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Hyperbaric Oxygen Therapy in the Management of Crush Injuries: A Randomized Double-Blind Placebo-Controlled Clinical Trial

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Hyperbaric Oxygen (HBO) therapy is advocated for the treatment of severe trauma of the limbs in association with surgery because of its effects on peripheral oxygen transport, muscular ischemic necrosis, compartment syndrome, and infection prevention. However, no controlled human trial had been performed until now to specify the role of HBO in the management of crush injuries. Thirty-six patients with crush injuries were assigned in a blinded randomized fashion, within 24 hours after surgery, to treatment with HBO (session of 100% O_2 at 2.5 atmosphere absolute (ata) for 90 minutes, twice daily, over 6 days) or placebo (session of 21% O_2 at 1.1 ata for 90 minutes, twice daily, over 6 days). All the patients received the same standard therapies (anticoagulant, antibiotics, wound dressings). Transcutaneous oxygen pressure ($Ptco_2$) measurements were done before (patient breathing normal air) and during treatment (HBO or placebo) at the first, fourth, eighth, and twelfth sessions. The two groups (HBO group, n = 18; placebo group, n = 18) were similar in terms of age; risk factors; number, type or location of vascular injuries, neurologic injuries, or fractures; and type, location, or timing of surgical procedures. Complete healing was obtained for 17 patients in the HBO group vs. 10 patients in the placebo group ($p < 0.01$). New surgical procedures (such as skin flaps and grafts, vascular surgery, or even amputation) were performed on one patient in the HBO group vs. six patients in the placebo group ($p < 0.05$). Analysis of groups of patients matched for age and severity of injury showed that in the subgroup of patients older than 40 with grade III soft-tissue injury, wound healing was obtained for seven patients (87.5%) in the HBO group vs. three patients (30%) in the placebo group ($p < 0.05$). No significant differences were found in the length of hospital stay and number of wound dressings between groups. For the patients with complete healing, the $Ptco_2$ values of the traumatized limb, measured in normal air, rose significantly between the first and the twelfth sessions ($p < 0.001$). No significant change in $Ptco_2$ value was found for the patients whose healing failed. The Bilateral Perfusion Index (BPI = $Ptco_2$ of the injured limb/$Ptco_2$ of the uninjured limb) at the first session increased significantly from 1 ata air to 2.5 ata O_2 ($p < 0.05$). In patients with complete healing, the BPI was constantly greater than 0.9 at 2.5 ata O_2 during the following sessions, whereas the BPI in air progressively rose between the first and the twelfth sessions ($p < 0.05$), reaching normal values at the end of the treatment. In conclusion, this study shows the effectiveness of HBO in improving wound healing and reducing repetitive surgery. We believe that HBO is a useful adjunct in the management of severe (grade III) crush injuries of the limbs in patients more than 40 years old.

Hyperbaric oxygen (HBO) therapy is being increasingly employed for patients with partial or total tissue ischemia, such as acute ischemia of the limbs, burns, radiation-induced necroses, and cases of diabetic foot.[1-4] In severe injuries of the extremities, acute ischemia may occur, either after disruption of large arterial vessels, requiring immediate vascular repair, or as microcirculatory insufficiency resulting from crush injuries with frank disruptions of soft tissues, and combinations of these. Because of hypoxia, complications related to soft-tissue damage occur, such as ischemic necrosis, anerobic infection, edema, and compartment syndrome.[5] Furthermore, exploration of wounds and surgical modalities to obtain stable osteosynthesis may require large skin incisions that aggravate soft-tissue ischemia. Thus, compound open fractures involving extensive bone and soft-tissue devascularization result in a high percentage of complications and amputations that can reach 16 to 60% as a result of infection or ischemia.[6-10]

The immediate effect of HBO therapy is hyperoxygenation, which results from increasing the amount of physically dissolved oxygen in plasma, which is directly proportional to the partial pressure of inhaled oxygen.[11] Hyperoxia can be of great benefit because of its multiple effects: improvement of oxygen delivery and preservation of tissue viability in ischemic areas,[12,13] vasoconstriction reducing vasogenic edema in compartment syndrome,[14,15] prevention of infection notably due to anerobic microorganisms,[16,17] and enhancement of the wound-healing process.[18] Thus, HBO therapy directly counteracts the factors contributing to wound-healing compromise.[5]

Reports on several small series of patients suggest the usefulness of HBO therapy in crush injuries.[1,17,19-21] However, no comparison has been made between standard treatments and the adjunct use of HBO therapy in the management of severe injuries of the limbs. This prospective and randomized study was carried out to evaluate the effect of HBO in crush injuries of the limbs and its use as an adjunct measure.

