

## Treatment of Ulcers on the Legs With Hyperbaric Oxygen

BOGUSLAV H. FISCHER, M.D.

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*Skin ulcerations present in patients being treated for other conditions with hyperbaric oxygen were noted to heal at an accelerated rate. This observation led to the concept of topical exposure to hyperbaric oxygen as a treatment of intractable cutaneous ulcers.*

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THIRTY PATIENTS, ranging in age from eight to ninety-five years with lesions that had been present from 15 days to six years, were treated with hyperbaric oxygen for ulcers on the leg. The majority had been previously treated by methods which had failed to improve or heal their conditions. The patients did not receive preferential treatment, attention, or care during oxygen therapy. Total healing was achieved in twenty eight patients. In two patients, hyperbaric oxygen treatment resulted in improvement of the ulcerations with suppression or elimination of pain, but failed to heal the lesions entirely. The treatment was very well tolerated by all patients and no untoward effects were noted.

A controlled study was performed in one case since the patient presented almost identical lesions on both feet. One foot was treated conventionally and the other foot was exposed to hyperbaric oxygen. The control side was subsequently exposed to hyperbaric oxygen. However, most of the patients served as their own controls since each had a history of past treatment with no improvement.

A hermetic chamber with controlled pressure sealing was constructed to allow uninterrupted exposure of the infected area to pure oxygen. A pressure not exceeding 22 mm Hg (1.03 atmosphere absolute) was applied to avoid obstruction of capillary blood flow (Figure 1). The oxygen drive was

maintained at four liters/minute with continuous humidification and discharged to free atmosphere. Oxygen treatment was performed twice daily, each session lasting from two to three hours. Normal saline dressing was applied during the oxygen-off hours. After the second or third day of oxygen therapy the demarcation line between the necrotic and viable tissue was clearly visible and debridement was performed when necessary. The patients were treated in a regular ward and customary precautions with the use of oxygen were observed. The oxygen was delivered from tanks or wall outlets. Photographic documentation and serial bacterial cultures were performed in the majority of patients throughout the course of treatment.

### RESULTS

#### *Burns: five cases*

The patients presented circumscribed, third degree burns of the feet and legs, not exceeding eight cm. in diameter. The lesions had been present from fourteen days to six weeks and showed no signs of improvement. On the second day of oxygen treatment there was evidence of pinkish coloration and stimulation of granulation. All lesions healed within five to nineteen days of oxygen treatment (Figures 2 and 3).

In one case a controlled trial was performed since the patient presented similar burns, of six weeks duration, on both feet. The left foot was treated with hyperbaric oxygen

Dr. Fischer is an Assistant Professor of Clinical Neurology at New York University Medical Center, New York City.

Send reprint requests to Dr. B.H. Fischer at 400 East 34th St., New York, N.Y. 10016.

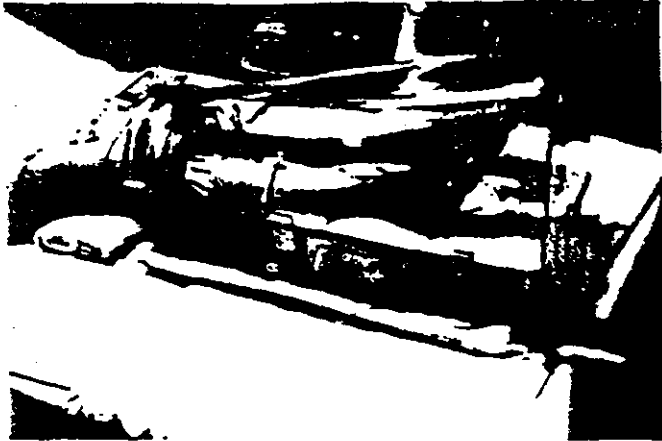


Figure 1 Topical Hyperbaric Oxygen Chamber for lower extremity featuring controlled pressure sealing and automatic pressure regulation control.

and healed within nineteen days while the ulceration on the right foot was only reduced by 30%. The right foot was then treated with hyperbaric oxygen and the ulceration closed within ten days.

*Pressure Sores: sixteen cases*

Pressure sores were present on the ankle, heel, and medial knee area. The duration of the lesions ranged from two months to four and one half years, and all had a past history of vigorous treatment including hospitalization, bed rest, and leg elevation. Total healing was achieved in all cases in a period of two weeks to two months of oxygen treatment. The regenerated epithelial tissue was well vascularized, elastic, and appeared healthy (Figures 4, 5 and 6).

*Ulceration due to venous stasis: three cases*

The lesions had been present from six months to thirty-five years. In all cases, a prompt arrest of inflammatory

reaction, subsidence of oedema, and an enormous stimulation of granulation was observed. The lesions closed within two to seven weeks of hyperbaric oxygen therapy.

