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# Hyperbaric Oxygen Therapy in Plastic Surgery

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**ABSTRACT**
**Objective:** In this review the literature aims to familiarize the reader with several of the more common plastic surgery problems and to review the appropriate use of HBO for each condition. Plastic surgery is a broad specialty encompassing a variety of areas including the treatment of traumatic soft tissue injury, burn care, hand surgery, microsurgery and limb replantation, problem wound management, flap reconstruction, cosmetic surgery, and congenital deformity. In recent years an emphasis on less anecdotal and more scientific research has provided a better understanding of the role of hyperbaric oxygen therapy (HBO) for many plastic surgical conditions. Patient selection remains the key to successful outcome. The rationale for HBO treatment of a specific plastic surgery problem should be based on scientific research if possible. If supportive research is not available or applicable to a specific case, then the decision to treat should be based on sound physiological principles.
**Keywords:** hyperbaric oxygen, plastic surgery, applications.

## INTRODUCTION

The most important effects of hyperbaric oxygen (HBO), for the surgeon, are the stimulation of leukocyte microbial killing, the enhancement of fibroblast replication, and increased collagen formation and neovascularization of ischemic tissue. Preoperative hyperbaric oxygen induces neovascularization in tissue with radionecrosis. Refractory osteomyelitis and necrotizing fasciitis appear to respond to adjunctive hyperbaric oxygen. Crush injury and compartment syndrome appear to benefit through preservation of ATP in cell membranes, which limits edema. Hyperbaric oxygen in burn injury permits shorter hospital stays, a reduced number of surgeries, and less fluid replacement. Skin grafts and flaps are reported to take more completely and more rapidly. The same mechanisms may apply in ischemic problem wounds such as infected diabetic extremities. Contraindications and side effects are described. Hyperbaric oxygen will not heal normal wounds more rapidly but may, under certain circumstances, induce problem wounds to heal more like normal ones.Here we will discuss the role of HBO in different palstic surgery issues:

### Acute traumatic ischemia

High-energy trauma to the extremities may be associated with major arterial injury or compartment syndrome resulting in varying degrees of tissue ischemia. Perhaps the most devastating form of acute traumatic ischemia is limb amputation.

The primary treatment for these injuries is restoration of blood flow in an attempt at limb salvage. Even if surgical revascularization is successful, reestablishment of blood flow may actually cause accelerated tissue damage due to reperfusion injury [1].

This is particularly true in cases of limb replantation in which the reperfused extremity has a large muscle mass. Muscle is very sensitive to ischemia-reperfusion (I-R) injury and some degree of permanent necrosis will occur with ischemia times greater than 4 h.

Concern over use of HBO in the face of 1-R injury revolved around the hypothesis that providing extra oxygen would increase free radical production and tissue damage. This concern has been resolved by recent studies which have shown that HBO actually antagonizes the ill effects of I-R injury in a variety of tissues [2] . Evaluation in a rat axial skin flap model of I-R injury has demonstrated that HBO treatment immediately upon reperfusion significantly improved skin flap survival following 8 h of global ischemia . This finding was opposite this author's original hypothesis that HBO would exacerbate reperfusion injury.

In a follow-up study the same skip flap model was used to show that HBO therapy increased microvascular blood flow during reperfusion compared with untreated ischemic controls. Subsequent models of I-R injury have also demonstrated improved blood flow and capillary density with HBO treatment .

Studies have shown that HBO treatment during reperfusion significantly improved the survival of free skin flaps following microvascular reattachment and ischemia times of up to 24 h. The skin flap studies have been corroborated by skeletal muscle experiments,



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which are more important from a clinical point of view in traumatic ischemias, due to the sensitivity of skeletal muscle to I-R injury. Early studies demonstrated a beneficial effect of HBO on ischemic skeletal muscle by reduction of edema and necrosis in rat hindlimb and dog compartment syndrome models.

**Compromised flabs**

The three keys to successful treatment of compromised flaps with HBO are (a) an accurate diagnosis of the specific flap problem, (b) the timing of the initiation of hyperbaric oxygen treatment, and (c) uninhibited communication between surgeon and hyperbaric physician [3] . In order for the hyperbaric physician to properly select and appropriately treat a compromised flap, an awareness of the different etiologies of flap compromise is necessary. A random flap with distal necrosis is completely different from a free flap with total venous occlusion; the etiology of compromise is different and so should be the treatment.

