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New research on decompression procedures

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The object of a new research grant from the National Institutes of Health to the Institute of Applied Physiology and Medicine, in Seattle, under the direction of Merrill P. Spencer, M.D., is to develop pathophysiological information which will lead to the increase in safety of hyperbaric exposures and in the prevention and treatment of decompression injuries.

A three-year program is planned for the study of major physiological factors of hyperbaric decompression and problem areas of currently used decompression tables, saturation decompressions, counter diffusion studies, and acute respiratory crisis.

In spite of the other hypotheses, the bubble hypothesis is believed by most workers in the field of hyperbaric physiology to be essentially correct despite the lack of specific facts concerning the origin of bubbles and their site of action. Gersch and Catchpole (1951) pointed out evidence for intravascular bubbles as the primary agent in both altitude and hyperbaric decompression sickness. The action of the bubbles was postulated to produce stagnation anoxia. Experiments in which guinea pigs were decompressed from atmospheres consisting of either helium, argon, or nitrogen and oxygen, demonstrated that intravascular gas bubbles were present in all tissues and organs, but were more numerous in those rich in fat. Extravascular bubbles were found in lipoid-rich tissues, such as fat, adrenal cortex, and myelin sheath, or nerve fibers, but were absent from fat-poor tissues, such as liver, skeletal muscles, or tendon (Figure 1).

The hypothesis that bubbles are primarily responsible for decompression sickness leads directly to questions of the exact role of these bubbles in the pathophysiology. Intravascular bubble theories (Figure 2) propose that bubbles form in the blood stream and block venous, or arterial, or capillary channels causing stagnation anoxia (Powell, 1972, 1978).

Of crucial importance to bubble formation is the hypothesis of a metastable limit for bubble formation. This was first proposed by Haldane, and the "Haldane Method" of rapid ascent with shallow decompression stops resulted from it (Boycott, et al, 1908). This hypothesis states that blood can tolerate a considerable degree of supersaturation without the formation of a separated gas phase. Furthermore, this supersaturation limit is a fixed constant: Below the postulated limit, bubbles will not form, while above it gas phase separation will occur. In contemporary decompression models, this metastable limit is proposed to be different for each tissue and to depend upon the tissue half-time. Whether a true metastable limit exists has been a difficult question to answer.

A major problem has thus been to determine (1) the presence or absence of bubbles in decompressed subjects, and (2) the relationship of these bubbles to the signs, symptoms, and injury which may later develop. Bubbles were not always observed if autopsy was delayed. Furthermore, if the problem was caused by small bubbles in the microcirculatory system, gross dissection or imaging techniques would not reveal them.

A voice was given to "silent bubbles" with the

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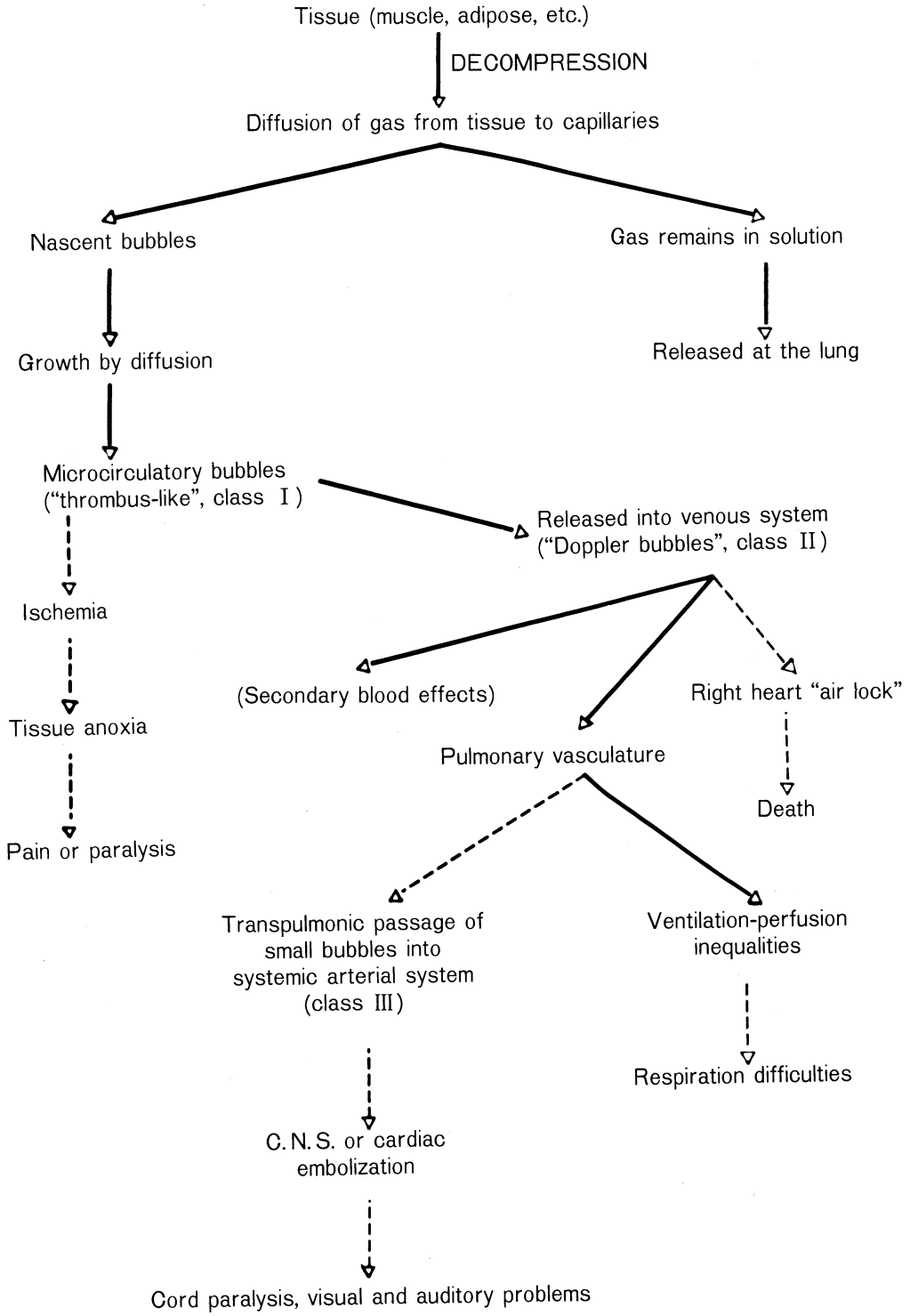

