HYPERBARIC OXYGEN THERAPY
An Evidence-Based Analysis

“Problem Wound Healing”
The first requirement for wounds to progress along anticipated time lines is healthy vascular tissue

* wounds in ischemic tissue heal poorly, or not at all

Hunt TK, et al. 1969
* Amer. J. Surgery 118

This suggests that one or more vascular-derived substrates drive the wound repair process
Indians living at high altitude* have long believed that their wounds heal more quickly if they descend

~ * lower pO2’s than at sea level

French divers reported that wounds seemed to heal faster in their undersea habitats* (Conshelf I and II)

~ * higher pO2’s than at sea level
Oxygen tensions are low in the area of injury.

If inspired oxygen levels are increased, actively healing wound dead space pO2 increases by a fraction of the increase in arterial pO2.

- *as more oxygen becomes available, more appears to be consumed during wound repair*

Hunt TK, et al. 1969
Amer. J. Surgery 118

Zederfeldt B, Hunt TK, 1969
Curr. Topics Surg. Research
Alveolar Gas Equation

...Alveolar O2 - Normobaric Oxygen Conditions

\[ PA\ O2 = (PBTPS \cdot Fi\ O2) - (Pa\ CO2 \div R) \]

Assuming:
PATM = 760 mmHg, FiO2 = 21%
PaCO2 = 40 mmHg, Respiratory Quotient = 0.8

Then:
\[ PAO2 = (713 \cdot 0.21) - (40 \div 0.8) \]
\[ = 100 \text{ mmHg} \]
Alveolar Gas Equation

...Alveolar O2 - Hyperbaric Oxygen Conditions

\[ PA\ O2 = (PBTPS \cdot Fi\ O2) - (Pa\ CO2 \div R) \]

Assuming:
PATM = 2280 mmHg, FiO2 = 100%
PaCO2 = 40 mmHg, Respiratory Quotient = 0.8

Then:
PAO2 = (2233 \cdot 1.0) - (40 \div 0.8)
= 2183 \text{ mmHg}
Oxygen Content

...Hemoglobin Binding

\[
\text{O}_2 \text{ Content} = \frac{1.34 \text{ ml } \text{O}_2}{\text{gm Hgb}} \times \frac{\text{gms Hgb}}{\text{dl}}
\]

Assuming:

\( \text{Hgb} = 16 \text{ gms/dl}, \quad \text{SaO}_2 = 98\% \)

Then:

\[
\text{Oxygen Content} = 1.34 \times 16 \times 0.98
\approx 20 \text{ ml/dl}
\]
Arterial and Venous O2 Content (1 ATA)

ΔA-V O2 ≈ 6 vol%

Arterial O2 Content

Venous O2 Content

Arterial Oxygen Pressure (PaO2)
Oxygen Content

...Dissolved in Blood (normobaric air)

\[
\text{Dissolved O}_2 = 0.0031 \frac{\text{ml O}_2}{\text{PaO}_2 \text{ mmHg} \cdot \text{dl} \cdot \text{mmHg PaO}_2}
\]

Assuming:
1 ATA, \( \text{PaO}_2 \approx 95 \text{ mmHg} \)

Then:
\[
\text{Dissolved O}_2 = 0.0031 \cdot 95 \\
= 0.29 \text{ ml/dl}
\]
Oxygen Content

...Dissolved in Blood (HBO)

\[
\text{Dissolved } O_2 = 0.0031 \frac{ml \ O_2}{mmHg \ PaO_2} \cdot dl \cdot mmHg \ PaO_2
\]

