HYPERBARIC MEDICINE SERVICE 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.

An 84-year-old female was urgently referred from her surgeon's office for evaluation and treatment recommendations. The previous day the patient had undergone a Mohs surgery and excision of a basal cell carcinoma from the right side of her nose. A rotational flap repaired the resulting soft tissue defect. Within 24 hours, the flap appeared dusky and its viability threatened. (Fig.1)

Significant Medical and Surgical History: Arthritis, previous Mohs surgical procedure for skin cancer at the right side of her nose; partial hysterectomy.

Review of Systems: Essentially unremarkable.

Assessment:
i. Status-post Mohs procedure and excision of a basal cell carcinoma
ii. Previous similar procedure at the same site
iii. Early evidence of skin flap compromise, periorbital and perilesional edema
iv. No patient-specific risks to hyperbaric oxygen therapy

Recommendations:
i. Immediate application of hyperbaric oxygen therapy, per compromised skin flap protocol
ii. Closely follow clinical progress
iii. Reevaluation following 10 hyperbaric oxygen treatments

The patient was agreeable to the treatment plan, as was her surgeon. Following completion of the informed consent process, hyperbaric treatments were initiated, on a BID basis. The first treatment was complicated by minor right ear discomfort, relieved with decongestants.

Following four treatments, delivered over approximately 48 hours, there was a marked reduction in edema. (Fig.2) After four treatments, the flap appeared to be improving. By treatment number nine periorbital edema had resolved and the sutures had been removed. (Fig.3)

Upon completion of the planned 10 treatments the flap appeared healthy and viable. Superficial eschar was greatly diminished. (Fig.4) It was considered that the patient was at the point of maximum benefit and hyperbaric oxygen therapy could be discontinued. The patient was discharged from hyperbaric service to the continued care of her primary physician.

Follow-up at 18 days post-hyperbaric oxygen therapy was significant for a completely healed flap, resolved edema and a good cosmetic result. (Fig.5)

Fig. 1

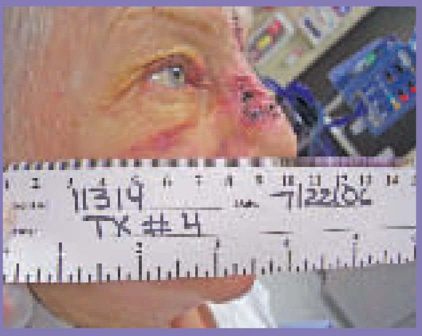

Fig. 2

Fig. 3

Fig. 4

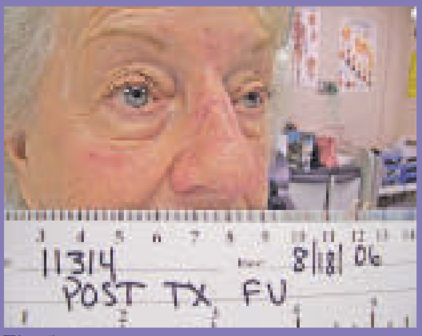

Fig. 5

Discussion:

A wide variety of skin flap options are available for repair of soft tissue defects. For every defect there will usually be one or two management options. The common approach is to begin with the least complex. With each type of flap there will be potential complications, ranging from the poor quality of host tissues to technical shortcomings related to flap design and implementation. In most cases the problem, once identified, can be corrected surgically. Examples include evacuation of thrombus, repositioning of a kinked pedicle or easing tension of an overly tight suture line. Venous outflow compromise may require leech therapy.(1)

Where surgical correction does not fully resolve the threatened state, or the etiology of flap compromise is random pattern ischemia, hyperbaric oxygen therapy has been successfully employed. Mechanisms include delivery of large volumes of oxygen and antagonism of ischemia-reperfusion injury via down regulation of leukocyte receptor sites.(2)

In the case presented above, the patient's flap compromise may have resulted from any combination of recipient bed issues (prior surgery), increased diffusing distances from functioning capillaries to the body of the flap (secondary to edema) and random pattern ischemia (the area of perfusion exceeding arterial inflow capacity).

In larger flaps, the degree of hypoxia and its reversibility secondary to hyperbaric oxygenation can be assessed via transcutaneous oxygen monitoring.(3) The size and location of the flap in this particular case precluded such assessment. The hyperbaric treatment course was, therefore, guided by clinical appearance.

No 'Level I' evidence is available for any of the options available to overcome skin flap compromise, including hyperbaric oxygen therapy. This may not be too surprising given a general lack of high level evidence across the medical and surgical continuum of diagnostic and therapeutic options. However, the present role of hyperbaric oxygen therapy in plastic surgery has been recently and comprehensively reviewed.(4) This review notes, "…there is enough animal evidence and observational data to warrant the application of hyperbaric oxygen in selective cases…"

In summary, the prompt referral of a threatened flap repair to hyperbaric medicine resulted in a good outcome. The patient required few treatments, provided on an outpatient basis, and were well tolerated, with the exception of minor and temporary ear discomfort.

References: *(Available upon request)*

1) Kubo T, Yano K, Hosokawa. Management of Flaps with Compromised Venous Outflow in Head and Neck Microsurgical Reconstruction. Microsurgery 2002; 22:391-395.

2) Buras J. Basic Mechanisms of Hyperbaric Oxygen in the Treatment of Ischemia-Reperfusion Injury. Internal Anesthesiology Clinics 2000; 38(1):91-108.

3) Clarke D. An Evidence-Based Approach to Hyperbaric Wound Healing. Blood Gas News 1998; 7(2):14-20.

4) Friedman HIF, Fitzmaurice M, Lefaivre JF, et al.: An Evidenced-Based Appraisal of the Use of Hyperbaric Oxygen on Flaps and Grafts. Plast. Reconstr. Surg. 2006; 117(SUPPL):175S-192S.

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 dismutase superoxide and perioxide 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 dismutase superoxide and perioxide production
Compromised skin flaps	Support marginally perfused/oxygenated tissues; antagonize ischemic-reperfusion injury; accelerate angiogensis
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	Induce bacteriostasis of anaerobes; (fasciitis and cellulitis) potentiate leukocytic dismutase superoxide and perioxide 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