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TABLE 1. Classification of soft-tissue injuries (derived from Gustillo)[10]

Type I	Wound less than 1 cm long and clean
Type II	Laceration more than 1 cm long without extensive soft-tissue damage, flaps, or avulsions
Type IIIA	Adequate soft-tissue coverage despite extensive soft-tissue laceration or flaps, or high-energy trauma irrespective of the size of the wound
Type IIIB	Extensive soft-tissue injury with periosteal stripping and bony exposure
Type IIIC	Arterial injury requiring repair

PATIENTS AND METHODS

During a 48-month period, patients with a severe limb injury were dispatched to the hyperbaric unit within 24 hours after the initial evaluation and surgical procedure. Patients were enrolled in the study according to the following criteria: acute injury of the limb classified as type II or III depending on soft-tissue injury (Table 1); surgical management within 6 hours after the injury; no history of peripheral arterial occlusive disease; and written informed consent from the patient.

Patients were excluded from the trial if any of the following conditions was present: enrollment in another trial; suspected pregnancy; or neurologic, pulmonary, or otorhinolaryngologic diseases contraindicating hyperbaric treatment.

The surgical procedures were performed by the same team and included adequate debridement and irrigation of the wound, primary closure without tension whenever possible with regard to the severity of the fracture and the soft-tissue injury, and stabilization procedures (internal, external, or conservative) used at the discretion of the surgeon. In addition to arterial repairs or venous reconstruction, fasciotomies were performed unless the injury had adequately decompressed all fascia compartments. The vascular reconstructions were covered with available local soft-tissue or muscle rotation flaps. Wounds were formally re-examined in the operating room, depending on the initial appearance of the wound and the ensuing clinical status of the patient. The medical treatment was standardized: Antibiotics commonly used were cloxacillin and ornidazole and were changed according to sensitivity patterns, and preventive antithrombotic treatment was performed with tedelparin.

On admission to the hyperbaric unit, patients were randomly assigned to receive HBO therapy or placebo. HBO therapy was performed in a multiplace hyperbaric chamber compressed with air at a pressure of 2.5 atmospheres absolute (ata). At this pressure, the patient breathed 100% oxygen via a facial mask. The HBO therapy protocol included 90 minutes oxygen breathing at 2.5 ata, twice daily, over 6 days. Placebo consisted of sessions in the hyperbaric chamber at a pressure of 1.1 ata in order to simulate compression and its effects on the ears, the patient breathing normal air via a facial mask. The placebo treatment included 90 minutes air breathing at 1.1 ata, twice daily, over 6 days. The surgeons and the patients were not informed of the treatment protocol (HBO therapy or placebo) performed during the study. Reevaluation of the injured extremities was carried out before each session, including examination of skin color, edema, motor and sensory function, and palpation of peripheral pulses. Wound dressings were performed in the surgical unit for all the patients included in the study.

The four primary study end points were wound healing without tissue necrosis requiring surgical excision; new major surgical procedures in relation to progressive and massive revitalization after entry in the trial, time of healing, and length of hospitalization.

In order to evaluate the effects of the treatment, transcutaneous oxygen pressure ($Ptco_2$) measurements were done by the use of miniature Clark electrodes (Siemens monitor, System Sirecust 400, Erlangen, Germany). Simultaneous readings were recorded from two sensors placed bilaterally on the extremities, mirror-like, on the site of skin injury. The electrodes were placed by the surgeon, at the end of the initial intervention, as near to the wound as possible, with the site remaining constant for all measurements. The measurements were done before (patient breathing ambient air) and during treatment exposure (HBO therapy or placebo) at the first, fourth, eighth, and twelfth sessions. A transcutaneous oxygen bilateral perfusion index (BPI) was calculated to quantify perfusion deficits by comparison with the uninjured (contralateral) limb as proposed by Kram.[22] The BPI is the ratio between the $Ptco_2$ of the injured limb and the $Ptco_2$ of the uninjured limb.

The study protocol was submitted and approved by the Institutional Review Board for human experimentation of the University Hospital of Angers. Written informed consent was obtained from the patient, a relative, or the patient's legally authorized representative.

