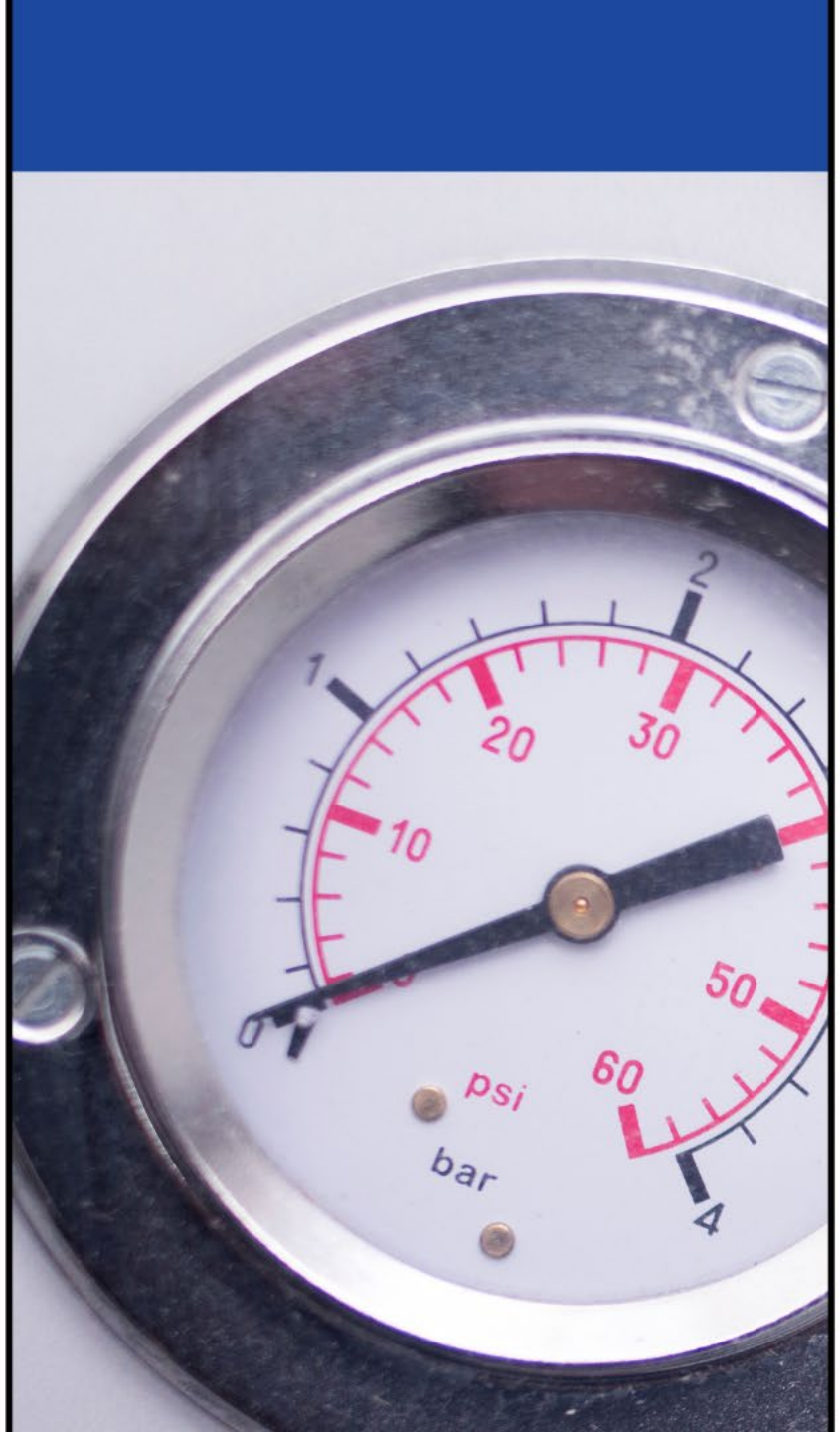
OCTOBER 2022 MONTHLY HBOT WEBINAR

Physiology of HBOT for Each Indication and When to Consider Continuation

PRESENTED BY THE INSPIRA HEALTH - ELMER PROGRAM



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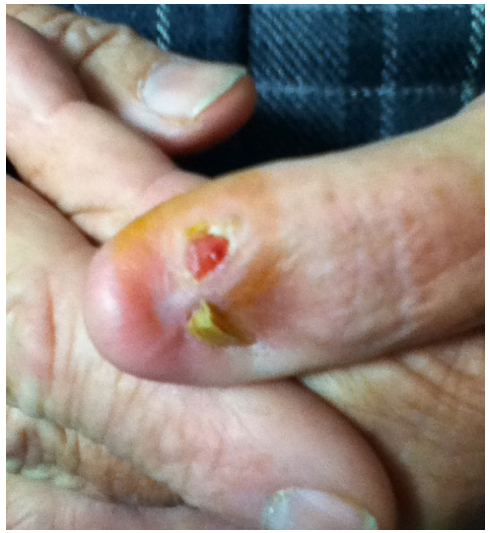
Chronic, Refractory Osteomyelitis

- Initial evidence for a beneficial therapeutic effect of HBOT in managing osteomyelitis stemmed from reports that demonstrated that the decreased oxygen tensions typically associated with bony infections can be returned to normal or above normal levels during HBOT.
- Neutrophils require tissue oxygen tensions of 30-40 mmHg to destroy bacteria by oxidative killing mechanisms. Leukocyte mediated killing of aerobic Gram-negative and Gram-positive organisms, including Staphylococcus aureus, is restored when the low oxygen tensions intrinsic to osteomyelitic bone are increased to physiologic or supra-physiologic levels. Additionally, HBOT therapy has been noted to exert a direct suppressive effect on anaerobic infections.
- In addition to enhanced leukocyte activity, HBOT can enhance the transport and augment the efficacy of antibiotic action. (cefazolin and HBOT therapy produced a 100-fold greater reduction in bacterial counts than either antibiotics or HBOT alone.) Comparable effects are also seen with HBOT in mitigating localized soft tissue infections. A research study