The effect of hyperbaric oxygen in crush injuries and skeletal muscle-compartment syndromes

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ABSTRACT / RATIONALE

Crush injuries represent a spectrum of injury to body parts as result of trauma. Presentations vary from minor contusions to limb-threatening damage.

Typically, the injury involves multiple tissues, from skin and subcutaneous, to muscle and tendons, to bone and joints. In their most severe presentations, predictable complications – including osteomyelitis, non-union of fractures, amputations and failed flaps – occur in approximately 50 percent of the cases with standard of practice surgical and medical interventions [1-3].

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 can 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 SMCS. The SMCS, especially in its incipient stages before a fasciotomy is required, is a therapeutic challenge since no means to arrest its progression exist other than hyperbaric oxygen (HBO₂).

Unfortunately, HBO₂ is woefully neglected as an adjunct for managing crush injury and SMCS. Strong arguments exist for its use based on evidenced-based information and how HBO₂ mitigates the pathology of these conditions.

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 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 [4-6]. Oxygen tensions in the tissue fluids greater than 30 mmHg are required for these responses to occur [7].

The second biochemical event is that of reperfusion injury [8]. 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 HBO₂:** The immediate justifications for using HBO₂ in crush injuries and compartment syndromes are twofold: First, HBO₂ supplements oxygen availability to hypoxic tissues during the early post-injury period when perfusion is most likely to be inadequate. Second, HBO₂ increases tissue oxygen tensions to sufficient levels for the host responses mentioned above to function. Hyperbaric oxygen exposures at two atmospheres absolute (atm abs) increase the blood oxygen content (the combination of hemoglobin and plasma carried oxygen) by 125 percent. The oxygen tensions in plasma as well as tissue fluids is increased tenfold (1000%) [9-11]. Sufficient oxygen can be physically dissolved in plasma under HBO₂ conditions to keep tissues alive without hemoglobin-borne oxygen [11]. Increased tissue oxygen tensions result in a three-fold driving force (mass effect) for oxygen to diffuse through tissue fluids [12,13]. This helps to compensate for the hypoxia resulting from the increased oxygen diffusion distance from the capillary to the cell through the surrounding edema.

Edema reduction is a secondary effect of tissue hyperoxygenation. Hyperbaric oxygen induces vasoconstriction, which reduces blood flow by 20 percent [10, 14]. Since inflow is decreased by 20 percent through vasoconstriction while outflow is maintained, the net effect is edema reduction of 20 percent [14-18]. 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 HBO₂-induced vasoconstriction [10,19,20].

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, HBO₂ interrupts the self-perpetuating, edema-ischemia vicious circle cycle to prevent progression of the injury.

Mitigation of the reperfusion injury is another effect of HBO₂ for crush injuries and compartment syndromes [21-23]. It interrupts the interactions between toxic oxygen radicals and cell membrane lipids by perturbing lipid peroxidation of the cell membrane and inhibiting the sequestration of neutrophils on post-capillary venules [24-26]. The biochemical mechanism that accounts for this latter effect is that HBO₂ interferes with the adherence of neutrophils elaborated through the beta₂ integrin (Cluster-Designation-11) on the sensitized capillary endothelium [22]. The result is interruption of the superoxide anion interaction with nitric oxide that produces the highly reactive peroxynitrite radical [27]. Another benefit of HBO₂ 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 [28,29].

**PATIENT SELECTION CRITERIA**

**Crush injuries:** Objective criteria coupled with accepted grading systems should be used to make decisions as to when to use HBO₂ for crush injuries and compartment syndromes. Not only must the seriousness of the injury be considered, but the ability of the host to respond to the injury needs to be factored into the decision-making process. Obviously, better criteria for using HBO₂ in crush injuries must be employed than saying the injury is very severe, or that HBO₂ is needed because a complication such as sloughing of a flap has arisen.

This objective can be met by utilizing the internationally accepted Gustilo open-fracture crush injury grading system, coupled with an innovative five-criteria 0-to-10 point objective host evaluation (Tables 1 and 2, facing page) [2,3,30]. For the host evaluation, five assessments considered most useful for making decisions as to whether the patient’s physiological status warrants using interventions to avoid amputation are graded on a 2 (best) to 0 (worst) analogue scale. The mangled extremity severity score (MESS) can also be used to provide objective criteria for HBO₂ decision-making with crush injuries, but will not be discussed further in this guideline in light of the almost exclusive utilization of the Gustilo system in the orthopedic community [31-33].

