INDICATIONS FOR HBOT

- Air Embolism
- Carbon Monoxide Poisoning
- Decompression Sickness
- Gas Gangrene
- Intracranial Abscess
- Necrotizing Soft Tissue Infections like
  - a) Cellulitis
  - b) Abscesses
  - c) Fasciitis
- Osteomyelitis (Refractory)
- Crush Injury / Compartment Syndrome
- Arterial Insufficiencies like
  - a) PVD
  - b) Venous Ulcer
  - c) Diabetic Ulcer
  - d) Central Retinal Artery Occlusion
- Delayed Radiation Injury like
  - a) Osteoradionecrosis
  - b) Radiation Cystitis
  - c) Radiation Proctitis
- Compromised Graft and Flap
- Acute Thermal Burn Injury
- Sudden Idiopathic Sensorineural Hearing Loss

The Hyperbaric Team

Many doctors are also recommending conditions like Traumatic Brain Injuries Cranial Vascular Injuries (stroke, clot & bleeding), Autism and Fibromyalgia.

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THE HBOT PULSE

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GODREJ MEMORIAL HOSPITAL
HYPERBARIC OXYGEN TREATMENT CENTER
HYPERBARIC MEDICINE & HBOT

DEPARTMENT OF HYPERBARIC & DIVING MEDICINE

GODREJ MEMORIAL HOSPITAL
COMPASSIONATE CARE FOR ALL
(NABH & NABL Accredited)

CRUSH INJURY, COMPARTMENT SYNDROME & OTHER ACUTE TRAUMATIC ISCHEMIAS

Introduction and Definitions : Crush injuries represent a spectrum of injury to body parts as a result of trauma. Presentations vary from minor contusions to limb threatening damage. Typically, the injury involves multiple tissues from skin and subcutaneous tissue to muscle and tendons to bone and joints. In their most severe presentations, predictable complications including osteomyelitis, non-union of fractures, failed flaps and amputations occur in approximately 50 percent of the cases with "standard of practice" surgical and medical interventions.

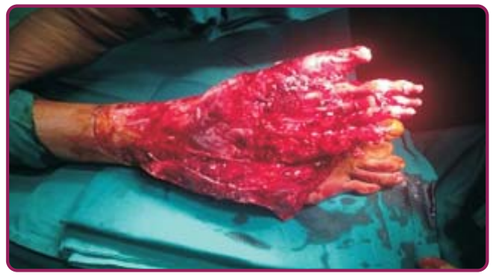
The skeletal muscle compartment syndrome (SMCS) is another consequence of trauma, but in this situation the target tissues are muscles and nerve. Edema and/or bleeding within the confines of the fascial envelope increase the pressure within the skeletal muscle-compartment. When the tissue fluid pressure within the compartment exceeds the capillary perfusion pressure to the muscles and nerves in the compartment, these tissues are rendered ischemic and manifest the signs and symptoms of a SMCS. The SMCS, especially in its incipient stages before a fasciotomy, is a therapeutic challenge since no means to arrest its progression other than hyperbaric oxygen (HBOT) exist. Unfortunately, HBOT is woefully neglected as an adjunct for managing crush injury and SMCS.

Pathophysiology : Trauma plus tissue hypoxia are the common denominators of crush injuries and SMCS. This leads to two consequences; first, a continuum of injury from normal to irreversibly damaged, and second, a self-perpetuating (i.e. vicious circle) progression of edema contributing to tissue ischemia and vice versa. Consequences of trauma include visible damage to tissue, injury at the cellular level and biochemical alterations. If the trauma and consequent energy transfer to the tissues is great enough, the tissues will immediately die. The only options in these circumstances are debridement if the site of involvement is small or major limb amputation if large.

