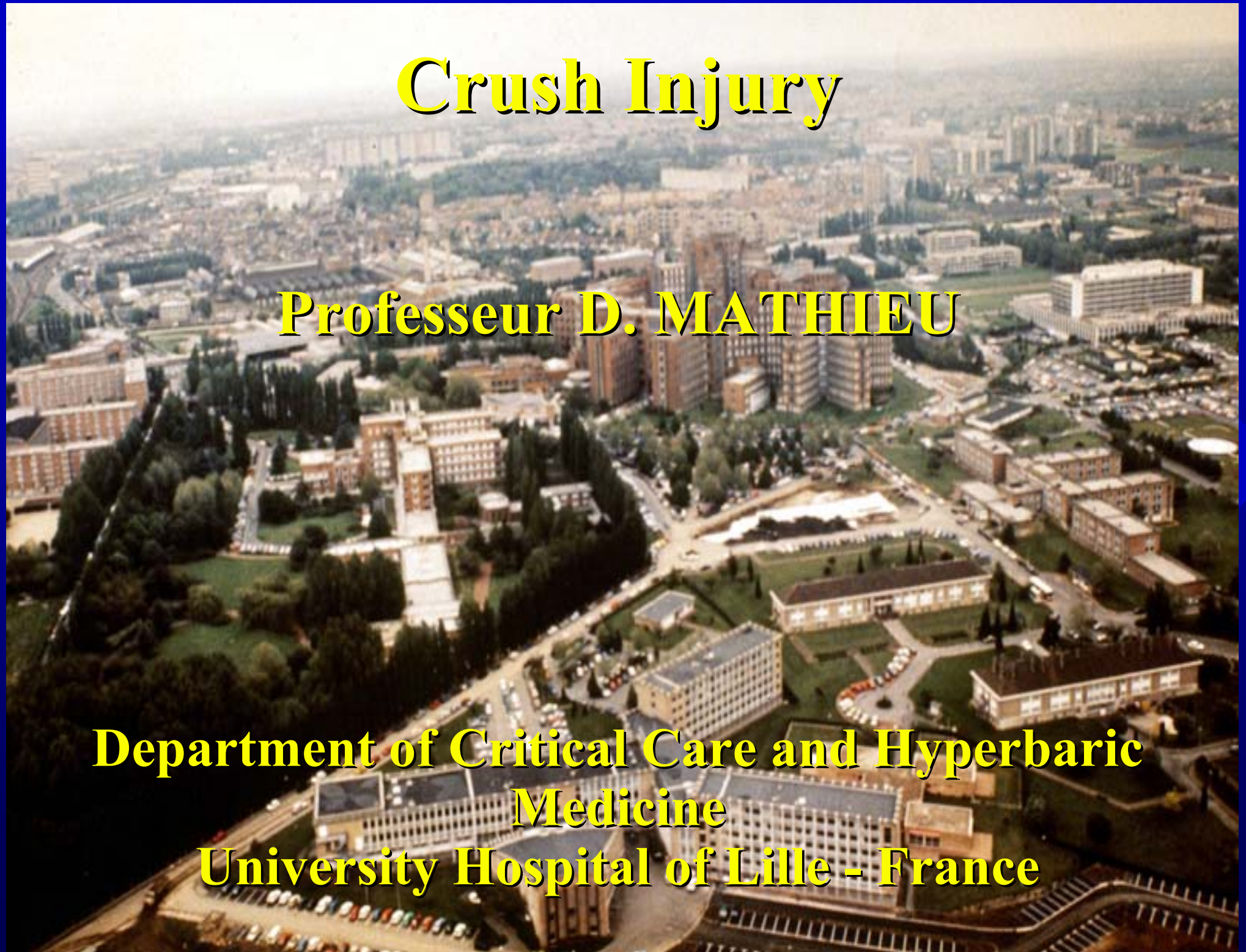


# Crush Injury

**Professeur D. MATHIEU**

**Department of Critical Care and Hyperbaric  
Medicine**

**University Hospital of Lille - France**



# Definitions

## Crush Injury

- An injury sustained when a body part is subjected to a high degree, or prolonged presence of a force or pressure
- Acute traumatic ischemia, with or without associated injuries, describes actual insult to tissues

## Crush Syndrome

- Term used to describe the systemic manifestations of crush injury after reperfusion of affected body part(s)

## Compartment Syndrome

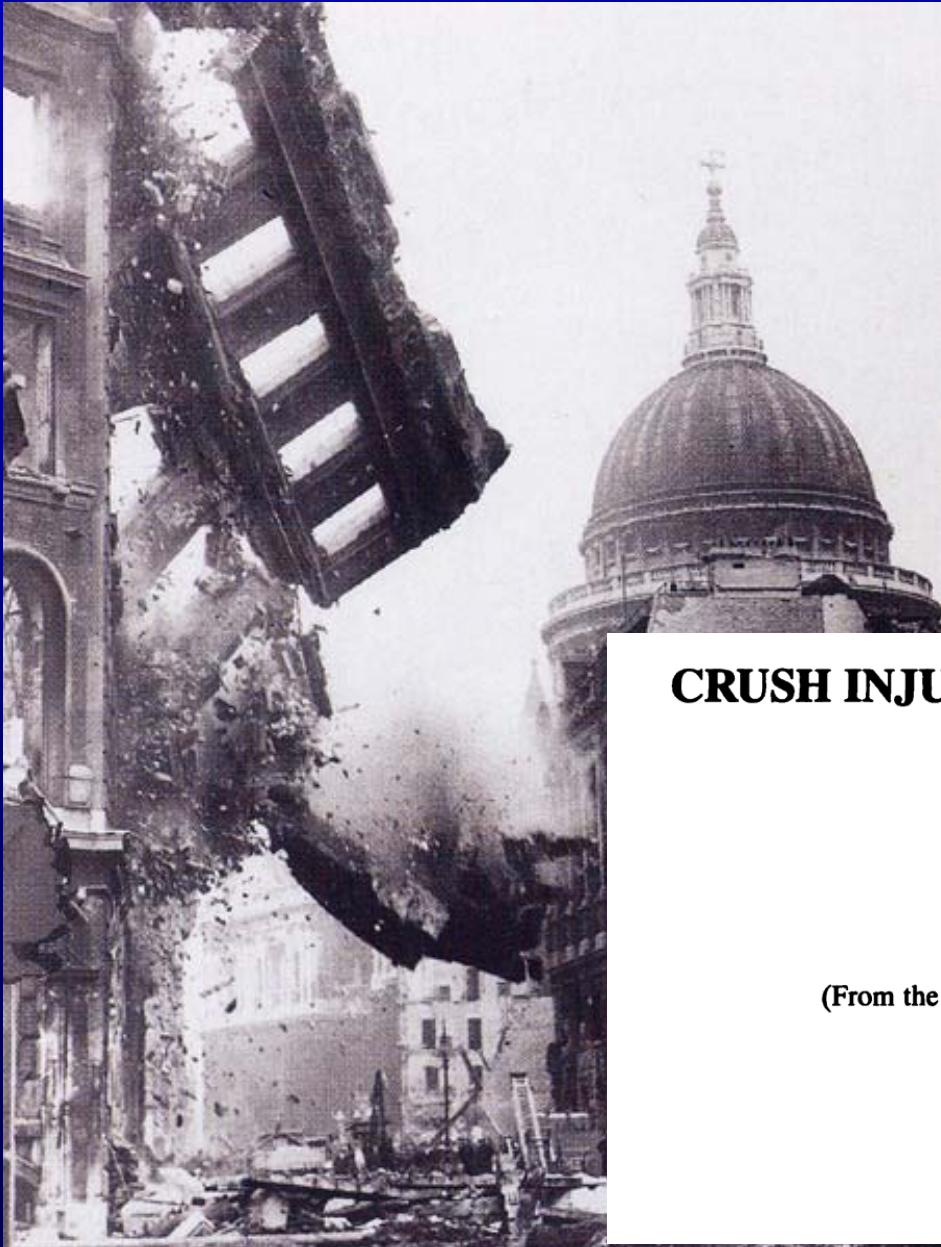
- is a collection of localized signs and symptoms that result when the perfusion pressure falls below the tissue pressure in a closed anatomic space for sufficient time that compromise of circulation and function of tissues involved occurs