Unfortunately, too often HBO₂ is requested only after complications from a crush injury have arisen, such as slough of a flap, wound dehiscence, threatened flap after delayed coverage/and or closure is done, muscle necrosis from residuals of a skeletal muscle-compartment syndrome and/or osteomyelitis. The time to start HBO₂ is with the initial management in those crush injuries where complications are predictable, such as the Gustilo III-B and C fractures and in lesser Gustilo grades in impaired and decompensated hosts (Table 2).
### TABLE 1: The five assessment host function score

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Grade</th>
<th>2 points</th>
<th>1 point</th>
<th>0 points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Activities of daily living</td>
<td>full</td>
<td>some</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>Ambulation</td>
<td>community</td>
<td>household</td>
<td>none</td>
<td></td>
</tr>
<tr>
<td>Co-morbidities*</td>
<td>not impaired</td>
<td>impaired</td>
<td>decompensated/ end-stage</td>
<td></td>
</tr>
<tr>
<td>Smoking/steroid use*</td>
<td>none</td>
<td>past</td>
<td>current</td>
<td></td>
</tr>
<tr>
<td>Neurological impairment</td>
<td>none</td>
<td>some</td>
<td>severe</td>
<td></td>
</tr>
</tbody>
</table>

* Use 1/2 point if findings are mixed or intermediate between two findings
* Subtract 1/2 point if walking aids used

**Note:** To determine host function score, add up the points for each assessment; score interpretations are provided on the right

* Whichever gives the lower score

**INTERPRETATION**

- **Healthy host**: 8 to 10 points
- **Impaired but compensated host**: 4 to 7 points
- **Decompensated host**: 0 to 3 points

### TABLE 2: Guide for the use of HBO₂ in open fracture, crush injuries (per the Gustilo classification)²,³ with consideration for host function

<table>
<thead>
<tr>
<th>Type (Gustilo)</th>
<th>Injury Characteristics</th>
<th>Anticipated Outcomes in Healthy Hosts</th>
<th>Indications for HBO₂ vs Host Status²³</th>
<th>Healthy</th>
<th>Impaired</th>
<th>Decompensated</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Small (&lt;1 cm wide) puncture wound from inside to out</td>
<td>Usually no different from a closed fracture</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Laceration with minimal deep soft tissue damage</td>
<td>Same as above</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Crush injuries</td>
<td>Depends on Sub-type</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes³</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>Sufficient soft tissue to close the wound</td>
<td>Complications² – 10%</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes³</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>Flaps needed for coverage</td>
<td>~ 50% incidence of complications²</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes³</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Major vascular injury</td>
<td></td>
<td>Yes</td>
<td>Yes</td>
<td>Yes³</td>
<td></td>
</tr>
</tbody>
</table>

**Notes:**
1. Refer to Table 1, the Host-Function Score
2. Complications include infection, failed flaps, delayed/nonunion, intractable pain, non-function and amputation
3. Consider primary amputation in decompensated hosts with Grade II-B and C open fracture, crush injuries; Hyperbaric oxygen may be needed to help with primary healing of the amputation flaps

**Compartment syndromes:** The skeletal muscle-compartment syndrome, like the crush injury, represents a continuum of severities divided into suspected, impending and established stages. The unifying pathophysiological feature of compartment syndromes is the self-perpetuating edema-ischemia cycle. In the suspected stage, the compartment syndrome is not actually present, but the severity of the injury or the circumstances (i.e., prolonged ischemia time) raises suspicions that a compartment syndrome could develop. In this stage HBO₂ is not recommended, but frequent neurocirculatory checks of the injured extremity are required to recognize the earliest possible progression to the impending stage.
FIGURE 1a: Indications for hyperbaric oxygen (HBO₂) in the skeletal muscle-compartment syndrome

**Findings**
1. Post-traumatic edema
2. Infiltrations
3. Venous outflow obstruction
4. Prolonged ischemia
5. Comatose posturing, “crunch” syndrome
6. Snare bite (unusual)
7. Shock (hyperperfusion) plus injury to soft tissues

**Management**
A. High “index of suspicion” for a SMCS
B. Frequent neurocirculatory checks (The “5 P’s”)
   - Pain
   - Parasthesias
   - Paralysis
   - Pulse
   - Paresthesia
C. Treat primary injuries

- Suspected Stage
  - (HBO₂ not Indicated)
  - Edema
  - Ischemia

- Impending Stage
  - HBO₂ Indicated
  - 3 or more clinical findings and/or 1 or more pressure measurement findings

**Compartment pressure measurements (If available)**
- Increasing serial measurements
  - Up to 45 mmHg in a Healthy Host, 30-40 mmHg in an Impaired Host, 20-30 mmHg in a Decompensated and/or Shocky host (See Table 1)

**Legend:** The Skeletal muscle-compartment syndrome starts with an injury or an insult (see findings above). If the edema-ischemia “vicious circle” progresses, the Suspected Stage evolves into the Impending Stage. Hyperbaric oxygen is the only known intervention that will mitigate the edema-ischemia “vicious circle” progression.