**Assuming:**
3 ATA, \( PaO_2 \approx 2140 \ \text{mmHg} \)

**Then:**
\[
\text{Dissolved } O_2 = 0.0031 \cdot 2140 = *6.6 \ ml/dl
\]
Arterial O2 Content (3.0 ATA)

**Oxygen Saturation (SaO2)**

- 100
- 80
- 60
- 40
- 20

**Oxygen Content (ml/dl)**

- 1000
- 2000
- 600
- 240
- 120
- 60

**Hgb O2 Content**

**Dissolved O2 Content**

- 6 vol% Dissolved

**Arterial Oxygen Pressure (PaO2)**

- 20
- 40
- 60
- 80
- 100
- 1000
- 2000

Graph shows the relationship between oxygen saturation (SaO2), oxygen content (ml/dl), and arterial oxygen pressure (PaO2) at 3.0 ATA, with highlighted areas indicating specific oxygen concentrations.
THE WOUND HEALING SEQUENCE

• **Coagulation**
  - sets the stage for healing by platelet activation and fibrin deposition

• **Inflammation**
  - activation of complement and kinins, attracting inflammatory cells (macrophage dominates)

• **Fibroplasia**
  - slowed by ischemia
THE WOUND HEALING SEQUENCE

- **Collagen production**
  - extremely sensitive to ischemia and arterial hypoxia

- **Angiogenesis**
  - first visible evidence of granulation tissue
  - an oxygen-dependent process

- **Epithelialization**
  - slowed or halted by ischemia
Raising arterial pO2 from 82 to 200 mmHg increases oxygen transport by about 1%.

- corresponding increase in collagen accumulation and its synthesis is greater than 50%.

A fall in arterial pO2 from 82 to 42 mmHg results in less than a 20% reduction in transport.

- corresponding depression in collagen synthesis of some 50%.

Hunt TK, Pai MP, 1972
Hypoxic tissue gradient mandatory for wound healing
When this gradient is destroyed capillary growth ceases
Higher inspired oxygen concentrations increase rate and density of capillary growth
Changes in oxygen tension significantly influence revascularization and epithelialization in a variety of animals.

- **hypoxia slows and hyperoxia accelerates closure rates in full thickness wounds**

Rate of epithelialization is correlated to oxygen’s availability.

Pai MR, Hunt TK, 1972

Silver IA, 1972
*Epiderm. Wound Heal.* 17
Angiogenesis is proportional to ambient pO2

- Hypoxia within the wound stimulates VEGF release
- Endothelial cell response to VEGF requires normoxia, and increases with hyperoxia

Surgical Forum; 48
Proponents first believed that it acted as a respiratory metabolite

- however, hyper-oxygenation is only achieved for 1.5-2.0 hrs daily in otherwise constantly hypoxic tissue

It was subsequently proposed that HBO increased the wound oxygen gradient and, therefore, the healing stimulus

- this issue remained controversial because of a paucity of analytically supportive experimentation
More recently, it is apparent that hyperbaric oxygen therapy acts by means of a signal transduction pathway:

- stimulating growth factors and/or their receptor sites through what appears to be a nitric oxide dependent process
1.0 ATA Air

\[ \Delta = 10 - 20 \text{ mmHg} \]
HBO at 2.4 ATA

\[ \Delta = 230 \text{ mmHg} \]
Proliferative Phase

$\Delta = 10 \text{ mmHg}$
Plateau Phase
HYPERBARIC OXYGEN

Effect on Angiogenesis

~ in-vivo murine model

- Three dimensional study
- Implanted fibrin-containing chambers
- Four chambers per rat with 4 rats in each group
- Groups
  - Controls .................atmospheric air
  - Study Group.............2.5 ATA x90 mins bid, x 14 days
  - Assessment.............total vascularized area, bud height, vessels diameter per bud