# Crush Syndrome



First described in 1940  
during the London  
bombing

# Crush Syndrome



First described in 1940  
during the London  
bombing

## **CRUSH INJURIES WITH IMPAIRMENT OF RENAL FUNCTION**

BY

**E. G. L. BYWATERS, M.B., B.S., M.R.C.P.**

*Beit Memorial Fellow*

AND

**D. BEALL, Ph.D. Toronto**

(From the Departments of Medicine and Pathology, British Postgraduate Medical School)

[WITH SPECIAL PLATE]

with comments by

**ERIC G. L. BYWATERS AND JAMES P. KNOCH**

Reprinted from *Br. Med. J.* 1: 427-432, 1941

# Crush Syndrome

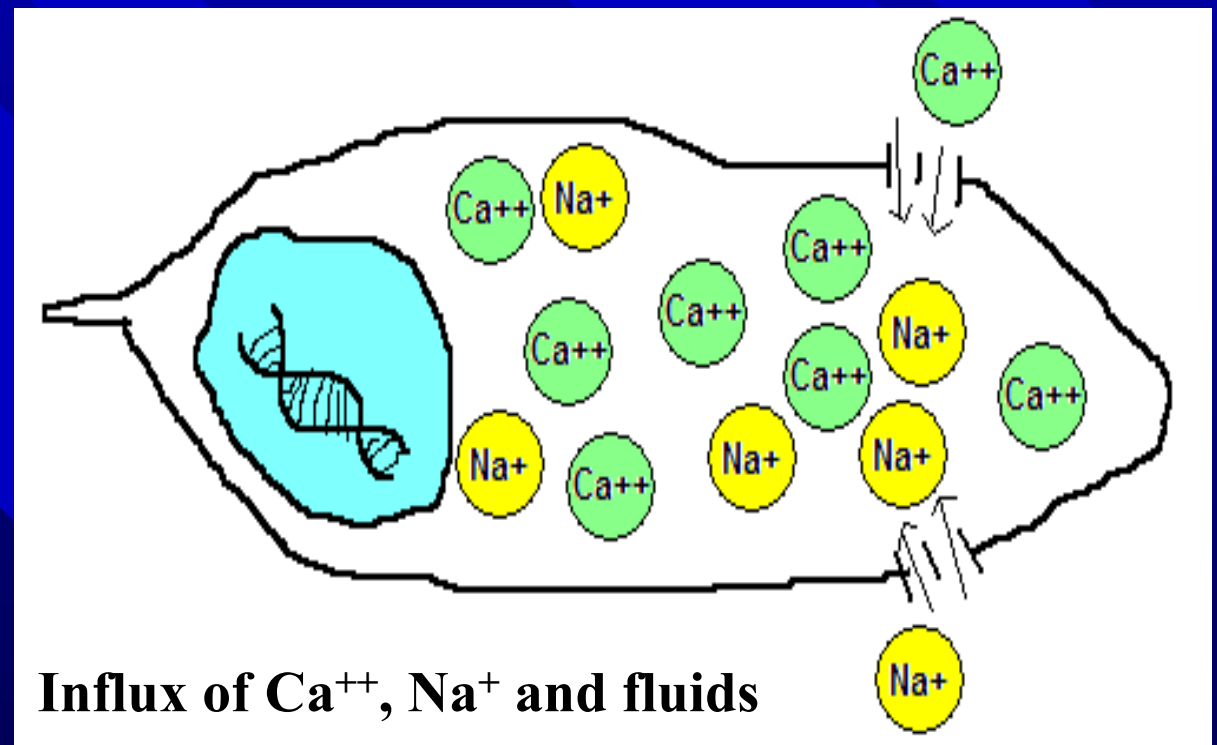
- **Systemic effects due to rhabdomyolysis and reperfusion of hypoxic and damaged tissues and is the major cause of early mortality**
- **Results most of the time from trauma or prolonged immobilization ( coma, poisoning,...)**
- **May occur in absence of trauma and evolve in the absence of early signs or symptoms**
  - **Arterial thrombosis or embolism**
  - **Drugs and toxic products ( i.e. carbon monoxide)**
  - **Toxins and envenomation**
  - **Severe anemia**

# Crush Syndrome: Pathophysiology

## Rhabdomyolysis

### ■ Efflux from damaged muscle cells of:

- Potassium
- Purines
- Lactic Acid
- Phosphate
- Myoglobin
- Thromboplastin
- Creatine



# Crush Syndrome : Pathophysiology

## ■ Reperfusion

- **Skeletal muscle damage greatest after reperfusion**
- **Superoxide radicals produced during reperfusion attacks free fatty acids, producing cellular edema, death, and necrosis**
- **Na-K-ATP pump exchanges intracellular sodium for calcium with further derangement of intracellular metabolism**

# Crush Syndrome: Pathophysiology

## Resultant effects of derangements due to rhabdomyolysis and reperfusion

■ Potassium	→ Hyperkalemia	→ Arrhythmias
■ Calcium	→ Hypocalcemia	→ Arrhythmias
■ Phosphate	→ Hyperphosphatemia	→ Renal damage
■ Myoglobin	→ Myoglobinemia	→ Renal damage
■ Fluid shifts	→ Hypovolemia	→ Renal failure
■ Reperfusion	→ Free radicals	→ Renal damage
■ Purines	→ Hyperuricemia	→ Renal damage
■ Hypoxemia	→ Lactic acid	→ Acidosis
■ Thromboplastin	→ Complement system	→ DIC
■ Creatinine	→ Elevated serum levels	
■ Sodium	→ Azotemia	

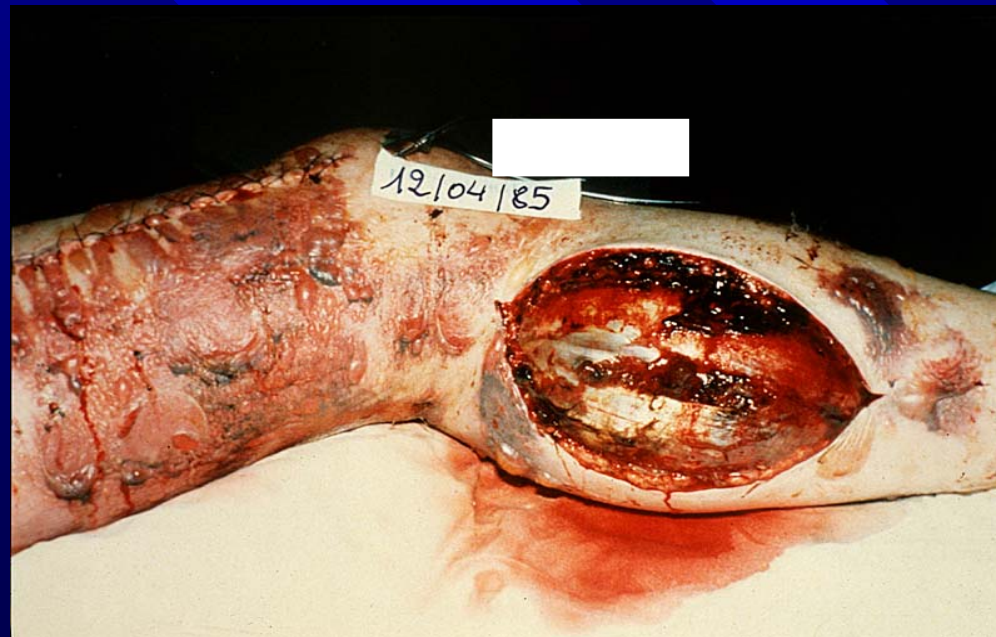
# Crush Syndrome: Treatment

- **The greatest danger is initial, during and after release of crushed limb from entrapment with restoration of circulation**
- **Mainstay of treatment is aggressive fluid resuscitation and brisk diuresis ( $> 200$  mL/h)**
- **Amount of tissue damage correlates with need for dialysis**
  - **Cannot determine actual tissue damage based on area of affected body part**
- **Delay in treatment associated with greater morbidity and mortality**
  - **50% renal failure at 6 hours**
  - **100% renal failure at 12 hours**
- **Rhabdomyolysis induced renal failure has 40% mortality**

# Compartment Syndrome

# Compartment Syndrome

- Is a collection of localized signs and symptoms that result when the perfusion pressure falls below the tissue pressure in a closed anatomic space for sufficient time that compromise of circulation and function of tissues involved occurs
- Can lead to crush syndrome systemic effects if left untreated or inadequately treated.



