Clinical Policy Bulletin: Hyperbaric Oxygen Therapy (HBOT)

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Policy

I. Aetna considers systemic hyperbaric oxygen therapy (HBOT) medically necessary for any of the following conditions:

- Acute air or gas embolism
- Acute carbon monoxide poisoning
- Acute cerebral edema
- Acute peripheral arterial insufficiency (i.e., compartment syndrome)
- Acute traumatic peripheral ischemia (including crush injuries and suturing of severed limbs) when loss of tissue is threatened and HBOT is used in combination with standard therapy
- Chronic refractory osteomyelitis, unresponsive to conventional medical and surgical management
- Compromised skin grafts and flaps
- Cyanide poisoning (with co-existing carbon monoxide poisoning)
- Decompression illness ("the bends")
- Exceptional blood loss anemia only when there is overwhelming blood loss and transfusion is impossible due to lack of suitable blood available, or religion does not permit transfusions
- Gas gangrene (Clostridial myositis and myonecrosis)
- Idiopathic sudden deafness, acoustic trauma or noise-induced hearing loss, when HBOT is initiated
- Non-healing infected deep ulcerations (reaching tendons or bone) of the lower extremity in diabetic a patient

Standard wound care in persons with diabetic wound includes:

- Evaluation of vascular status and correction of any vascular problems in the affected limb if possible
- Optimization of glucose control
- Debridement by any means to remove devitalized tissue
- Mainstreaming of granulation tissue with appropriate moist dressings
- Appropriate off-loading, and
- Neces

Failure to respond to standard wound care occurs when signs of healing for at least 30 consecutive days. Wounds must be evaluated at least every 30 days of HBOT. Continued treatment with HBOT is not considered medically necessary if measurable sign of healing is not demonstrated within any 30-day period of treatment. Note: HBOT is not considered medically necessary for any of the following conditions:

- Pneumatoses cystoides intestinalis
- Progressive necrotizing soft tissue infections, including mixed aerobic and anaerobic infections (Mele fasciitis)
- Prophylactic pre- and post-treatment for members undergoing dental surgery of a radiated jaw
- Radiation-induced hemorrhagic cystitis
- Radiation necrosis (brain radionecrosis, myoradionecrosis, osteoradionecrosis, and other soft tissue...
II. Aetna considers the use of systemic HBOT experimental and investigational for the following conditions (no because there is insufficient evidence in the medical literature establishing that systemic HBOT is more effe therapies:

- Actinic skin damage
- Actinomycosis and other mycoses
- Acute coronary syndrome
- Acute or chronic cerebrovascular insufficiency/accident (including thrombotic or embolic stroke)
- Acute renal arterial insufficiency
- Acute thermal and chemical pulmonary damage, i.e., smoke inhalation (e.g., carbon tetrachloride, hyp pulmonary insufficiency
- Aerobic septicemia and systemic aerobic infection
- Anaerobic septicemia and infection other than clostridial
- Anoxic brain injury
- Arthritic diseases
- Arthritis
- Aseptic necrosis of the femoral head and neck
- Autism
- Bell's palsy
- Bone grafts or fracture healing (e.g., nonunion fractures)
- Calciphylaxis (calcific uremic arteriolopathy)
- Cancer
- Cardiogenic shock
- Cerebral palsy
- Chronic peripheral vascular insufficiency
- Closed head and/or spinal cord injury
- Cognitive impairment (e.g., senility, senile dementia)
- Cystic acne
- Diabetic foot ulcers that are not infected
- Diabetic superficial wounds
- Facial neuritis
- Fibromyalgia
- Frostbite
- Glioblastoma
- Hepatic artery thrombosis
- Hepatic necrosis
- HIV infection
- Infective polyneuritis
- Inflammatory bowel disease (Crohn's disease and ulcerative colitis)
- Interstitial cystitis
- Intra-abdominal abscess, pseudomembranous colitis (antibiotic-induced colitis)
- Intracranial abscesses
- Ischemia due to lupus vasculitis
- Legg-Calve Perthes disease
- Lepromatous leprosy
- Lyme disease
- Lymphedema
- Melasma
- Meningitis
- Methicillin-resistant Staphylococcus aureus (MRSA) infections
- Migraine or cluster headaches
- Multiple sclerosis
- Myocardial infarction
- Myofascial pain syndrome
- Necrotizing arachnidism
- Non-compromised skin grafts and flaps
- Non-diabetic cutaneous, decubitus, pressure and venous stasis ulcers
- Non-vascular causes of chronic brain syndrome (e.g., Alzheimer's disease, Korsakoff's disease, Pick
- Ophthalmologic diseases (including central retinal artery occlusion, central retinal vein occlusion, dia
- glaucoma, keratoconjunctivitis, radiation injury to the optic nerve, retinal detachment)
- Organ transplantation and storage
- Osteonecrosis of the jaw
- Osteoporosis
- Otitis externa
- Parkinson's disease
- Post-organ transplantation re-vascularization
- Pulmonary emphysema
- Pyoderma gangrenosum
- Radiation-induced cholangitis, myelitis, enteritis, sarcoma
- Raynaud's syndrome
- Recto-vaginal fistula
- Reflex sympathetic dystrophy (complex regional pain syndrome)
- Seizure disorders
- Sickle cell crisis or hematuria
- Skin burns (thermal)
- Superficial and/or non-infected diabetic ulcers
- Surgical wound dehiscence
- Systemic inflammatory response syndrome
- Tetanus
- Tinnitus
- Traumatic brain injury
- Vesicocutaneous fistula
- Xerostomia/salivary gland dysfunction

III. Aetna considers systemic HBOT experimental and investigational for members with any of the following con
- HBOT, as the safety of systemic HBOT for persons with these contraindications to HBOT has not been esta
- Concurrent administration of doxorubicin, cisplatin, or disulfiram
- Premature infants (birth prior to 37 weeks gestation)
- Untreated pneumothorax

IV. Aetna considers topical HBOT directly administered to the open wound, and limb-specific hyperbaric oxygen
- limb-encasing devices experimental and investigational because its efficacy has not been established throu
- trials.
Background

Hyperbaric oxygen therapy (HBOT) is defined as systemic treatment in which the entire patient is placed inside a pressure chamber and breathes 100% oxygen under a pressure greater than 1 atmosphere (atm). It is used to treat certain diseases and improve when an increased partial pressure of oxygen is present in perfused tissues.

The literature states that HBOT should not be a replacement for other standard successful therapeutic measures. The response of the individual patient and the severity of the original problem, treatment may range from less than 1 week duration, the average being 2 to 4 weeks. Hyperbaric oxygen therapy for more than 2 months is usually not necessary.

Hyperbaric oxygen therapy has been shown to be an effective method for treating diabetic foot wounds in carefully selected extremity lesions. Although the results of multiple retrospective studies involving a significant number of patients have shown a high success rate in patients who had been refractory to other modes of therapy, several recent prospective, randomized controlled trials have supported the adjunctive role of systemic hyperbaric oxygen therapy in the treatment of non-healing infected deep wounds in patients with diabetes. Such evidence is lacking, however, for superficial diabetic wounds and non-diabetic cutaneous venous stasis ulcers.

A number of technology assessment organizations, including the Cochrane Collaboration, the Wessex Institute, the Foundation for Medical Research, and the Agency for Healthcare Research and Quality (AHRQ), have systematically reviewed the use of hyperbaric oxygen for each of the indications for which it has been used.

An evidence review conducted by the Alberta Heritage Foundation for Medical Research (Hailey, 2003) concluded that there is insufficient evidence to support the use of HBOT in brain injury. The assessment concluded that "The balance of benefits and harms of HBOT for brain injury, cerebral palsy, or stroke has not been adequately studied."

Denton et al (2004) systematically reviewed the evidence regarding HBOT for radiation cystitis. Of the 19 studies that were included, all the reports were case series and only 1 was a prospective series. The authors stated that "[t]he level of evidence is essentially IIIC (weak evidence), apart from one prospective case series of forty patients." The latter study was graded IIC (prospective study without calculation of sample size and without accurate and standard definition).

In a Cochrane review, Bennett et al (2005) concluded that for people with acute coronary syndrome, individual small studies add little information, but did not reduce mortality. They noted that in view of the modest number of patients, methodological shortcomings suggest that the results should be interpreted cautiously, and an appropriately powered trial of high methodological rigor is justified to determine whether HBOT can be expected to derive most benefit from HBOT. The routine application of HBOT to these patients cannot be justified.

A Cochrane review (Bennett et al, 2005) assessed the evidence of effectiveness of HBOT for long-term radiation injury to the anus and rectum. The investigators found HBOT significantly improved chance of healing for radiation proctitis (relative risk interval [CI]: 1.2 to 6.0). The investigators concluded that small trials suggest that HBOT is useful for treatment of radiation proctitis.