Mechine A, et al. 1999
Ann Chirurgie; 53(4)
HYPERBARIC OXYGEN

Effect on Angiogenesis

~ a three dimensional study of capillary budding

Plexiglass ring
Calibrated holes
Granulation tissue bud

Rohr S, et al 1992
XVIII EUBS Meeting
# HYPERBARIC OXYGEN

## Effect on Angiogenesis

### Histological Examination

<table>
<thead>
<tr>
<th>Vasc. Area</th>
<th>Bud height</th>
<th>Bud diameter</th>
<th># Buds</th>
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<tbody>
<tr>
<td>(mm²)</td>
<td>(mm)</td>
<td>(mm)</td>
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<tr>
<td>Controls</td>
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<tr>
<td>19.73</td>
<td>0.312</td>
<td>0.79</td>
<td>11.75</td>
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<tr>
<td>(12.34)</td>
<td>(.06)</td>
<td>(.06)</td>
<td>(6.10)</td>
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<tr>
<td>HBO</td>
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<tr>
<td><em>40.16</em></td>
<td><em>0.288</em></td>
<td><em>0.86</em></td>
<td><em>16.85</em></td>
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<tr>
<td>(24.87)</td>
<td>(.03)</td>
<td>(.09)</td>
<td>(11.1)</td>
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</table>

* 5% confidence level

Mechine A, et al. 1999
A single HBO exposure

Increased endothelial cell proliferation, within 15 mins.

* Fibroblasts required 120 mins. to begin proliferation

*A second same day exposure produced no further increase

* An increase from 2.4 to 4.0 ATA O2 did not enhance response

Int.J.Oral Max. Surg. 26
HYPERBARIC OXYGEN

Fibroblast response to a single treatment


Int.J.Oral Max. Surg. 26
HYPERBARIC OXYGEN

Induced Angiogenesis

~ researching its mechanism

- HBO observed to induce angiogenesis
- VEGF critical to new vessel formation

* VEGF activity only initiates formation of immature vessels
* VEGF must work in concert with angiopoietins to produce mature functioning vessels

In this model, HBO selectively enhanced Ang2 gene expression through an eNOS pathway

~ inhibition of eNOS blocked this process

Lin S, et al. 2002
Biochem Biophys Res Comm; 296

in-vitro dosing
HYPERBARIC OXYGEN
Severely Ischemic Diabetic Foot Wounds

~ a randomized trial

70 consecutive pts. randomized to receive HBO, or not: standard aggressive medical/surgical care in both groups

Both groups well matched from clinical, vasculopathy and neuropathy perspectives

Faglia E, et al. 1996 Diabetes Care; 19(12)
## MAJOR AMPUTATION RATES

<table>
<thead>
<tr>
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<th>HBO(35)</th>
<th>Non-HBO(33)</th>
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<tbody>
<tr>
<td>After revascularization</td>
<td>2/13(15.4)</td>
<td>4/13(30.8)</td>
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<tr>
<td>No revascularization</td>
<td>1/22(4.5)</td>
<td>7/20(35.0)</td>
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<tr>
<td>All major amputations</td>
<td>3/35(8.6)*</td>
<td>11/33(33.3)</td>
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</table>

*p= 0.016

Faglia E, et al. 1996
*Diabetes Care*; 19(12)
### HYPERBARIC OXYGEN

#### Crush Injuries

~~randomized double-blind placebo-controlled trial~~

36 pts. evaluated within 24 hours of initial surgery

<table>
<thead>
<tr>
<th>Group</th>
<th>Pts.</th>
<th>Details</th>
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</thead>
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<tr>
<td>Tx. Group</td>
<td>18</td>
<td>2.5 ATA O2 x 90 mins. BID x 6 days</td>
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<tr>
<td>Controls</td>
<td>18</td>
<td>1.1 ATA Air x 90 mins. BID x 6 days</td>
</tr>
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</table>

~ HBO utilized to improve viability of ischemic tissues

~ All other medical and surgical measures maintained

*Journal of Trauma;* 41(2)
HYPERBARIC OXYGEN

Crush Injuries

<table>
<thead>
<tr>
<th>GROUP</th>
<th>HBO(18)</th>
<th>Placebo(18)</th>
<th>p-value</th>
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<tbody>
<tr>
<td>Complete healing</td>
<td>17</td>
<td>10</td>
<td>0.009</td>
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<tr>
<td>Tissue necrosis</td>
<td>1</td>
<td>8</td>
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<tr>
<td>New surgical proc.</td>
<td>2</td>
<td>8</td>
<td>0.03</td>
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<tr>
<td>Skin flaps/grafts</td>
<td>1</td>
<td>6</td>
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</tr>
<tr>
<td>Vascular surgery</td>
<td>1</td>
<td>0</td>
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<tr>
<td>Amputation</td>
<td>0</td>
<td>2</td>
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</tr>
</tbody>
</table>

Time to healing (d.)