Statistical Analysis

The criteria used to judge the outcome of the traumatized limb was wound healing without tissue necrosis requiring further surgery. Comparisons of qualitative data between the HBO group and the placebo group were made with the chi-squared test and the Fisher's exact probability test when an expected n value was less than five. Quantitative values were compared between groups using the Student's test. Paired or unpaired t tests were also used, as appropriate, for comparisons between $Ptco_2$ or BPI values. All data are presented as the mean ± standard deviation. A p value of less than 0.05 was considered significant.

RESULTS

A total of 36 patients were enrolled in the trial: 18 in the HBO group and 18 in the placebo group. In all patients neither signs of cerebral oxygen toxicity were observed nor were other adverse effects of pressurization. The HBO group and the placebo group were similar in terms of age (respectively 45.8 (±16.1) years and 51.5 (±20.9) years) and risk factors: diabetes mellitus (respectively two and four patients) and previous neurologic deficit (one patient in each group). Mechanisms of injuries and bones involved are summarized in Tables 2 and 3. No difference was found in the severity of fractures and soft-tissue injuries between the two groups (Table 4). Five patients in each group had crush injuries without bone lesions. Two patients in the HBO group had

TABLE 2. **Mechanisms of injury**

Groups	HBO	Placebo	Total
Motorcycle crashes	5	4	9
Motor vehicle crashes	3	4	7
Pedestrian accidents	1	2	3
Machine tools	2	2	4
Farm injuries	4	2	6
Other (fall, railway injury)	3	4	7

TABLE 3. **Bones involved**

Bones Involved	HBO	Placebo	Total
Tibia	9	8	17
Metatarsus	3	2	5
Radius and ulna	1	1	2
Metacarpus	—	2	2

TABLE 4. **Clinical data of the 36 patients with crush injuries on admission**

Groups	HBO (n = 18)	Placebo (n = 18)
Arterial injury	2	0
Neurologic injury	2	1
Fractures	13	13
Open fractures[a]		
Type II	5	5
Type III	6	8
Type IV	2	0
Soft-tissue injury[b]		
Grade II	4 (1)	7 (1)
Grade IIIA	9 (1)	8 (2)
Grade IIIB	3 (2)	3 (2)
Grade IIIC	2 (1)	0

Results were not different between the placebo group and the HBO group.

[a] For classification of open fractures, see ref. 9.

[b] Classification of soft-tissue injuries derived from Gustillo[10]; number of patients without fractures are in parentheses.

TABLE 5. **Stabilization procedures and skin flaps or grafts performed on admission, before randomization**

Skeletal fixation	HBO	Placebo	Total
External	1	3	4
Internal	9	9	18
Conservative (traction, cast)	3	1	4
Skin flaps or grafts	2	3	5

injuries of the cubital artery requiring end-to-end arterial repair in one case and vein graft in the other case. None of the patients was managed by primary amputation. Fracture stability was achieved initially in each case. Stabilization procedures were not different between the two groups (Table 5). Skin flaps and grafts were performed initially on two cases in the HBO group and on three cases in the placebo group. Type, location, or timing of surgical procedures were not statistically different between the two groups.

TABLE 6. **Patients characteristics by treatment outcome**

Groups	HBO (n = 18)	Placebo (n = 18)	*p*
Complete healing	17	10	0.009
Tissue necrosis	1	8	
New surgical procedures	2 (1 patient)	8 (6 patients)	0.03 (0.04)
Skin flaps and grafts	1	6	
Vascular surgery	1	0	
Amputation	0	2	
Wound dressings	15.8 (±9.4)	16.3 (±12.1)	0.45
Time of healing (days)	50.2 (±21.1)	55.8 (±19.9)	0.21

Data are presented as mean ± SD. The *p* values were obtained using the chi-squared test, the Fisher's exact test, and the Student's test.

Complete wound healing without tissue necrosis requiring surgical excision was obtained for 17 patients in the HBO group vs. 10 patients in the placebo group ($p < 0.01$; Table 6). A new surgical procedure was performed on one patient in the HBO group because of early ischemia of an initial flap coverage requiring debridement and coverage with a second flap and an end-to-side arterial anastomosis. Six patients in the placebo group were managed by repeated debridement because of progressive necrosis of tissues. They all had secondary flap coverage. Two patients developed flap loss affecting coverage of the fracture and required amputation. Repetitive surgical procedures were statistically higher in the placebo group compared to the HBO group (respectively 33.3 vs. 5.6%; $p < 0.05$). Because the patients were not matched for age and severity of injury before inclusion in the study, the results of the outcomes, taking into account these two criteria, are summarized in Table 7. In the subgroup of patients older than 40, with grade III soft-tissue injury, wound healing was obtained in seven patients (87.5%) in the HBO group vs. three patients (30%) in the placebo group ($p < 0.005$).