Fig.2 Working hypothesis of the pathophysiological consequences of a gas phase in the body following decompression (after Powell, 1977).

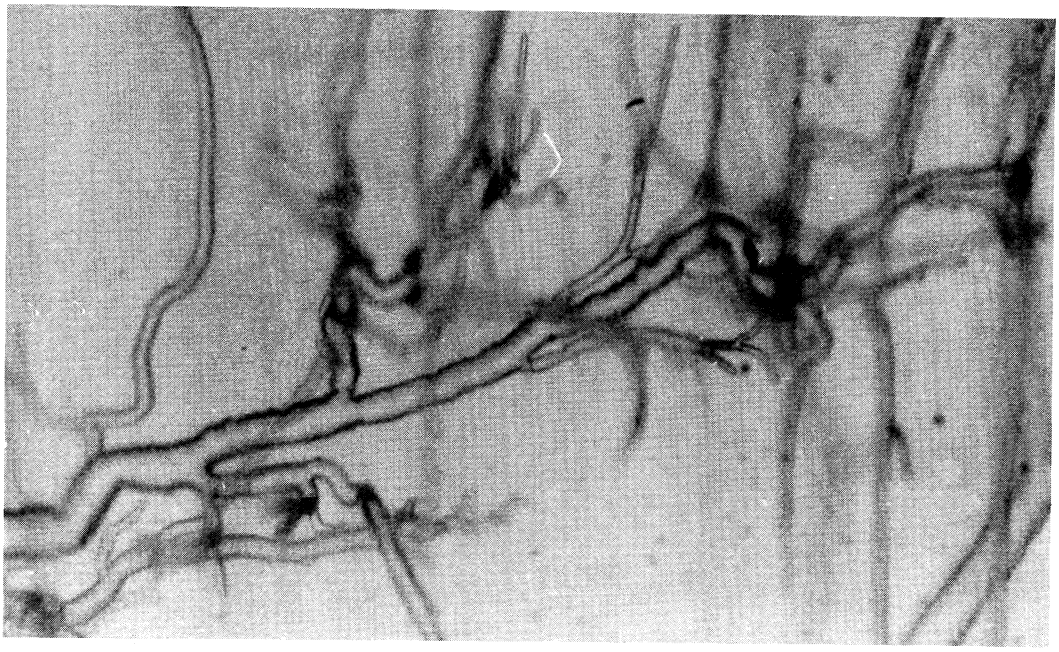

Fig.1 Gas bubbles in the capillaries and venules of rat muscle tissue (Powell, 1975).

AIR DIVES (Human)

LIMB BENDS / BUBBLE GRADE	NONE	MILD	MODERATE
0	100% (n=85)		
I・II	88% (n=30)	12% (n=4)	
III	60% (n=9)	33% (n=5)	7% (n=1)
IV	67% (n=14)	14% (n=3)	19% (n=4)

Fig.3 Probability of decompression sickness (limb pain) for various precordial bubbles grades, air dives.