The use of HBO therapy is appropriate only if the following criteria are met:

1. The flap problem must be defined
2. There must be some documented perfusion of the flap
3. The treatment should make physiological sense and be based on a knowledge of research and clinical experience
4. Treatment should be initiated only after appropriate surgical measures of salvage have been considered
5. The time to the initiation of treatment should be minimal

A wealth of literature evaluating the effect of hyperbaric oxygen in compromised flaps has been published. Generally the majority of these articles have shown a beneficial response. The problem is that most of the published experimental studies evaluated random flap necrosis and generally showed a 15-30% improvement in survival with HBO compared with no treatment.

With the advances in flap design and knowledge of blood supply, random flaps are rarely used in modern plastic surgery, due to the availability of axial flaps and free tissue transfer. The fact that the random flaps in most animal studies are intentionally elevated at a length that exceeds the known blood supply in order to obtain necrosis brings into question the clinical relevance of these experiments.

A plastic surgeon would not intentionally elevate a flap longer than its width would allow and count on adjunctive means to help the distal part of the flap survive .

Only in recent years has animal research evaluating the effect of HBO on axial flaps come to light. Although this recent information has contributed significantly to the understanding of the role of HBO in current flap surgery, there is still a need for further research to fully understand the effect, mechanism and timing of RBO treatment. The remainder of this section focuses on the appropriate use of RBO for specific flap problems based on research, physiological principles, and this author's experience. The timing of treatment and protocols used is also discussed.

**Random ischemia**

Random flap necrosis almost invariably occurs at the distal end

of the flap. An axial flap that is inadvertently elevated at a length that exceeds the blood supply of a named artery will create the situation where a random extension of an axial flap is produced.

Loss of this distal random portion of the flap, which is often the most critical part of the flap, can be minimized by HBO' treatment [4] . Random ischemia is usually identified within the first 24 h by a progressively dusky appearance that becomes fairly well demarcated and may be associated with epidermolysis.

This may not be recognized until 24-48 h after surgery. Hyperbaric oxygen therapy should be initiated as soon as visible signs of random compromise are noticed.

**Wound healing**:

The rationale for the use of hyperbaric oxygen therapy for problem wounds stems from its beneficial effects on the wound microenvironment.

Animal studies have shown that elevation of oxygen tension in a hypoxic wound stimulates fibroblast proliferation and collagen synthesis, enhances the leukocyte killing and bacterial clearance of infected wounds, and promotes epithelialization .

Although a moderate degree of wound hypoxia provides a stimulus for angiogenesis, it was shown that it is actually the oxygen gradient at the wound site that provides the stimulus [5] .

The most common wounds referred for HBO treatment are diabetic foot ulcers. Less common wound referrals include venous stasis ulcers, nondiabetic arterial insufficiency wounds, decubitus ulcers, and vasculitic wounds. Although all of the above are considered problem wounds, each type has a different etiology and is subject to unique management principles [6] .

Problem wounds associated with connective tissue diseases represent a special class of ulcers in which adjunctive HBO appears to be beneficial. These wounds are often long standing, painful, slow to granulate, and refractory to usual wound care measures. Although a few case reports have appeared illustrating successful use of HBO for pyoderma gangrenosum , information about connective tissue related ulcers is generally sparse.

The presence of some supportive clinical research in this group of ulcers warrants a trial of HBO treatment especially in wounds without deep structure exposure as a last -ditch effort to try to stimulate granulation tissue and healing prior to proceeding with amputation.

**Burn injury**

Acute thermal injury is characterized by a central zone of coagulation (necrosis), an intermediate zone of stasis (ischemia), and a peripheral zone of erythema (inflammation). The burn wound is dynamic in that progression from stasis to coagulation can occur up to 48 h after injury. In addition to local changes, a generalized capillary leak occurs resulting in intravascular depletion and total body edema formation[7].

The rationale for the use of HBO for burn injury comes from animal studies, which have shown that this treatment maintains the integrity of the microcirculation, reduces fluid extravasation and generalized edema, and preserves ATP levels and tissue viability [8].

In a recent study **Bucky**, *et al*. used a rabbit model of burn injury to show that use of a monoclonal antibody, which blocks CDI8-mediated neutrophil adhesion, improved re-epithelialization and reduced progressive tissue destruction in the zone of stasis.

This may provide another mechanism for HBO in burn injury, because it is known from ischemia-reperfusion studies that HBO inhibits neutrophil adhesion function which resulted in improved microcirculatory flow.

Retrospective studies have not shown a significant reduction in mortality with the use of HBO in larger burns [9].

Future studies should also identify specific populations of burn injury patients that may or may not benefit from HBO with regard to body surface area and depth with and without inhalation injury.