FIGURE 1b: Requirements for fasciotomy in the skeletal-muscle compartment syndrome and indications for HBO₂ post-fasciotomy

**Clinical Findings**
1. Increasingly severe pain
2. Anesthesia
3. Paralysis
4. Extreme discomfort with passive stretch
5. Tautness (rigid swelling) of muscle compartment
6. Encephalopathy, myelopathy and/or traumatic neuropathy

**Impending Stage**
- Edema
- Ischemia

**Established Stage**
- Fasciotomy Required
  - 3 or more clinical findings and/or 1 or more pressure measurement findings

**Indications for HBO Post-fasciotomy**
- Ischemic muscle
- Demarcation of viable and non-viable muscle is unclear
- Massive swelling/prolonged ischemia
- Threatened skin flap or graft
- Residual neuropathy
- Markedly impaired or decompensated host

**Manometrics**
(If available)
- >45 mmHg in a Healthy Host;
- 30-40 mmHg in an Impaired Host;
- 20-30 mmHg in a Decompensated and/or Shocky host (See Table 1)

**Legend:** If the Impending Stage progresses onto the Established Stage, HBO must not be used as a substitute for fasciotomy. However, HBO should be used post-fasciotomy if one or more residual findings are present.
If the edema-ischemia cycle perpetuates itself, the condition may evolve into the impending stage. In this stage, signs include:
1. increasing pain;
2. hypesthesias;
3. muscle weakness;
4. discomfort with passive stretch and/or
5. tenseness in the compartment.

If any of these signs exist, compartment pressure measurements should be made. If the compartment pressure(s) and clinical finding are such that fasciotomy is not required at this time, HBO2 should be started to prevent progression from the impending stage to the established stage. If pressure testing is not available and the compartment syndrome is not in the established stage, three or more clinical findings is sufficient indication to initiate HBO2 treatments (Figure 1-a, facing page). A second indication for HBO2 in the impending stage, if pressure testing is available, is increasing compartment pressures with repeated measurements. As in crush injuries, the host-function status needs to be considered when making decisions to use HBO2 for the impending stage of the SMCS.

In the established stage of the SMCS, symptoms, signs and/or pressure measurements confirm the diagnosis and dictate immediate fasciotomy be done (Figure 1-b, facing page). Hyperbaric oxygen must not be used as reason to defer surgery in the above situations. However, after fasciotomy HBO2 should be used as an adjunct to wound management if significant residual problems remain, such as ischemic muscle, threatened flaps, unclear demarcation between viable and non-viable muscle, residual neuropathy, massive swelling, prolonged (more than six hours) ischemia time and/or significant host impairment as determined by the Host-Function Score.

The term “lag phase” refers to the time interval from the injury or insult to the time symptoms of SMCS are severe enough to make the diagnosis. It may vary from an hour or two, with bleeding into the compartment, to 24 hours or more with blunt trauma. The lag phase is a manifestation of the self-perpetuating events that precede the impending and established phases of the SMCS.

**CLINICAL MANAGEMENT**

**Crush injuries:** Early application of HBO2, preferably within four to six hours of the injury, is recommended. Treatment schedules for crush injuries should be tailored to mitigate the suspected pathophysiology – for example: three or more treatments in a 24-hour period for critical ischemias; twice a day for threatened flaps and oxygenating an environment so host factors can function; and once a day for dealing with infections, remodeling or resorption of calcified tissues (Table 3, above). For the isolated reperfusion injury after revascularization or thrombectomy of an extremity that otherwise has sustained minimal physical trauma, a single HBO2

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**TABLE 3: Treatment recommendations and peer review when using HBO2 for crush injury and compartment syndrome**