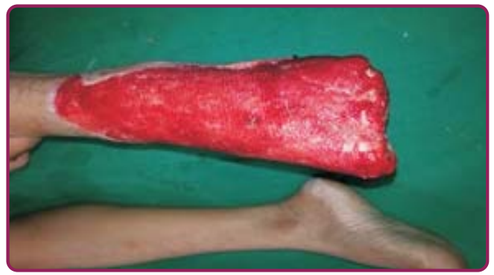
At the cellular level the self-perpetuating aspects of these injuries manifest themselves. Trauma to blood vessels, especially at the microcirculation level, leads to transudation of fluid (i.e. edema formation), interstitial bleeding, sluggish flow, stasis, slugging, rouleau formation, and

MARVELOUS RECOVERY

Treated at Godrej Memorial Hospital by the Department of Surgery
(In all these cases HBOT played a vital role in the treatment plan.)

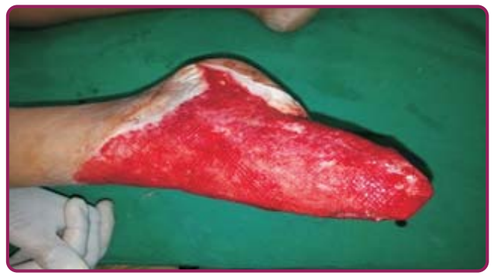


First day at GMH



10 days with HBOT

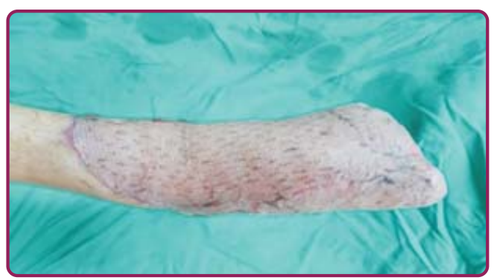
This worker from Godrej & Boyce Manufacturing Company Ltd. was brought to Godrej Memorial Hospital when a fully loaded fork-lift went over his left foot. Proficient multiple surgeries under **Dr. Pankaj Ahire** and good use of HBOT has certainly saved his foot from amputation.



20 days with HBOT

30 days with HBOT - ready for grafting

Post grafting



Discharged after miraculous recovery

Edema reduction is a secondary effect of tissue hyperoxygenation. Hyperbaric oxygen induces vasoconstriction which reduces blood flow by 20 percent. Since inflow is decreased by 20 percent through vasoconstriction while outflow is maintained, the net effect is edema reduction of 20 percent. Edema reduction occurs because of decreased filtration of fluid from the capillary to the extracellular space as a consequence of vasoconstriction while resorption of fluid at the capillary level is maintained.

Hyperoxygenation of the plasma maintains oxygen delivery to tissues in the presence of HBOT-induced vasoconstriction. Another consequence of decreasing the interstitial fluid pressure through edema reduction is improved blood flow through the microcirculation. The reason for this is that once the interstitial fluid pressure is reduced below the capillary perfusion pressure, the collapsed microcirculation can again open-up and allow perfusion to resume. By reducing edema while supplementing tissue oxygenation, HBOT interrupts the self-perpetuating, edema-ischemia "vicious circle" to prevent progression of the injury.

Mitigation of the reperfusion injury is another effect of HBOT for crush injuries and compartment syndromes. It interrupts the interactions between toxic oxygen radicals and cell membrane lipids by disturbing lipid peroxidation of the cell membrane and inhibiting the sequestration of neutrophils on post-capillary venules. The biochemical mechanism that accounts for this latter effect is that HBOT interferes with the adherence of neutrophils on the sensitized capillary endothelium. The result is interruption of the superoxide anion interaction with nitric oxide that produces the highly reactive peroxynitrite radical. Another benefit of HBOT for reperfusion injury is the help in providing an oxygenated environment for the generation of oxygen radical scavengers (such as superoxide dismutase, catalase, peroxidase and glutathione) that detoxify reactive oxygen species.

Treatment Protocol : HBOT should be started as soon as is feasible, ideally within 4-6 hours from time of injury to receive the greatest benefits from this therapy. After emergent surgical intervention, UHMS recommends that the patient undergo HBOT at 2-2.5 ATA for 90 - 120 minutes. For the first 2 – 3 days perform HBOT two times daily and then once daily for the next 7-15 days.