No significant differences were found in the time of healing and number of wound dressings between the two groups (Table 6) and between groups of patients matched for age and severity of injury. Length of hospitalization was similar in the two study groups: 22.4 (±12.4) days in the HBO group and 22.9 (±16.3) days in the placebo group, and in groups of patients matched for age and severity of injury. Characteristics of the patients requiring a new major surgical procedure are reported in Table 8.

TABLE 7. **Results of treatment in groups of patients matched for age and severity of trauma**

Age (years)	HBO Group				Placebo Group			
	< 40		≥ 40		< 40		≥ 40	
Soft-tissue injury[a]	Grade II	Grade III	Grade II	Grade III	Grade II	Grade III	Grade II	Grade III
Success	2	6	2	7	3	1	3	3
Failure	0	0	0	1	0	0	1	7
Totals	2	6	2	8	3	1	4	10

Success, wound healing; failure, tissue necrosis requiring surgical excision and/or a new surgical procedure.

[a] Classification of soft-tissue injuries derived from Gustillo.[10]

TABLE 8. **Characteristics of the patients requiring new surgical procedures**

Groups	HBO (n = 1)	Placebo (n = 6)
Age (years)	52	53.2 (±8.5) range 42–67
Diabetes mellitus	0	1
Fractures	1	4
Soft-tissue injury[a]		
Grade IIIA	—	3
Grade IIIB	—	3
Grade IIIC	1	0
Timing of new surgical procedures (days)	4	12.2 (±6.4) range 4–20

[a] Classification of soft-tissue injuries derived from Gustillo.[10]

During the study no severe infection appeared at the site of injury requiring changes of antibiotics or a new surgical procedure. There were no thromboembolic complications.

Systematic evaluation of the injured extremities before each session showed the following results: Edema was present, at the first session, in 12 patients in the HBO group and 11 patients in the placebo group; whereas, at the end of treatment, three patients in the HBO group and six patients in the placebo group had edema of the injured extremities. Cyanosis was present initially in four patients in the HBO group and in five patients in the placebo group. During the following HBO therapy sessions, only one patient had persistent skin cyanosis; whereas, in the placebo group two additional patients developed skin cyanosis at the injury site ($p < 0.05$). No pulse deficit was noted during the study protocol.

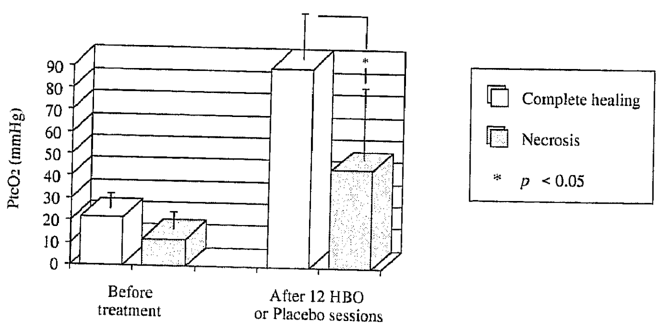

FIG 1. **$Ptco_2$ of the injured limbs at 1 ata. Bars represent mean ± SD. $Ptco_2$ values increased significantly from the first to the twelfth sessions in patients with complete healing ($p < 0.001$).**

In normal air, before the first session, the $Ptco_2$ values in the traumatized limbs were not statistically different between the HBO group (15.3 ± 6.6 mm Hg) and the placebo group (18.5 ± 5.7 mm Hg), and between patients with complete healing and with failed healing (Fig. 1). In patients with complete healing, the $Ptco_2$ values of the traumatized limb, measured in normal air at 1 ata, rose significantly between the first and the twelfth sessions (respectively 21.6 (±5.7) mm Hg and 90 (±20.1) mm Hg; $p < 0.001$). No significant change in the $Ptco_2$ value was found for the patients who failed to heal. At the twelfth session the $Ptco_2$ values were statistically lower in patients who failed to heal (Fig. 1).

