Brain Damage and Hyperbaric Oxygenation

It is common knowledge that divers risk the classical joint pain of the "bends," but they also risk damage to the nervous system from bubbles. The problem is also encountered in military aircraft flying at very high altitude and in extravehicular activity on space missions. What has recently emerged from a more detailed understanding of the pathphysiology of circulating bubbles is relevant to many other areas of medicine. Bubbles can enter the circulation of divers in two ways. If the lung tears on a rapid ascent, because of the expansion of trapped gas, large bubbles can enter the systemic arterial circulation. If they reach the brain, they may cause death (Figure 1) or may result in stroke. Similar problems occur from bubbles in cardiopulmonary bypass surgery in the "post-pump syndrome." Although air bubbles have been regarded as occlusive agents simply obstructing blood flow, their effects are much more complex. Some tissue necrosis may be caused by the ischemia, but it is now known that, as in stroke, a much larger volume of brain tissue is affected. This zone, identified as the ischemic penumbra, is associated with Edema, because of increased permeability of the blood-brain barrier. This may be severe enough to be associated with diapedetic hemorrhage, as is shown in Figure 1. Brain lesions may evolve over many years and a pathological study of a gunshot wound to the brain after a survival of 22 years has shown changes still continuing with damaged, but preserved, neurons present.

Hyperbaric therapy is well established as the definitive treatment for air embolism and was originally based simply on reducing the size of bubbles by increasing barometric pressure. However, since 1966, pure oxygen rather than air has been introduced in hyperbaric therapy with greater success. It has now been recognized that the improved outcome is due to the greatly increased plasma oxygen content constricting dilated blood vessels and restoring the blood-brain barrier. In addition, from studies of the benefit of hyperbaric oxygenation of reperfusion injury in re-implantation surgery, it has been shown that a high plasma oxygen concentration reduces leukocyte adhesion and improves endothelial function. In the second mechanism where gas enters the circulation in divers, it is derived from excess nitrogen content or super saturation on decompression and so is known as decompression sickness. Most dives of any significance form some bubbles, which are generally microscopic. They arise on the venous side of the circulation pass through the right heart and are normally filtered by the lung. However, trans-pulmonary passage may occur and transfer to the systemic arterial circulation is also possible via an atrial septal defect. In transit through the vasculature of the CNS, micro bubbles cause an immediate focal disturbance of the blood brain barrier, which, paradoxically, affects the veins principally of the white matter. In very severe cases, this may present clinically as an acute leuco-encephalomyelitis, but decompression sickness can also mimic the individual neurological syndromes, such as transverse myelitis, Bell's palsy, or optic neuritis, which collectively are known as multiple sclerosis (MS).

Figure 2 (not on e-mail) shows the appearance of the lower thoracic spinal cord in experimental decompression sickness. The associated edema causes a reduction in oxygen transport because of the increased tissue water content and the extra-vasation of proteins causes inflammation. The presence of hypoxia has yet to be demonstrated in the lesions of decompression sickness, However, has been found in acute plaques in MS using magnetic resonance spectroscopy.

The well-established success of hyperbaric oxygen therapy in the immediate treatment of the focal CNS lesions of decompression sickness is relevant to the treatment of other neurological diseases associated with disturbance of the blood brain barrier.

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