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MECHANISM AND DETECTION OF DECOMPRESSION SICKNESS

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MECHANISM AND DETECTION OF DECOMPRESSION SICKNESS

This report describes the work and results found during studies conducted to elucidate the basic physiology, biochemistry and biophysics involved in the etiology of decompression sickness. We have investigated gas transport in living tissue by means of in vivo probes and mass spectrometric analysis, bubble growth in the tissue microcirculatory system using through-transmission ultrasound, bubble distribution in the major circulatory pathways with both doppler ultrasound flowmeters and visual inspection, and the changes in serum biochemistry following varying degrees of decompression injury. Experiments have been conducted on rats, miniature pigs, and men as the subjects.

The principal methodology employed has been to use dive profiles which result in minimal to no observable signs of decompression sickness in our animal subjects to approximate the situations which obtain in manned diving. This allows an easier transcription of results found from animal experimentation to man; we do not use death as an endpoint in animals, since that data would not apply generally to man.

Through-Transmission Ultrasound

Through-transmission mode ultrasound, at a frequency of 5.7MHz., was employed to sutdy gas-liquid phase separation in thigh muscle of rats following decompression. Pressures were varied to give differing degrees of decompression sickness. The time course for the separation was compared to the time course for the development of decompression sickness signs in other rats, decompressed from similar pressures, exercising on a treadmill. The parameters of

the ultrasound signal attenuation--i.e., time to attain an effect, rate of change of effect, and magnitude of effect--paralleled the manifestations of corresponding signs of decompression sickness.

It was found that there was a period after reaching the "surface" when no gas phase could be detected. The gas phase then slowly grew, either in size and/or numbers of "bubbles", persisted for a period of often three hours for the case of nitrogen as the compression gas, and slowly decayed.

In manned diving trials where through-transmission ultrasound was monitored, an attenuation was not found in the absence of symptoms of decompression sickness; no cases of decompression sickness were encountered for comparison.

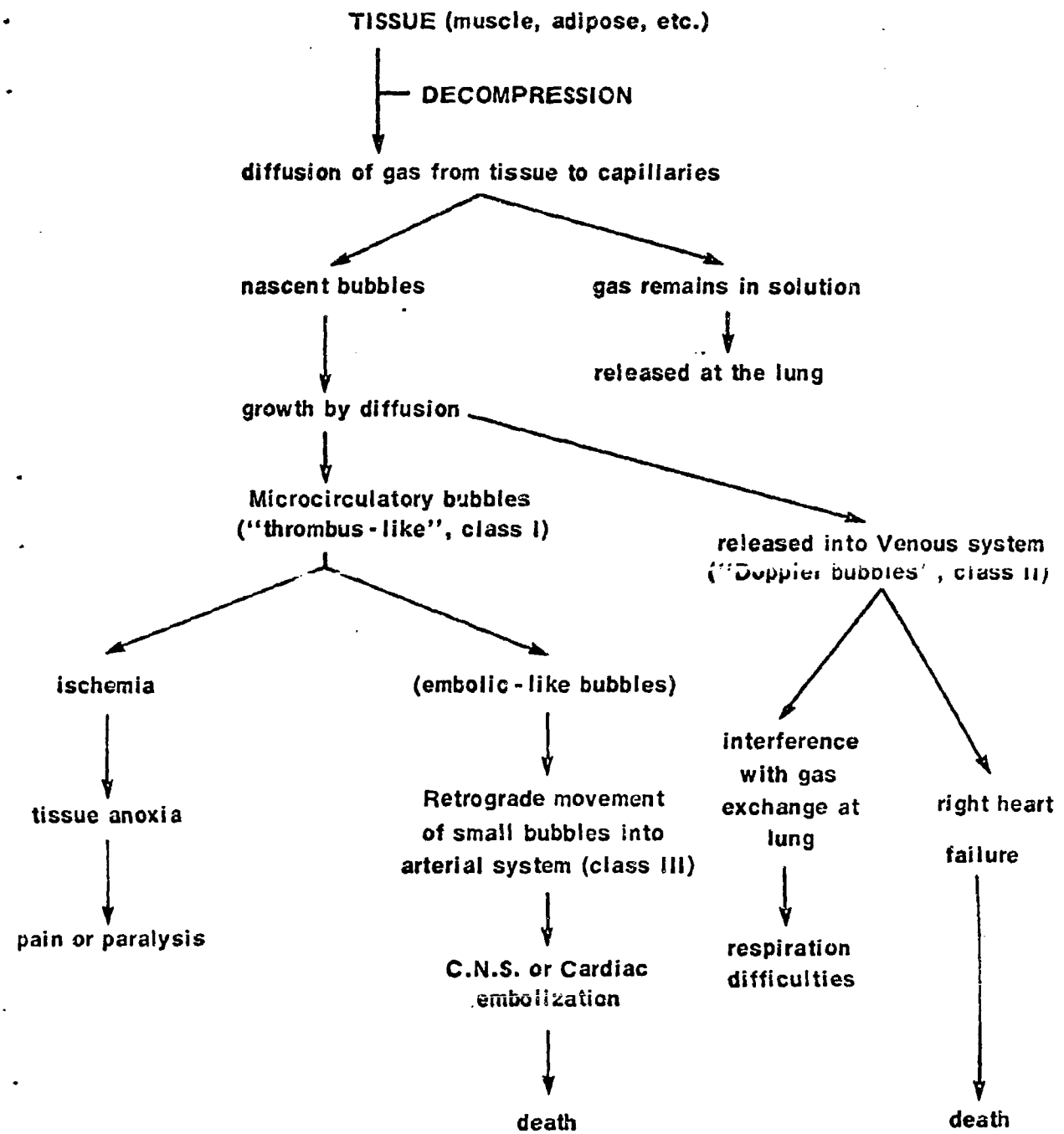
Doppler Ultrasound and Visual Monitoring

Studies were made of the distribution of the gas phase in the various venous and arterial channels in the rat following decompression. A doppler ultrasound flowmeter, operated in the transcutaneous mode was employed to detect the presence of bubbles in the posterior vena cava before surgical manipulation. By a visual inspection of the major venous and arterial tracts, the size, number and location of bubbles was determined. Inspections were made on groups of rats decompressed on profiles which produced 100% mortality; the profiles were then reduced in severity, by reducing the time spent at pressure, to a point where individual rats were free of the signs of decompression sickness. Even in this group, bubbles were easily found, and they came from tissue areas not associated with decompression sickness in the rat, e.g., abdominal tissue.

