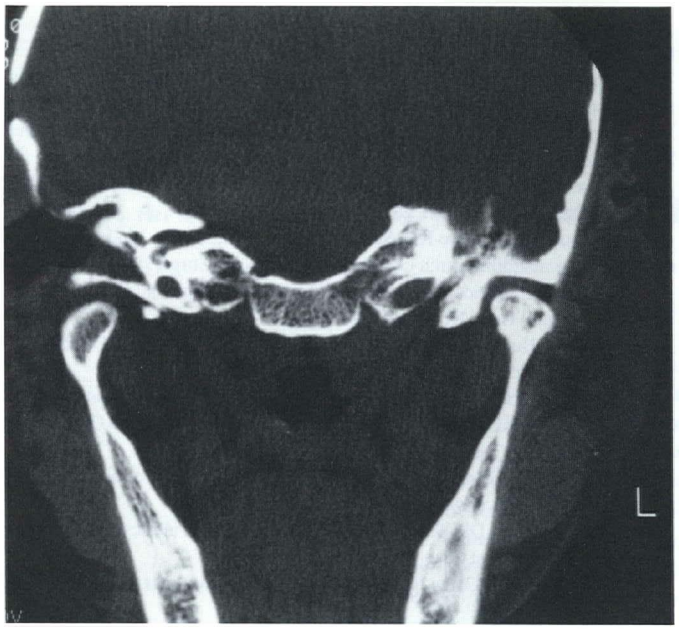


Fig 2C—Post HBO therapy: high-resolution CT after extended radical mastoidectomy, split-thickness skin graft of the temporal bone defect, ten of 20 HBO treatments, and oral ciprofloxacin. Less bone irregularity of anterior external auditory canal wall.

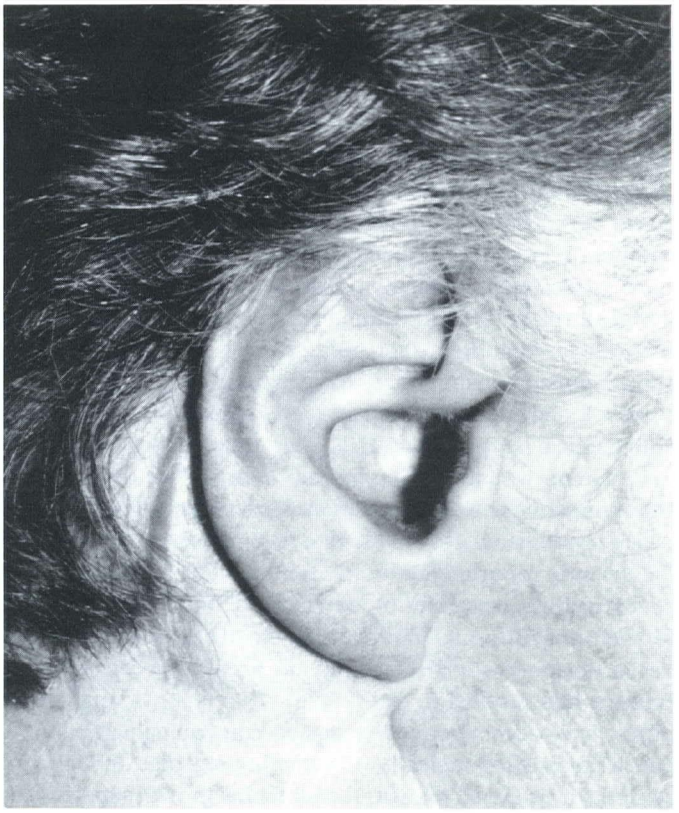


Fig 3—Operative site following 20 HBO treatments. Completely epithelialized mastoid defect. Healed postauricular mastoid-cutaneous fistula. No granulation tissue. Fever, pain, and abnormal sedimentation rate now resolved.

sia, and mental confusion; and 4) otologic complications, such as auricular cellulitis, suppurative labyrinthitis, and subperiosteal abscess (25).