demonstrated a 46% reduction in infection resolution time from a mean of 13 to only 6 days when HBOT was added to antibiotics in the management of soft tissue infections. As infected soft tissues often act as conduits for initiating and sustaining cortical bone infections, HBOT's benefit in ameliorating soft tissue infections may be critical to its overall efficacy in refractory osteomyelitis.
- There is evidence that HBOT enhances osteogenesis. Remodeling of bone by osteoclasts is an oxygen-dependent function. As previously noted, HBOT can restore physiologic or provide supra-physiologic oxygen tension in hypoxic bone environments, thus osteoclast function in infected bone can be improved.
- The pathophysiology of chronic osteomyelitis is characterized by both acute and chronic sources of ischemia. HBOT has been shown to be effective in reducing tissue edema, lowering intra-compartmental pressures and ameliorating the detrimental effects of inflammatory reactions. Over the longer term, HBOT can be used to promote new collagen formation and capillary angiogenesis in both hypoxic bone and surrounding tissues. By creating a sustained increase in the arterial perfusion of previously hypoxic bone and soft tissues, HBOT can reduce the susceptibility of these tissues to recurrent infection and necrosis.

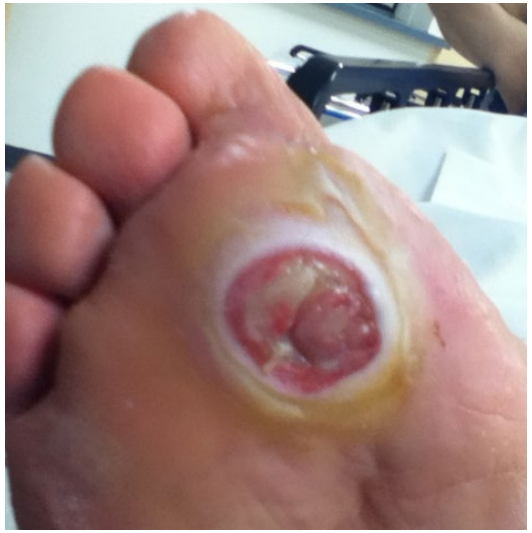
cool… but I'm not a scientist. WHAT DOES THAT MEAN?