# Compartment Syndrome

## Suggestive clinical findings

- **Similar settings to crush injury, but may also occur with subacute trauma**
- **Bone fractures**
- **High velocity penetrating injury to muscles in closed compartment with extensive tissue disruption**
- **Can also occur in subacute fashion due to prolonged immobilization on hard surface**

# Compartment Syndrome: Pathophysiology

- **Significance of muscle mass damage**
- **Typically occurs in major muscle groups enclosed by inelastic, fibrous sheaths**
- **Tissue/muscle damage results in edema in a closed volume space**
  - **Progressive cycle of edema, perfusion compromise, tissue hypoxia and cellular derangement, further edema, etc.**
  - **Untreated, will produce same effects as crush injury**

# Compartment Syndrome: Clinical Presentation

- **The 5 P's**
  - **Pain**
  - **Pallor**
  - **Paresthesia**
  - **Paralysis**
  - **Pressure**
- **Progression of symptoms**
  - (sometimes the 6<sup>th</sup> P)

# Compartment Syndrome: Treatment

## Prehospital

- **Primary survey and initial stabilization (ABC's)**
- **Suspect compartment syndrome**
- **Immobilize affected part**
- **Treat other injuries**

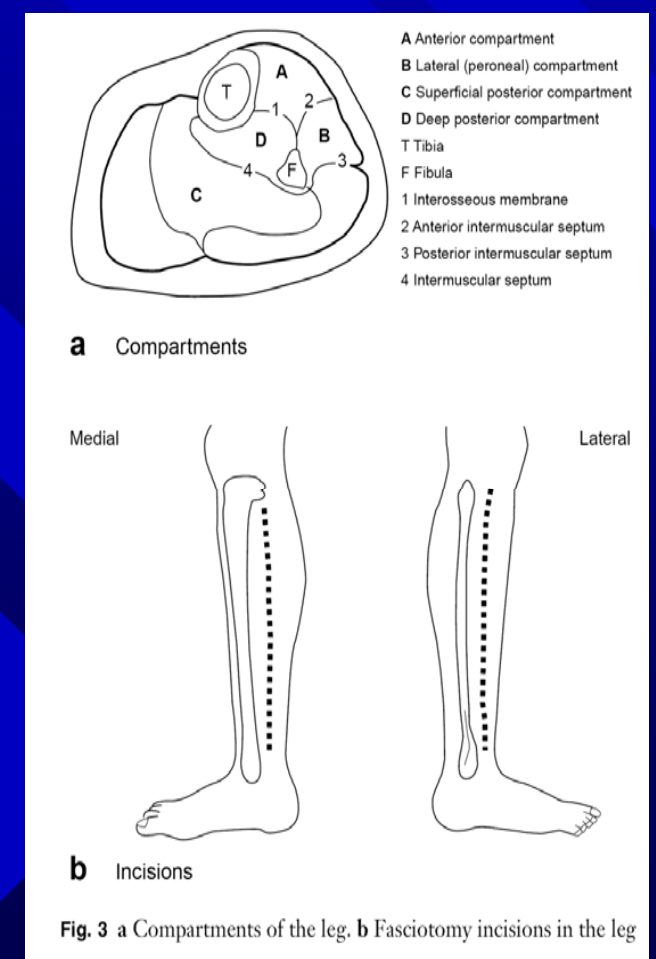
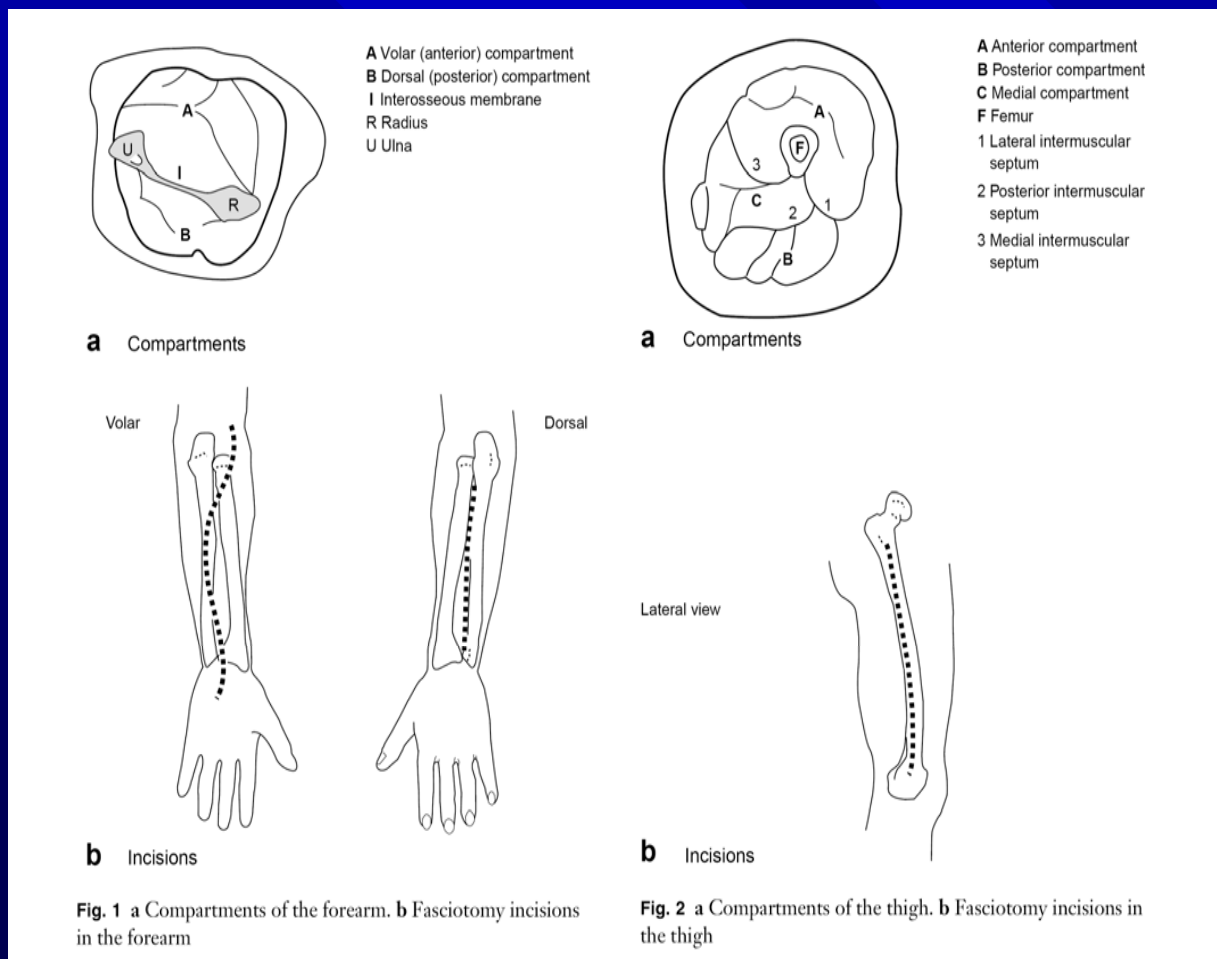
# Compartment Syndrome: Treatment

## Hospital

- **Primary survey, stabilization and resuscitation, secondary survey**
- **Diagnosis through examination**
- **Treat systemic effects of compartment syndrome similar to crush injury treatment**