Absolute contraindications to HBOT include: untreated pneumothorax, concurrent administration of disulfiram (Antabuse), administration of the antineoplastic agents doxorubicin and cisplatinum; and administration to premature infants (d
fibroplasia). Relative contraindications to the use of HBOT include prior chest surgery, lung disease, viral infection surgery, optic neuritis, seizure disorders, high fever, congenital spherocytosis, and claustrophobia.

Topical HBOT administered to the open wound in small limb-encasing devices is not systemic HBOT and its efficacy established due to the lack of controlled clinical trials. In addition, in vitro evidence suggests that topical HBOT does not increase oxygen tension beyond the superficial dermis. Examples of topical HBOT devices are TOPOX portable hyperbaric sacral chambers (Jersey City, NJ), Oxyboot and Oxyhealer from GWR Medical, L.L.P. (Chadds Ford, PA).

The Undersea and Hyperbaric Medical Society issued the following policy statement on topical oxygen, often referred to as "hyperbaric oxygen therapy" (Feldmeier et al, 2005): "1. Topical oxygen should not be termed hyperbaric oxygen since the term "hyperbaric" intentionally or unintentionally suggests that topical oxygen treatment is equivalent or even identical to hyperbaric oxygen therapy. Mechanisms of action or clinical study results for hyperbaric oxygen can not and should not be co-opted to support hyperbaric oxygen therapy and topical oxygen have different routes and probably efficiencies of entry into the wound and biochemistry are necessarily different. 3. The application of topical oxygen cannot be recommended outside of a clinical setting based on the volume and quality of scientific supporting evidence available, nor does the Society recommend third-party reimbursement. 4. Before topical oxygen can be recommended as therapy for non-healing wounds, its application should be subjected to the same intense scientific scrutiny to which systemic hyperbaric oxygen has been held".

There is insufficient evidence of the effectiveness of hyperbaric oxygen as a treatment for autism. Rossignol (2007) reported on a neurodevelopmental disorder currently affecting as many as 1 out of 166 children in the United States. Numerous individuals have revealed evidence of cerebral hypoperfusion, neuro-inflammation and gastrointestinal inflammatory oxidative stress, relative mitochondrial dysfunction, neurotransmitter abnormalities, impaired detoxification of toxins, and production of porphyrins. Many of these findings have been correlated with core autistic symptoms. For example, autistic children have been correlated with repetitive, self-stimulatory and stereotypical behaviors, and impairments in perception, and social interaction. Hyperbaric oxygen therapy might be able to improve each of these problems in autistic children. Specifically, HBOT has been used with clinical success in several cerebral hypoperfusion conditions and can complicate blood flow by increasing the oxygen content of plasma and body tissues. Hyperbaric oxygen therapy has been reported to have anti-inflammatory properties and has been shown to improve immune function. There is evidence that oxidative stress can decrease oxygen tension at the wound site and improve neurotransmitter abnormalities. In addition, HBOT up-regulates enzymes that can help reduce oxidative stress, reduce mitochondrial dysfunction and improve neurotransmitter abnormalities. Dysbiosis is common in autistic children and HBOT can improve the gut microbiome. HBOT has been shown to mobilize stem cells from the bone marrow to the systemic circulation. Recent studies in human subjects have shown that HBOT can improve cognitive function and reduce inflammation. However, the application of HBOT for autism should, for now, be considered an experimental treatment modality. Consequently, this treatment should be limited to research projects.

An systematic evidence review of hyperbaric oxygen therapy for autism (Moqadem and Pineau, 2007) prepared for a technology assessment agency, concluded: "In light of its assessment, AETMIS concludes that there is insufficient evidence for the efficacy of hyperbaric oxygen therapy in the management of autistic disorders. In these circumstances, further studies are needed to evaluate the results of the current and future studies. In short, for the management of autism, hyperbaric oxygen therapy should, for now, be considered an experimental treatment modality. Consequently, this treatment should be limited to research projects."

Rossignol et al (2009) carried out a multi-center, randomized, double-blind, controlled study to evaluate the efficacy of HBOT with autism. A total of 62 children with autism recruited from 6 centers, aged 2 to 7 years (mean of 4.92 +/- 1.21) were randomized to receive 40 hourly treatments of either HBOT at 1.3 atm and 24 % oxygen ("treatment group", n = 33) or slightly pressurized 21 % oxygen ("control group", n = 29). Outcome measures included Clinical Global Impressions (CGI) scale, Aberra (ABC), and Autism Treatment Evaluation Checklist (ATEC). After 40 sessions, mean physician CGI scores significantly differed between treatment and control groups (p = 0.0008), receptive language (p < 0.0001), social in eye contact (p = 0.0102); 9/30 children (30 %) in the treatment group were rated as "very much improved" or "much improved".
2/26 (8 %) of controls (p = 0.0471); 24/30 (80 %) in the treatment group improved compared to 10/26 (38 %) of controls. Parental CGI scores significantly improved in the treatment group compared to controls in overall functioning (p = 0.0168), and eye contact (p = 0.0322). On the ABC, significant improvements were observed in the score, irritability, stereotypy, hyperactivity, and speech (p < 0.03 for each), but not in the control group. In the treatment group, mean changes on the ABC total score and subscales were similar except a greater number of children demonstrated improvements. On the ATEC, sensory/cognitive awareness significantly improved (p = 0.0367) in the treatment group. Post-hoc analysis indicated that children over age 5 and children with lower initial autism severity demonstrated significant improvements. Hyperbaric treatment was safe and well-tolerated. The authors reported that children with autism received hyperbaric oxygen for 40 hourly sessions had significant improvements in overall functioning, receptive language, and eye contact, and sensory/cognitive awareness compared to children who received slightly pressurized room air.

Rossignol et al (2009) concluded that "given the positive findings of this study, and the shortage of proven treatments for autism, parents who pursue hyperbaric treatment for their child with autism can be assured that it is a safe treatment used in this study (1.3 atm), and that it may improve certain autistic behaviors. Further studies are needed by other researchers to confirm these findings; we are aware of several other planned or ongoing studies of hyperbaric treatment in children with autism, but the positive results of this study and those of several previous studies, the use of hyperbaric treatment appears to be promising for children with autism".

The study by Rossignol et al (2009) had several major limitations. First, there were no significant differences between treatment and control groups for most of the primary outcomes. In the treatment group compared to the control group, mean changes in total ABC score and subscales for social withdrawal, social interaction, and eye contact; there were no significant differences between treatment and control groups in the expressive language, sleep pattern, attention span, self-stimulatory behaviors, social interaction, play skills, self-injurious behavior, mood, anxiety level, aggression, general health, gross motor skills, and mean physician CGI scores significantly improved in the treatment group compared to controls in overall functioning, receptive language, and eye contact; there were no significant differences between treatment and control groups in the expressive language, sleep pattern, attention span, self-stimulatory behaviors, social interaction, play skills, self-injurious behavior, mood, anxiety level, aggression, general health, gross motor skills. Moreover, while mean parental CGI scores significantly improved in the treatment group compared to controls in receptive language, and eye contact; there were no significant differences between the treatment group and controls only in the sensory/cognitive awareness subscale. There were significant differences between treatment and control groups in total score, and in the subscales for social withdrawal, social interaction, and eye contact; there were no significant differences between treatment and control groups in total score, and in the subscales for speech, socialization, and expressive language.

Another important issue that was not fully addressed was the adequacy of blinding. The study states that 6 adults (children and parents) and investigators themselves to ascertain if they are able to distinguish between treatment and control better than would be expected by chance, which was not done in this study. The important issue is whether or not the persons in the study were able to distinguish between treatment and control better than would be expected by chance, and significance are employed in this analysis.

The most critical issue that was not addressed in this study was the durability of results. These investigators measured initiation and immediately upon completion of 40 HBOT sessions. However, the treatment and control groups were substantial period of time after the study was completed to determine whether significant differences between treatment persisted. In other words, does HBOT result in durable benefits, or do any improvements dissipate after completion of treatment?

It should also be noted that autism is not approved as an indication for HBOT neither by the Undersea and Hyperbaric Medicine Committee (2009) nor by the European Committee for Hyperbaric Medicine (Yildiz et al, 2008). Furthermore, in a review on autism, Levy et al. (2009) concluded that popular biologically based treatments include anti-infectives, chelation medications, gastrointestinal medication immunoglobulins. Non-biologically based treatments include auditory integration therapy, chiropractic therapy, cranial osteopathy, and acupuncture. However, the evidence for the efficacy of these treatments is limited, and further research is needed to establish their effectiveness.
interactive metronome, and transcranial stimulation. However, few studies have addressed the safety and effective treatments.