- HBO(18): 50.2(+/-21)
- Placebo(18): 56.3(+/-20)
- p-value: 0.21

*Journal Trauma*; 41(2)
**HYPERBARIC OXYGEN**

*Therapeutic Dosing for Chronic Wounds*

- **Treatment Pressure:** 2.0 – 3.0 ATA oxygen
- **Treatment Time:** Commonly 90 mins. oxygen for a total exposure period of 120 mins.
- **Treatment Frequency:** Once daily, five to six times weekly
- **Treatment Duration:** Point of maximum benefit vs. complete secondary intention healing
- **Treatment Algorithm:** Based on tissue oximetry
HYPERBARIC OXYGEN

Technology Assessments – Diabetic Foot Wounds

Alberta Heritage: CAN 1998
Strong evidence to support its use. Better healing rates, fewer amputations.

BCBS Tech Evaluation. USA 1999
Sufficient evidence to support its use in chronic wounds. Better healing rates, fewer amputations.

American Diabetes Ass: USA 1999
Reasonable to use it to treat severe and life/limb threatening wounds, particularly if regional ischemia cannot be corrected.
Our assessment on this type of diabetic wound (chronic refractory) concurs with the previous positive assessments of related technical reviews.

Clinical Evidence: UK 2000

Likely to be beneficial. Reduces absolute risk of amputation.

MSAC: AUS. 2000

Statistical homogeneity of controlled studies provided some confidence of HBO’s effects. Major amputations less likely.

AHRQ, Boston: USA 2001

Our assessment on this type of diabetic wound (chronic refractory) concurs with the previous positive assessments of related technical reviews.
“Evidence is adequate to conclude that HBO therapy is clinically effective...in the treatment of certain types of limb-threatening diabetic wounds.”
HYPERBARIC OXYGEN
Mechanistic Basis And Resulting Indications

Neovascularization

- Large oxygen gradient required for initiation of angiogenesis
- Minimum level of oxygen required as substrate for:
  - Fibroblast proliferation
  - Collagen formation
Infection

White cells ingest bacteria under a wide range of oxygen tensions

_~ but they cannot kill them under anaerobic conditions_

Phagocytosis is followed by a burst of oxygen consumption

_~ coincident with death of bacteria_

Hunt TK, et al. 1972
Surgical Forum; 23
HYPERBARIC OXYGEN
A Signal Transducer

~ up-regulation of PDGF-B receptor sites in ischemic rabbit ear wounds

HBO therapy results in a statistically significant increase in expression of PDGF-beta receptor

~ ”results clearly demonstrate that HBO therapy is able to modulate and regulate gene expression via a signal transduction pathway”

“Represents a mechanism whereby relatively brief intermittent episodes of hyperoxia favorably modify healing deficit ischemia”

Bonomo SR, et al. 1998 UHM; 25 (4)
**Hypothesis:**
HBO increases growth factor receptor levels via a nitric oxide dependent mechanism

Skin fibroblasts treated with a single HBO treatment had significantly faster (p<0.01) proliferation rates than controls.

~ this rate was reversed when NO was inhibited

**Conclusion**
HBO treated fibroblasts have increased proliferation and responsiveness rates to growth factors because of GF receptor expression and signaling, via a NO pathway