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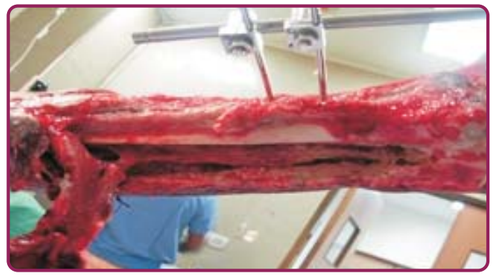
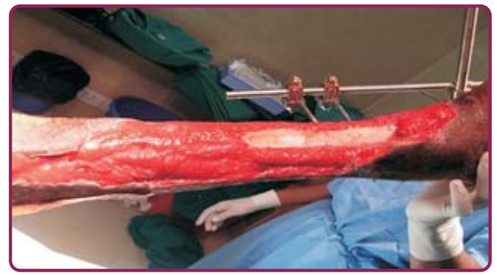
MD, DMM, DHA
Consultant Hyperbaric & Diving Medicine

MIRACLE AT WORK

Treated at Godrej Memorial Hospital by the Department of Surgery
(In all these cases HBOT played a vital role in the treatment plan.)

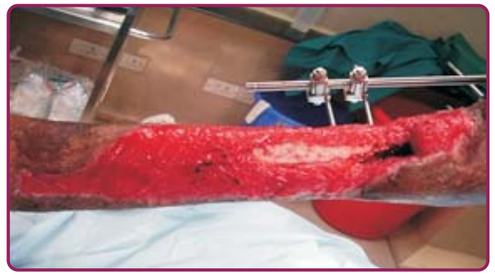


First day at GMH

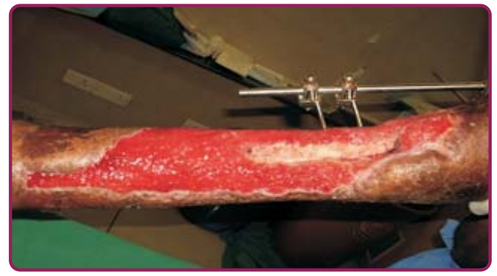


10 days with HBOT

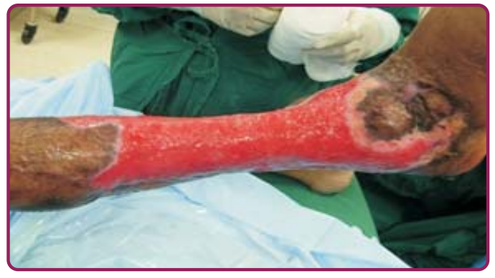
A tractor ran over the right leg of this youngster from Orissa.
He reached GMH 45 days later with a fear of imminent amputation.
Kudos to the Surgical team under Dr. Kishore Manek for saving his leg.



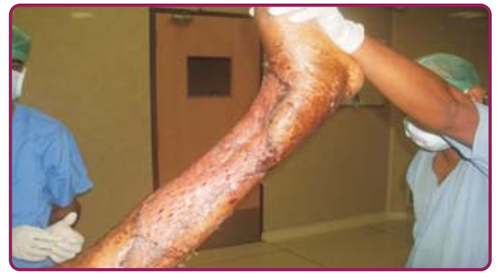
20 days with HBOT



30 days with HBOT



40 days with HBOT - ready for grafting



Discharged after miraculous recovery

obstruction. The consequences are ischemia and hypoxia to the tissues perfused by the damaged vasculature. When this occurs, cells are no longer able to maintain their metabolic functions such as retaining their intracellular water. This further contributes to edema and third spacing of fluid. If the edema occurs in a closed space the increased pressure will collapse the microcirculation, eliminate oxygen transfer across the capillary endothelium and further contribute to the hypoxic insult.

