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1. The Present Circumstances of Hyperbaric Medicine in China

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China has been developing her hyperbaric medicine including both aspects; clinical application of hyperbaric oxygen treatment of disorders and reasearch into their mechanisms, medical and physiological research during saturation driving experiments.

I. Clinical application of hyperbaric oxygen.

Hyperbaric oxygen treatment in which a patient breathes pure oxygen intermittently while the pressure of the treatment chamber is increased to a point higher than sea level in either a mono- or multiplace chamber.

In China, the special committee on hyperbaric oxygenation was appointed in 1984 under the committee on surgery of the Chinese society of medicine. Then the Chinese society of hyperbaric oxygenation was founded in 1992 to faster exchange of date on the hyperbaric medicine and physiology. At present the society has grown to nearly 2000 members and reported the results of research and clinical aspects of hyperbaric medicine annually at scientific meeting and in Chinese journal of hyperbaric oxygenation medicine. From 1963 to 1992 in China were held 7 national conferences of hyperbaric oxygenation medicine, and 1000 representatives were present and reported 600 articles. The XI International Congress on Hyperbaric Medicine was held in Fuzho of China in 1993 by the host of professor Li Wen-ren.

In 1980 the special committee of hyperbaric oxygenation medicine recommended 44 indications to hyperbaric oxygen treatment; 28 of them are refind clinical efficacy, the others are experimental. In the former case there are cases of acute carbon monoxide poisoning, cerebral edema, acute high altitude sickness, cardiac infarction, acute coronary insufficiency, cerebral infarction, shock, unconsciousness due to head and intracranial surgery, severe hypoxia with cerebral disorders, retinal artery occlusion, idiopatic sudden deafness, anemia, exceptional blood loss, meningitis, radiation, myelitis, skin grafts and flaps, and radionecrosis etc.

In our institute, between April 1989 and May 1994 using the hyperbaric oxygen we have to-tally treated 2351 cases and 65705 courses wiht 470 cases/year, 13141 courses/year, and 27.9 courses/case, averagely. These cases included decompression sickness, acute carbon monoxide poisoning, gas gangrene, air embolism, cerebral ischemia, idiopatic sudden deafness and cerebrovascular diseases, etc. The results were remarkable.

In China first hyperbaric chamber was built by professor Li Wen-ren in 1964 for clinical use in Fujian Provincial Hospital. From then on, hyperbaric oxygen has become an useful treatment of clinical disorders; 560 monoplace chambers, they have been designated by Chinese state medical burean to produce medical hyperbaric oxygen chambers.

II. Hyperbaric medical and physiological research during saturation diving experiments.

Since 1975 in China a series of experiments including air saturation diving, nitrogen-

oxygen, helium-nitrogen-oxygen and helium-oxygen saturation diving have been carried out. In addition to these Chinese scientists also performed a 5000m high altitude saturation diving experiment. In the experiments mentioned above we concentrated our researches on hyperbaric medicine, physiology and decompression table, the influence of hypobaric pressure and hyperbaric pressure and of hyperoxia and hypoxia on physiological changes as well as on the underwater working capability and sleep function, etc.

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2. The Role of Complement and Neutrophils in Air Bubble-Induced Lung Injury

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Pulmonary air embolism (PAE) occurs in diving and aviation during acute reduction of ambient pressure. It is also a complication in clinical procedures, such as in an upright patient during neurological surgery, gunshot wound to the chest, or accidental introduction through intravascular catheters. Pulmonary arterioles and capillaries trip air bubbles, thereby prevents the cerebral and coronary circulation from fatal embolization. This non-respiratory function of lung, however, leads to acute lung injuries, which may be a consequence of mechanical obstruction, altered biochemical environment, or both. While there is no doubt that the ischemia and deformation effects of bubbles (or other solid emboli) play a role, bubbles can produce a number of indirect effects leading to tissue damage. Hemodynamic consequences of PAE and the involvement of the complement system and leukocytes are the focal points of this presentation.

Hemodynamics, PAE-induced circulatory changes in rats, cats, rabbits, dogs, sheep, and humans are similar in that PAE causes pulmonary hypertension, systemic hypotension, a decreased cardiac output, and poor gas exchanges. The complement system is involved because decomplementation, by pretreatment with cobra venom factor CVF^h or repeated compression-decompression cycles, reduces hemodynamic effects of PAE

in the rat. The involvement of leukocytes in pulmonary hypertension was suggested, in an isolated and perfused lung model, that thromboxane B_2 and chemiluminescence rose in the perfusate following PAE.

Lung Injury. PAE alters biochemical environment of the lung by activating complement system and blood cell components. Release of vasoactive substances, mediators, oxygen radicals may cause lung injury. Besides microanatomical demonstrations, lung injury is indicated when capillary permeability to fluid and protein rises. In the isolated rat lung model, infusion of 0.25ml air at rates ranging from 0.03 to 0.5ml/min, raised the PAP indifferently, but lung weight increased with infusion rates. Infusion of 0.25ml air over a 1-min period raised permeability coefficient, K_f, from a baseline of 0.21 ± 0.05 to 1.28 ± 0.26 $g\cdot min^{-1}\cdot cmH_2O^{-1}$ ($n=8$, $p<0.001$). Protein concentration in lung lavage fluid also rose, indicating lun injury. Leukocyte counts in the perfusate were unaffected by the embolization, but chemiluminescence activity was increased, indicating a possible role for activated leukocytes in lung injury induced by PAE, Addition of activated leukocytes to the perfusate drastically raised K_1, lung weight gain, and chemiluminescence, further suggesting the involvement of leukocytes in the PAE-induced lung injury.

Conclusion. Direct and indirect dffects of PAE lead to pulmonary hypertension abd lung injury, which involve the activation of the complement system and leukocytes. It is known that prior decomplementation reduces the risk of decompression sickness. Whether deactivation of leukocytes would lead to the same conclusion deserves further investigation. (Supported in part by a grant from NOAA, U.S.Department of Commerce, NA36RG0507 R/HP6)