Ghanizadeh (2012) stated that there is a controversy regarding the effectiveness of HBOT for the treatment of autism. Systematically reviewed the current evidences for treating of autism with HBOT. According to PRISMA guidelines from the databases of MEDLINE/PubMed, Google Scholar, and Randomized Controlled Trials in Hyperbaric Medicine were searched. In addition, medical subject heading terms and text words for hyperbaric oxygen therapy and autism were inclusion criteria were published studies that reported the original data from the trials conducted on the patients with outcomes with a valid and reliable instrument. A quality assessment was also conducted. The electronically searched publications. Two studies were randomized, double-blind, controlled-clinical trials. While some uncontrolled and suggested that HBOT is effective for the treatment of autism, these promising effects are not replicated. The authors controlled studies with rigorous methodology are needed to provide scientific evidence-based HBOT for autism treatments.

Although a recent article (Butler et al, 2008) included ischemic central retinal vein and artery occlusions among indications for HBOT. Folio et al (2007) described a case of frostbite to all fingers of a mountain climber, treated with HBOT. All fingers were function, with only some cosmetic deformity to the tip of the most severely affected finger. Because few cases of frostbite have been reported, these researchers hoped that such case reports will stimulate future research in this area. It is anecdotal cases may help guide future research in this area. Sequential digital photographs were taken at various pressures. They raised the possibility of photographic techniques and standards that may facilitate planning of therapy treatment comparisons, resulting in more consistency in the future. For example, a graphical software that allows morphing of sequent images to demonstrate healing progress in a concise movie format. The morphed demonstration of healing to the referring provider and patient and helps in teaching and research on frostbite treatment.

Kiralp et al (2009) evaluated the effects of HBOT on myofascial pain syndrome (MPS). A total of 30 patients with pain syndrome were divided into HBOT (n = 20) and control groups (n = 10). Patients in the HBOT group received a total of 10 HBOT treatments, and patients in the control group received placebo treatment in a hyperbaric chamber. Pain threshold and visual analog measurements were performed immediately before and after HBOT and 3 months thereafter. Additionally, Pain Disability Index (PDI), Mental and Physical Health Short Form 12 Health Survey (SF-12) evaluations were done before HBOT and after 3 months. Hyperbaric oxygen therapy was well tolerated with no complications. In the HBOT group, pain threshold significantly increased and VAS scores significantly improved after 3 months compared with pre-treatment values. In the control group, pain thresholds, VAS score, and Mental Health SF-12 scores improved after 3 months compared with placebo treatment; however, significant improvement was observed in the Physical Health SF-12. They concluded that HBOT may be a valuable alternative to other methods in the management of MPS. They stated that further randomized, double-blinded and placebo-controlled studies to evaluate the possible role of HBOT in the management of MPS are needed.

Urade (2009) stated that bisphosphonates (BPs) are effective in the treatment of hypercalcemia of malignancy, musculoskeletal events associated with metastatic breast cancer and prostate cancer, and osteoporosis. Despite these benefits, bisphosphonates-related osteonecrosis of the jaws (BRONJ) becomes a growing and significant problem in a subset of patients, especially those receiving intravenous preparations. Bisphosphonate-related osteonecrosis of the jaws has also been reported in patients receiving intravenous BP treatments, although the incidence is extremely low. Most of BRONJ cases occur after dental treatments such as tooth extraction, and dental implants, and are refractory to conventional treatment modalities such as debridement, antibiotics compared to EU and USA, the number of BRONJ cases is still small in Japan, but it is exactly increasing year by year. The number of BRONJ in patients receiving oral BPs to that in patients receiving intravenous BPs is higher in Japan than other countries due to the difference of time of approval. In this communication, the practical guidelines for prevention of BRONJ recently released from USA and Canada were introduced. Although no effective therapy for BRONJ has importance of oral hygiene, patient education and treatments suitable for clinical stage was emphasized.

Freiberger (2009) stated that BPs suppress bone turnover by disrupting osteoclast signal transduction, maturation, and migration. It has been hypothesized that suppressed turnover can impair oral wound healing, leading to BRONJ. However, as an adjunct to surgery and antibiotics, might have utility in the treatment of BRONJ because it produces reactive
species that positively modulate the redox-sensitive intracellular signaling molecules involved in bone turnover. The treatment of BRONJ is currently under investigation in randomized controlled trials (RCTs) at Duke University Minnesota, and the early results have been encouraging. This report discussed osteoclast biology, how HBOT has bone turnover by way of the signaling effects on osteoclasts, the available clinical data on HBOT in the treatment of RCTs of HBOT, and the study-associated efforts to find biomarkers to characterize an individual's risk of developing BRONJ.

Vescovi and Nammour (2010) stated that BRONJ is an area of uncovered bone in the maxillo-facial region that did after identification by health care provider, in a patient who was receiving or had been exposed to BP therapy (BPT) radiation therapy to the craniofacial region. Low-grade risk of ONJ is connected with oral BPT used in the treatment of multiple myeloma and bone metastases (from 0.8% to 12%). The management of BRONJ currently has yet been developed and interrupting BPT does not seem to be beneficial. Temporary suspension of benefit, while long-term discontinuation (if systemic conditions permit it) may be beneficial in stabilizing sites of ON symptoms. The use of oral anti-microbial rinses in combination with oral systemic antibiotic therapy -- penicillin, m clindamycin, doxycycline, erythromycin -- is indicated for stages I and II of Ruggiero's staging. The role of HBOT is benefits of this treatment have recently been described in association with discontinuation of BPT and conventional surgical).

In a Cochrane review, Eskes and colleagues (2010) examined the effects of HBOT as a treatment for acute wound surgery and trauma. Randomized controlled trials comparing HBOT with other interventions or comparisons between regimens were selected. Two review authors conducted selection of trials, risk of bias assessment, data extraction independently. Any disagreements were referred to a third review author. A total of 3 trials involving 219 subjects studies were clinically heterogeneous, therefore a meta-analysis was inappropriate. One trial (48 participants with split skin grafts) compared HBOT with usual care and reported a significantly higher complete graft survival association healthy graft area risk ratio [RR] 3.50; 95% CI: 1.35 to 9.11). A second trial (36 participants with crush injuries) rep wounds healed with HBOT than with sham HBOT (RR 1.70; 95% CI: 1.11 to 2.61) and fewer additional surgical procedures were reported. A third trial (48 participants undergoing flap grafting) reported no significant differences in complete graft survival with HBOT compared with debridement (RR 0.95 to 1.38) or heparin (RR 1.21; 95% CI: 0.99 to 1.49). Many of the pre-defined secondary outcomes, mortality, pain scores, quality of life, patient satisfaction, activities daily living, increase in transcutaneous oxygen p amputation, length of hospital stay and costs, were not reported. All 3 trials were at unclear or high risk of bias. There is a lack of high quality, valid research evidence regarding the effects of HBOT on wound healing. While some HBOT may improve the outcomes of skin grafting and trauma, these trials were at risk of bias. They stated that further high quality RCTs is needed.

The Canadian Agency for Drugs and Technologies in Health's review on the use of HBOT for difficult wound (Boud 7 health technology assessments, 5 systematic reviews, and 1 RCT. Overall, the authors of the identified studies f clinically effective as well as cost-effective when it was used to treat patients with diabetes who have lower extremity wounds. Some positive evidence to suggest that HBOT was clinically effective when it was used to treat radiation proctitis was considered insufficient to promote the routine use of HBOT for non-diabetic pressure ulcers, delayed radiation burns, as well as skin grafts and flaps. No evidence was identified on the use of HBOT in post-organ transplantatio authors concluded that overall, the best evidence on the use of adjunctive HBOT was associated with the treatment wounds. The evidence that supported its use, however, was not reliable. Although there were many recommenda HBOT as adjunctive treatment for specific indications, there is little evidence on its clinical and economic benefits.

