特別講演1 SL1

Traumatic Ischemias and the Roles of Hyperbaric Oxygen

Focusing on Crush Injuries & A Physiological Model for DCS Presentations

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Crush Injury is the most recognized of the spectrum of traumatic ischemias. Hyperbaric oxygen is an "approved" use by most governing authorities for the traumatic ischemias. Three features unify crush injuries and the other conditions in the spectrum of traumatic ischemias. **First**, there is a trauma cause for the condition that leads to ischemia at the injury site. For example, energy exchange to tissues is the cause of the pathology for crush injuries while thermal injury i.e., heat or cold, is the trauma source for burns and frostbite. Trauma inherit in every surgery is the etiology for threatened flaps & grafts, failing amputations, and reimplantation concerns. **Second**, the trauma plus ischemia to the injured tissue contributes to a self-perpetuating cycle of edema and hypoxia in the post-injury stage of the traumatic ischemias. **Third**, a spectrum of severities of the traumatic ischemias range from non-existent/mild- to moderate-to severe/tissue death. Hyperbaric oxygen through its mechanisms is able to mitigate the pathology for each of the three unifying features of the traumatic ischemias. This presentation focuses on the crush injury component of the traumatic ischemias with discussion of its pathology, classification, treatment protocol, outcomes, and rationale for using hyperbaric oxygen.

Decompression sickness (DCS) is a syndrome due to its varied presentations & locations. Scientific work on DCS focuses on more than 20 models for predicting bubble formations using tissue halftimes. Four index cases & awareness of the variability of perfusion provide a physiological explanation why signs & symptoms of DCS occur as they do. These cases raised questions about the predictability of tissue half times (& even Wienke's free gas phase concept) for explaining these patients' outcomes. Interruption of autoregulation of perfusion coupled with off-gassing gradients provide an explanation. With over a 96,500 Km in line length of our vascular tree, its capacity is, at a minimum, 20-times greater than our 5-liter blood volume. Thus, blood flows where it is needed due to exacting regulation by the sympathetic nervous system & chemical mediators. The index cases: 1) Deaths after precipitous ascent in hard hat gear; 2 & 3) Severe neurological residuals after transient loss of consciousness upon dive completions; and 4) Cardiac arrest on the bottom with intravascular bubbles upon ascent even with CPR led us to generate a 3-compartment model based on physiology rather than tissue half times Transient interruption of perfusion to instantaneously saturated tissues (lungs, blood, heart, and brain-spinal cord) can cause severe neurological residuals from autochthonous bubble formation. A "protected" highly perfusion-regulated group of tissues (muscle, viscera, bone, skin, & subcutaneous tissues) only have perfusion proportional to their activity/metabolic needs and rarely cause symptoms with decompression. Avascular connective tissues (ligaments, tendons, fascia, joint capsules, & membranes) on- and offgas slowly by diffusion from tissue fluids associated with long or deep dives. My 3-compartment perfusion-gradient, physiologically-based model explains why DCS presentations appear as they do.