<table>
<thead>
<tr>
<th>Condition</th>
<th>HBO2 Treatments and Peer Review</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Conditions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Reperfusion Injury</td>
<td>1</td>
<td>Minimal tissue trauma; e.g. after revascularizations, free flaps and transient ischemias</td>
</tr>
<tr>
<td>2. Crush Injury</td>
<td>8 (TID 2 days, BID 2 days and daily 2 days)</td>
<td>If deterioration noted when HBO treatments are decreased, resume the previous schedule</td>
</tr>
<tr>
<td>3. Compartment Syndrome</td>
<td>3 (BID day 1 and a single HBO Rx day 2)</td>
<td>HBO is not a substitute for fasciotomy; use HBO for the impending stage of the SMCS</td>
</tr>
<tr>
<td><strong>Residual Problems and/or Complications</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Threatened flaps and grafts</td>
<td>10 (BID for 5 days)</td>
<td>If site remains tenuous, consider daily HBO treatments an additional 5 days</td>
</tr>
<tr>
<td>2. Problem wounds/infected wounds</td>
<td>21 (BID for 7 days, daily for 7 days)</td>
<td>See problem wounds section</td>
</tr>
<tr>
<td>3. Refractory osteomyelitis</td>
<td>21 (Daily for 3 weeks)</td>
<td>HBO must be integrated with a combined antibiotic and surgical strategy</td>
</tr>
<tr>
<td>4. SMCS post-fasciotomy concerns</td>
<td>14 (BID for 7 days)</td>
<td>Concerns include massive swelling, threatened flaps, unclear demarcation, neuropathy, etc. (see text)</td>
</tr>
</tbody>
</table>

**Notes:** Peer review should be done by two or more of the following: 1) HBO consulting physician, 2) Trauma/orthopaedic surgeon, 3) Plastic/reconstructive surgeon and/or 4) primary care physician

**Abbreviations:** BID = Twice a day, e.g. = for example, HBO = Hyperbaric Oxygen, SMCS = Skeletal muscle-compartment syndrome, TID = Three times a day
treatment, based on animal studies and limited clinical observations, is probably adequate [22,26]. Typically, treatment pressures range from 2 atm abs in monoplace chambers to 2.4 atm abs in multiplace chambers, with oxygen breathing periods of 90 minutes for two or more treatments a day to 120 minutes for single daily treatments.

**Compartment syndromes**: For the impending stage of the SMCS, HBO2 treatments should be given twice a day for 24 to 36 hours, the time that the self-perpetuating edema-ischemia cycle would be expected to end. Symptoms and signs of pain reduction, absence of neurological abnormalities, and less tautness in the compartment should be used in deciding to stop HBO2.

For residual complications after fasciotomy has been performed for an established compartment syndrome, HBO2 should be given twice a day for a seven- to 10-day period or when the problems have stabilized enough that no benefit is being realized from HBO2. Treatment durations and pressures are the same for crush injuries: that is, 90- to 120-minute durations at 2.0 to 2.4 atm abs.

**SUPPORTING LITERATURE AND EVIDENCE-BASED INDICATIONS**

**Crush injury literature review**: More than 600 clinical cases reported in more than 20 publications attest to the usefulness of HBO2 in crush injuries [33,34]. Although most of the reports describe the benefits of HBO2 in subjective terms such as HBO2 treatments were helpful, good results were achieved, or from past experiences problems of similar magnitude would have resulted in amputations, overall outcomes were positive in about 80 percent of the reports [35].

The more important observation was that, as the frequency of treatments increased, the outcomes improved [36-38]. Specifically, in traumatic ischemias, Schramek reported 100 percent salvage rates with six HBO2 treatments a day, Loder reported 80 percent complete or partial recoveries with three HBO2 treatments a day, and Slack reported that 59 percent responded well with one HBO2 treatment a day. In 2005 Lisardo et al. published an evidenced-based approach regarding the use of HBO2 in the management of crush injuries and traumatic ischemias [39]. They found nine reports comprising approximately 150 patients that met their inclusion criteria. Eight of nine studies showed a beneficial effect of HBO2 with only one reported major complication. They concluded that HBO2 as an adjunct to managing crush injuries and traumatic ischemias is not likely to be harmful and could be beneficial if administered early.

The one randomized controlled trial in their report was that of Bouachour’s crush injury-fracture study [40]. Bouachour and his co-authors reported complete healing in 94 percent of the HBO2 group vs. 33 percent in the controls (p<0.01) while the need for additional surgeries was 6 percent in the HBO2 group as compared to 33 percent in the controls (p<0.05). The hyperbaric oxygen arm also demonstrated benefits when age was used as a marker for host-function status.

**Compartment syndromes literature review**: In the 1980s the effects of HBO2 on the SMCS were reported in a series of articles using a canine model [15-17,42,42]. The HBO2-treated group had significantly less skeletal muscle necrosis than the controls when radiopharmaceutical and histological methods were used to study outcomes. When HBO2 treatments were delayed, more injury was observed in the HBO2 group, but was still significantly less than the controls. In animals rendered shocky by exsanguination, HBO2 provided protective benefits as measured by muscle necrosis and edema reduction as compared to the controls. Nylander’s studies with tourniquet ischemia showed similar benefits in the HBO2-treated animals [14]. Bartlett et al. reported significantly improved electrophysiological muscle function in a canine SMCS with a combination of fasciotomy and HBO2 versus the fasciotomy group alone [43]. These findings are consistent with several hundred reported clinical experiences using HBO2 for SMCS [44-46].

