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The Effects of Rapid Decompression on Total Plasma Phospholipids and Selected Hematological Parameters in Dogs

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THE EFFECTS OF RAPID DECOMPRESSION ON TOTAL PLASMA PHOSPHOLIPIDS AND SELECTED HEMATOLOGICAL PARAMETERS IN DOGS

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This thesis is approved as a creditable and independent investigation by a candidate for the degree, Master of Science, and is acceptable as meeting the thesis requirements for this degree, but without implying that the conclusions reached by the candidate are necessarily the conclusions of the major department.

Thesis Adviser — Date

Head, Entomology-Zoology Department — Date

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LJF

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Decompression sickness is probably one of the least understood maladies of altered pressure environments.(47) It was first described by Robert Boyle in 1660 when he wrote: "....That upon the sudden removal of the wonted pressure of the ambient Air, the warm Blood of those Animals was brought to an Effervescence or Ebullition, or at least so vehemently expanded as to disturb the Circulation of the Blood, and so disorder the whole Oeconomy of the body."(24, 48)

Today, over three centuries since Boyle's statement investigators are still attempting to ascertain additional knowledge into the etiology of decompression sickness.(5) However, to date, no steadfast explanation as to the cause, or causes, of decompression sickness have been proposed.

The necessity of elucidating the causative factors of decompression sickness is of major importance because of the increasing occurrence of man's exposure to altered pressure environments. Modern man is constantly attempting to invade new territories, in the form of outerspace and the ocean floor, in an attempt to find new homes, additional food, and more living space. As a consequence, he is exposing himself to the possibility of dysbarism.

Decompression sickness, also referred to as dysbarism, "bends", "chokes", and many others, manifests itself in several ways depending on the severity of the case. Signs and symptoms range from mild headache to muscular cramps, paralysis, hemorrhage of the subcutaneous tissue, ischemia, visual disturbances, pulmonary edema, hemoconcentration, asphyxiation, shock, and, quite often, death.[19, 36, 50, 52] If man is to succeed in his attempts to conquer new territories he must find some means to eradicate these problems involved with decompression sickness.

The purpose of this project was twofold: (1) measure the change in total plasma phospholipids due to rapid decompression, and (2) measure the effects of rapid decompression on selected hematological parameters.

An attempt has been made to show that an increase in phospholipids following rapid decompression does occur. This attempt was made to substantiate the Disseminated Intravascular Coagulation (DIC) Theory of the etiology of decompression sickness. DIC involves massive coagulation throughout the capillary vasculature and is supposedly initiated by a "coagulation factor". Guyton has stated that the "coagulation factor" is a phospholipid or number of phospholipids.[28] Therefore, if an increase in the total phospholipids is found, evidence in support of DIC may be indicated.

The hematological parameters measured were red blood cell count, hematocrit, and total white blood cell count. These parameters were measured to derive a more definitive role of the effects of rapid decompression upon these blood constituents.

REVIEW OF LITERATURE

History of Decompression Sickness

As was stated in the introduction, Boyle was probably the first individual to observe decompression sickness. In addition, he foresaw the possibility of placing man inside of a "Reciever" to observe his responses to an altered pressure environment.(24) This "Reciever" may have been a forerunner of our modern decompression chamber.

The 17th-19th centuries were filled with attempts by man to use hyperbaric pressure confinements. For example, the diving bell and diving suits were used.(3) The first reference made to dysbarism during this period was by Liddell in 1842. Liddell observed that divers were afflicted with rheumatism after having been exposed to a hyperbaric environment. This observation has striking similarity to what is referred to as "bends" today. The latter case is a confirmatory sign of decompression sickness.(24)

Junod, in 1834, was the first to use a vessel of the decompression chamber type. His vessel was made of a copper material and measured about 4 feet in diameter. He experimented on himself and others in his artificially produced hypobaric and hyperbaric environments. His notes do not report anything that resembled decompression

sickness. Therefore, it is assumed that the working pressures of his vessel were not large enough to produce dysbarism.(48)