*Injected post-surgical wounds: two cases*

A deep ulceration of the medial area of the right ankle after a venous cut-down was present. The lesion of a month's duration had failed to heal despite treatment including bed rest and elevation of the leg. Within eight days of hyperbaric oxygen treatment, there was a prompt arrest of inflammation, subsidence of oedema and suppression of bacterial growth. The deep necrotic crater was filled with granulation permitting re-application of sutures followed by healing.

A great toe became infected after an operation for an ingrown toe nail. The ulcer, of sixteen days duration, was steadily deteriorating. An amputation was considered. After twelve hours of hyperbaric oxygen therapy the severe necrotic reaction with cellulitis was totally under control. Healing was achieved within the next twenty days (Figures 7, 8).

*Ulcerations associated with rheumatoid arthritis: three cases*

The patients presented ulcerations of the lower tibia. One ulceration had been present for one year and one for four months. Both patients healed within six to twenty days of oxygen treatment.

The third patient had an ulceration of two months duration on the great toe. Oxygen treatment resulted in suppression of pain with contraction of the ulcer and mild stimulation of granulation, but failed to heal the ulceration after four weeks of therapy.

*Ulcerations associated with hyper-gamma-globulinemia: one case*

The lesion affected both sides of the right ankle for six years. Severe infection and oedema were brought under control within ten days of oxygen treatment. However,



Figure 2 Third degree burn of the right foot of 15 days duration in an eight year old child. Start of oxygen treatment.

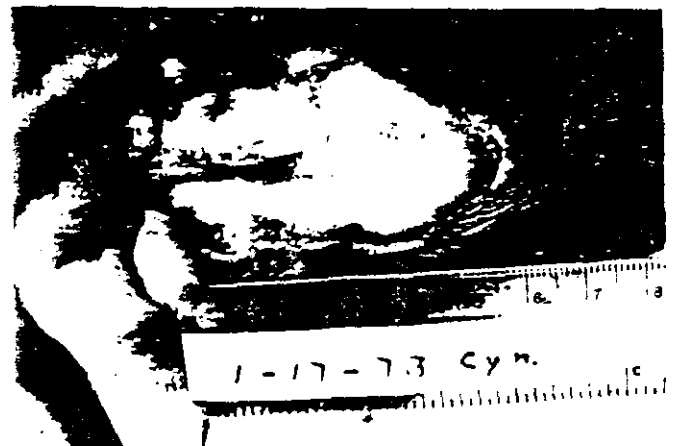


Figure 3 Status on sixth day of oxygen treatment.



Figure 4 Pressure sore of the right heel of two months duration in a fourteen year old girl.

Start of hyperbaric oxygen therapy, four hours daily, pressure 22 mm Hg.

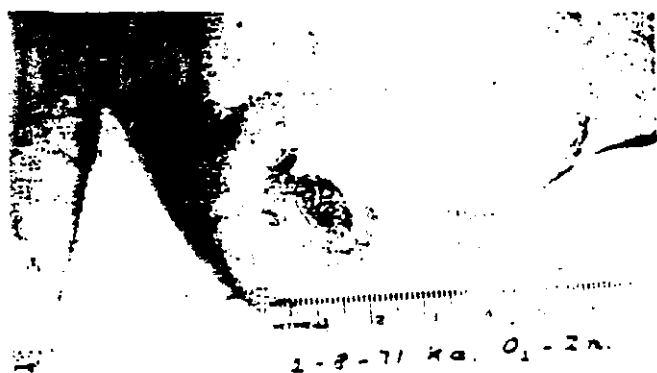


Figure 5 Status after 48 days of oxygen treatment. (Treatment was interrupted for 10 days due to the patient's holiday leave.)

Ulceration reduced to a small, superficial skin defect with good granulation. Oxygen treatment discontinued. Lesion was entirely closed 7 days later.

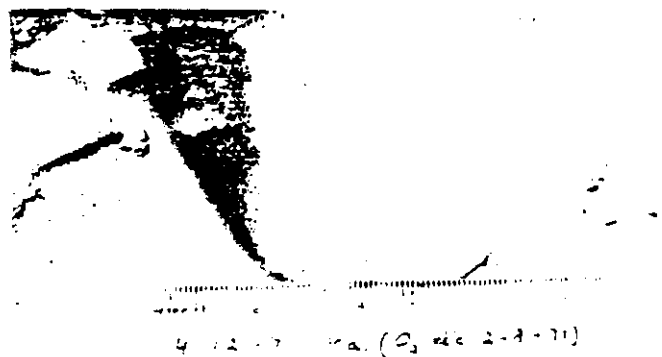


Figure 6 Follow up: appearance of the site of previous ulceration 2 months later. Note excellent quality of reconstituted epithelium.

there was a total absence of granulation and epithelium formation. Treatment was discontinued after ten days.