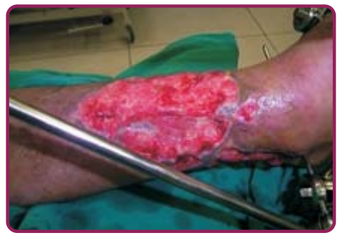
Events at the biochemical level, the ultimate determinants of outcome, are manifested in two ways. First, oxygen is required for all cellular metabolic functions. If oxygen tensions are insufficient, wound healing and angiogenesis responses as elaborated through the fibroblast and bacterial killing by the neutrophil are thwarted. Oxygen tensions in the tissue fluids greater than 30 mmHg are required for these responses to occur. The second biochemical event is that of the reperfusion injury. Once perfusion is temporarily interrupted, occurring in varying degrees with crush injuries and compartment syndromes, the endothelium becomes sensitized to the hypoxic insult. This results in activation of adhesion molecules leading to the attachment of neutrophils to the endothelium. The consequence is a cascade of biochemical events arising from the neutrophil releasing its reactive oxygen species. These oxygen radicals damage tissue beyond repair and cause severe vasoconstriction, defining the reperfusion injury and the no reflow phenomenon associated with it.

Mechanisms of HBOT: The immediate justifications for using HBOT in crush injuries and compartment syndromes are twofold: First, HBOT supplements **oxygen availability to hypoxic tissues** during the early post-injury period when perfusion is most likely to be inadequate. Second, HBOT increases tissue oxygen tensions to sufficient levels for the host responses mentioned above to function. Hyperbaric oxygen exposures at 2.4 atmospheres absolute (ATA) increase the blood oxygen content (the combination of hemoglobin and plasma carried oxygen) by 150 percent. The oxygen tensions in plasma as well as tissue fluids is increased 15-fold (1500 %). Sufficient oxygen can be physically dissolved in plasma under HBOT conditions to keep tissues alive without hemoglobin-borne oxygen. Increased tissue oxygen tensions result in a three-fold "driving force" (mass effect) for oxygen to diffuse through tissue fluids. This helps to compensate for the hypoxia resulting from the increased oxygen diffusion distance from the capillary to the cell through the surrounding edema.

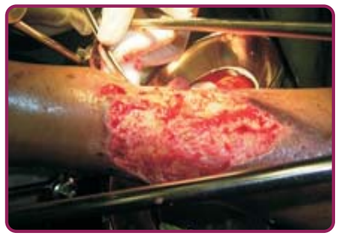
INTERESTING TRAUMA CASES

Treated at Godrej Memorial Hospital by the Department of Surgery
(In all these cases HBOT played a vital role in the treatment plan)

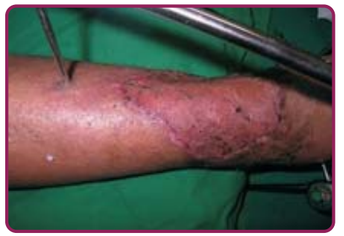
This teacher came to the Hospital when a car ran over her left leg. She reached GMH with a severely crushed injury requiring amputation below the knee. 20 days of HBOT and surgical intervention under **Dr. Harold D'souza** led to good healing and **allowing her to walk out of the hospital.**



First day at GMH

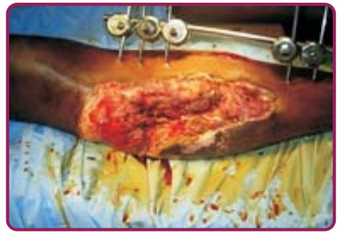


10 days with HBOT

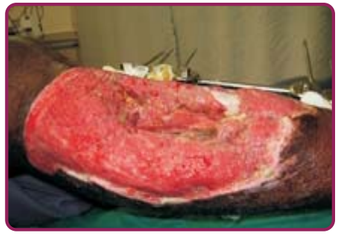


20 days with HBOT & skin grafting

This college student had a bike accident and shattered his right lower limb. Skillful surgery under **Dr. Jiten Chaudhary** and 20 days of HBOT at GMH led to prevention of amputation below the knee. His crush injury healed well and his lower limb was salvaged.



First day at GMH



10 days with HBOT

20 days with HBOT & skin grafting