# Compartment Syndrome Fasciotomy

Based on compartment pressure measurement



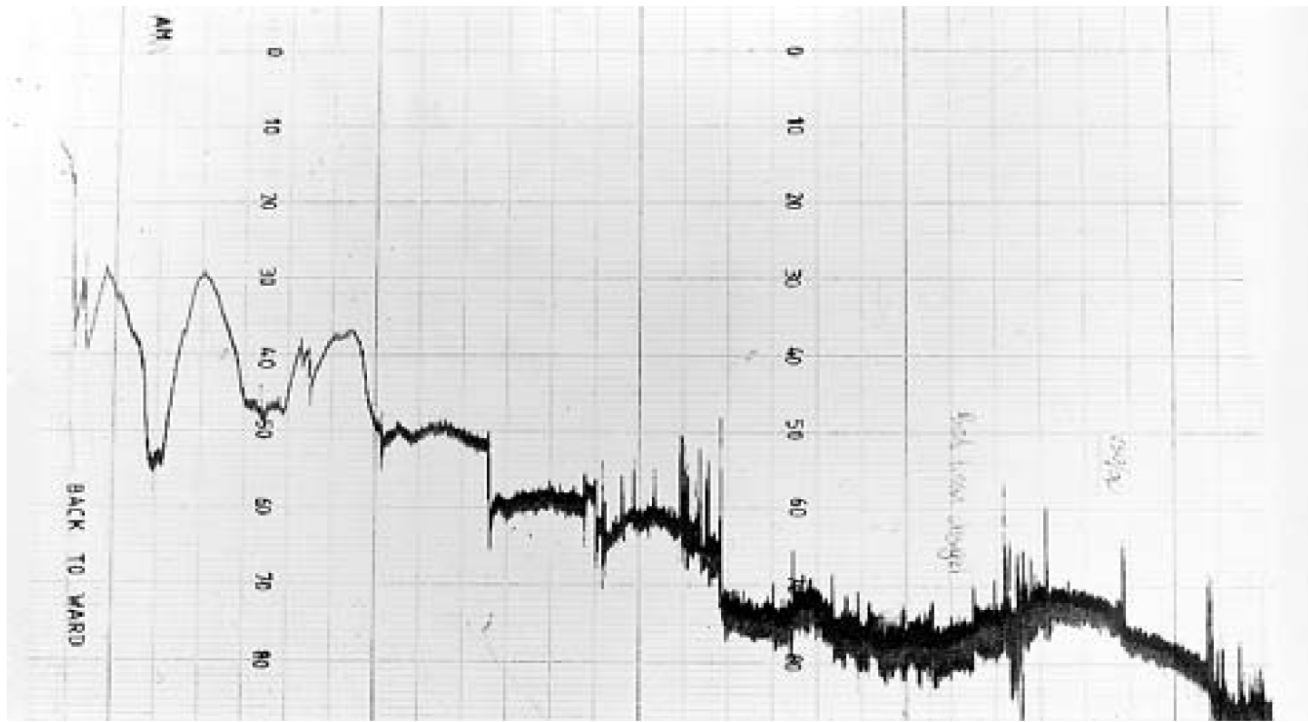


Fig. 1

Case 3. Compartment monitoring in a case of acute compartment syndrome showing the pressure increasing to 75 mmHg.



# Compartment Syndrome

## Fasciotomy

- Fasciotomies are a definitive treatment, but tissue pressure at which it is required is controversial.
  - Absolute compartment pressure  $>50$  mm Hg
  - Compartment perfusion pressure ( diastolic BP minus compartment pressure) lower than 30 mm Hg
- Fasciotomy has to be done early as delayed fasciotomy (beyond 48-72 hours) increases risk of sepsis and death due to extensive necrotic tissues

# HYPERBARIC OXYGEN THERAPY



# Hyperbaric Oxygen Therapy

## Mechanisms of action

- **Increase in tissue oxygen delivery**
- **Effects on tissue vascularization**
- **Effects on anaerobic bacteria and host defences against infection**
- **Effects on wound healing**

# Hyperbaric Oxygen Therapy

**Mechanisms of action**

**The « new » perspective**

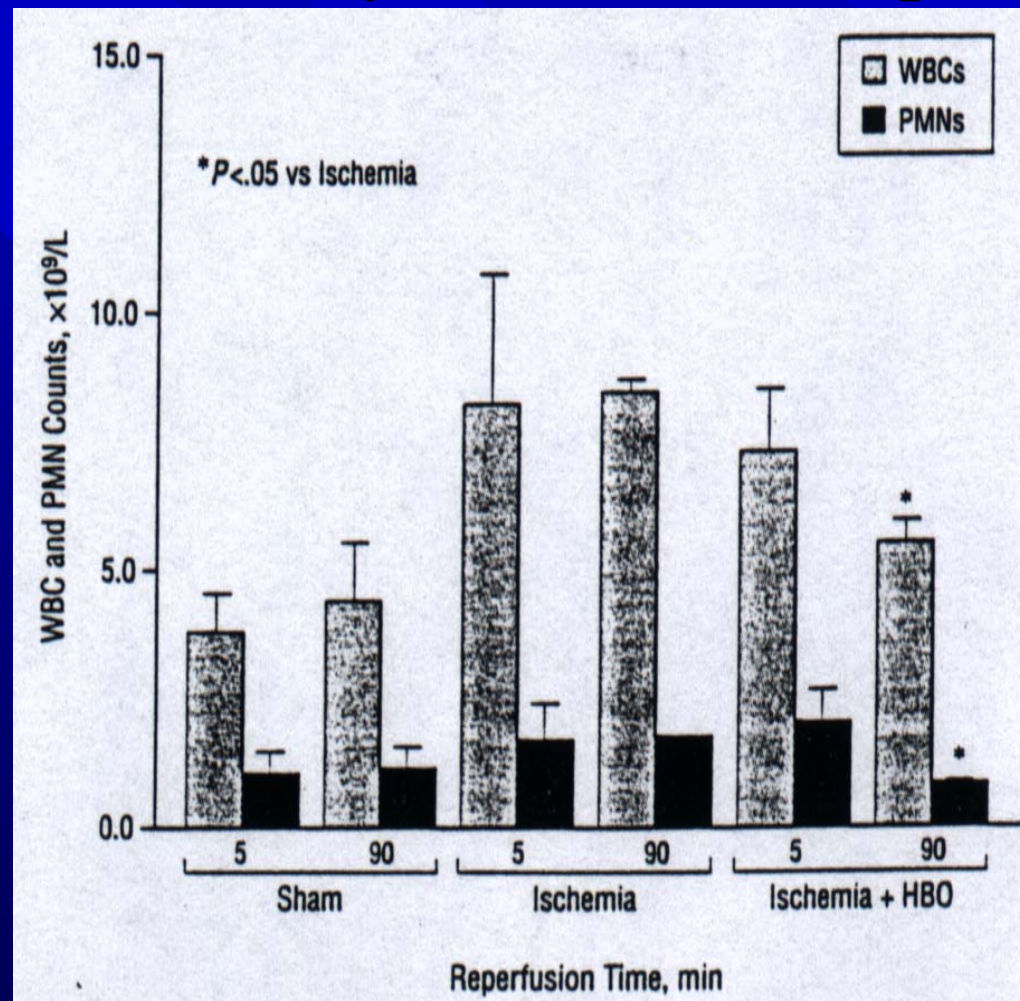
- **ischemia-reperfusion phenomenon**
- **leukocyte-endothelium interaction**
- **inflammatory reaction**
- **anti oxidant defences**
- **post aggressive apoptosis.**

# HBO and ischemia-reperfusion

**Model :** musculo-cutaneous flap

**HBO :** - improves flap survival

- reduces leukocyte and PMN sequestration in the flap



(Zamboni, 1996)

- reduces myeloperoxidase activity in the flap

(Hong, 2003)

# Hyperbaric Oxygen and leukocyte-endothelium interaction

**Model :**            **musculo-cutaneous flap.**

- HBO :**
- **decreases the number of leukocyte adherent to the endothelium**
  - **does not modify the expression of CD18**
  - **decreases the expression of ICAM-1 and ICAM-1 mRNA**  
(Hong, 2003)
  - **this decrease in ICAM-1 expression may be in part due to induction of eNOS**  
(Buras, 2000)

