招待講演1

The Effects of Hyperbaric Oxygen on Soft Tissue Injury

Michael B. Strauss

Department of Hyperbaric Medicine Long Beach Memorial Medical Center

Introduction There is excellent justification for using Hyperbaric oxygen (HBO) therapy as an adjunct for managing soft tissue injuries (STI) when healing is not occurring as expected and/or complications for STI is as an adjunct for managing diabetic foot infections. Other STI for which HBO is a useful adjunct include threatened flaps and grafts, crush injuries and compartment syndromes, necrotizing soft tissue infections, radiation injury of soft tissue and burns. This paper describes the physiological effects of HBO that mitigate the above soft tissue injuries.

Primary Mechanisms of Hyperbaric Oxygen Physics and physiological data confirm why HBO is a useful adjunct for managing STI. Mechanisms of HBO are either primary or secondary. The primary mechanisms have immediate and direct effects on pathological conditions, are transient, that is disappearing rapidly after the HBO treatment is completed, and initiate the secondary mechanisms. Hyperoxygenation and bubble reduction are the two primary mechanisms of HBO. Secondary mechanisms promote healing and minimize soft tissue injury. They give HBO its durable results.

Hyperoxygenation Hyperoxygenation is the essential primary mechanism of HBO that influences STI. At a pressure of 2 atmospheres absolute (ATA) which is equivalent to 10 meters of sea water pressure, HBO increases plasma and tissue oxygen tensions 10-fold, blood oxygen content by 125 percent and diffusion of oxygen through tissue fluids 3-fold. This adds approximately five volumespercent of oxygen to the plasma and nearly matches the normal arterial-venous oxygen extraction that occurs at the capillary level (Figure 1). This HBO effect from the physically dissolved oxygen in the plasma permits survival and function of tissues in the absence of red blood cell carried oxygen and was so confirmed by Boerma, et al. in their classic "Life without Oxygen" study. 1

Oxygen is physically forced into the plasma in direct proportion to the inhaled partial pressure of oxygen in the breathing medium. For each millimeter increment of oxygen partial pressure in the breathing medium, 0.003 volumes-percent of oxygen is forced into the plasma and secondarily diffused into the tissue fluids. At 2 ATA this results in almost a five volumes percent increment in blood oxygen content and ten-fold increases in plasma and tissue oxygen tensions. Since oxygen diffusion through tissue fluids is proportional to the square root of the increase in oxygen tension in the plasma, at 2 ATA the 10-fold increase in plasma oxygen content increases the diffusion distance approximately 3-fold (the square root of the 10-fold increase).

Secondary Mechanisms Secondary mechanisms of HBO occur because of the transient effects HBO has on target tissues and microorganisms. Target cell types include fibroblasts, smooth muscle cells of arteries, neutrophils, endothelial cells, microorganisms and possibly red blood cells. Secondary mechanisms initiate effects on host factors essential for wound healing, vasoconstriction for edema reduction, inhibition and death of microorganisms, perturbation of the reperfusion injury and possibly augmentation of red blood cell deformation. Whereas, the hyperoxygenation effect is immediate and transient and directly proportional to the partial pressure of inhaled oxygen, the secondary effects usually take days to weeks to be fully achieved, are directed towards cell function rather than cell survival as is the case with the primary effect of hyperoxygenation, seem to be most effective in pulsed doses once or twice a day and at pressures of two atmospheres absolute. Finally, there does not appear to be toxicity to the cells responsible for the secondary effects of HBO in contrast to oxygen toxicity from the direct effects of hyperoxygenation.

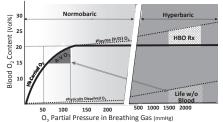
Soft tissue injuries Mitigated by HBO There are documented benefits of hyperbaric oxygen for at least six different soft tissue injury types. 4) Hyperbaric oxygen should be considered for them when healing concerns are observed and/or anticipated. These injures include diabetic foot infections, threatened flaps and grafts, crush injuries and compartment syndromes, necrotizing soft tissue infections, radiation injury of soft tissues and burns. Specific secondary mechanisms have precise effects for each soft tissue injury (Table 1). Usually there is one most important secondary mechanism for mitigating each soft tissue injury with other mechanisms contributing in varying degrees to the healing process.