- Bone infections cause a decrease in oxygen in the bone, HBOT increases the oxygen in the bone to normal or even above normal levels.
- HBOT increases the body's natural ability to fight infections and can suppress infection.
- HBOT can enhance the transport and augment the efficacy of antibiotic. (basically, making the antibiotic work better and get to where we need it to go)
- Osteomyelitis causes ischemia in the bone, causing necrosis. HBOT stimulates osteoclast function, making the bone stronger and rebuilding damage caused by osteomyelitis, essentially creating osteogenesis.
- HBOT can be used to promote new collagen formation and capillary angiogenesis in both the bone and surrounding tissues.
- By improving the health of the bone and surrounding soft tissues, the likelihood of Osteomyelitis recurring is reduced.

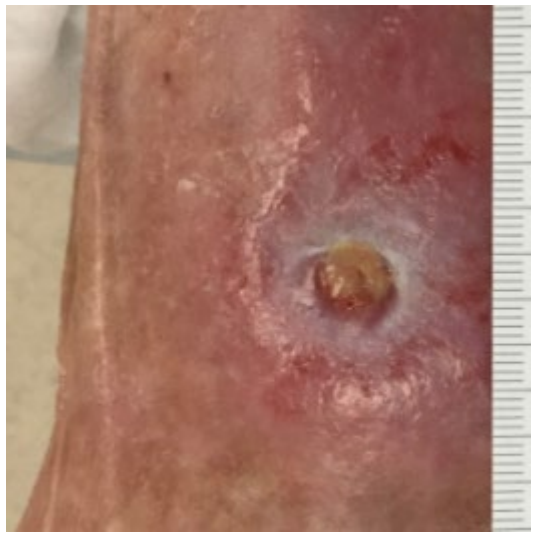
Chronic Refractory Osteomyelitis Examples