# Hyperbaric Oxygen and inflammatory reaction

**Model :** Human blood-derived monocyte-macrophages stimulated by LPS, lipid A, phytohaemagglutinin A

**HBO :**

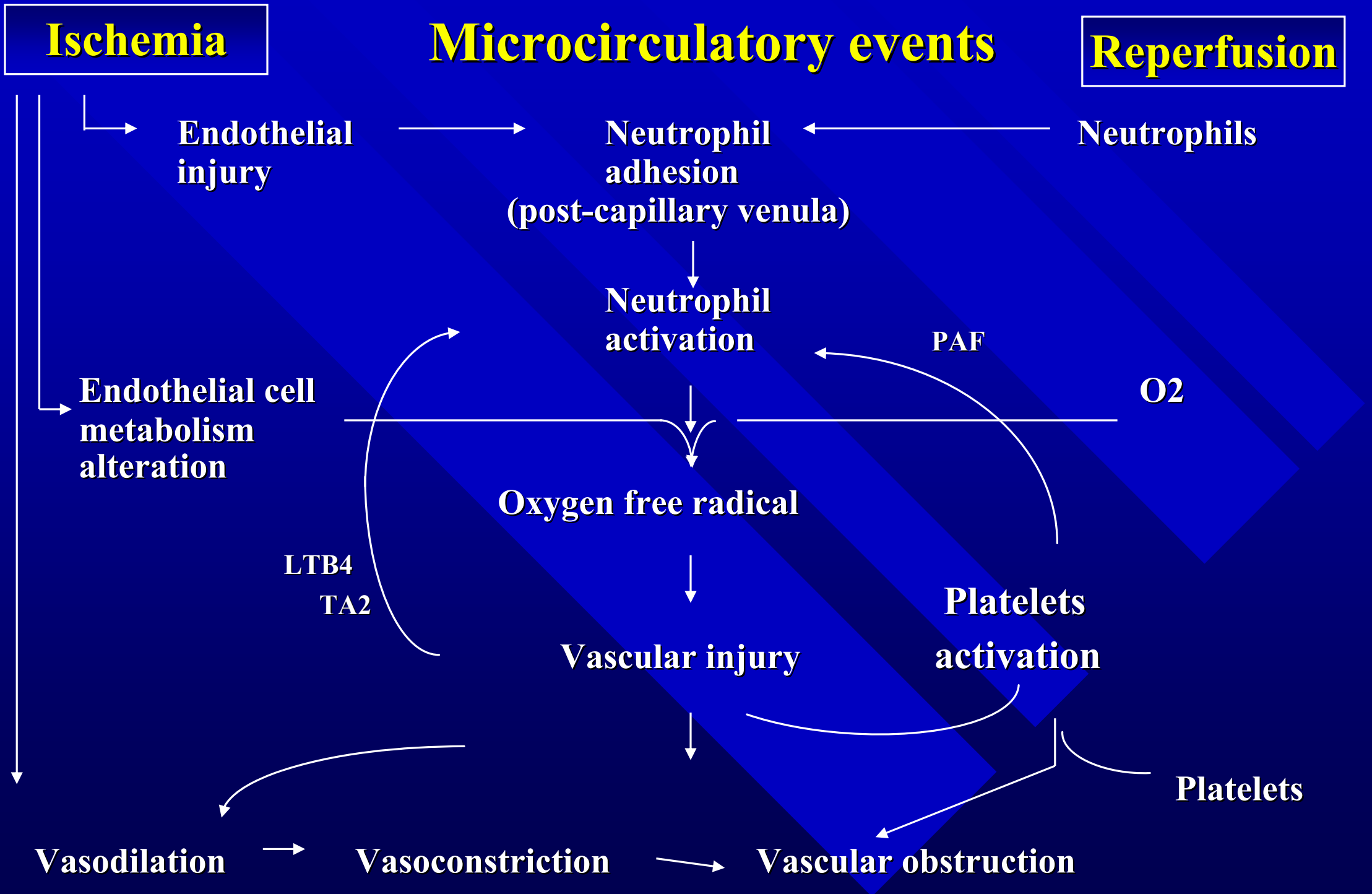
- decreases TNF alpha production
- decreases IL 1 beta production
- decreases IL 1 beta mRNA

(Benson, 2003)

- decreases interferon gamma secretion

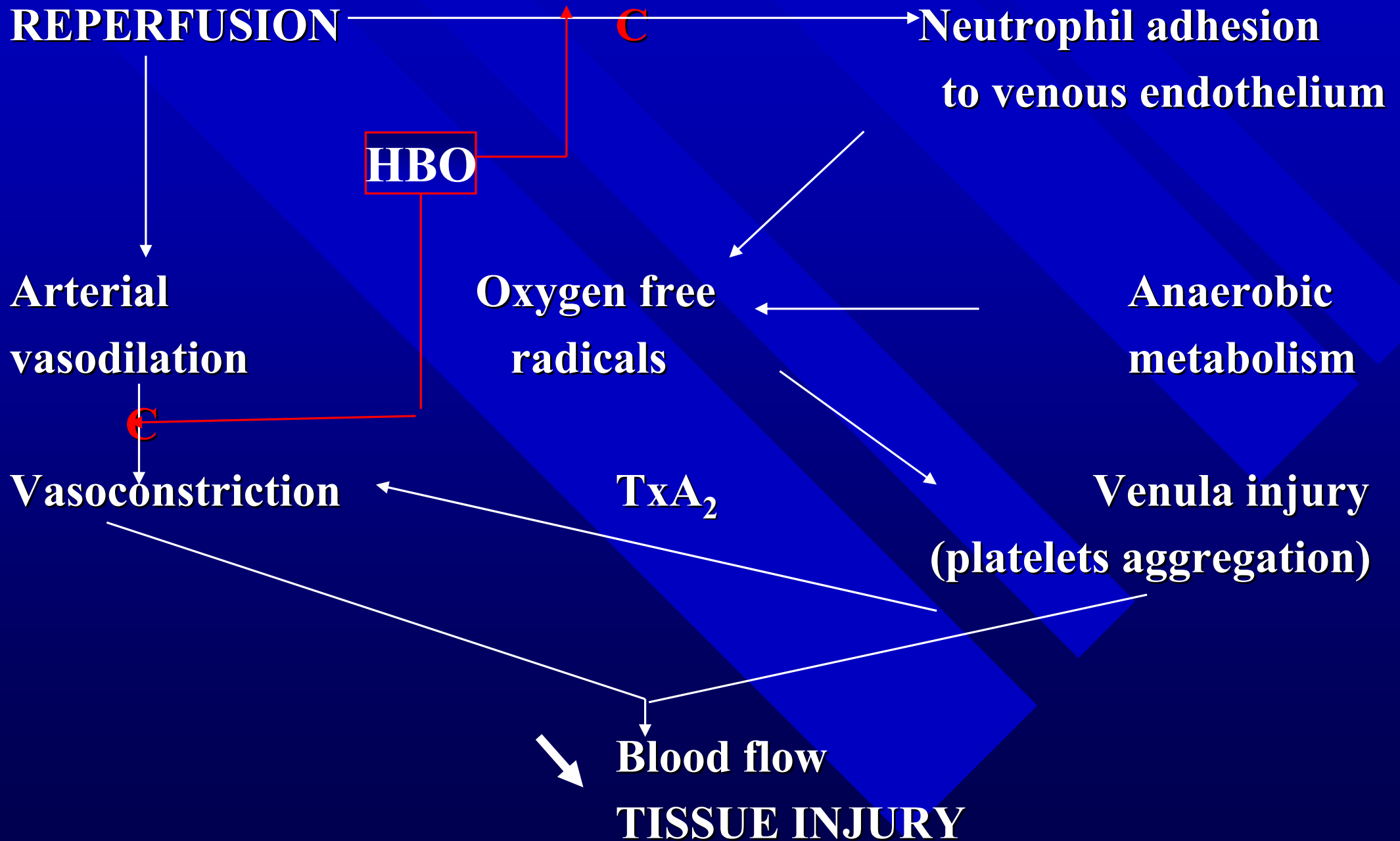
(Granowitz, 2002)