The BPI (BPI = $Ptco_2$ of the injured limb/$Ptco_2$ of the uninjured limb) in normal air was not different, before the first session, between the HBO group (0.3 ± 0.1), and the placebo group (0.4 ± 0.1). At the first HBO session, the BPI increased significantly from 1 ata air to 2.5 ata O_2 (respectively 0.4 ± 0.1 vs. 0.6 ± 0.1; $p < 0.05$). During the following HBO sessions, in patients with complete healing, the BPI at 2.5 ata O_2 was constantly greater than 0.9, whereas the BPI in air rose progressively between the first and the twelfth sessions (from 0.4 (±0.1) to 0.8 (±0.1); $p < 0.05$). At the twelfth session, the BPIs were not different at 1 ata air and 2.5 ata O_2 (Fig. 2). In patients with failed healing in the placebo group, the BPI did not change significantly between the first and the twelfth sessions (respectively 0.3 (±0.2) vs. 0.3 (±0.1)). In the HBO group, values of the BPI, in normal air, in the patient with failed healing were 0.36 before the first session, 0.65 before the fourth session, and 0.19 at the eighth session (fourth day of inclusion in the study).

One patient in each group required a new surgical procedure at the fourth day of the treatment protocol (Table 7). The re-evaluation of the injured extremities at each session was helpful for these two patients. It is worth noting that these two patients had low initial $Ptco_2$ values that did not increase during the HBO or placebo sessions, whereas the common clinical signs were absent.

DISCUSSION

Ischemia of soft tissue in crush syndrome remains a difficult condition to treat, and tissue oxygenation is a difficult component to monitor clinically. In this randomized placebo-

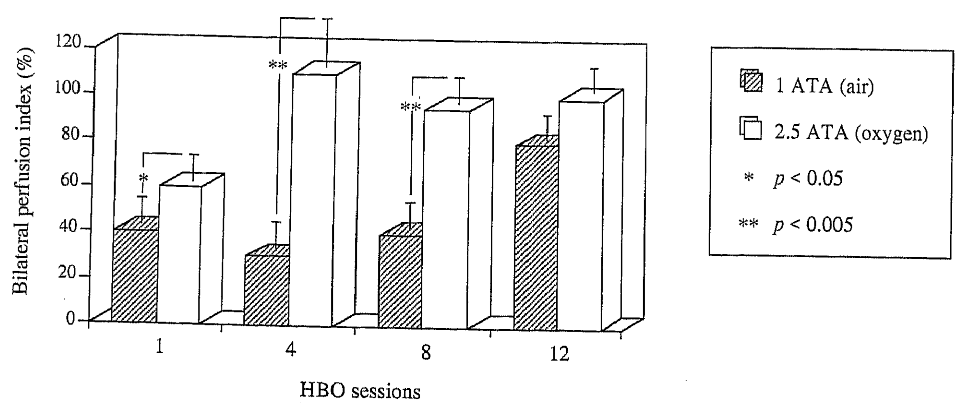

FIG 2. **Changes in BPI in patients with complete healing in the HBO group. Bars represent mean ± SD.**

controlled study, HBO therapy was used in an attempt to improve the viability of injured tissue after initial surgical management. This trial shows that HBO therapy improves wound healing and reduces repetitive surgery with results significantly different from the placebo in patients older than 40 with grade III soft-tissue injury. Lack of differences between the HBO group and the placebo group, and between subgroups of patients matched for age and severity of injury, in terms of time of healing and length of hospitalization, might be explained by the fact that radical intervention such as amputation in the placebo group eliminates the time of the progressive healing process.

The immediate effect of HBO is hyperoxygenation. For example, a 10-fold oxygen tension in blood is achieved at 2 ata.[11] Hyperoxia has beneficial effects through four mechanisms. First, hyperoxia increases delivery of oxygen to ischemic tissue. Survival of tissues is directly dependent on oxygen tension.[23] The diffusion distance of oxygen through tissue fluids is increased 3-fold by HBO.[13,24] Thus, in the area with damaged microcirculation, as a result of crush injury, the higher diffusion of oxygen to the tissue allows sufficient oxygen tension to avoid tissue ischemia. However, HBO may help compromised tissue to survive only if no occlusion of major arterial vessels occurs, because tissue oxygen delivery is directly dependent on blood flow.[25] It is therefore essential that effective perfusion persist in order to sustain increased oxygen transport, explaining the protective effect of HBO in acute ischemia of the limbs.[26,27] Second, hyperoxygenation has a direct vasoconstricting effect.[28,29] Vasoconstriction results in a diminution of edema in ischemic tissues by reduction of the capillary transudation flow rate.[14] Microcirculation is improved as the interstitial pressure is reduced. In an experimental model of canine compartment syndrome, HBO reduced the formation of edema by 20% in injured muscles as well as muscular necrosis estimated on the uptake of technetium-99m pyrophosphate.[15,30] Third, hyperoxia acts against infection in different ways. HBO induces the production of toxic oxygen radicals that have a direct lethal effect on strict anerobic organisms such as Clostridia species.[30] This direct bactericidal effect is of little importance on aerobic microorganisms. The major effect of oxygen on infection is its role in bacterial host defence. Hyperoxia enhances the oxygen-dependent intracellular killing mechanisms of the polymorphonuclear leukocytes and affects bacterial clearance.[32] An oxygen tension of 45 mm Hg reverses the depressed white blood cell–killing capacity in infected ischemic tissues.[33] Last, oxygen is an important factor in wound healing. Hyperoxygenation is mandatory to promote collagen production by fibroblasts whose functions are altered when an inadequate oxygen tension of less than 10 mm Hg is present in the ischemic area.[34] HBO enhances fibroblast differentiation, collagen synthesis,[18] and angiogenesis,[35] leading to increased wound closure rate in hypoxic tissues.[36]