The HBO protocol for adjunctive HBO treatment of burn injury as outlined by the UHMS Committee report should be followed.

**Low arterial inflow**

Situations of low arterial inflow occur in pedicle and free flaps when the artery is subject to intermittent vasospasms. This situation can also occur if edema in tissues around the pedicle is partially obstructing inflow or if a hematoma around the pedicle is present. The latter presents clinically different from the vasospasm as a pale pink color, and slow capillary refill is persistent, rather than intermittent [10] .

The primary treatment for this problem is surgical reexploration to insure that a hematoma, anatomical kinking, or twisting of the pedicle is not present. If a surgical condition for the compromise is not found and the flap continues to show signs of low arterial inflow, then HBO treatment is indicated.

In vasospasm situations, even although hyperbaric oxygen has the potential for vasoconstriction within the chamber, the author feels that the beneficial effects of oxygenating the compromised tissue outweigh this potential effect. The treatment protocol is similar to that for random ischemia.

**Total arterial occlusion**

Total arterial occlusion is usually easily recognized by a pale color of the flap that demonstrates absent capillary refill. This can occur from intraoperative damage to the arterial pedicle of an axial flap, or may result from mechanical compression of the artery (hematoma), or anatomical kinking or twisting of the pedicle.

Total arterial occlusion can also occur following free tissue transfer in which the arterial anastomosis becomes thrombosed. Again, the primary treatment of this condition is immediate surgical reexploration. Primary HBO treatment without surgical reexploration is not indicated. If the arterial problem is surgically corrected and the flap has had a period of warm ischemia [11] , then HBO treatment is indicated as soon as possible after reestablishment of arterial inflow.

Hyperbaric oxygen should be used to treat any free flap complicated by arterial thrombosis that

requires revision anastomosis and is associated with a secondary ischemia time.

**Partial venous congestion**

Partial venous congestion is the most common etiology of compromise in
pedicle axial flaps. Again, the usual reasons for partial venous occlusion must be ruled out including compression on the pedicle from hematoma, anatomical kinking, and twisting of the pedicle vein. Often, however, the inherent venous drainage, of an axial flap is inadequate due to the choke system between the capillary beds. In addition, flaps such as the reverse radial forearm flap have a reverse venous drainage, which creates a transient congestion of the flap until the venous outflow can be established through branches and valve blowout of the venacommicantes. Primary treatment for venous congestion in the latter situations is medicinal or chemical leeching.

The addition of HBO can be a useful adjunctive treatment to leeching to help oxygenate and support the flap until pedicle or peripheral venous channels can be reestablished [12] , which usually takes 7-10 days.

Animal experiments evaluating the effect of HBO in situations of partial venous congestion are nonexistent.

**Total venous occlusion**

Total venous occlusion in pedicle flaps can occur from the previously mentioned reasons including hematoma compression, twisting, and kinking of the pedicle.

With regard to free tissue transfer, total venous occlusion by vessel thrombosis is the most common cause of flap failure. It is often the venous anastomosis that thromboses first which backs up the microcirculation and results ultimately in arterial thrombosis and complete necrosis of the flap. The use of HBO alone for total venous occlusion is ineffective.

This has been demonstrated in a rat axial skin flap model of total venous occlusion where the administration of HBO did not alter the 100 % necrosis of the flap . This makes sense because it is known that the effects of HBO are systemic and require blood flow to the injured tissue in order to be effective.

Further research is needed, however, to determine whether HBO can be effective in situations of partial venous occlusion. The primary treatment for total venous occlusion is surgical reexploration. However, in situations where patient deterioration or refusal to reoperation are present, then leeching to provide venous outflow, provided that the artery remains patent in combination with HBO therapy, may result in flap salvage.

**Free tissue transfer**

Hyperbaric oxygen therapy is extremely valuable in the salvage of compromised free tissue transfers. Free flaps are axial flaps, and therefore necrosis often results from a long primary ischemia time or any secondary ischemia time [13] . The necrosis is generally the result of I-R injury, which has already been discussed. This injury is usually heterogeneous (rather than global) and reversible in the early stages.

Research in axial skin flap models of ischemia reperfusion injury have shown that HBO significantly improves survival when administered before and during reperfusion following 8 h of ischemia . This experience has also been demonstrated in animal free-flap models . In a clinical study the salvage of free flaps with secondary ischemia times was significantly enhanced by HBO treatment . This becomes very important to the microsurgeon, because even in the best of hands the accepted pedicle thrombosis rate is 5 -10 %.