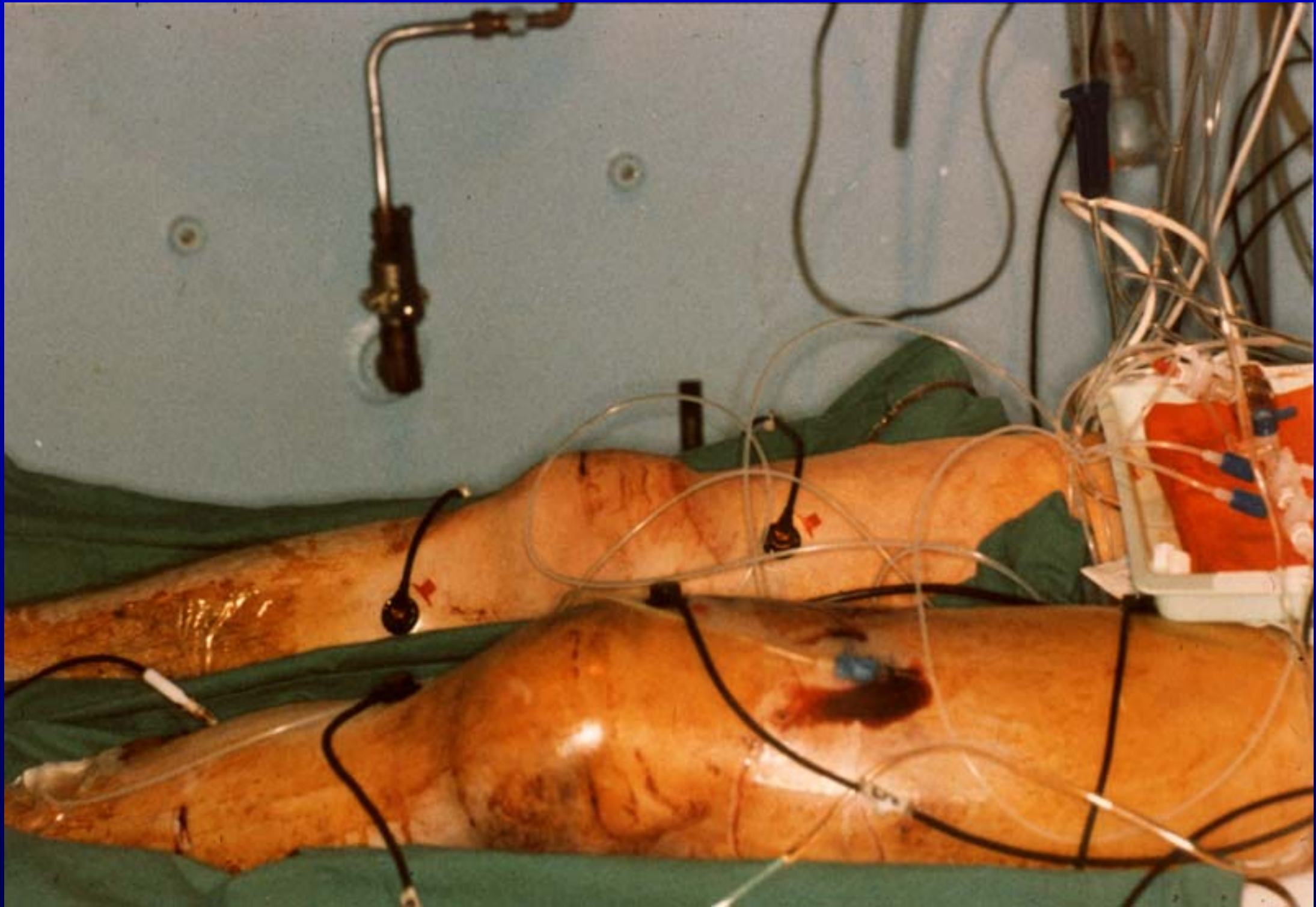
# ISCHEMIA REPERFUSION INJURY



# ISCHEMIA REPERFUSION INJURY

## HBO Effects





# LIMB CRUSH INJURY

## Effect of HBO

	1 ATA	1 ATA	2.5 ATA
	air	100 % O <sub>2</sub>	100 % O <sub>2</sub>
Compartment Pressure (mmHg)	30	26	20
P <sub>tc</sub> O <sub>2</sub> (mmHg)	20	240	810
Tissue PO <sub>2</sub> (mmHg)	12	40	80
Laser Doppler flow (PU)	35	80	82
Vasomotion	-	-	+

# Microcirculatory effect of HBO

## Hyperoxic blood flow redistribution

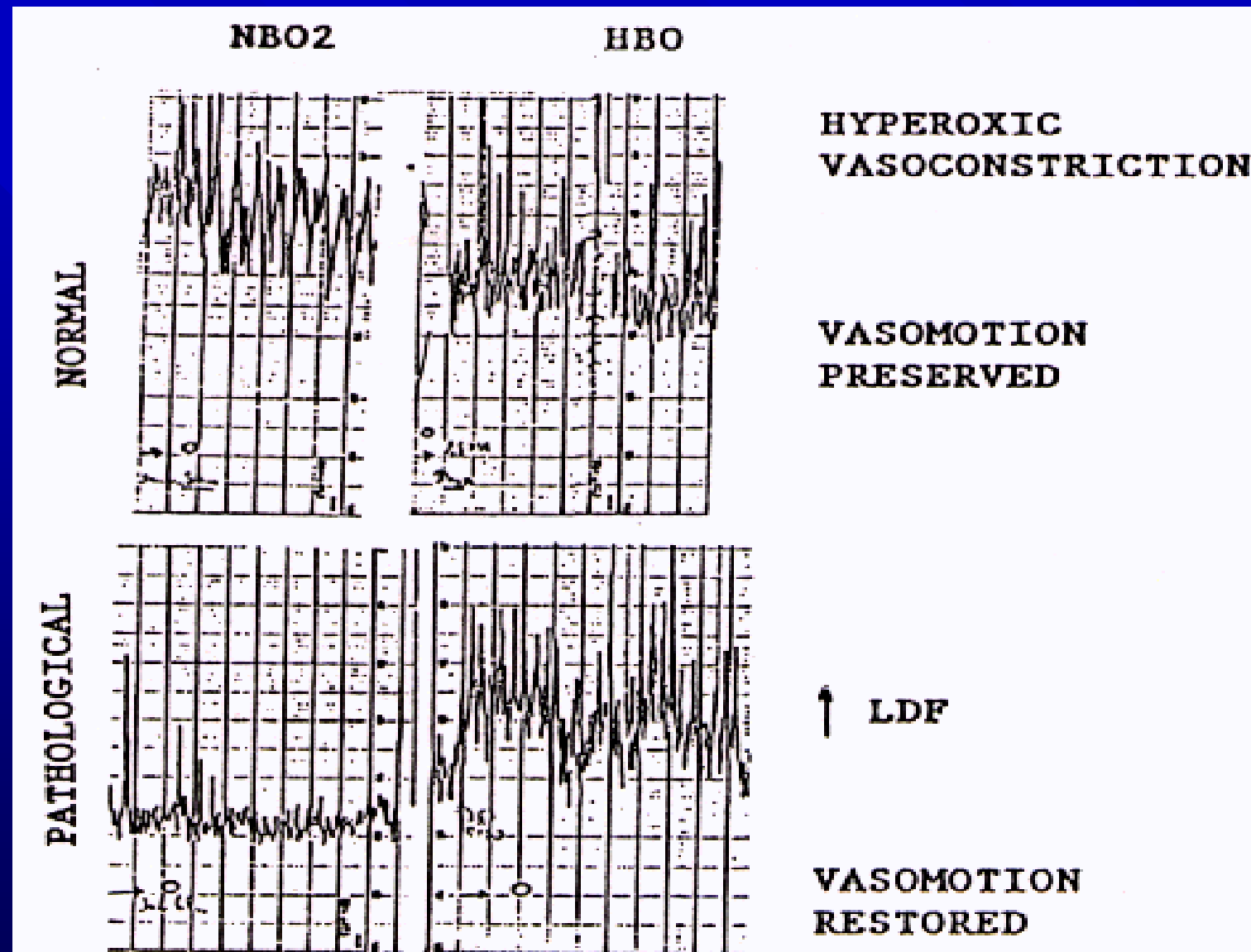
Transcutaneous pressures of oxygen			
Area	1 ata air	1 ata O <sub>2</sub>	2.5 ata O <sub>2</sub>
Subclavicular	38 ± 15	* 336 ± 94	* 1059 ± 166
Ischemic	23 ± 17	* 75 ± 40	* 405 ± 214
Contralateral	33 ± 22	* 218 ± 75	* 817 ± 192
Doppler laser blood flow			
Area	1 ata air	1 ata O <sub>2</sub>	2.5 ata O <sub>2</sub>
Ischemic	17 ± 11	ns 17 ± 9	ns 16 ± 9
Contralateral	15 ± 5	* 12 ± 4	* 9 ± 3

