# HYPERBARIC MEDICINE PROGRAM CASE OF THE MONTH

*A series of case presentations identifying the improved clinical and cost outcomes that characterize the addition of hyperbaric oxygen therapy to standard medical and surgical measures, in carefully selected patients.*

A 51 yowf is referred for evaluation and treatment recommendations. Her primary diagnosis is recurrence of right face and neck melanoma. She presents with post-operative incision dehiscence and further soft tissue breakdown within the radiotherapy portal.

**Significant Medical History:** Recurrent malignant melanoma, first diagnosed in 1988 as a 1.3 mm lesion in the right pre-auricular region. This has necessitated multiple surgeries including neck dissection with right-facial nerve sacrifice and 6300 cGy external beam radiation (early 2003).

**Other Medical History:** Insomnia; controlled seasonal allergies; laparoscopic D&C, and bladder surgery.

**Review of Systems:** Essentially unremarkable

**Physical Examination:** WDWN 51 yowf. Two 2.0 cm x 0.5 cm adjacent lesions along the right neck. (Fig. 1)

**Assessment:**

i Radiation-induced soft tissue necrosis, refractory to standard care
ii No evidence of local recurrence
iii Lesions likely to remain problematic and may further deteriorate, given the progressive obliterative endarteritis that characterizes late radiation tissue injury
iv No patient-specific risks to hyperbaric oxygenation

**Recommendations:**

i Institute hyperbaric oxygen therapy, per soft tissue radionecrosis protocol (once daily treatments at 2.5 atmospheres absolute pressure five days per week)
ii Reevaluation following 20 treatments
iii Maintain present wound care

These recommendations are agreed to by the patient and her referring physician. The informed consent process is completed.

Hyperbaric treatments commence, and are tolerated without complaint or apparent side-effect. At treatment number 12 the patient notes decreased wound drainage and the development of tingling sensations around the lesions. There is early evidence of wound repair. (Fig. 2)

By treatment number 21 both lesions are clearly of healing. There is decreased erythema and minimal drainage. (Fig. 3)

By treatment number 36 the lesions are almost completely healed. The surrounding skin is now pink and healthy in appearance. There is no further drainage. (Fig. 4) The patient appears close to the point of maximum benefit. The plan now is to hold hyperbaric oxygen treatments and follow to determine subsequent clinical course, which is anticipated to be favorable.

The wounds heal completely over the following 21 days. The patient reports improved motor function on the right side of her face and her mouth. Perilesional sensory perceptions have returned.

Follow-up, two months after completion of the hyperbaric treatment course is significant for an enduring soft tissue healing response. Unfortunately, the patient has developed yet another local recurrence, this time with probably pulmonary metastiatic disease. The patient undergoes neck dissection at this time, with no soft tissue healing complications, as one would anticipate with the improved vascular density that hyperbaric treatments afford. (Fig. 5) ENT surgery completely attributes this ability to re-operate on HBO's effects in tissues previously declared inoperable if further tumor found.