**American Heart Association level of classification**: When the American Heart Association (AHA) criteria are used for crush injuries, HBO2 meets the criteria for a 1-b evidenced-based indication. This is based upon a high benefit-to-complication ratio, the mechanisms of HBO2 are appropriate for mitigating the pathophysiology, and a published randomized control trial exists. If SMCS is considered as a separate entity rather than in the traumatic ischemia group, the indication drops to a 2-a level because of the absence of a randomized control trial.

**Rationale-based, evidence-appropriate indications**: With less than 20 percent of the decisions made in clinical medicine meeting the criteria of evidenced-based indications, a more pragmatic system is recommended for making decisions as to whether to use HBO2 in general and crush injuries, and compartment syndromes in particular [47]. Consequently, a rationale-based, evidence-appropriate (RBEa) evaluation system is recommended [48] (Table 4, facing page). Five assessments considered most useful for making RBEa decisions as to whether to use interventions to avoid amputation are graded on a 2 (best) to 0 (worst) analogue scale. This generates a 0 to 10 score. Scores of 5 or greater are considered justification for using the intervention. For crush injuries the RBEa score is 7, and for compartment syndrome it is 6.
Utilization Review

Crush Injuries: Recommendations for utilization review have been listed previously (Table 3). Because of the various timelines in the progression of complications that arise from open-fractures and crush injuries, utilization review must be carefully considered. For crush injuries and compartment syndromes, utilization review should be initiated by the consulting hyperbaric medicine physician, and the decision whether to extend HBO2 treatments or stop them should be a joint opinion of the hyperbaric physician, the trauma/orthopedic surgeon, the plastic/reconstructive surgeon and/or the primary care physician.

Compartment Syndromes: For the impending stage of compartment syndromes, utilization review is recommended after three HBO2 treatments. For managing complications post-fasciotomy, utilization review should be done after seven days or 14 HBO2 treatments.

Threatened Flaps or Grafts: If subsequent skin grafting or delayed closure is done for the fasciotomy site, and the graft or flaps are threatened, then HBO2 treatments should be instituted per these latter protocols.

Cost Impact

Crush Injuries: The additional expenses associated with HBO2 treatments when used in crush injuries weigh favorably against the costs of dealing with 50 percent complication rates associated with Gustilo Type III-B and C open-fracture crush injuries [1-3]. In 1977 Brighton estimated that in the United States, $140,000 was the average cost per patient required to resolve the 100,000 open fracture-crush injuries that failed to heal primarily each year [49]. Costs would be manyfold higher today.

Even with new technologies, the complication rates are predictable for the most severe open-fracture crush injuries (Gustilo II-B and C) [2,3]. Because of the large number of open-fracture crush injuries that occur in the United States each year, a reduction in complications and the morbidity associated with them could have a substantial impact on healthcare costs and far outweigh additional expenses associated with HBO2 treatments.

Not to be dismissed are the intangible benefits that primary healing and avoidance of amputations have for the patient’s mental outlook, ability to function independently and return to gainful employment [50].

Experiences with HBO2 from the supporting literature and evidenced-based indications show when this modality is used as an adjunct for managing open-fracture crush injuries, complication rates of the severest injuries are approximately 20 percent. This compares favorably to the 50 percent complication rates reported in the literature when HBO2 was not used [1-3].

Compartment Syndromes: In a review, total costs were reported to be 75 percent less when HBO2 was initiated in the impending stage of the SMCS to prevent its progression to the established stage (and the need for fasciotomy) than for treating complications with HBO2 after a fasciotomy had been done [51].
Postscript: Although frostbite injuries, burns, threatened flaps and grafts and in-jeopardy replantations are not specifically mentioned in this section, they all have the unifying factor of trauma plus tissue hypoxia. The insults are what differentiate this group – cold injury for frostbite, heat injury for burns, etc. – from crush injuries and compartment syndromes. Their pathophysiology is similar to these two conditions with a continuum of injury responses and the self-perpetuating edema-ischemia vicious circle. For other conditions HBO₂ is indicated because they are, in reality, acute traumatic peripheral ishmias [52,53].

REFERENCES

49. Brighton CT. Quotation. Hosp Tribune (May 9, 1977); p5.