\* p < 0.05

Transcutaneous pressure of oxygen and amplitude of laser Doppler blood flow in various oxygen breathing pressures in 10 patients with unilateral limb traumatic ischemia

# LIMB CRUSH INJURY

## Effect of HBO



LDF - Laser Doppler  
Flowmetry

# HBO AND LIMB CRUSH SYNDROM

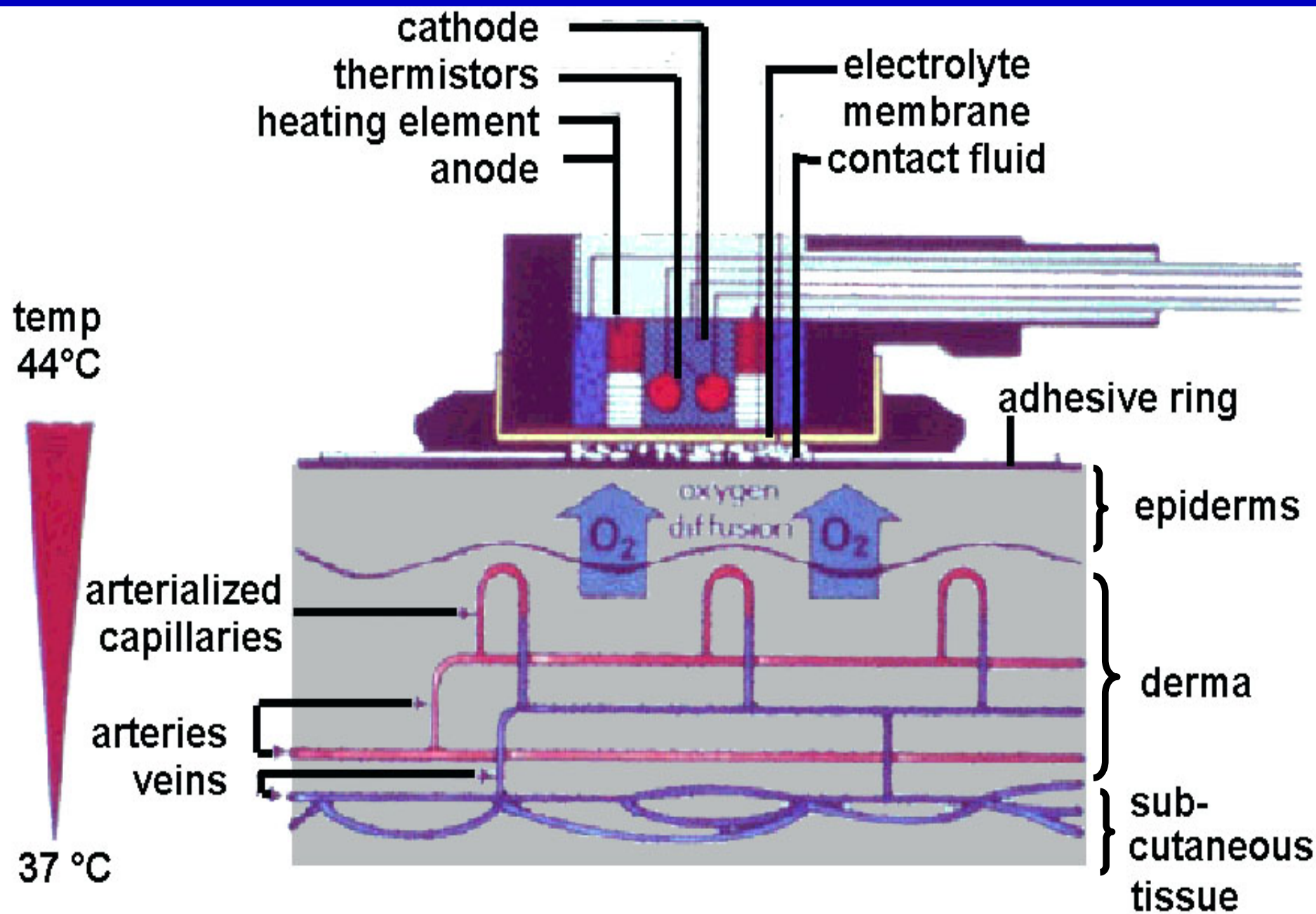
	<b>Control ( n=15 )</b>	<b>HBO ( n=16 )</b>
<b>Arterial trauma</b>	<b>0</b>	<b>2</b>
<b>Nerve trauma</b>	<b>0</b>	<b>2</b>
<b>Fractures</b>	<b>11</b>	<b>12</b>
<b>Open fractures ( Type III)</b>	<b>6</b>	<b>7</b>
<b>Soft tissue injury</b>		
<b>Type II</b>	<b>5</b>	<b>3</b>
<b>Type III</b>	<b>10</b>	<b>13</b>
<b>Injury Severity Score</b>	<b>9.3 +/- 1</b>	<b>9.5 +/- 1.4</b>
<b>Hospital length of stay (days)</b>	<b>23.3 +/- 16.8</b>	<b>22.4 +/- 12.4</b>

**Bouachour et al, 1994**

# HBO AND LIMB CRUSH SYNDROM

	<b>Control ( n=15 )</b>	<b>HBO ( n=16 )</b>	<b>p</b>
<b>Complete healing</b>	<b>7</b>	<b>15</b>	<b>0.004</b>
<b>Tissue necrosis</b>	<b>8</b>	<b>1</b>	
<b>Surgical procedures</b>	<b>8 (6 patients)</b>	<b>2 (1 patients)</b>	<b>0.015 (0.025)</b>
<b>Skin grafts and flaps</b>	<b>6</b>	<b>1</b>	
<b>Vascular surgery</b>	<b>0</b>	<b>1</b>	
<b>Amputation</b>	<b>2</b>	<b>0</b>	
<b>Dressing</b>	<b>16.8 +/- 9.5</b>	<b>16.9 +/- 13.2</b>	<b>n.s.</b>
<b>Time to complete healing (days)</b>	<b>58.7 +/- 19.1</b>	<b>49.3 +/- 21.6</b>	<b>n.s.</b>