Gallego et al (2011) evaluated the effectiveness of HBOT as a potential treatment for patients with hemorrhagic rad (RADC). This prospective study included 38 patients, 21 men and 17 women, mean age of 66.5 years (46 to 75), pelvic radiotherapy, with the diagnosis of RADC with or without radio-induced proctitis (RADP), gross hematuria an symptoms. Hyperbaric oxygen therapy was applied in a multi-place chamber; patients breathed pure oxygen (100 atmospheres of pressure (ATAs). Patients received an average of 31.2 sessions (10 to 48 sessions) and the medians months (4 to 72 months). Hematuria was completely resolved in 34 of the 38 patients. After HBOT, 6 patients req
anemic hematuria and 1 for acute obstructive pyelonephritis. In general, patients tolerated treatment well; however, barotrauma requiring myringotomy. The authors concluded that HBOT can be used to satisfactorily treat RADC, le improvements that begin during the initial sessions in the majority of cases, and with a more than acceptable level

Shao and colleagues (2012) compared the efficacy of intravesical hyaluronic acid (HA) instillation and HBOT in the induced hemorrhagic cystitis (HC). In total 36 patients who underwent radiotherapy for their pelvic malignancies an from HC were randomly divided into an HA group and an HBOT group. Symptoms of hematuria, frequency of void scale of pelvic pain (range of 0 to 10) were evaluated before and after the treatment with follow-up of 18 months. study and no obvious side effects of intravesical HA were recorded. The improvement rate showed no statistical d groups at 6, 12 and 18 months after treatment. Decrease of frequency was significant in both groups 6 months afte significant in the HA group 12 months after therapy. The improvement in the visual analog scale remained significant months. The authors concluded that intravesical instillation of HA was as effective in treating radiation-induced HC tolerated and resulted in a sustained decrease of bladder bleeding, pelvic pain and frequency of voiding for at least

Parra et al (2011) assessed the efficacy of HBOT in HC cases. A retrospective analysis of patients with HC after p receiving HBOT at the authors’ center between January 2002 and January 2010 was performed. Their protocol in HBOT in a multi-place hyperbaric chamber with 90 mins of 100 % oxygen breathing at 2.2 ATAs. Success was eva partial stop of bladder bleeding. Telephone follow-up was updated at the time of submission in all cases. A total o (21 males, 4 females); the mean age was 66.7 years. Twenty men were irradiated for prostate cancer and 1 for bla had cervix cancer and 1 endometrial cancer. In all cases previous conservative treatment had failed and HBOT w other measures failed. All the patients responded to HBOT and none recurred after end of treatment at a mean fol There were no serious complications. The authors concluded that HBOT is a highly effective and safe, non-invas secondary to pelvic radiation; it should be considered as first line alternative in these difficult cases.

Savva-Bordalo et al (2012) stated that late-onset HC after allogeneic hematopoietic stem cell transplantation (HSC with BK virus (BKV). Anti-viral drugs are of limited efficacy and the optimal treatment for HC has not yet been esta oxygen therapy may benefit these patients. These researchers retrospectively evaluated the effectiveness of HBO after allogeneic HSCT. All 16 patients had macroscopic hematuria and BKV infection. Patients received 100 % ox chamber at 2.1 ATAs for 90 mins, 5 days per week, with a median 13 treatments (range of 4 to 84). Fifteen patien resolution of hematuria. Median urinary DNA BKV titers declined after HBOT (p < 0.05). Patients started on HBOT HC responded sooner (p < 0.05). The authors concluded that HBOT was generally well-tolerated and proved to b difficult to manage condition.

Craighead et al (2011) reviewed the evidence regarding HBOT for late radiation tissue injury in gynecologic malgn Embase, Cochrane Library, National Guidelines Clearinghouse, and Canadian Medical Association Infobase datab June 2009 for clinical practice guidelines, systematic reviews, randomized controlled trials, or other relevant eviden evaluate soft tissue necrosis, cystitis, proctitis, bone necrosis, and other complications were excluded. Two randonized studies, and 5 supporting documents comprise the evidence base. In addition, information on the harm with HBOT were reported in 3 additional sources. There is modest direct evidence and emerging indirect evidence broadly effective for late radiation tissue injury of the pelvis in women treated for gynecologic malignancies. The au based on the evidence and expert consensus opinion, HBOT is likely effective for late radiation tissue injury of the efficacy specifically for radiation damage to the anus and rectum; the main indication for HBOT therapy in gynecol management of otherwise refractory chronic radiation injury; HBOT may provide symptomatic benefit in certain clin soft-tissue necrosis, and osteonecrosis); and HBOT may reduce the complications of gynecologic surgery in patien removal of necrosis.

Also, an UpToDate review on “Cystitis in patients with cancer” (Moy, 2011) states that “[h]yperbaric oxygen therapy but is limited to stable patients and those with access to a hyperbaric chamber”.

Matchett et al (2009) stated that numerous studies have demonstrated a protective effect of HBOT in experimental many physiological and molecular mechanisms of HBOT-related neuro-protection have been identified. These res pertaining to HBOT and cerebral ischemia in the National Library of Medicine and National Institutes of Health data
mechanisms of HBOT-related neuro-protection. Hyperbaric oxygen therapy has been shown to ameliorate brain in models including focal cerebral ischemia, global cerebral ischemia, neonatal hypoxia-ischemia and subarachnoid trials of HBOT in focal ischemia have not shown benefit, although 1 trial of HBOT before cardiopulmonary bypass d neuropsychological and inflammatory outcomes with hyperbaric oxygen therapy. Hyperbaric oxygen therapy is as cerebral oxygenation, reduced blood-brain barrier breakdown, decreased inflammation, reduced cerebral edema, d pressure, reduced oxidative burden, reduced metabolic derangement, decreased apoptotic cell death and increase The authors concluded that on a molecular level, HBOT leads to activation of ion channels, inhibition of hypoxia ind regulation of Bcl-2, inhibition of MMP-9, decreased cyclooxygenase-2 activity, decreased myeloperoxidase activity superoxide dismutase and inhibition of Nogo-A (an endogenous growth-inhibitory factor). Ongoing research will co mechanisms of HBOT-related neuro-protection, and possibly expand HBOT use clinically.

Michalski et al (2011) stated that high socioeconomic burden is attributed to acute ischemic stroke, but treatment s Normobaric oxygen therapy (NBOT) and HBOT were frequently investigated in pre-clinical studies following acute with predominantly beneficial effects in different outcome measurements. Best results were achieved in transient HBOT early after artery occlusion, and by using relatively high pressures. On molecular level, oxygen application l stabilization, reduction of excito-toxic metabolites, and inhibition of inflammatory processes. Therefore, NBOT and hopeful in salvaging impaired brain cells during ischemic stroke. However, harmful effects have been noted contrib properties, e.g., vasoconstriction and free oxygen radicals. In the clinical setting, NBOT provided positive results i HBOT failed to show efficacy in 3 randomized trials. To date, the translation of numerous evidentiary experimenta implementation remains open. Recently, oxygen became interesting as an additional therapy to neuro-protective o combine positive effects. The authors concluded that further preclinical research is needed exploring interactions and key factors with multi-phasic roles in acute damaging and delayed inflammatory processes after cerebral ische proteinase’s and hypoxia-inducible factor-1α.

Calciphylaxis, also referred to as calcific uremic arteriolopathy (CUA), is a syndrome associated with end-stage ren necrotic skin ulcers, often leading to a fatal outcome. Hyperbaric oxygen has been used to enhance wound healing treatment of calciphylaxis is unclear. Rogers and Coates (2008) stated that CUA is a rare but important cause of patients with chronic kidney disease. The prevalence of CUA is increasing in patients with renal failure, and the co recognized in non-uremic patients. There has been increasing understanding of the molecular basis of vascular c the important role of the uremic microenvironment in the factors implicated in the differentiation of vascular smooth osteoblasts. New options for treatment of hyperphosphatemia and secondary hyperparathyroidism in patients with have become available in the last few years and these have begun to be used in patients with CUA. These includ non-calcium/non-aluminum-containing phosphate binders and case reports of use of cinacalcet. Other treatments targeted directly at calcium/phosphate homeostasis include HBOT and the antioxidant cation chelator sodium thios concluded that clinicians managing patients with CUA should consider a combination approach of treating derange newer therapeutic agents and promoting wound healing with other older modalities such as HBOT and sodium thio stated that randomized controlled trials for treatments in CUA are still lacking.