HELIUM or NEON DIVES (Human)

LIMB BENDS / BUBBLE GRADE	NONE	MILD	MODERATE
0	100% (n=1)		
I・II	93% (n=13)		7% (n=1)
III	73% (n=8)	9% (n=1)	18% (n=2)
IV	39% (n=9)	13% (n=3)	48% (n=11)

Fig.4 Probability of decompression sickness (limb pain) for various precordial bubble grades, helium or neon dives.

advent of the Doppler Ultrasound Flowmeter (Franklin, et al., 1961). Early flowmeter workers noted that small air bubbles in the calibrating liquids produced very large signal artifacts as they passed through the transducer ultrasound beam. Exploiting this observation, Spencer (1968) subjected a Doppler-implanted sheep to a simulated dive to 200 feet in a medical decompression chamber. During decompression, bubble signals consisting of sharp clicks, and whistles were heard. Probes on the vena cava of sheep demonstrated that bubbles could be detected in this system, and venous bubbles did not result in extreme symptoms such as convulsions and death.

There have been numerous observations that gas emboli are frequently produced in hyperbaric chamber exposures on U.S. Navy tables, and there are few researchers today who would seriously consider that venous gas bubbles are found only in bends-producing dives. Spencer and Johanson (1974) and Pilmanis (1977) found there was a great variability in divers in bubble formation, and those divers with a greater propensity to bubble formation had a greater tendency to decompression sickness. Limited predictive success in large animal diving experiments so far has come from calculation probabilities of decompression outcome to bubble frequency detected precordially. In studies utilizing miniature swine dived on nitrogen, helium, or neon-oxygen mixtures, or humans dived on neon-or helium-oxygen mixtures (Powell 1977), there was a definite tendency for bends following a high degree of bubbles detected over the precordial region (Figures 3 and 4).

The specific aim of one portion of the research project is to clarify by means of venous gas embolism (VGE) detection (Spencer and Clark, 1972) the role of four major physiological factors which may alter the incidence of decompression sickness. These factors are the response to submersion, cold exposure, exercise when submerged in cold water, and the effect of straining the musculoskeletal system during decompression.

The specific aims of another portion are: 1) to determine the longest controlling half-time in the body that limits the rate of decompression from hyperbaric exposures, 2) to determine the individual variability in the longest limiting half-time, and 3) to gather information concerning the predictability of bends from Doppler ultrasonic venous gas embolism detection.

Various decompression profiles from hyperbaric saturation exposures abound based on each investigator's or diving company's pet theory. Generally, they are constructed from a model which utilizes a series of half-time compartments representing the bodies' distribution of compartments in which the inert gas is dissolved and released. The data that has been gathered to test the models consist mainly of the incidence of "bends" which might develop. The variability between individuals and various diving situations has caused a confusing range of results.

The use of the Doppler ultrasonic detection of venous gas emboli during decompressions offers an opportunity to determine objectively the optimum profile for decompression exposures with minimal risk of producing "bends." We plan to determine this optimum profile in sheep as a demonstration for what can be done in humans. Following the animal experiments and utilizing the information gathered, a series of pilot human experiments to determine the applicability of the procedure to human saturation decompressions.

While there may be differences between animals and humans, the principal differences are mainly represented by longer half-time compartments of larger animals. Direct decompression studies have previously indicated that the longest probable half-time compartment of human subjects is less than 200 minutes (Spencer and Johanson, 1974; Spencer, 1976).

While Doppler ultrasonic detection of venous gas emboli (VGE) has been successful in investigating decompressions from non-saturation exposures to hyperbaric air, it has been applied in a limited way to saturation exposures. Some investigators have doubted that VGE detection has an application in saturation decompression on the basis that the slow decompression rates utilized challenge only the slowest decompression compartments which may not have access to the blood. Failure to observe VGE during saturation decompression, however, may be due to: 1) slower

than optimum decompression rates, 2) lack of training of observers, and 3) poor sensitivity of the transcutaneous Doppler detector. Doppler ultrasonic detection of circulating bubbles during decompression from helium saturation at a pressure of 300 meters of sea water in both humans and in animals has been demonstrated (Guillerm, et al., 1975).