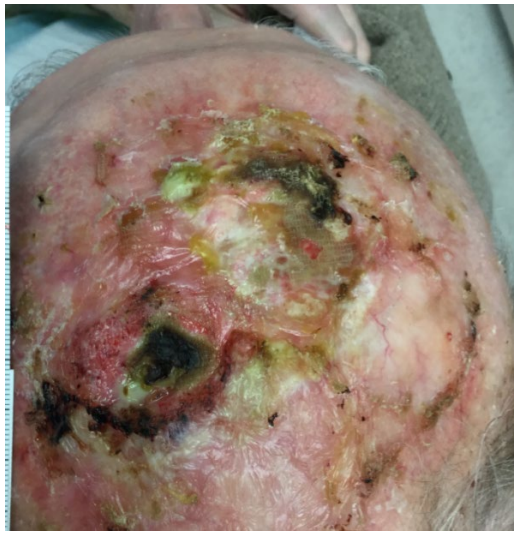
Thumb



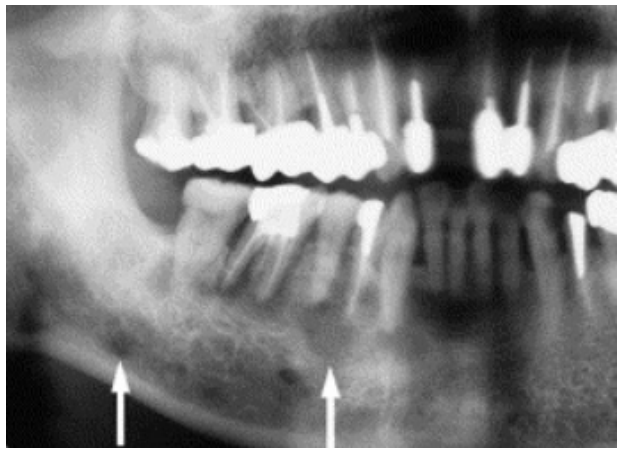
Foot



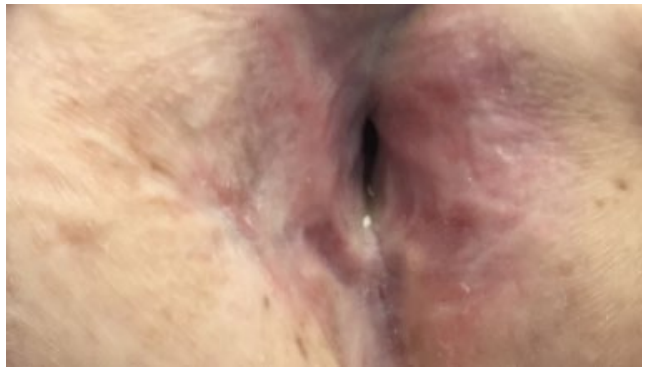
Ankle



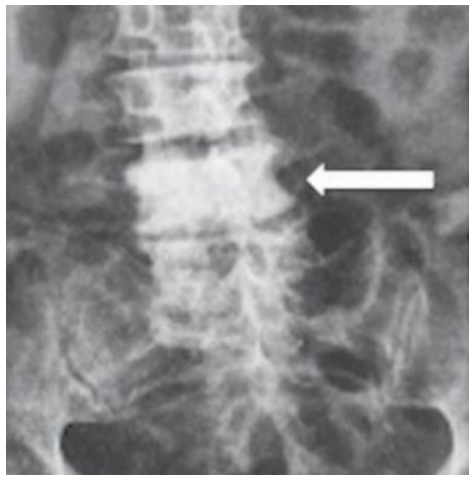
Skull



Mandible



Ischium



Vertebrae

CROM & HBOT / When to Continue

Matt’s suggestions

- Order 30 HBO treatments with reassessment every 10 treatments
- Continuation – during the reassessment, if the ulcer is continuing to improve continue HBOT
- If at the reassessment the ulcer is NOT continuing to improve, you must discontinue HBOT
- Utilization Review at treatment #60 to continue

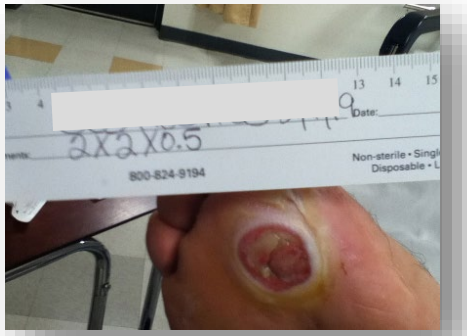
Chronic Refractory Osteomyelitis

After 29 Hyperbaric Treatments

Length: 2 cm

Width: 2 cm

Depth: 0.5 cm

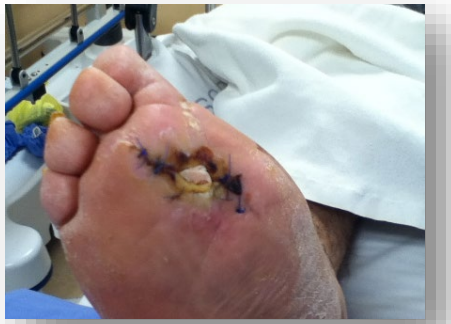


After 48 Hyperbaric Treatments & Bone Debridement

Length: 1.8 cm

Width: 1 cm

Depth: 1.7 cm



Complete Closure

4 Months After Hyperbaric

Length: 0 cm

Width: 0 cm

Depth: 0 cm

Delayed Radiation Injury (soft tissue & bony)

- Because a consistent cause and manifestation of radiation injury is vascular obliteration and stromal fibrosis, the known impact of hyperbaric oxygen in stimulating angiogenesis is an obvious and important mechanism whereby HBOT is effective in radiation injury. HBOT induces neovascularization in hypoxic tissues. Marx has demonstrated the enhanced vascularity and cellularity in heavily irradiated tissues after hyperbaric oxygen therapy by comparing histologic specimens from patients pre- and post- hyperbaric oxygen.
- The impact of hyperbaric oxygen in terms of its beneficial effects in irradiated tissues: 1) Hyperbaric oxygen stimulates angiogenesis and secondarily improves tissue oxygenation; 2) Hyperbaric oxygen reduces fibrosis

Let’s break it down!