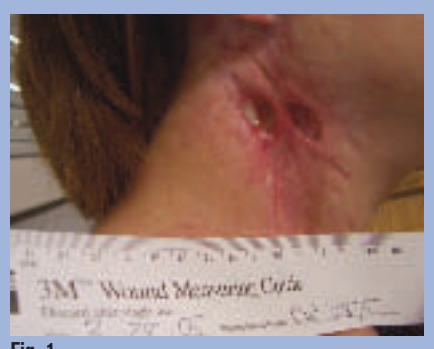

Fig. 1

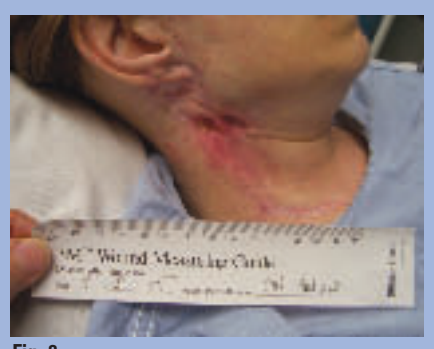
Fig. 2

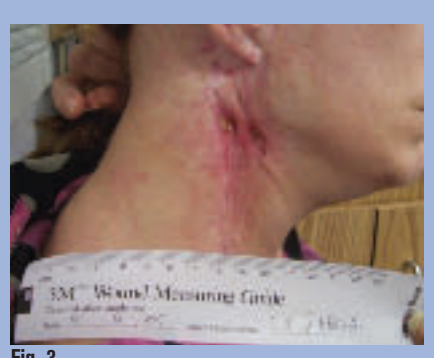
Fig. 3


Fig. 4


Fig. 5

## Discussion

Damaging side-effects to bone and soft tissue are a well recognized "late" complication of therapeutic radiation.[1] Several recent advancements have served to minimize but no eliminate such damage to non-target tissues. The injury produced by therapeutic radiation is progressive in nature. It has long been considered to commence, and become clinically manifest, many months to several years after exposure. More recently, it has been suggested that this process is actually the continuum of an injury that begins at the time of radiotherapy.[2] The end pathophysiologic result is a proliferative and obstructive endarterits, occurring at the small vessel level. Resulting break down of bone or soft tissue is invariably the result of trauma to the site (surgical wounding; unintentional injury; dental extraction, are examples).[3] Less commonly, the tissue bed breaks down spontaneously.

Resulting lesions are complex and frequently represent a significant clinical challenge. There is some similarity to the "whittling away" syndrome that characterizes foot lesions in the diabetic patient.

In contrast to more standard medical therapies and surgical interventions, hyperbaric oxygen but not normobaric oxygen therapy directly reverses the underlying pathophysiologic process.[4] Hyperbaric oxygen therapy has proven clinically helpful[5,6] and formal trials involving several anatomic sites are underway.[7]

Hyperbaric medicine benefited this patient both directly and indirectly. The wounds healed with hyperbaric oxygen alone, thereby avoiding a significant surgical repair. Further, subsequent surgical incisions necessary to address local recurrence healed quickly and completely. This was certainly in large part a function of the greatly improved vascular density generated within irradiated tissue by hyperbaric oxygenation.

**References:**

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3 Curi MM, Dib LL: J Oral Maxillofac Surg 1997;55:540-544
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7 www.baromedicalresearch.org

## INDICATIONS AND RATIONALE FOR HBO THERAPY *

| Indications | Rationale |
|---|---|
| Acute carbon monoxide poisoning | Relieve hypoxia; hasten elimination of CO; antagonize brain lipid peroxidation. |
| Acute exceptional blood loss anemia | Increase physically dissolved oxygen; treat hypoxia; support marginally perfused tissues. |
| Acute thermal burns | Relieve hypoxia; decrease fluid losses; limit burn wound extension and conversion; treat edema; promote wound closure. |
| Cerebral arterial gas embolism | Overcome free gas volume; relieve hypoxia; antagonize leukocyte mediated ischemia-reperfusion injury |
| Chronic osteomyelitis | Augment host antimicrobial defenses; induce angiogenesis; potentiate leukocytic superoxide and peroxide production; relieve hypoxia; augment antibiotic therapy; extend post-antibiotic effect; augment osteoclast activity. |
| Clostridial gas gangrene | Reduce size of gaseous bullae; inactivate clostridial alpha toxin; inhibit alpha toxin production; induce bacteriostasis; potentiate leukocytic superoxide and peroxide production |
| Compromised skin flaps | Support marginally perfused/oxygen-ated tissues; antagonize ischemic-reperfusion injury; accelerate angio- gensis. |
| Crush injury; acute ischemia | Provide interim tissue oxygenation in relative states of ischemia; reduce edema; reduce compartment pressures; antagonize ischemic-reperfusion injury; augment limb salvage. |
| Decompression sickness | Overcome free gas volume- induced ischemia; relieve hypoxia; hasten elimination of offending inert gas; treat edema |
| Late radiation tissue injury | Re-establish wound oxygen gradients; relieve hypoxia; induce angiogenesis; prepare for definitive coverage |
| Late radiation tissue injury prophylaxis | Re-establish wound oxygen gradients; induce angiogenesis prior to surgical wounding. |
| Necrotizing soft tissue infections (fasciitis and cellulitis) | Induce bacteriostasis of anaerobes; potentiate leukocytic superoxide and peroxide production; relieve hypoxia; more closely demarcate potentially viable tissue |
| Non-healing marginally perfused wounds | Re-establish wound oxygen gradients; relieve hypoxia; reduce edema; induce angiogenesis; correct diabetic-induced leukocyte changes; prepare for definitive coverage. |

*UNDERSEA AND HYPERBARIC MEDICAL SOCIETY, 2003