In a randomized study, Gothard et al (2010) examined effect of HBOT on arm lymphedema following adjuvant radi cancer. A total of 58 patients with greater than or equal to 15 % increase in arm volume after supraclavicular +/- ax (axillary surgery in 52/58 patients) were randomized in a 2:1 ratio to HBOT (n = 38) or to best standard care (n = 2 breathed 100 % oxygen at 2.4 ATAs for 100 mins on 30 occasions over 6 weeks. Primary endpoint was ipsilateral a percentage of contralateral limb volume. Secondary endpoints included fractional removal rate of radioisotopic tr extracellular water content, patient self-assessments and UK SF-36 Health Survey Questionnaire. Of 53/58 (91.4 assessments, 46 had 12-month assessments (86.8 %). Median volume of ipsilateral limb (relative to contralateral) (IQR 126.0 to 152.3 %) in the control group, and 135.5 % (IQR 126.5 to 146.0 %) in the treatment group. Twelve m median (IQR) volume of the ipsilateral limb was 131.2 % (IQR 122.7 to 151.5 %) in the control group and 133.5 % the treatment group. Results for the secondary endpoints were similar between randomized groups. The authors evidence has been found of a beneficial effect of HBOT in the treatment of arm lymphedema following primary sur radiotherapy for early breast cancer.
Radiotherapy is generally used in the treatment of malignant tumors in the head and neck region. It causes a hypo-vascular environment that leads to injury to surrounding normal tissue, both acute and chronic, ranging from osteoradionecrosis. These side effects are debilitating and greatly influence quality of life in these patients. Hyperbaric oxygen therapy (HBOT) is clinically used to prevent or treat these side effects by enhancing oxygen pressure and thereby regeneration. Although its mechanism of action is still poorly understood, and controversy exists in the literature about its clinical utility, it is widely used. A systematic review on HBOT in the management of radiation-induced injury in the head and neck (2010) conducted a review on HBOT in the management of radiation-induced injury in the head and neck. A systematic review performed in PubMed for experimental and clinical studies conducted regarding the use of HBOT in previously irradiated tissue from January 1990 to June 2009. Experimental research is scarce, and clinical studies are especially lacking in this field, but the recent discussions on the subject are ongoing, most studies suggest a beneficial role for HBOT in previously irradiated tissues. Furthermore, a systematic review of salivary gland hypo-function and xerostomia induced by cancer therapies, on behalf of the Salivary Gland Hypo-function/Xerostomia Section; Oral Care Study Group; Multinational Association of Supportive Care in Cancer (MASCC)/International Society of Oral Oncology, assessed the literature for management strategies and salivary gland hypo-function and xerostomia induced by cancer therapies and to determine the quality of evidence-based recommendations. The electronic databases of MEDLINE/PubMed and EMBASE were searched for articles published 1989 NIH Development Consensus Conference on the Oral Complications of Cancer Therapies until 2008 inclusive. Independent reviewers extracted information regarding study design, study population, interventions, outcomes, and conclusions. A total of 72 interventional studies met the inclusion criteria. In addition, 49 intensity-modulated radiotherapy (IMRT) studies were included as a management strategy aiming for less salivary gland damage. Management guidelines were drawn up for IMRT, amifostine, muscarinic agonist stimulation, oral mucosal lubricants, acupuncture, and submandibular gland transfer, acupuncture, HBOT, management strategies in pediatric cancer populations, and the economic concept of gland hypo-function and xerostomia.

Also, UpToDate reviews on “Treatment of Sjögren’s syndrome” (Fox and Creamer, 2012) and “Hyperbaric oxygen therapy” (MeChem and Manaker, 2012) do not mention the use of HBOT for the treatment of xerostomia.

An UpToDate review on “Hyperbaric oxygen therapy” (MeChem and Manaker, 2012) does not mention the use of induced cholangitis.

The Cancer Care Ontario’s clinical practice guideline on “The management of head and neck cancer in Ontario” (Gough et al, 2012) mention the use of HBOT for radiation-induced sarcoma of the scalp. UpToDate reviews on “Treatment protocols for sarcoma” (Brenner et al, 2012) and “Local treatment for primary soft tissue sarcoma of the extremities and chest wall” do not mention the use of HBOT. Furthermore, the National Comprehensive Cancer Network’s clinical practice guidelines for sarcoma (Version 3.2012) does not mention “hyperbaric oxygen therapy”.

In a Cochrane review, Bennett et al (2012a) evaluated the effects of adjunctive HBOT for traumatic brain injury (TBI) and searched CENTRAL, MEDLINE, EMBASE, CINAHL and DORCTHIM electronic databases. They also searched the articles, hand-searched relevant journals and contacted researchers. All searches were updated to March 2012. Comparing the effect of therapeutic regimens that included HBOT with those that did not, for people with TBI were included in this review (285 receiving HBOT and 286 in the control group). The results of 2 studies indicated the use of HBOT resulted in a decrease in the proportion of people with an unfavorable outcome 1 month after treatment using the Glasgow Outcome Scale (GOS) score of 5; a ‘unfavorable’ outcome was considered as a score of 1, 2, or 3. Pooled data from final follow-up showed a significant reduction in the risk of dying when HBOT was used (RR 0.69, 95% CI: 0.54 to 0.88, p = 0.003) and suggested the
patients to avoid 1 extra death (number needed to treat (NNT) 7, 95 % CI: 4 to 22). Two trials suggested favorably pressure in people receiving HBOT and in whom myringotomies had been performed. The results from 1 study su (MD) with myringotomy of -8.2 mmHg (95 % CI: -14.7 to -1.7 mmHg, p = 0.01). The Glasgow Coma Scale (GCS) and 2 small trials reported a significant improvement in GCS for patients treated with HBOT (MD 2.68 points, 95 % 0.0001), although these 2 trials showed considerable heterogeneity (I(2) = 83 %). Two studies reported an incident pulmonary impairment in the HBOT group versus 0 % in the non-HBOT group (p = 0.007). In general, the studies significant risk of bias. None described adequate randomization procedures or allocation concealment, and none o staff was blinded to treatment. The authors concluded that in people with TBI, while the addition of HBOT may red improve the final GCS, there is little evidence that the survivors have a good outcome. The improvement of 2.68 p interpret. This scale runs from 3 (deeply comatose and unresponsive) to 15 (fully conscious), and the clinical impo of approximately 3 points will vary dramatically with the starting value (e.g., an improvement from 12 to 15 would re clinical benefit, but an improvement from 3 to 6 would leave the patient with severe and highly dependent impair that the routine application of HBOT to these patients cannot be justified from this review. Given the modest numb methodological shortcomings of included trials and poor reporting, the results should be interpreted cautiously. An trial of high methodological rigor is required to define which patients, if any, can be expected to benefit most from H

In a Cochrane review, Phillips and Jones (2013) evaluated the effectiveness of adjunctive HBOT for malignant otiti investigators searched the Cochrane Ear, Nose and Throat Disorders Group Trials Register; the Cochrane Central Trials (CENTRAL); PubMed; EMBASE; CINAHL; Web of Science; ICTRP and additional sources for published and date of the most recent search was April 4, 2013. Randomized controlled trials, involving adults, undergoing hype malignant otitis externa were selected for analysis. No identified articles described RCTs of HBOT in the treatmen externa. The authors concluded that no clear evidence exists to demonstrate the effectiveness of HBOT when com antibiotics and/or surgery. They found no data to compare rates of complication between the different treatment m is required.

Margolis et al (2013) compared the effectiveness of HBOT with other conventional therapies administered in a wou treatment of a diabetic foot ulcer and prevention of lower-extremity amputation. This was a longitudinal observatio address treatment selection bias, these investigators used propensity scores to determine the "propensity" that an receive HBOT. They studied 6,259 individuals with diabetes, adequate lower limb arterial perfusion, and foot ulcer dermis, representing 767,060 person-days of wound care. In the propensity score-adjusted models, individuals rec likely to have healing of their foot ulcer (hazard ratio 0.68 [95 % CI: 0.63 to 0.73]) and more likely to have an ampu Additional analyses, including the use of an instrumental variable, were conducted to assess the robustness of ths confounding. Hyperbaric oxygen therapy was not found to improve the likelihood that a wound might heal or to de amputation in any of these analyses. The authors concluded that the use of HBOT neither improved the likelihood nor prevented amputation in a cohort of patients defined by Centers for Medicare and Medicaid Services eligibility c the usefulness of HBOT in the treatment of diabetic foot ulcers needs to be re-evaluated.

Limb-specific HBOT entails sealing an individual's arm or leg into an air-tight plastic container that is sealed with pl exposing the limb to pure oxygen greater than 1 atm of pressure. Much of the research on this form of therapy ha wounds arising in individuals with diabetic foot ulcers. However, there is currently insufficient evidence from RCTs effectiveness of limb-specific HBOT.

In a prospective and controlled study, Lisagors et al (2008) evaluated the feasibility of HBOT as an efficient and sa standardized treatment protocol and its possible immunomodulatory impact of 44 patients with diagnosed acute pa course of the disease was accompanied by systemic inflammatory response syndrome in all the patients on admis and HBOT on homeostasis, the number of performed operations, mortality rates, the levels of 2 cytokines, intra-ab effects caused by HBOT were evaluated. A treatment group consisted of 22 patients receiving HBOT for 3 days (tw place chamber under pressures of 1.7 to 1.9 ATA. Patients (n = 22) in the control group were managed in accorda treatment protocol. The authors found more stable homeostasis, decreased mortality rate, and the number of oper This type of additional therapy, possibly contributed to the decrease of intra-abdominal pressure within the first 6 da
authors concluded that these findings suggested HBOT can affect an inflammatory response, by decreasing the lev
cytokines and increasing those of anti-inflammatory ones.

An UpToDate review on "Hyperbaric oxygen therapy" (Mechem and Manaker, 2014) states that "A number of pote
poorly validated and require more rigorous evaluation. Future indications for HBO may be derived from its apparent
reperfusion injury and inflammation. Preliminary animal and human studies evaluating uses in syndromes as dispa
infection, the systemic inflammatory response syndrome, traumatic brain or spinal cord injury, sickle cell crisis, fibr
stroke have been conducted, with variable results. Further investigation will need to be conducted before HBO can potential indications".