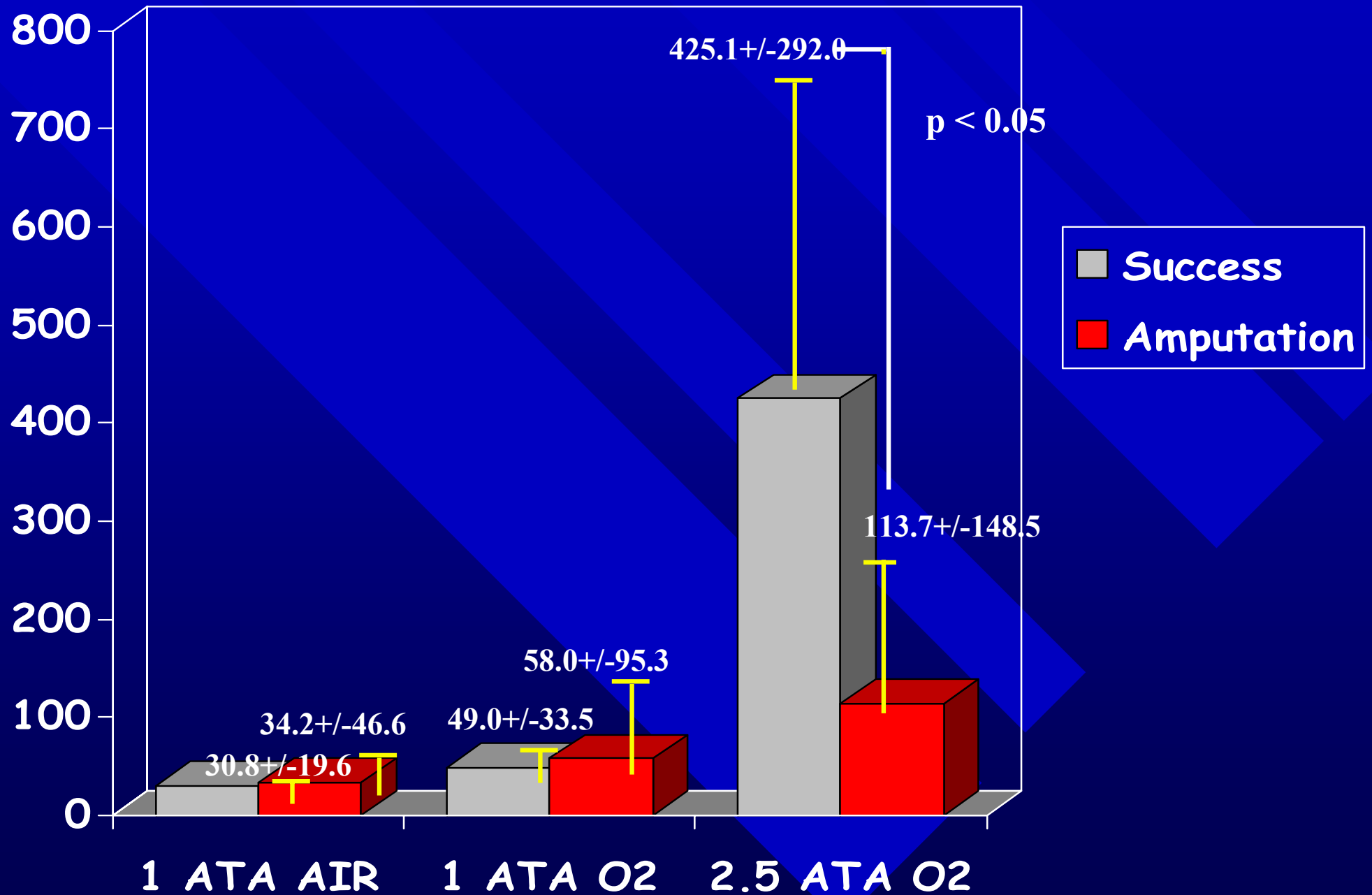
**Bouachour et al, 1994**

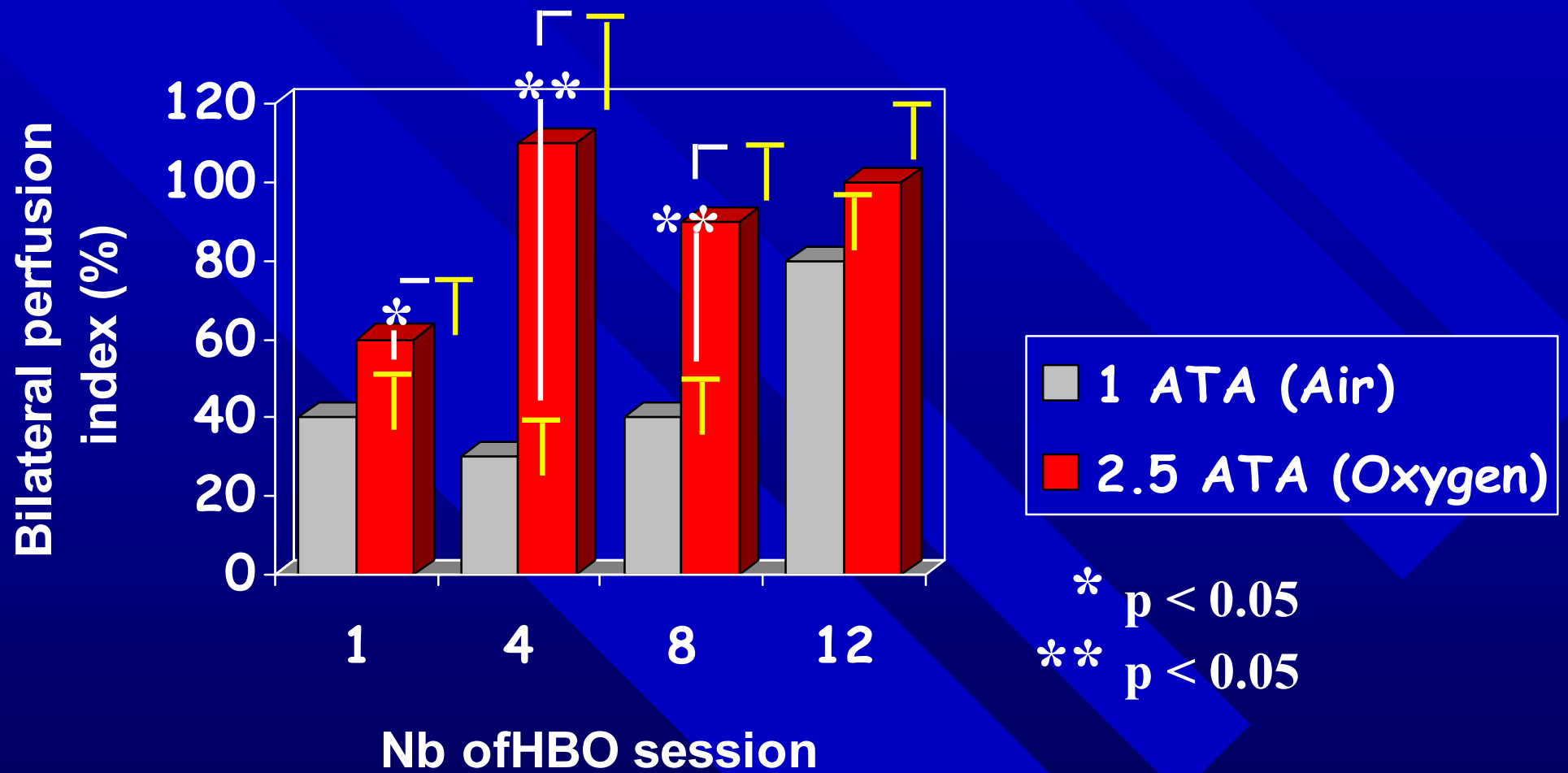


Principle of cutaneous pO<sub>2</sub> measurement by heated oxygen sensor

# Transcutaneous Oxygen Pressure

## Arterial traumatic ischemia





# Crush syndrome

- **Clinical efficacy evidenced for the number of surgical procedures and complete healing time (Bouachour et al, J Trauma, 1996).**
- **Indication : open fracture grade III B and III C of Gustillo classification.**
- **Recommendation type I level 2 (Milano, 1996 – Lille, 2004).**

Type	Mechanism	Infection rate	Recommendation
<b>I</b>	Small laceration < 1 cm	Minimal	No HBO
<b>II</b>	Large laceration, but minimal soft tissue damage	3%	No HBO
<b>III</b> Sub type <b>A</b>	Crush injury : Sufficient soft tissue to close wound (primary or delayed)	Infection 4 % amputation 0 %	No HBO except high risk patients
Sub type <b>B</b>	Flaps or grafts required to cover bone	Infection 52 % amputation 16 %	HBO
Sub type <b>C</b>	Major vessel injury	Infection 42 % amputation 42%	HBO