Clinical studies indicating the use of HBO therapy in the surgical management of severe limb injuries are limited. The first case report considering the beneficial adjunct of HBO[19] was followed by small series of patients where HBO was considered useful in 60 to 85% of cases.[1,17,20] Indications considered for HBO involved vascular injuries,[1,37] soft-tissue injuries, and anerobic infection associated with open fractures.[17,20] In some cases HBO reversed a desperate ischemic situation despite earlier successful vascular repair.[1] A recent study reported an improvement in prognosis of post-traumatic acute ischemia in the lower extremities after reconstructive surgery.[21] Complete limb salvage was accomplished in 8 out of 13 cases and in 3 cases the level of amputation was lowered.[21] In our study, HBO, in patients older than 40, clearly reduces the need for repetitive and aggressive debridement of tissues compromised by progressive necrosis. Similar results were reported concerning necrotizing fasciitis.[38] HBO therapy started as soon as possible stimulates the regenerative potential of the microcirculation in compromised but still viable tissues. Moreover, in advanced situations with start of distal necrosis, HBO therapy may be helpful to separate viable from nonviable tissues and to limit surgical excision.[5]

Surgery of complex fractures may disturb local soft-tissue vascularization. In addition, the presence of metallic osteosynthesis material represents a risk factor of infection. The choice of the fixation device depends on the type and location of the fracture, the severity of soft-tissue damage and subsequent soft-tissue coverage requirement. External fixation is used generally in massive soft-tissue injury to achieve fracture stability in conjunction with screws.[10] The site of the fixation may interfere with the possibilities of flap coverage. HBO could limit the soft-tissue consequences of surgical procedures more aggressive than expected, due to difficulties encountered during intervention. While avoiding soft-tissue necrosis, HBO prevents secondary exposure of joints, fractures, blood vessels, and neural structures, which radically modifies trauma prognosis.[5]

Certainly not every patient with trauma of the extremities requires HBO therapy. In this study the benefits of HBO therapy for improving healing and limb survival are in close relationship with patient age and severity of the injury. It is well known that elderly patients, compared with younger persons similarly injured, have worse prognoses. The results of the present study suggest that HBO therapy, in the group of patients over 40 years old with the most severely injured limbs, is a useful adjunct in the management of crush injuries. HBO therapy should be reserved for those patients with recognized risk factors, clinical compromise, or a history of impaired wound healing.

Efficient evaluation of tissue oxygenation is necessary to supply information concerning the degree of ischemia of the tissue immediately adjacent to the injury. To measure intratissue oxygen tension, one may use invasive methods such as intramuscular electrodes or noninvasive procedures such as transcutaneous pressure electrodes that reflect oxygen tension of tissues located just below the electrodes.[22] Transcutaneous oxygen pressure is linked to oxygen delivery, which is the result of oxygen content and blood flow.[39] Any limitation of local blood flow such as arterial disruption, thrombosis, or external compression of vessels leads to a reduction in $Ptco_2$. This noninvasive method of exploration was validated during HBO therapy.[40] The interest in measuring transcutaneous pressures in HBO has been underscored in treated patients with posttraumatic limb ischemia to predict the final outcome of the limb.[37] It is possible to predict with 100% sensitivity and 94% specificity whether or not secondary amputation of the traumatized limb should be performed. Moreover, during HBO therapy $Ptco_2$ monitoring seems to be useful to evaluate the evolution of the traumatized limb. In this study, at the end of the treatment, the $Ptco_2$ values were statistically lower in patients with failed healing. The increase of the BPI, as early as the first HBO session, highlights the effectiveness of HBO therapy. As the HBO therapy continued, the BPIs in air rose progressively to reach normal values, similar to those achieved during HBO, showing the re-establishment of normal tissue perfusion and oxygenation. These results suggest that HBO therapy improves tissue viability in two ways. HBO, diminishing edema, permits blood flow to improve, and increasing arterial oxygen content permits oxygen delivery to the tissue to increase. Moreover, when there is a sudden drop in $Ptco_2$ values, re-evaluation of the injured limb becomes imperative. Thus, this study shows that transcutaneous oxygen monitoring is a valuable method of estimating the response to HBO therapy in patients with crush injuries of the limbs.