In a phase II clinical trial, Ogawa al (2012) analyzed the long-term results of radiotherapy given immediately after H
chemotherapy in adults with high-grade gliomas. Patients with histologically confirmed high-grade gliomas were ad
daily 2 Gy fractions for 5 consecutive days per week up to a total dose of 60 Gy. Each fraction was administered im
with the time interval from completion of decompression to start of irradiation being less than 15 minutes. Chemot
procarbazine, nimustine, and vincristine and was administered during and after radiotherapy. A total of 57 patients
glioblastoma and 18 patients with Grade 3 gliomas) were enrolled from 2000 to 2006, and the median follow-up of
62.0 months (range of 43.2 to 119.1 months). All 57 patients were able to complete a total radiotherapy dose of 60
HBOT with 1 course of concurrent chemotherapy. The median overall survival times in all 57 patients, 39 patients
patients with Grade 3 gliomas, were 20.2 months, 17.2 months, and 113.4 months, respectively. On multi-variate a
alone was a significant prognostic factor for overall survival (p < 0.001). During treatments, no patients had neutro
hemorrhage, and no serious non-hematologic or late toxicities were seen in any of the 57 patients. The authors co
delivered immediately after HBOT with multi-agent chemotherapy was safe, with virtually no late toxicities, and see
patients with high-grade gliomas. Moreover, they stated that this treatment strategy seemed promising and merited

Furthermore, the National Comprehensive Cancer Network’s clinical practice guideline on “Central nervous system
does not mention the use of HBOT as a therapeutic option.

Dulai et al (2014) stated that although there is experience using HBOT in Crohn's disease and ulcerative colitis, th
effectiveness of HBOT in inflammatory bowel disease (IBD) is unknown. These researchers quantified the safety a
for Crohn's disease (CD) and ulcerative colitis (UC). The rate of adverse events with HBOT for IBD was compared
adverse events with HBOT. MEDLINE, EMBASE, Cochrane Collaboration and Web of Knowledge were systemat
PRISMA standards for systematic reviews. A total of 17 studies involving 613 patients (286 CD, 327 UC) were inc
response rate was 86 % (85 % CD, 88 % UC). The overall response rate for perineal CD was 88 % (18/40 compe
healing). Of the 40 UC patients with endoscopic follow-up reported, the overall response rate to HBOT was 100 %
treatments, there were a total of 9 adverse events, 6 of which were serious. The rate of adverse events with HBOT
seen when utilizing HBOT for other indications (p < 0.01). The risk of bias across studies was high. The authors c
relatively safe and potentially effective treatment option for IBD patients. Moreover, they stated that to understand in
IBD, well-controlled, blinded, randomized trials are needed for both CD and UC.

The Infectious Diseases Society of America’s clinical practice guideline on “The diagnosis and treatment of diabeti
al, 2012) stated that “For specifically treating DFO [diabetic foot osteomyelitis], the developers do not currently sup
treatments such as hyperbaric oxygen therapy .... Consider providing empiric therapy directed against methicillin-r
aureus (MRSA) in a patient with a prior history of MRSA infection; when the local prevalence of MRSA colonization
the infection is clinically severe”.

Also, an UpToDate review on “Treatment of invasive methicillin-resistant Staphylococcus aureus infections in adult
mention the use of HBOT as a therapeutic option.

The European Society of Clinical Microbiology and Infectious Diseases Fungal Infection Study Group and the Euro
Medical Mycology’s joint clinical guidelines on “The diagnosis and management of mucormycosis” (Cornely et al, 2
“Hyperbaric oxygen is supported with marginal strength only”. Furthermore, an UpToDate review on “Hyperbaric o
and Manaker, 2014) states that “HBO has been advocated for use in other severe invasive infections such as cuta
CPT Codes / HCPCS Codes / ICD-9 Codes

CPT codes covered if selection criteria are met:

99183  Physician attendance and supervision of hyperbaric oxygen therapy, per session

HCPCS codes covered if selection criteria are met:

C1300  Hyperbaric oxygen under pressure, full body chamber, per 30 minute interval

HCPCS codes not covered for indications listed in the CPB:

A4575  Topical hyperbaric oxygen chamber, disposable

E0446  Topical oxygen delivery system, not otherwise specified, includes all supplies and ac

Other HCPCS codes related to the CPB:

J9000  Injection, doxorubicin HCl, 10 mg

J9060  Injection, cisplatin, powder or solution, 10 mg

Q2050  Injection, doxorubicin hydrochloride, liposomal, not otherwise specified, 10 mg

ICD-9 codes covered if selection criteria are met:

038.3  Septicemia due to anaerobes [progressive necrotizing soft tissue anaerobic infection

040.0  Gas gangrene [Clostridial myositis and myonecrosis]

250.70 - 250.71  Diabetes with peripheral circulatory disorders [non-healing infected deep ulcerations bone] of the lower extremity unresponsive to at least 1 month of meticulous wound c debridement, maximal antibiotic therapy, tight glycemic control, and appropriate trea insufficiency, including revascularization if necessary]

250.80 - 250.81  Diabetes with other specified manifestations [non-healing infected deep ulcerations ( bone) of the lower extremity unresponsive to at least 1 month of meticulous wound c debridement, maximal antibiotic therapy, tight glycemic control, and appropriate trea insufficiency, including revascularization if necessary] [not covered for diabetic super

280.0  Iron deficiency anemia secondary to blood loss (chronic) [overwhelming and transfus because there is no suitable blood available or religion does not permit]

285.1  Acute posthemorrhagic anemia [overwhelming and transfusion is impossible because blood available or religion does not permit]
348.5  Cerebral edema [acute]
388.10 - 388.12  Noise effects on inner ear [noise-induced hearing loss when HBOT is initiated within
388.2  Sudden hearing loss, unspecified [idiopathic when HBOT is initiated within 3 months
440.20 - 440.9  Atherosclerosis of native arteries and bypass graft of extremities [non-healing infecte
                      (reaching tendons or bone) of the lower extremity unresponsive to at least 1 month o
                      including aggressive debridement, maximal antibiotic therapy, tight glycemic control,
                      treatment of arterial insufficiency, including revascularization if necessary]
442.0 - 442.3  Other aneurysm of extremities
443.0 - 443.1  Other peripheral vascular disease [acute peripheral arterial insufficiency]
443.81 - 443.9  Other specified peripheral vascular diseases [acute peripheral arterial insufficiency]
444.21 - 444.22  Arterial embolism of the extremities [acute peripheral arterial insufficiency]
444.81  Arterial embolism and thrombosis of the iliac artery [acute peripheral arterial insufficie
454.0  Varicose veins of lower extremities with ulcer [non-healing infected deep ulcerations
                      bone) of the lower extremity unresponsive to at least 1 month of meticulous wound c
                      debridement, maximal antibiotic therapy, tight glycemic control, and appropriate trea
                      insufficiency, including revascularization if necessary]
454.2  Varicose veins of lower extremities with ulcer and inflammation [non-healing infected
                      (reaching tendons or bone) of the lower extremity unresponsive to at least 1 month o
                      including aggressive debridement, maximal antibiotic therapy, tight glycemic control,
                      treatment of arterial insufficiency, including revascularization if necessary]
459.81  Venous (peripheral) insufficiency, unspecified [venous stasis ulcer - non-healing infec
                      (reaching tendons or bone) of the lower extremity unresponsive to at least 1 month o
                      including aggressive debridement, maximal antibiotic therapy, tight glycemic control,
                      treatment of arterial insufficiency, including revascularization if necessary]
526.4  Inflammatory conditions of the jaws [radiation necrosis of jaw]
526.89  Other specified diseases of jaw [prophylactic pre- and post-treatment for members un
                      a radiated jaw]
595.82  Irradiation cystitis
673.00 - 673.04  Obstetrical air embolism
728.86  Necrotizing fasciitis
730.10 - 730.19  Chronic osteomyelitis [unresponsive to conventional medical and surgical managem
733.45  Aseptic necrosis of bone, jaw
733.49  Other aseptic necrosis of bone [osteoradionecrosis]
885.0 - 887.7  Traumatic amputation thumb, finger(s), arm and hand [when loss of function or life is
                      used in combination with standard therapy]
<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>895.0 - 897.7</td>
<td>Traumatic amputation toe(s), foot, leg(s) [when loss of function or life is threatened a combination with standard therapy]</td>
</tr>
<tr>
<td>902.53</td>
<td>Injury to the iliac artery [acute peripheral ischemia when loss of function, limb, or life is used in combination with standard therapy]</td>
</tr>
<tr>
<td>903.00 - 903.01</td>
<td>Injury to axillary blood vessels [acute peripheral ischemia when loss of function, limb HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>903.4</td>
<td>Injury to palmar artery [acute peripheral ischemia when loss of function, limb, or life is used in combination with standard therapy]</td>
</tr>
<tr>
<td>903.8</td>
<td>Injury to other specified blood vessels of upper extremity [acute peripheral ischemia when limb, or life is threatened and HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.0</td>
<td>Injury to common femoral artery [acute peripheral ischemia when loss of function, limb and HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.1</td>
<td>Injury to superficial femoral artery [acute peripheral ischemia when loss of function, limb, or life is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.41</td>
<td>Injury to popliteal artery [acute peripheral ischemia when loss of function, limb, or life is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.51</td>
<td>Injury to anterior tibial artery [acute peripheral ischemia when loss of function, limb, or HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.53</td>
<td>Injury to posterior tibial artery [acute peripheral ischemia when loss of function, limb, HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>904.7</td>
<td>Injury to other specified blood vessels of lower extremity [acute peripheral ischemia when limb, or life is threatened and HBOT is used in combination with standard therapy]</td>
</tr>
<tr>
<td>925.1 - 929.9</td>
<td>Crush injuries [when loss of function, limb, or life is threatened and HBOT is used in standard therapy]</td>
</tr>
<tr>
<td>951.5</td>
<td>Injury to acoustic nerve [acoustic trauma when HBOT is initiated within 3 months after]</td>
</tr>
<tr>
<td>958.0</td>
<td>Air embolism [acute]</td>
</tr>
<tr>
<td>958.90 - 959.99</td>
<td>Compartment syndrome</td>
</tr>
<tr>
<td>986</td>
<td>Toxic effect of carbon monoxide [acute]</td>
</tr>
<tr>
<td>987.7</td>
<td>Toxic effect of hydrocyanic acid gas [with co-existing carbon monoxide poisoning]</td>
</tr>
<tr>
<td>989.0</td>
<td>Toxic effect of hydrocyanic acid and cyanides [with co-existing carbon monoxide poisoning]</td>
</tr>
<tr>
<td>990</td>
<td>Effects of radiation, unspecified [radiation necrosis (osteoradionecrosis, myoradionecrosis, and other soft tissue radiation necrosis) or proctitis] [not covered for radiation-induced cholangitis, myelitis, enteritis, or optic nerve injury] [not covered for radiation-induced]</td>
</tr>
<tr>
<td>993.3</td>
<td>Caisson disease [decompression illness]</td>
</tr>
<tr>
<td>ICD-9 Code</td>
<td>Description</td>
</tr>
<tr>
<td>------------</td>
<td>-------------</td>
</tr>
<tr>
<td>996.52</td>
<td>Mechanical complications due to graft of other tissue, not elsewhere classified [compr flaps]</td>
</tr>
<tr>
<td>996.55</td>
<td>Mechanical complications due to artificial skin graft and decellularized allografts [co and flaps]</td>
</tr>
<tr>
<td>996.69</td>
<td>Infection and inflammatory reaction due to other internal prosthetic device, implant, a skin grafts and flaps</td>
</tr>
<tr>
<td>996.79</td>
<td>Other complications due to other internal prosthetic device, implant, and graft [compr flaps]</td>
</tr>
<tr>
<td>998.59</td>
<td>Other postoperative infection [non-healing infected deep ulcerations (reaching tendon extremity unresponsive to at least 1 month of meticulous wound care, including aggr maximal antibiotic therapy, tight glycemic control, and appropriate treatment of arteri revascularization if necessary)]</td>
</tr>
<tr>
<td>998.83</td>
<td>Non-healing surgical wound [non-healing infected deep ulcerations (reaching tendon extremity unresponsive to at least 1 month of meticulous wound care, including aggr maximal antibiotic therapy, tight glycemic control, and appropriate treatment of arteri revascularization if necessary)]</td>
</tr>
</tbody>
</table>