Delayed Radiation Injuries – explained

- Radiation kills everything in its path, good or bad.
- HBOT stimulates angiogenesis, growing new blood vessels. This improves tissue oxygenation.
- HBOT improves vascularity in irradiated bone.
- HBOT reduces fibrosis (fibrosis is thickening or scarring of tissue.)

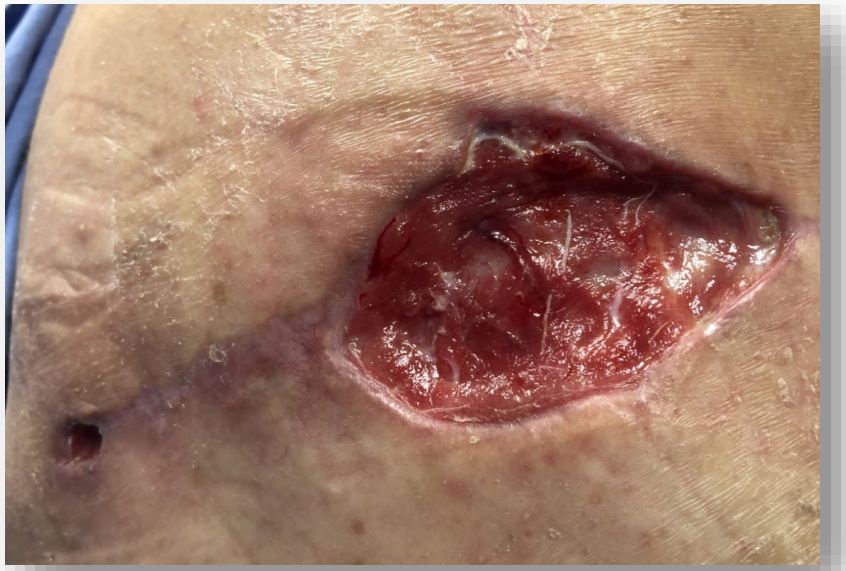
Soft Tissue Radiation & Osteoradionecrosis with HBOT / When to Continue

Matt's Suggestions

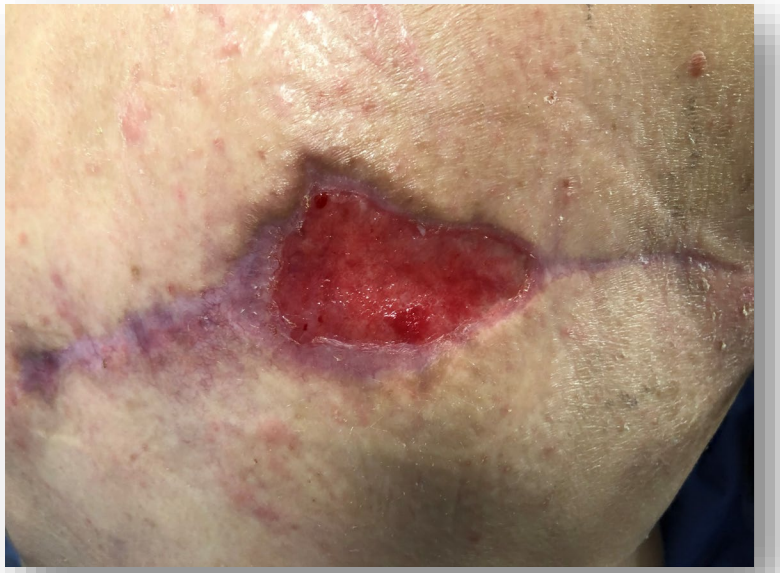
- ORN 20/10 and reassess for continuation
- STRI-30 and reassess for continuation
- STRI-Continue treatment if patient is improving- STRI-blood should be slowing and pain should be lessening
- ORN-Dental carries and bone coverage, fistulae drainage
- Utilization Review-if clinically there is no improvement by treatment #60

Soft Tissue Radionecrosis

The start of HBOT treatments.