Pure oxygen applied under ambient pressure has little effect on a skin lesion, as evidenced by Peters<sup>1</sup> and Fischer<sup>2</sup>. Once pressure was raised to 12 mm Hg and higher, the acceleration of healing was observed in practically all cases, however some did not heal entirely. In order to avoid any interference with the capillary blood circulation the therapeutic pressure never exceeded 22-25 mm Hg., equivalent to 1.03 ata.

The formidable obstacle of proper and reliable control of the sealing pressure was resolved by the development of an automatically controlled pressure seal, working on a flutter valve principle. This permitted the application of pressurized oxygen directly to the affected site without any danger of circulatory embarrassment over a prolonged period of time.

An analysis of cases clearly indicates the importance of the vascular status of the treated ulceration. Pressurized oxygen failed to heal ulceration associated with severe ischemia<sup>3,4</sup>. The beneficial effects of hyperbaric oxygen can be explained by several factors inherent in this type of treatment. Oedema is either eliminated or diminished by external pressure distributed evenly over the lower extremity while in the chamber. The relative hypoxia, present in practically all ulcerations, was effectively eliminated as evidenced by capillary blood pO<sub>2</sub> measurements performed serially by collecting capillary blood directly from oxygen-exposed lesions. Blood collected at the same time from fingertips served as a control.

As anticipated, the capillary pO<sub>2</sub> in the ulcerated area was in the range of 76 to 79 mm Hg, as opposed to 95-95 in a healthy fingertip. After one hour of oxygen exposure at 22 mm Hg., the capillary pO<sub>2</sub> rose to 115 mm Hg., and after two hours to 120 mm Hg. The finger tip pO<sub>2</sub> remained on a stable level of 96-97 mm Hg.

This finding indicated an absorption of oxygen through an ulceration without influencing the systemic capillary pO<sub>2</sub>. Oxygen permeability of the skin is dependent upon skin thickness and oxygen pressure<sup>5</sup>. Normal skin absorbs very little oxygen under ambient pressure. The rapid saturation of ulcerated tissue with oxygen stimulates the proliferation of granulation and epithelium, provided the ulceration has an adequate blood supply. In several cases the stimulation of granulation tissue was so effective that it proliferated above the skin level, necessitating periodic removal to permit the spread of newly formed epithelium.

The saturation of ulcerated tissue with oxygen also resulted in elevation of the Redox potential, which is the main active barrier against bacterial proliferation. Studies in vitro showed a moderate suppressive action of oxygen at 22 mm Hg. on bacterial growth. The suppression of bacterial growth increases rapidly with raised oxygen pressure and reaches a peak at approximately 2 atmospheres absolute.



Figure 7 In-grown toenail operation with subsequent severe infection and cellulitis. Status sixteen days after the operation. Start of hyperbaric oxygen treatment.

This pressure could never be applied topically since it would result in total arrest of blood circulation. Yet, in a series of cases, the bacterial growth was visibly suppressed by exposing the lesions to oxygen at 22 mm Hg. It is postulated that oxygen applied at lower pressure indirectly inhibits the bacterial growth by raising the Redox potential and stimulating the white cell front.

Four to six hours of oxygen exposure in the majority of lesions was sufficient to stimulate the healing response. In several cases, a short course in hyperbaric oxygen lasting only three to four days sufficed to rejuvenate the ulceration and stimulate healing without further oxygenation. Lesions associated with small vessel disturbances proved to be the most stubborn to topical hyperbaric oxygen treatment. The unpredictability of therapeutic response is well illustrated by the total healing of two ulcerations associated with rheumatoid arthritis, while the third lesion failed to heal. Equally mixed results were observed in lesions associated with hyper-gamma-globulinemic purpura and peripheral vascular occlusive disease.

The treatment is relatively simple, safe and inexpensive. It does not require any special installations and is easily tolerated by patients. It shortens the patient's suffering, eases the nursing effort and returns the patient to gainful life. No adverse reactions were observed. It must be kept in mind, however, that hyperbaric oxygen is not a "miracle"



Figure 8 Status one day later after six hours of hyperbaric oxygen treatment. Almost total arrest of inflammatory reaction with incipient contraction of the lesion. Oxygen treatment continued for the next three days until total control of the infection was clearly evident. Lesion healed unevenly within six weeks with no further treatment.

procedure. It is limited to small areas and needs prudent application to achieve full capacity healing.

Further clinical research is in progress involving diabetic ulcerations and lesions requiring surgical intervention. Topical hyperbaric oxygen may serve as a preparation technique or a post-operational auxiliary method to control certain complications.

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