In conclusion, this study demonstrates that HBO therapy is a useful therapeutic adjunct in the management of severe trauma of the limbs in older patients with grade III soft-tissue injuries. HBO improves wound healing and reduces repetitive surgery necessary in cases of aggravation of crushing tissue damage. At the time of initial assessment, limb viability may be expected to improve with the conjunction of surgical procedures and HBO therapy, sparing extensive excision of damaged soft tissue or primary amputation. According to the results of this clinical trial, HBO therapy should be recommended in the management of severe crush injuries in patients over 40 years old.

REFERENCES

1. Schranek A, Hashmonai M: Vascular injuries in the extremities in battle casualties. *Br J Surg* 64:644, 1977
2. Cianci P, Sato R: Adjunctive hyperbaric oxygen therapy in the treatment of thermal burns: A review. *Burns* 20:5, 1994
3. Kindwall EP: Hyperbaric oxygen's effect on radiation necrosis. *Clin Plast Surg* 3:473, 1993
4. Baroni G, Porrot T, Faglia E, et al: Hyperbaric oxygen in diabetic gangrene treatment. *Diabetes Care* 10:81, 1987
5. Strauss MB: Role of hyperbaric oxygen therapy in acute ischemias and crush injuries: An orthopedic perspective. *HBO Review* 2:87, 1981
6. Drapanas T, Hewitt RL, Weichert RT, et al: Civilian vascular injuries. A critical appraisal of three decades of management. *Ann Surg* 172:351, 1970
7. Keeley SB, Sydner WH, Weigelt JA: Arterial injuries below the knee: Fifty one patients with eighty two injuries. *J Trauma* 23:285, 1983
8. Lange RH, Bach AW, Hansen ST, et al: Open tibial fractures with associated vascular injuries: Prognosis for limb salvage. *J Trauma* 25:203, 1985
9. Byrd HS, Spicer TE, Cierny G: Management of open tibial fractures. *Plast Reconstr Surg* 76:719, 1985
10. Gustillo RB, Mendoza RM, Williams DN: Problems in the management of type III (severe) open fractures: A new classification of type III open fractures. *J Trauma* 24:742, 1984
11. Basset BE, Bennett PB: Introduction to the physical and physiological bases of hyperbaric therapy. In Davis JC, Hunt TK, (eds): *Hyperbaric Oxygen Therapy*. Bethesda, Md, Undersea Medical Society, 1977, pp 11–24
12. Nylander G, Nordstrom H, Lewis D, Larsson J: Metabolic effects of hyperbaric oxygen in postischemic muscle. *Plast Reconstr Surg* 79:91, 1987
13. Bowersox JC, Strauss MB, Hart GB: Clinical experience with hyperbaric oxygen therapy in the salvage of ischemic skin flaps and grafts. *J Hyperbaric Med* 1:141, 1986
14. Nylander G, Lewis D, Nordstrom H, Larson J: Reduction of postischemic edema with hyperbaric oxygen. *Plast Reconstr Surg* 76:595, 1985
15. Strauss MB, Hargens AR, Gershuni D, et al: Reduction of skeletal muscle necrosis using intermittent hyperbaric oxygen in a model of compartment syndrome. *J Bone Jt Surg* 65A:656, 1983
16. Demello FG, Haglin JJ, Hitchcock CR: Comparative study of experimental *Clostridium perfringens* infection in dogs treated with antibiotics, surgery and hyperbaric oxygen. *Surgery* 73:936, 1973
17. Loder RE: Hyperbaric oxygen therapy in acute trauma. *Ann R Coll Surg Engl* 61:472, 1979
18. Hunt TK, Nunikoski J, Zederfeldt BH, Silver IA: Oxygen in wound healing enhancement: Cellular effects of oxygen. In: Davis JC, Hunt TK (eds): *Hyperbaric Oxygen Therapy*. Bethesda, Md, Undersea Medical Society, 1977, pp 111–122
19. Smith G, Stevens J, Griffiths JC, et al: Near avulsion of foot treated by replacement and subsequent prolonged exposure of patients to oxygen at two atmospheres pressure. *Lancet* 2:1122, 1961
20. Szekely O, Szanto G, Takats A: Hyperbaric oxygen therapy in injured subjects. *Injury* 4:294, 1973
21. Shupak A, Gozal D, Ariel A, et al: Hyperbaric oxygenation in acute peripheral post-traumatic ischemia. *J Hyperbaric Med* 2:7, 1987
22. Kram HB, Shoemaker WC: Diagnosis of major peripheral arterial trauma by transcutaneous oxygen monitoring. *Am J Surg* 147:776, 1984
23. Gottrup F, Firmin R, Hunt TK, Mathes SJ: The dynamic properties of tissue oxygen in healing flaps. *Surgery* 95:527, 1984
24. Pierce EC II. Pathophysiology, apparatus and methods, including the special techniques of hypothermia and hyperbaric oxygen. In Pierce EC II (ed): *Extracorporeal Circulation for Open Heart Surgery*. Springfield, Ill, Charles C Thomas, 1969, pp 83–84
25. Guyton AC, Ross JM, Corrier D, et al: Evidence for tissue oxygen demand as the major factor causing autoregulation. *Circ Res* 15(suppl):60, 1964
26. Meijne NG: Flow distribution changes during extracorporeal circulation at two atmospheres absolute. In Ledingham IMCA (ed): *Hyperbaric Oxygenation*. Edinburgh, Livingston, 1965, pp 136–148
27. Wang MCH, Reich T, LesHo WH, et al: Hyperbaric oxygenation: Oxygen exchange in an acutely ischemic vascular bed. *Surgery* 59:94, 1966
28. Bird AD, Telfer ABM: Effect of hyperbaric oxygen on limb circulation. *Lancet* 1:355, 1965
29. Sullivan SM, Johnson PC: Effect of oxygen on blood flow autoregulation in cat sartorius muscle. *Am J Physiol* 241:H807, 1981
30. Skyhar MJ, Hargens AR, Srauss MB, et al: Hyperbaric oxygen reduces edema and necrosis of skeletal muscle in compartment syndromes associated with hemoraghic hypotension. *J Bone Jt Surg* 68A:1218, 1986
31. Hill GB, Osterhout S: Experimental effects of hyperbaric oxygen on selected *clostridial* species. *In vitro* studies. *J Infect Dis* 125:17, 1972