After just 7 treatments!



Diabetic Foot Ulcer

- A basic pathway to non-healing is the interplay between tissue hypoperfusion, resulting hypoxia, and infection. A large body of evidence exists which demonstrates that intermittent oxygenation of hypo-perfused wound beds, mitigates many of these impediments and sets into motion a cascade of events that leads to wound healing. Physiologically, this produces a directly proportional increase in the plasma volume fraction of transported oxygen that is readily available for cellular metabolism. Availability of substrate for oxygen dependent enzymatic reactions critical to repair and resistance to infection is even more important than normalization of metabolic rate. Furthermore, oxidants appear to be among the most important signals that control the healing process, and this may be another mechanism for the benefits of HBOT in hypoxic wounds. Oxygen diffusion varies in a direct linear relationship to the increased partial pressure of oxygen present in the circulating plasma caused by HBOT. This significant level of hyperoxygenation allows for the reversal of localized tissue hypoxia, which may be secondary to ischemia or to other local factors within the compromised tissue.
- In the hypoxic wound, HBOT acutely corrects the pathophysiology related to oxygen deficiency and impaired wound healing. A key factor in hyperbaric oxygen therapy's enhancement of the hypoxic wound environment is its ability to establish adequate oxygen availability within the vascularized connective tissue compartment that surrounds the wound. Proper oxygenation of the vascularized connective tissue compartment is crucial to the efficient initiation of the wound repair process and becomes an important rate-limiting factor for the cellular functions associated with several aspects of wound healing.
- Neutrophils, fibroblasts, macrophages, and osteoclasts are all dependent upon an environment in which oxygen is not deficient in order to carry out their specific inflammatory or repair functions. Improved leukocyte function of bacterial killing and antibiotic potentiation, have been demonstrated. Suppression of synthesis of many bacterial toxins occurs when tissue oxygen values are sufficiently elevated during treatment. Blunting of systemic inflammatory responses and prevention of leukocyte activation and adhesion following ischemic reperfusion are effects that may persist even after completion of hyperbaric oxygen treatment.
- Stimulation of tissue growth supporting wound healing has also been demonstrated by a variety of mechanisms: 1) Vascular endothelial growth factor (VEGF) release is stimulated, and platelet derived growth factor (PDGF) receptor appearance is also induced. 2) Persistent increases in nitric oxide in wound fluid in diabetic ulcers associated with increased granulation tissue formation and wound closure when patients are exposed to HBOT.
- The net result of HBOT is improved local host immune response, clearance of infection, enhanced tissue growth and angiogenesis leading to progressive improvement in local tissue oxygenation and healing of hypoxic wounds.

To Make a Long Story Short…

Diabetic Foot Ulcers – explained

- Increase oxygenation in the blood plasma and subsequently the wound, reducing hypoxia.
- All of the following are dependent upon an environment in which oxygen is not deficient in order to carry out their specific inflammatory or repair functions…
  - Neutrophils (white blood cells that are your body's first line of defense in an immune response)
  - Fibroblasts (the most common cell in connective tissue – collagen),
  - Macrophages (white blood cell that surrounds and kills microorganisms, removes dead cells, and stimulates the action of other immune system cells.)
  - Osteoclasts (initiate normal bone remodeling and mediate bone loss in pathologic conditions by increasing their resorptive activity) Increase the body's natural ability to fight infection.
- Stimulation of tissue growth, creating an increase in granulation tissue and wound healing.
- Overall, improved immune response, clearance of infection, enhanced tissue growth and angiogenesis leading to improvement in wound healing.