The time course for the appearance and disappearance of venous bubbles was studied. They reached a maximum in number shortly after reaching "surface" and were generally eliminated before the severest degree of decompression sickness was encountered. This is in contrast to bubbles found by the through-transmission ultrasound method in the leg. Here the time course for the appearance and disappearance paralleled the time course of decompression sickness signs.

Correlations between numbers of venous bubbles and the severity of decompression sickness were poor. Arterial bubbles were only found to be present in rats which were moribund; in such cases, cardiac output was reduced to so low a level that the gas phase moved down the arterial tree from the tissues. Retrograde motion of bubbles in the arterial system in these instances allowed a redistribution of gas phase by the arterial system. It must be emphasized, however, that bubbles were never found in the arterial system of rats which had only "limb bends"; arterial bubbles appeared only shortly before death.

The following working hypothesis was developed to explain the various pathophysiological consequences of a gas phase in a animal following decompression. We attribute the effects of decompression in the rat to the presence of three bubble "classes." Class I is a "thrombus-like" gas phase which grows in the microcirculatory system and is responsible for limb pain and possible paralysis when present to a sufficient extent. Venous bubbles, Class II, are generally asymptomatic if limited in number. In larger quantities, this phase will produce respiratory problems and even death through right heart


TISSUE (muscle, adipose, etc.)
DECOMPRESSION
diffusion of gas from tissue to capillaries
nascent bubbles
gas remains in solution
released at the lung
growth by diffusion
Microcirculatory bubbles
("thrombus-like", class I)
released into Venous system
("Doppler bubbles", class II)
ischemia
(embolic-like bubbles)
interference with gas exchange at lung
right heart failure
tissue anoxia
Retrograde movement of small bubbles into arterial system (class III)
pain or paralysis
C.N.S. or Cardiac embolization
respiration difficulties
death
death

failure (or pulmonary edema if death does not quickly follow decompression). Arterial bubbles, Class III, occur only when cardiac output has been reduced by a large number of venous bubbles entering the right heart. Arterial bubbles can produce death by embolizing the central nervous system or heart although death generally occurs from right heart failure as the predominant mechanism. The hypothesis is diagramed in the figure.

In Vivo Tissue Gas Analysis

To measure the rate of gas uptake in living tissue, a mass spectrometer analyzer was assembled. Gas was sampled from the tissue by means of a 22-gauge stainless steel tube covered with Teflon. This allowed a sufficient amount of gas to enter the system without depletion of the surrounding tissue. Probes were inserted into the calf muscles of rats; air was used as the compression gas.

No dependence of the uptake rate on pressure (40, 50, and 70 psi.) was found. The uptake halftimes were found to range from 3.8 to 7.8 minutes for the various rats tested. It is not known at this time if this represents a true subject-to-subject difference or it simply reflects a difference in probe position.

There was an indication that the elimination rate was not equal to the uptake rate for decompressions which would not produce signs of decompression sickness. This could be explained as a change in the perfusion rate for the whole tissue as a result of blockage of capillaries by a separated gas phase.

Studies of Serum Enzyme Level Changes

Miniature pigs were subjected to simulated dives on helium-oxygen, neon-oxygen, and nitrogen-oxygen (air) mixtures and decompressed to produce different degrees of decompression sickness. Changes in the serum levels of creatine phosphokinase and lactate dehydrogenase were sought. No difference in enzyme levels between pre-dive and after short air bounce dives were found. Large elevations of these two enzymes were found when nitrogen-oxygen was used as the compression gas and where mild symptoms developed. However no changes were found when helium-or neon-oxygen mixtures were used even if definite severe signs of decompression sickness ("limb bends") were evident. Similar experiments conducted earlier with rats indicated myocardial tissue as the damaged site and the origin of the enzymes. We interpret these findings in pigs as effects on myocardial tissue caused by bubbles in the vena cava and right heart. The more fat-soluble nitrogen would release bubbles into the venous system from adipose tissue. Heart problems would be seen (serum enzyme level changes) in the absence of changes in severity of limb "bend" decompression sickness. In terms of the earlier model, fat-soluble nitrogen would contribute more Class II bubbles than helium or neon; limb pain, the result of Class I bubbles was not the site of tissue damage and origin of CPK and LDH.

Dysbaric Osteonecrosis Studies

Three of the Hormel miniature pigs used in these and other decompression studies have been autopsied and examined for evidence

of bone necrosis. Long bones were X-rayed, sectioned and fixed, are are being processed for further study. This work is being carried out in cooperation with Dr. Kent H. Smith of the Virginia Mason Research Center. The animals were full grown in 1967 and have been subjected to numerous decompressions since 1969. If lesions are found they will be correlated with the diving history of the animals.

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1. Powell, M.R. Mechanism and detection of decompression sickness. Technical Memorandum UCRI-673, Tarrytown, N.Y.: Ocean Systems, Inc., 1971.

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