32. Knighton DR, Halliday B, Hunt TK: Oxygen as an antibiotic. A comparison of the effects of inspired oxygen concentration and antibiotic administration on in vivo bacterial clearance. *Arch Surg* 121:191, 1986
33. Mader JT, Brown GI, Guckian JC, et al: A mechanism for the amelioration by hyperbaric oxygen of experimental staphylococcal osteomyelitis in rabbits. *J Infect Dis* 142:915, 1980
34. Hunt TK, Pai MP: The effect of varying ambient oxygen tensions on wound metabolism and collagen synthesis. *Surg Gynecol Obstet* 135:561, 1972
35. Manson PN, Im MJ, Myers RAM: Improved capillaries by hyperbaric oxygen in skin flaps. *Surg Forum* 31:564, 1980
36. Kivisaari J, Niiniskoski J: Effects of hyperbaric oxygenation and prolonged hypoxia on the healing of open wounds. *Acta Chir Scand* 141:14, 1975
37. Mathieu D, Wattel F, Bouachour G, et al: Post-traumatic limb ischemia. Prediction of final outcome by transcutaneous oxygen measurements in hyperbaric oxygen. *J Trauma* 20:307, 1990
38. Riseman JA, Zamboni WA, Curtis A, et al: Hyperbaric oxygen therapy for necrotizing fasciitis reduces mortality and the need for debridements. *Surgery* 108:847, 1990
39. Shoemaker WC, Vidyssagar D: Physiological and clinical significance of $Ptco_2$ and $Ptcco_2$ measurements. *Crit Care Med* 9:698, 1981
40. Sheffield PJ, Workman WT: Noninvasive tissue oxygen measurements in patients administered normobaric and hyperbaric oxygen by mask. *Hyperbaric Oxygen Rev* 6:47, 1985