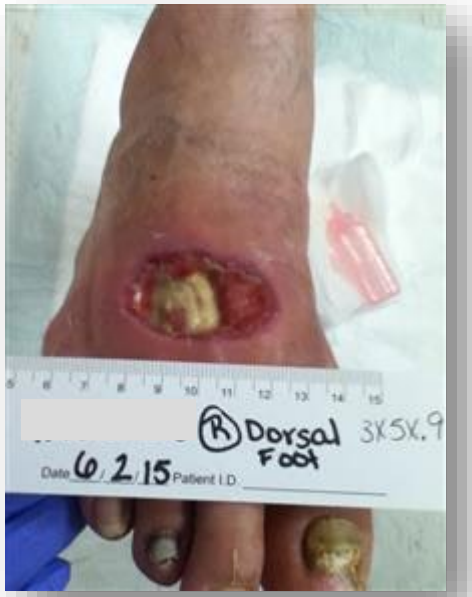
Wagner Grade 3 Diabetic Foot Ulcer

Pre-Hyperbaric

Length: 3 cm

Width: 5 cm

Depth: 0.9 cm

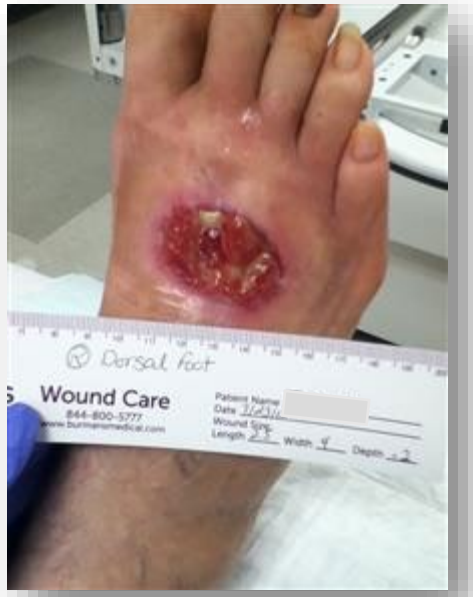


After 30 Hyperbaric Treatments

Length: 2.5 cm

Width: 4 cm

Depth: 0.2 cm



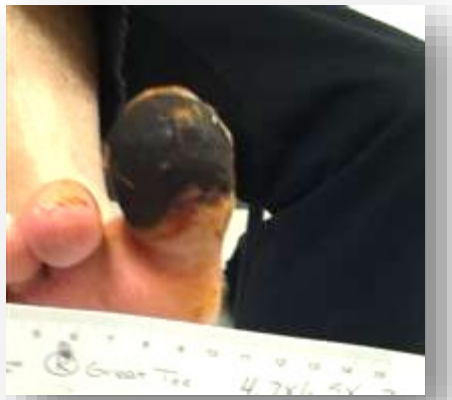
Wagner Grade 4 Diabetic Foot Ulcer

Initial Hyperbaric Consultation

Length: 4.7 cm

Width: 6.5 cm

Depth: 0.2 cm

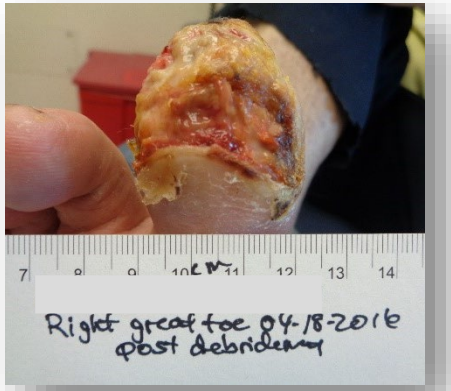


After 8 Hyperbaric Consultation

Length: 5 cm

Width: 5.7 cm

Depth: 0.5 cm



After 58 Hyperbaric Treatments

Length: 1 cm

Width: 1 cm

Depth: 0.1 cm

DFU & HBOT / When to Continue

Matt’s suggestions

- Order 30 HBO treatments with reassessment every 10 treatments
- Continuation during the reassessment, the ulcer is continuing to improve continue HBOT. If at treatment #30 there is no improvement HBOT must be stopped
- If at the reassessment the ulcer is not continuing to improve, you must discontinue HBOT
- Utilization Review at treatment #60 to continue