ICD-9 codes not covered for indications listed in the CPB (not all-inclusive):

<table>
<thead>
<tr>
<th>ICD-9 Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>003.21</td>
<td>Salmonella meningitis</td>
</tr>
<tr>
<td>008.45</td>
<td>Clostridium difficile [intra-abdominal abscess, pseudomembranous colitis (antibiotic-i</td>
</tr>
<tr>
<td>013.0</td>
<td>Tuberculous meningitis</td>
</tr>
<tr>
<td>030.0</td>
<td>Lepromatous (type L) [leprosy]</td>
</tr>
<tr>
<td>036.0</td>
<td>Meningococcal meningitis</td>
</tr>
<tr>
<td>037</td>
<td>Tetanus</td>
</tr>
<tr>
<td>038.0 - 038.2, 038.4 - 038.9</td>
<td>Septicemia [except anaerobic infection]</td>
</tr>
<tr>
<td>039.0 - 039.9</td>
<td>Actinomycotic infections</td>
</tr>
<tr>
<td>042</td>
<td>Human immunodeficiency virus [HIV] disease</td>
</tr>
<tr>
<td>088.81</td>
<td>Lyme disease</td>
</tr>
<tr>
<td>090.42</td>
<td>Congenital syphilitic meningitis</td>
</tr>
<tr>
<td>091.81</td>
<td>Acute syphilitic meningitis (secondary)</td>
</tr>
<tr>
<td>094.2</td>
<td>Syphilitic meningitis</td>
</tr>
<tr>
<td>098.82</td>
<td>Gonococcal meningitis</td>
</tr>
<tr>
<td>100.81</td>
<td>Leptospiral meningitis (aseptic)</td>
</tr>
</tbody>
</table>
110.0 - 118 Mycoses
140.0 - 208.92 Malignant neoplasm [cancer]
230.0 - 234.9 Carcinoma in situ [cancer]
275.49 Other disorders of calcium metabolism [calciphylaxis (calcific uremic arteriolopathy)]
282.62 Hb-SS disease with mention of crisis [sickle cell crisis]
290.0 - 290.9 Dementias [cognitive impairment]
294.8 Other persistent mental disorders due to conditions classified elsewhere [dementia N impairment]
299.00 - 299.01 Autistic disorder
310.1 Personality change due to conditions classified elsewhere [cognitive impairment]
310.8 Other specified nonpsychotic mental disorders following organic brain damage [cogn
320.0 - 322.9 Meningitis- bacterial, due to other organisms, and of unspecified cause
324.0 Intracranial abscess
331.0 - 331.9 Other cerebral degenerations [cognitive impairment]
332.0 Paralysis agitans
332.1 Secondary Parkinsonism (parkinsonism due to drugs)
337.20 - 337.29 Reflex sympathetic dystrophy [complex regional pain syndrome]
339.00 - 339.02 Cluster headaches
340 Multiple sclerosis
341.20 - 341.9 Acute (transverse) myelitis [radiation induced]
343.0 - 343.9 Infantile cerebral palsy
345.00 - 345.91 Epilepsy and recurrent seizures
346.00 - 346.93 Migraine
348.1 Anoxic brain damage
351.0 Bell's palsy
351.8 Other facial nerve disorders [facial neuritis]
357.0 Acute infective polyneuritis
360.00 - 379.99 Disorders of the eye and adnexa [ophthalmologic diseases]
380.10 Infective otitis externa, unspecified
388.30 - 388.32  Tinnitus
410.00 - 412  Myocardial infarction
433.00 - 434.91  Occlusion and stenosis of precerebral and cerebral arteries [acute or chronic cerebro insufficiency/accident including thrombotic or embolic stroke]
435.0 - 435.9  Transient cerebral ischemia [acute or chronic cerebrovascular insufficiency]
436  Acute, but ill-defined, cerebrovascular disease [acute or chronic cerebrovascular ins including thrombotic or embolic stroke]
437.0 - 437.9  Other and ill-defined, cerebrovascular disease [acute or chronic cerebrovascular insu including thrombotic or embolic stroke]
438.0  Late effects of cerebrovascular disease, cognitive deficits
444.89  Arterial embolism and thrombosis of other specified artery [hepatic]
447.6  Arteritis, unspecified [Lupus vasculitis]
457.0 - 457.1  Lymphedema
491.20 - 491.22  Obstructive chronic bronchitis [bronchitis with emphysema]
492.0 - 492.8  Emphysema
506.0 - 506.9  Respiratory conditions due to chemical fumes and vapors [Acute thermal and chem i.e., smoke inhalation (e.g., carbon tetrachloride, hydrogen sulfide) with pulmonary in
508.0 - 508.9  Respiratory conditions due to other and unspecified external agents [Acute thermal a damage, i.e., smoke inhalation (e.g., carbon tetrachloride, hydrogen sulfide) with pulm
527.7 - 527.9  Xerostomia and salivary gland dysfunction
555.0 - 555.9  Crohn's disease
558.1  Gastroenteritis and colitis due to radiation
567.22  Peritoneal abscess [intra-abdominal]
570  Acute and subacute necrosis of liver [hepatic]
576.1  Cholangitis [radioation-induced hemorrhagic]
595.1  Chronic interstitial cystitis
595.82  Irradiation cystitis
599.70 - 599.72  Hematuria
619.1  Digestive-genital tract fistula, female [rectovaginal fistula]
674.10 - 674.14  Disruption of cesarean wound
686.0 - 686.9 Other local infections of skin and subcutaneous tissues [except Meleney's ulcer] [infec
tive clostridial]