The preliminary conclusions from the pilot sheep experiments are: 1) The longest limiting tissues half-time to which attention must be given during saturation decompression procedures can be determined by monitoring the incidence of pulmonary artery gas emboli. 2) The numbers of VGE which are tolerable in a practical diving situation and optimize the decompression procedures are represented by Grade II embolization. 3) Decompression from saturation can be efficiently and safely performed by attention to a single theoretical half-time compartment utilizing either staged or continuous decompression. 4) The longest controlling tissue compartment tolerates an initial 50-foot "pull" for a 100-foot saturation dive. 5) Decompression stops shallower than 10 feet must be observed to prevent significant gas phase formation. 6) Tissue compartment surfacing pressures no greater than 25 feet of air pressure are necessary to prevent a significant VGE and bends. 7) "Bends" may be prevented in subjects when VGE is not allowed to rise above Grade III. 8) Experimental data from sheep may be used to predict the response of human subjects. In this regard, evaluation of human studies may well indicate the advisability of applying a safety factor to the animal data. 9) Decompression procedures based on theoretical limiting half-times greater than 200 minutes do not increase safety and appear to reduce the cost effectiveness of hyperbaric-exposed, saturation divers. 10) The procedure for determining the limiting half-time and subsaturation hyperbaric exposures indicates a similar applicability to human divers.

BUBBLE GRADING SYSTEM

GRADE	BUBBLES/SEC.
I. a. VERY OCCASIONAL (<50%) BUBBLE SIGNALS	<1
b. 50% OF HEART CYCLES CONTAIN BUBBLE SIGNALS	
II. a. MANY CYCLES, BUT NOT ALL, CONTAIN BUBBLE SIGNALS 1/2 TO 1 B/CYCLE (SEC.)	1-10
b. MAJORITY OF HEART BEATS 1-10 BUBBLES/CYCLE (SEC.)	
III. a. ALL HEART BEATS CONTAIN BUBBLE SIGNALS 10-100 B/SEC.	10-100
IV. ALL HAVE B's 100-1000 BUBBLES/SEC.	100-1000
V. a. SOME INDIVIDUAL CARDIAC SIGNALS IDENTIFIABLE >1000 BUBBLES/SEC.	
b. NORMAL HEART SOUNDS NOT IDENTIFIABLE AND NO INDIVIDUAL BUBBLE SIGNALS DISCERNIBLE	>1000

The aim of another study is to determine if the brain can produce counter-perfusion/diffusion venous gas bubbles and, if so, under what conditions. Bubble formation can occur in subcutaneous tissues and enter the venous return under isobaric conditions, when a subject is breathing nitrogen, and the skin is exposed to a helium atmosphere (Graves, et al., 1973). This phenomenon is explained on the basis of the differences in diffusivity and tissue thicknesses through which the two gases are diffusing in opposite directions. If suitable nuclei are present, bubbles will form and grow continuously (Idicula, et al., 1976).

D'Aoust (1977) has demonstrated that VGE are detected with Doppler ultrasound in goats following a rapid isobaric switch of the chamber inert gas environment from nitrogen to helium at 132 and 198 feet of sea water. Bubbles occurred with no change in hydrostatic pressure 30 to 60 minutes after the switch in chamber gas from normoxic nitrogen to normoxic helium and continued at low rates (less than 100 events per minute) for up to 12 hours.

It appears of great importance to determine whether or not any set of conditions can exist where gas switching can produce bubbles in the central nervous system.

The echocardiograph is a widely available and highly developed instrument for clinical diagnosis of cardiac pathology and physiology. The transducers can be aimed at various portions of the

heart and easily display the location of the right ventricular cavity and left ventricular cavity as well as other structures. By manual manipulation of the transducer over the chest, all the cardiac chambers and great vessels can be visualized.

We plan to (1) investigate the ultrasonic echocardiograph in the study of circulating decompression gas emboli, (2) to study cardiac dynamics in the acute respiratory crisis of decompression sickness, (3) identify the changes with right ventricular pressure changes and (4) to evaluate the diagnostic possibilities of the instrument in clinical detection and management of "chokes."

It will be of great interest to determine if the echocardiograph can detect more subtle quantities of venous gas embolism than can the Doppler precordial detector. We know by experience that numerous gas bubbles may be detected with Doppler in femoral veins of animals when no bubbles are detectable with the precordial monitoring. If clear bubble signals are seen on the echocardiograph, and they are not detected with the precordial bubble detector, we may then conclude that the echocardiograph is a more sensitive and more specific instrument for VGE precordial detection.

Further potential value of the echocardiograph in monitoring cardiac bubbles is its ability to specify the cardiac chamber, artery, or vein in which bubbles are situated. For example, it has potential in tracing the course and location of central bubbles as to whether they are present in the superior vena cava or inferior vena cava and they can be monitored for passage across pulmonary vasculature into the left ventricle. We could expect to see VGE in the left ventricle before or simultaneously with arterial signals with the echocardiograph and thus provide more accurate monitoring for the systemic arterial circulation.

Of special interest are recent reports from the British Royal Navy's Diving Institute at Alvestoke that they had observed a significant increase in erythrocyte sedimentation rate among all divers who experienced "bend" systems following deep mixed gas decompression profiles.

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