Let’s Recap – Indications and Rationale

- **Acute Carbon Monoxide Poisoning** – Relieve hypoxia; hasten elimination of CO; antagonize brain lipid peroxidation
- **Acute Exceptional Blood Loss Anemia** – Increase physically dissolved oxygen; treat hypoxia; support marginally perfused tissues
- **Acute Thermal Burns** – Relieve hypoxia; decrease fluid losses; limit burn wound extension and conversion; treat edema; promote wound closure
- **Arterial Gas Embolism** – Overcome free gas volume; relieve hypoxia; antagonize leukocyte mediated ischemia-reperfusion injury
- **Chronic Refractory Osteomyelitis** – Augment host antimicrobial defenses; induce angiogenesis; potentiate leukocytic dismutase superoxide and perioxide production; relieve hypoxia; augment antibiotic therapy; extend post-antibiotic effect; augment osteoclast activity
- **Clostridial Gas Gangrene** – Reduce size of gaseous bullae; inactivate clostridial alpha toxin; inhibit alpha toxin production; induce bacteriostasis; potentiate leukocytic dismutase superoxide and perioxide production
- **Compromised Skin Grafts/Flaps** – Support marginally perfused/oxygenated tissues; antagonize ischemic reperfusion injury; accelerate angiogenesis
- **Crush Injury; Acute Ischemia** – Provide interim tissue oxygenation in relative states of ischemia; reduce edema; reduce compartment pressures; antagonize ischemic-reperfusion injury; augment limb salvage
- **Decompression Sickness** – Overcome free gas volume-induced ischemia; relieve hypoxia; hasten elimination of offending inert gas; treat edema
- **Late Radiation Tissue Injury** – Re-establish wound oxygen gradients; relieve hypoxia; induce angiogenesis; prepare for definitive coverage
- **Necrotizing Soft Tissue Infections** – Induce bacteriostasis of anaerobes; (fasciitis and cellulitis) potentiate leukocytic dismutase superoxide and perioxide production; relieve hypoxia; more closely demarcate potentially viable tissue
- **Non-Healing Marginally Perfused Wounds** – Re-establish wound oxygen gradients; relieve hypoxia; reduce edema; induce angiogenesis; correct diabetic-induced leukocyte changes; prepare for definitive coverage

*UNDERSEA AND HYPERBARIC MEDICAL SOCIETY, 2008

Source

HBO Indications - Undersea & Hyperbaric Medical Society (uhms.org)


QUIZ

Question 1

- Define angiogenesis.

Answer 1

- The development of new blood vessels

Question 2

- Our goal is to use hyperbaric oxygen therapy until the diabetic foot ulcer closes completely. True or false?

Answer 2

- False!
- Our goal is to promote angiogenesis, improve immune response, promote clearance of infection, and enhance tissue growth – leading to improvement in wound healing. (Not necessarily closure.)

Question 3

- When looking at how hyperbaric oxygen therapy works for chronic, refractory osteomyelitis, which of the following is true?
  - A – HBOT increases oxygen in the bone.
  - B – HBOT stimulates osteoclast function.
  - C – HBOT augments antibiotics, increasing its efficacy.
  - D – All of the above.

Answer 3

- D – All of the above.

SerenaGroup HBOT Contact Information

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SerenaGroup Education

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Round Table Discussion

SerenaGroup Upcoming HBOT Educational Courses

Introduction to Hyperbaric Medicine = Wichita KS

November 3 - November 6

Ascension Via Christi St. Francis

Introduction to Hyperbaric Medicine = Omaha NE

January 12, 2023 - January 15, 2023

CHI Health Creighton University Medical Center Bergan Mercy

Introduction to Hyperbaric Medicine = West Palm Beach FL

March 23, 2023 - March 26, 2023

Perry Baromedical Corporation

Introduction to Hyperbaric Medicine = West Palm Beach, FL

August 17, 2023 - August 20, 2023

Perry Baromedical Corporation

Introduction to Hyperbaric Medicine = West Palm Beach, FL

November 9, 2023 - November 12, 2023

Perry Baromedical Corporation

Next Month's Presenter

DATE: Tuesday, November 15, 2022, at 12 pm eastern time.

PRESENTING: CHI Health - Mercy

TOPIC: Creating a Safe and Comfortable Environment