692.70 - 692.79 Contact dermatitis and other eczema due to solar radiation [actinic skin damage]

706.1 Other acne [cystic]

709.09 Other disorders of skin and subcutaneous tissues [melasma]

710.0 Systemic lupus erythematosus [ischemia due to lupus vasculitis]

711.00 - 716.99 Arthropathies

729.1 Myalgia and myositis, unspecified [myofascial pain syndrome]

732.1 Juvenile osteochondrosis of head of femur [Legg-Calve-Perthes disease]

733.00 - 733.09 Osteoporosis

733.10 - 733.19 Pathologic fracture [fracture healing]

733.42 Aseptic necrosis of head and neck of femur

733.45 Aseptic necrosis of bone, jaw

733.81 - 733.82 Malunion and nonunion of fracture

743.20 - 743.22 Buphthalmos [ophthalmologic diseases]

757.0 Hereditary edema of legs

770.2 Interstitial emphysema and related conditions

780.31 - 780.39 Convulsions

780.97 Altered mental status [cognitive impairment]

781.8 Neurologic neglect syndrome [cognitive impairment]

785.51 Cardiogenic shock

797 Senility without mention of psychosis [cognitive impairment]

800.00 - 829.1 Fractures [fracture healing (e.g., nonunion fractures)]

850.0 - 854.19 Intracranial injury, excluding those with skull fracture [cognitive impairment] [not cove
injury]

907.0 Late effect of intracranial injury without mention of skull fracture [cognitive impairmen

941.00 - 946.5 Burns of face, head, neck, trunk, upper limb, wrist and hand, lower limb, and multiple
thermal]

950.0 - 950.9 Injury to optic nerve and pathways [ophthalmologic diseases (including diabetic retino
detachment, central retinal artery occlusion, radiation injury to the optic nerve, glau
keroendotheliosis)]
952.00 - 952.9  Spinal cord injury without evidence of spinal bone injury
959.01  Head injury, unspecified [cognitive impairment] [closed head injury]
987.0 - 987.6  Toxic effects of other gases, fumes, or vapors [other than carbon monoxide] [Acute th pulmonary damage, i.e., smoke inhalation (e.g., carbon tetrachloride, hydrogen sulfid insufficiency]
989.5  Toxic effect of venom [necrotizing arachnidism ]
991.0 - 991.3  Frostbite [face, hand, foot, and other and unspecified sites]
996.40 - 996.49  Mechanical complication of internal orthopedic device, implant, and graft [bone grafts
996.67  Infection and inflammatory reaction due to other internal orthopedic device, implant o
998.30 - 998.33  Disruption of wound [dehiscence of operation wound]
V42.0 - V43.89  Organ or tissue replacement by transplant or other means [organ transplant or storag
V49.83  Awaiting organ transplant status [organ transplant or storage]
V54.10 - V54.29  Aftercare for healing fracture [fracture healing (e.g., nonunion fractures)]
V58.44  Aftercare following organ transplant [post organ transplant revascularization]

Other ICD-9 codes related to the CPB:
569.89  Other specified disorders of intestine [pneumatosis cystoides intestinalis]
707.00 - 707.9  Chronic ulcer of skin [see criteria for coverage in diabetic adults only]
E863.4  Accidental poisoning by other and unspecified insecticides [cyanide]
E863.8  Accidental poisoning by fumigants [cyanide]
E868.0 - E868.9  Accidental poisoning by other utility gas and other carbon monoxide

ICD-9 codes contraindicated for this CPB:
045.00 - 079.99  Viral infections and diseases
282.0  Hereditary spherocytosis [congenital]
460 - 519.9  Disease of the respiratory system [lung disease including 512.0 - 512.8 untreated pn
765.00 - 765.28  Disorders relating to short gestation and low birthweight [premature infants (birth prio
780.60  Fever [high]

The above policy is based on the following references:


54. Rosenbaum P. Controversial treatment of spasticity: Exploring alternative therapies for motor function in chi
Child Neurol. 2003;18 Suppl 1:S89-S94.
55. Patterson J. Hyperbaric oxygen therapy for central osteoradionecrosis. STEER: Succinct and Timely Evaluat
Bazian Ltd., eds. London, UK: Wessex Institute for Health Research and Development, University of Southa
56. Patterson J. Hyperbaric oxygen therapy for central retinal artery occlusion. STEER: Succinct and Timely Eva
Bazian Ltd., eds. London, UK: Wessex Institute for Health Research and Development, University of Southa
57. Dent THS. Hyperbaric oxygen therapy for carbon monoxide poisoning. STEER: Succinct and Timely Evaluat
Bazian Ltd., eds. London, UK: Wessex Institute for Health Research and Development, University of Southa
58. Ball CM. Hyperbaric oxygen therapy for multiple sclerosis. STEER: Succinct and Timely Evaluated Evidence
London, UK: Wessex Institute for Health Research and Development, University of Southampton; 2002;2(6
59. Bisset F. Hyperbaric oxygen therapy in people with necrotising fasciitis or Fournier's gangrene. STEER: Suc
60. Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. Cochr
61. Villanueva E, Bennett MH, Wasiak J, Lehm JP. Hyperbaric oxygen therapy for thermal burns. Cochrane Dat
62. Dent THS. Hyperbaric oxygen therapy for carbon monoxide poisoning. STEER: Succinct and Timely Evaluat
Bazian Ltd., eds. London, UK: Wessex Institute for Health Research and Development, University of Southa
64. van Ophoven A, Rossbach G, Oberpenning F, Hertle L. Hyperbaric oxygen for the treatment of interstitial cy
65. Bennett MH, Kertesz T, Yeung P. Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and t
67. Bennett M, Jepson N, Lehm P. Hyperbaric oxygen therapy for acute coronary syndrome. Cochrane Databa
69. Bennett M, Best TM, Babul S, Taunton J. Hyperbaric oxygen therapy for delayed onset muscle soreness an
72. Bennett MH, Trytko B, Jonker B. Hyperbaric oxygen therapy for the adjunctive treatment of traumatic brain i
77. Schwarz S, Leweling H, Meinck HM. Alternative and complementary therapies in multiple sclerosis. Fortsch
78. Taylor RS, Simpson IN. Review of treatment options for Lyme borreliosis. J Chemother. 2005;17 Suppl 2:3-
79. Carson S, McDonagh M, Russman B, Helfand M. Hyperbaric oxygen therapy for stroke: A systematic revie
Hyperbaric Oxygen Therapy (HBOT)

89. Helms AK, Whelan HT, Torbey MT. Hyperbaric oxygen therapy of cerebral ischemia. Cerebrovasc Dis. 200
103. Folio LR, Arkin K, Butler WP. Frostbite in a mountain climber treated with hyperbaric oxygen: Case report. 563.
130. Brenner T, duggal S, Natale J, Wirth SM. Treatment protocols for soft tissue and bone sarcoma. Last review UpToDate Inc., Waltham, MA.
131. Delaney TF, Harmon DC, Gebhardt MC. Local treatment for primary soft tissue sarcoma of the extremities reviewed December 2012. UpToDate Inc., Waltham, MA.
Washington, PA.
guideline: Sudden hearing loss. Otolaryngol Head Neck Surg 2012;146(3 Suppl):S1-S35. Available at:
episodic migraine prevention in adults: Report of the Quality Standards Subcommittee of the American Aca
135. Holland NJ, Bernstein JM, Hamilton JW. Hyperbaric oxygen therapy for Bell's palsy. Cochrane Database Sy
2012b;2:CD007288.
139. Bennett MH, Stanford RE, Turner R. Hyperbaric oxygen therapy for promoting fracture healing and treating
141. Phillips JS, Jones SE. Hyperbaric oxygen as an adjuvant treatment for malignant otitis externa. Cochrane D
2013;5:CD004617.
143. University of Michigan School of Medicine Clinical Cases Summary: Vesicocutaneous fistula. University of
2000. Available at:
http://www.med.umich.edu/lrc/coursepa ges/m1/anatomy2010/html/clinicalcases/vesicocutaneous_fistula/ve
(procarbazine, nimustine, and vincristine) for high-grade gliomas: Long-term results. Int J Radiat Oncol Biol
146. Lipsky BA, Berendt AR, Cornia PB, et al. 2012 Infectious Diseases Society of America clinical practice guide
Washington, PA.
150. Cornely OA, Arikan-Akdagli S, Dannaoui E, et al; European Society of Clinical Microbiology and Infectious
Study Group; European Confederation of Medical Mycology. ESCMID and ECMM joint clinical guidelines fo
151. Wigley FM. Treatment of the Raynaud phenomenon resistant to initial therapy. UpToDate Inc., Waltham, M
November 2014.
152. Mechem CC, Manaker S. Hyperbaric oxygen therapy. UpToDate Inc., Waltham, MA. Last reviewed Novem