The diagnosis of osteoradionecrosis, osteomyelitis, and malignant external otitis is primarily "clinical." Radiologic studies may demonstrate inflammatory disease of the temporal bone but often lag behind the severe early clinical signs. Radiologic bone changes demonstrating bone lysis and dissolution appear slowly even in acute osteolytic osteomyelitis. Bone imaging is not diagnostic until 30% to 50% of bone is lost (20). The three most reliable procedures for diagnosis and evaluation of the progress of chronic temporal bone inflammation are thin section, high-resolution CT in combination with gallium-67 citrate and triphasic ^{99m}technetium-labeled phosphate anion bone scintigraphy (18-22,26). Gallium scanning demonstrates the nonspecific binding of gallium to actively dividing cells and organelles (leukocytes, tumor cells, actively dividing osteoblasts) in soft tissue inflammation. The procedure reveals osteogenic activity from the stage of initial infection through phases of healing, persistence, or recurrence, indicating the degree of therapeutic response. ^{99m}Tc bone imaging demonstrates acute osteitis and osteomyelitis and is used to differentiate "healing" from active infection. Technetium scans can detect morphologic findings before structural changes are seen on CT and may reveal physiologic changes in advance of the healing process. Gallium scan findings improve or become normal when the infection is sterilized, while the technetium scan remains abnormal and indicates the extent of bone destruction.

Antimicrobial therapy is selected on the basis of cultures and antibiotic sensitivity. Ideally, antibiotic therapy should include an aminoglycoside plus an anti-Pseudomonal penicillin, a fluoroquinolone, or a carbapem.

Whenever possible, surgical management of chronic osteomyelitis of the temporal bone should be performed after preoperative tetracycline labeling. Operative plans include consideration of limited versus radical removal of nonviable bone, as well as postoperative management of the mastoid cavity. Tetracycline, 250 mg orally four times daily, is administered for seven to ten days prior to surgery. Use of ultraviolet light intraoperatively produces fluorescence of the viable bone and facilitates surgical removal of diseased tissue (10,16). Managing the residual mastoid cavity is a difficult problem, particularly when granulation tissue, drainage, and pain persist. Optimal healing may require obliteration of the cavity with abdominal fat, a rotational flap, or split-thickness skin graft.

HBO has been effective in treating experimentally-induced staphylococcal osteomyelitis in rabbit tibia (27). Compared to normal bone, osteomyelitic bone has decreased blood flow and markedly reduced partial pressure of oxygen. Reduced tissue oxygen impairs phagocytic and/or intracellular killing mechanisms (27).

The three variables of HBO therapy in the management of chronic osteomyelitis of the temporal bone are: 1) correlation with medical and surgical intervention, 2) duration of treatments

which is determined by clinical response and radiologic changes, and 3) frequency of "dives." Clinical response to HBO therapy is indicated by relief of pain, subsidence of otorrhea and fever, and fall to normal in the sedimentation rate. Resolution of pathological bone imaging and no further development of mastoid granulations six months following HBO therapy are evidence of remission. Although the absolute pressure used, the number of days, and frequency of HBO treatments may vary, standard treatment is usually conducted at 2.5 ATA (45 feet below sea level) for 90 to 120 minutes, one or two times daily. This schedule for high-dose, short-term, and intermittent 100% oxygen administration minimizes oxygen toxicity (2,4,15).

New experimental pharmacologic agents (in combination with medical, surgical, and HBO therapy) may prove effective in hastening wound healing by improving tissue oxygenation and stimulating neovascularization. Such pharmacologic agents include endothelial growth factors, free radical scavengers, and the recently reported methylxanthines. The methylxanthine, pentoxifylline, decreases blood viscosity, increases red blood cell deformability, and improves red blood cell flow properties. In combination with HBO therapy the agent has been shown to enhance flap survival in rats (28,29). Two types of angiogenic factors have been reported to enhance skin flap neovascularization when applied topically or when impregnated in Gelfoam carriers for sustained release: 1) endothelial growth supplement, a neural extract from bovine hypothalamus which is mitogenic to endothelial cells; and 2) endothelial growth cell factor, an acidic polypeptide from bovine neural tissue which induces endothelial cell replication (30). Certain free radical scavengers have been demonstrated to reduce flap ischemia and to delay the onset of HBO toxicity. These include superoxide dismutase, allopurinol, vitamin E, glutathione, and disulfiram (31). Future management of inflammatory disease of the head and neck may well involve the use of HBO in combination with such pharmacologic agents.

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