Reenstra WR, et al 2002
*UHM Suppl., Abst.84*
<table>
<thead>
<tr>
<th>Mechanism of Action</th>
<th>Clinical Effects</th>
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<tbody>
<tr>
<td>Hyperoxygenation</td>
<td>Increased O2 Diffusion</td>
</tr>
<tr>
<td>Neovascularization</td>
<td>Increased O2 Content</td>
</tr>
<tr>
<td>Antimicrobial Effects</td>
<td>Fibroblast Proliferation</td>
</tr>
<tr>
<td></td>
<td>Angiogenesis/Vasculogenesis</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>Direct Cidal Effects</td>
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<tr>
<td>Attenuation of Reperfusion Injury</td>
<td>Antibiotic Synergism</td>
</tr>
<tr>
<td></td>
<td>Toxin Inhibition / Inactivation</td>
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<tr>
<td></td>
<td>Edema Reduction</td>
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<td></td>
<td>Modulation of gene expression</td>
</tr>
<tr>
<td></td>
<td>Modulation of redox signaling</td>
</tr>
</tbody>
</table>
Hyperbaric Oxygen

Mechanistic Basis And Resulting Indications

Antimicrobial Effects

- Provides minimum level of oxygen required as substrate for leukocyte oxidative killing
- Toxin inhibition and inactivation
- Direct bactericidal effect on anaerobic bacteria
- Antibiotic synergism
HYPERBARIC OXYGEN
Mechanistic Basis And Resulting Indications

Antimicrobial Effects

- Gas gangrene (Bakker D, et al., 1996)
- Necrotizing soft tissue infections (Hollabaugh R, et al., 1998)
- Refractory osteomyelitis (Calhoun J, et al., 1991)
HYPERBARIC OXYGEN
Mechanistic Basis And Resulting Indications

Vasoconstriction

- Decreases edemagenic potential while maintaining oxygen delivery
- Enhances oxygen diffusion
HYPERBARIC OXYGEN
Mechanistic Basis And Resulting Indications

Vasoconstriction

- Acute thermal injury (Niezgoda J, et al., 1997)
- Compartment syndrome (Strauss M, et al., 1985)
- Compromised skin grafts (Zamboni W, 1996)
HYPERBARIC OXYGEN
Mechanistic Basis And Resulting Indications

Attenuation of Reperfusion Injury

- Modulates gene expression
- Modulates redox signaling
- Attenuates expression of Intracellular adhesion molecules (ICAMS)
- Decreases inflammatory response and secondary tissue injury
HYPERBARIC MEDICINE

Delivery Systems

Multiplace Chamber

Monoplace Chamber
The Multiplace Chamber
The Monoplace Chamber
HYPERBARIC OXYGEN

Transcutaneous Oxygen Algorithm

- Is wound healing complicated by hypoxia?
- Is any such hypoxia reversible?
- Is the patient responding to HBO therapy?
- Has a therapeutic endpoint been reached?
HYPERBARIC MEDICINE

Patient Assessment

Transcutaneous Oximetry

- Oxygen electrode which measures transcutaneous oxygen tension
- Provides an indication of vascular flow and capillary density
- Predictive value for wound healing based on oxygen dependent mechanisms
HYPERBARIC MEDICINE

*transcutaneous oximetry*

- Does not assess wound directly
- Limited by electrode size and patient factors
HYPERBARIC MEDICINE

Case Presentation

Case 1:

- 66 y/o WM with DM, HTN, CAD, CHF admitted with necrotizing infection of the right upper extremity with sepsis following a cat bite.
- Underwent 2 surgical debridements.
- HBO consulted as last resort for upper limb preservation.
**TRANSCUTANEOUS OXIMETRY ASSESSMENT**

**PATIENT'S NAME:** Clifford Day  **DIAGNOSIS:** Diabetic Wound

**DATE:** 4-20-00  **REFERENCE SITE:** 62 mmHg

**PULSE OX. SAT.:** 88%

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<th>Olene Pate</th>
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### Reference Site

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### Site #1

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<th>Natural Proximal Wrist</th>
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**RPI 0.45**

### Site #2

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**RPI 0.45**

### Site #3

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**RPI 0.37**

### Site #4

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**RPI 0.19**

### Site #5

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**RPI**

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**RPI**

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**RPI**

### Site #8

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**RPI**

(forms/tocomman)
Case 1:

- Tolerated HBO without complication
- Discharged to home with functional right upper extremity after skin grafting of right forearm
HYPERBARIC MEDICINE