This often results in a desperate situation, because a secondary ischemia time has occurred prior to revision anastomosis. Necrosis of a free tissue transfer is a significant loss, because the defect, which the free flap was used to close, is recreated along with the donor-site morbidity.

Free flaps compromised by prolonged primary or secondary ischemia have responded dramatically to HBO treatment with 100 % viability in most cases if the time to the initiation of treatment is minimal [14] . Patient selection includes free tissue transfer with a primary ischemia time of greater than 3-4 h especially if a composite flap with muscle is transferred. Any free flap which has to be reexplored to treat a vessel thrombosis and has an associated secondary ischemia time should be treated with HBO.

**Grafts**

Although skin grafts are often classified together with compromised flaps, i. e. "grafts and flaps;' they are completely different from a physiological point of view. All flaps have an inherent blood supply whereas a skin graft is avascular tissue that depends on the quality of the recipient bed for revascularization. The application of HBO to split thickness skin

grafting is to treat the recipient wound in order to provide adequate granulation to support the graft [15].

Unlike treating a compromised flap, which is often after the fact, the goal of successful skin grafting is preparing a hypoxic compromised recipient bed prior to placement of the graf [16] . The only controlled clinical study by Perrins and Cantab revealed a significant improvement in skin graft survival from 17 to 64 % with the addition of HBO treatment . It is unclear, however, why the overall graft survivals in this study were so low.

**Radiation tissue damage:**

The beneficial effects of HBO on radiated tissue have been demonstrated by Marx in both animal and clinical studies . The physiological basis for this beneficial effect is the promotion of angiogenesis in radiated tissue where the capillary bed is damaged. Small radiation-induced soft tissue ulcers can often be healed with HBO alone. If the radiation ulcer is large and will require a flap for closure, then the use for HBO depends on the amount of ulcer excision.

If the entire ulcer and radiated tissue can be excised and a vascularized flap is used to close the defect, then HBO is unnecessary. Often, however, the flap is placed to cover an excised radiated ulcer and sutured to radiated tissue. In this instance pre and postoperativeHBO therapy is critical to promote healing of the flap tissue to the surrounding radiated tissue.

The HBO therapy in this situation becomes invaluable to the plastic surgeon, because improving the vascularity to the radiated tissue allows a lesser debridement and resection of the radiated ulcer . This in turn allows for a smaller flap and reduces the associated donor morbidity [16].

**Necrotizing infections**

Necrotizing infections include a wide range of named diseases including gas gangrene, clostridial myonecrosis, Fournier's gangrene, necrotizing fascitis, Meleney's gangrene, anaerobic necrotizing cellulitis, etc. This can seem quite confusing especially when all of these conditions are variants of the same disease process.

From a practical point of view it makes more sense to classify necrotizing infections based on three criteria that are known to affect mortality:

(a) Bacteriology.(b) anatomy of the tissue involved, either skin, subcutaneous tissue, fascia or muscle, or a combination of all; and (c) the location of the disease process. This allows exact classification on an anatomical basis that is easily understood by all physicians involved in the treatment of this disease.

Despite a more aggressive surgical approach and advances in antibiotic therapy, reported mortalities from necrotizing infections remain as high as 75 % in some series.

Important components of care include aggressive nutritional support, meticulous wound care, and appropriate methods for wound closure, as well as HBO therapy [17,18] .

Infected tissue is hypoxic, which provides an environment for growth of anaerobic bacteria.

Hyperbaric oxygen, in addition to a direct inhibitory effect on certain bacteria, raises the tissue oxygen tension and enhances host responses such as neutrophil phagocytosis.A study of [19], showed that combination treatment with HBO and penicillin significantly reduced mortality and colony forming units per gram of muscle compared with penicillin therapy alone.

A prospective randomized clinical trial would be necessary to absolutely evaluate the effect of HBO on necrotizing infections [19].

**Summary**

The information in this review should provide a template regarding the use of HBO therapy by the practicing plastic surgeon and hyperbaric physician.

Hyperbaric oxygen therapy should no longer be viewed as a skeptical treatment modality, but rather "the plastic surgeon's friend" for a variety of different clinical situations.

Although HBO research in recent years has become more scientific and less anecdotal, there is still a need for further experimental and prospective clinical studies to more accurately define and confirm the specific role of HBO therapy for many plastic surgery conditions.

New frontiers currently being investigated including the effect of

HBO on peripheral nerve regeneration , electrical burn injury

various types of flap compromise, and mechanisms of ischemia-reperfusion injury all provide exciting potential future applications in the specialty of plastic surgery.

**The study was done according to the ethical board of King Abdulaziz university.**

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