Conclusions Whenever healing problems occur and/or are anticipated with soft tissue injuries, HBO should be used as a management adjunct. Secondary mechanisms of HBO in conjunction with hyperoxygenation result in the healing responses observed for STI.

References

- 1) Boerma, I, MG Meyne, WK Brummelkamp, et al., Life without blood. A study of the influence of high attmospheric pressure and hyperthermia on dilution of the blood., J Cardiovasc Surg, 1960; 1:133-146
- 2) Krogh, A, The number of distribution of capillaries in muscle with calculation of the oxygen pressure head necessary for supplying the tissue. J Physiol, 1919; 52:409-415
- 3) Pierce, EC II, Pathophsiology, apparatus and methods, including the special techniques of hypothermia and hyperbaric oxygen. Extracorporal Circulation for Open-Heart Surgery, 1969, Charles C. Thomas, Springfield, IL, 84-88
- 4) Strauss, MB, IV Aksenov, SS Miller, MasterMinding Wounds, Best Publishing Company, Flagstaff AZ, 2010

Figure 1 The Hyperoxygenation Effect of Hyperbaric Oxygen



Legend HBO increases the blood O2 content by adding O2 to the plasma. Normally 5 **Legend** HBO increases the blood O_2 content by adding O_2 to the plasma. Normally 5 volumes percent of O_2 is extracted from the capillary bed as blood passes from the arterial to the venous side of the capillary $(A-V \ O_2 \ difference)$. With HBO at > 2 ATA enough O_2 is physically dissolved in the plasma to meet the $A-V \ O_2$ extraction in the capillary bed. This was the physiological basis for Boerma's "Life without Blood" study. Once hemoglobin becomes fully saturated with O_2 (at an O_2 partial pressure of the breathing gas about 100 mmHg), no more O_2 is added to the hemoglobin carried O_2 in the blood $(A-V) \ O_2 = A$ arterial-Venous Oxygen $(A-V) \ O_2 = A$ hemoglobin, $(A-V) \ O_2 = A$ hemoglobin, $(A-V) \ O_2 = A$ hemoglobin, $(A-V) \ O_3 =$

Table 1 Secondary Mechanisms of Hyperbaric Oxygen Useful for Soft Tissue Injuries

	Host Factors Elbroblasts, WIIC killing, angiogenesis	Vaso- constriction	Microbes Killing, bacteriostasis	Reperfusion Injury	Enhance RBC Deformability	Comments	
Diabetic Foot Infections	**	_	*	_	+	Angiogenesis & WBC oxidative killing particularly important	
Threatened Flaps/Grafts	-	_	_	-	4	Hyperoxygenation (primary mechanism) with increased diffusion the main effect	
Crush Injury	-	**	-	*	-	Edema reduction & hyper- oxygenation also apply to compartment syndromes	
Necrotizing soft tissue infections	*	-	**	_	-	Initial effects on microbes— especially aerobes supplemented by effects on host factors	
Radiation Injury	**	_	4	_	_	Hyperoxygenation with increased diffusion provides environment to promote angiogenesis	
Burns	*	*	-	-	-	Hyperoxygenation helps margina tissues survive; edema reduction improves perfusion	

Legend Secondary mechanisms of HBO are largely responsible for the benefits observed when this modality is used as an adjunct for managing soft tissue injuries. In several injuries (e.g. threatened flaps &burns), the (primary)hyperoxygenation effect is the crucial mechanism that improves outcomes. Enhanced RBC deformability may improve oxygen delivery to tissues where sludging and low blood flow are present Key = strong effect (doubly so with 2 stars), = probable, but less important effect of the secondary mechanism, — = No effect from the secondary mechanism, HBO =

Hyperbaric Oxygen, RBC = Red Blood Cell, WBC = White blood cell (neutrophil)