Case Presentation

Case 2:

- 67 y/o WF with HTN, PVD, CREST syndrome s/p right fem-pop bypass with wound dehiscence and tissue necrosis.
- HBO consulted for support of chronic compromise wounds.
TRANSCUTANEOUS OXIMETRY ASSESSMENT

PATIENT’S NAME: STUCKEY, BESSIE
DIAGNOSIS: "Crest Syndrome"

DATE 2/24/00
TECH: A. WHITMORE

REFERENCE SITE: 165 mmHg
Pulse Ox. Sat. 84%

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<th>SITE #3</th>
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RPI 0.046
RPI 0.015
RPI 0.015
RPI 0.015
RPI 0.538
RPI 0.538
RPI 0.553
RPI 0.492
RPI 1.676
Continued clinical improvement was noted and HBO was discontinued at a perceived point of maximal benefit.

The patient ultimately underwent skin grafting for closure.
HYPERBARIC MEDICINE

Case Presentation

Case 3:

- 56 y/o male with h/o diabetes and a chronic non-healing wound over the lateral aspect of the right foot.
- HBO consulted for adjunctive therapy
Case 3:

- Patient reached point of maximal benefit and further HBO held
- Wound adequately prepared for grafting
Case 4:

- 72 y/o female with history of DM, PVD who is s/p arterial bypass surgery and fore foot amputation.
- Hyperbaric medicine consulted for compromised surgical site.
Patient: 4736 4-1-05 TX 18

Wound Length: 

Wound Width: 

Wound depth: 

Smith & Nephew, Inc.
Wound Management
Customer Care Center • 1 800 876-12
Case 4:

- Granulation tissue noted in ankle wound
- Significant improvement in fore foot amputation wound
Case 5:

- 64 y/o WF with h/o DM and a Wagner III wound of the right foot
- Transcuateous oximetry revealed dermal hypoxia of a degree which was not predictive of wound healing in the foot
- HBO offered for wound support following a digital amputation
HYPERBARIC MEDICINE

Case Presentations

Case 5:

- Significant improvement in tissue integrity
- Successful control of underlying infection
Case 6:

- 15 y/o WF s/p reconstructive facial surgery with a post operative compromised flap
- HBO consulted for urgent graft support
Case 6:

- Significant improvement in graft perfusion
- Successful intervention of ischemia-reperfusion injury
- Significant percentage of graft survival
S.S, a 56 y/o IDDM WM, referred for evaluation and tx. recommendations. Presents with poorly-healing left third toe ray amputation (revised 3 weeks earlier)

- Present wound care involves hydrogen peroxide irrigation, and wet to damp dressing
- PMH significant for right BKA (1998) and LLE bypass grafting (1992)
<table>
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<tr>
<th>Date</th>
<th>S.S</th>
<th>21% O₂</th>
<th>100% O₂</th>
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Essentially normal exam
Hold HBO therapy
Conservative care
Discontinue hydrogen peroxide
A 74 yo diabetic male: Left great toe amputation on 9.14.98, secondary to ischemia. Primary closure via a rotational flap

9.28.98
Wound F/U. Tenderness over first metatarsal and plantar surfaces. Erythema and edema noted, as are ischemic changes over the superior portion of the flap.

~ pt. admitted; further surgery anticipated; tcoms ordered.
Present level of amputation clearly compromised.

Revisions within the foot unlikely to be successful under the present flow circumstances.

~ recommend further LLE arterial work-up
9.29.98 Operative exploration

Attempts made to identify grafting opportunities

~ severely calcified vessels ruled out by-pass grafting or other flow augmentation options

10.1.98 Patient underwent a left BKA
RC, an 83 yobf, is referred for evaluation and treatment recommendations. Presents with a dehisced right BKA.

- surgery (9.4.94) secondary to severe and non-reconstructible PVD

- medical hx. complex, including IDDM; HTN; MI; CVA; CRF, and DVT
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<tr>
<td>10 cm above knee, medial</td>
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References