The compressed air technique for treating the adverse effects of high pressure was first used by Triger, a Frenchman, in 1839. Triger was given credit for being the first to observe the aberrant effects of high pressure on human beings.(24)

In 1854 two French physicians, Pol and Watella, conducted a study into the etiology of the illness that the Triger's workmen were experiencing. Fatalities were not a too uncommon occurrence at this time. Pol and Watella indicated congestion of the lungs and brain in addition to capillary oxygen toxicity as the causative factors of this intriguing malady.(12)

The next 40-50 years were heavily laden with various theories as to the cause of dysbarism according to Fryer.(24) He also stated that few were of value. Some of these attributed respiration and its effect on causing "vertical rising and falling of the brain" as the causative factor of decompression sickness. Guerard attributed the problem to increased pressure upon the surfact of the body.(36) In 1861, Brerton stated that there was a definite racial susceptibility factor in that the Irish were more prone to decompression sickness than any other

race.[24]

In 1869 it was shown by Mericourt that undersea divers experienced the same illness that tunnel workers experienced after having been exposed to prolonged elevated pressure. He also proposed that the body tissues became "saturated" with gases under a hyperbaric environment, and upon sudden removal of the ambient pressure, the saturated tissues began to "fizz". Mericourt is well-known for his statement "Man is really, from the physiological point of view, in the situation of a bottle of artificial Seltz water."[24]

Also at about this time Paul Bert was performing massive experimentation on such topics as hypoxia, asphyxia, oxygen poisoning, and decompression sickness. He suggested the need for a minimum time for decompression. However, his suggestions were not put to use in the tunnel operations. As a result, men were still succumbing to the effects of decompression sickness.[47]

In 1907, about 250 years after Boyle's original observation, Haldane, Boycott, and Damant presented a partial solution to alleviating decompression sickness.[24] They stated that the large factor causing decompression sickness after prolonged hyperbaric exposure was the rate at which the ambient pressure was removed. They used the ratio of initial to final pressure during decompression

to derive their forecasting ability. The numerical ratio that they suggested was 2:1, that is, if the individual was at 20 lbs/sq in gauge (psig), the next stop in decompression would be 10 psig, etc.

In the early 1950's new ideas as to how decompression sickness could be prevented were presented. Among these was Fulton's idea of removing the pressure by small increments rather than relying on the single ratio dictum of Haldane.(59) In addition, Van der Ave presented the idea of multiple critical ratios, rather than Haldane's single critical ratio.(24) Eyes were also turning to the extravascular aspects to decompression sickness rather than just the intravascular.

According to Fryer only two new concepts in decompression etiology have evolved since the early fifties. The first of these involves the trapping of liberated air bubbles in the pulmonary vasculature due to "pulmonary cysts, obstructed lung segments, or temporarily constricted airways."(24) The second has been presented by Le Messurier and Hills of Australia.(34) They propose that all tissues possess an "inherent unsaturation" by which can be calculated entirely different decompression tables than have been in use in the last two decades.

In addition to Fryer's inclusions of new concepts of decompression sickness etiology the author would like to

include these new concepts or theories. The one gaining much momentum at present, is called disseminated intravascular coagulation. It is proposed that with the formation of air bubbles following rapid decompression a series of events occurs which results in the formation of an intravascular blood clot.(1, 15, 20, 23, 29, 36, 50)

Another concept involves the release of a hormone-like substance from the pulmonary tissue following the formation of air bubbles. This hormone supposedly causes constriction of the air conduction pathways resulting in asphyxiation of the individual.(18, 19, 37, 41, 43, 71, 72)

Still another concept in the etiology of decompression sickness is the mobilization of unstable lipids from the body tissue in response to trauma caused by multiple air emboli. As a result of the trauma, the unstable lipids are released from the bone marrow, liver, and adipose tissue as lipid emboli into the cardiovascular system. These emboli then continue to circulate until they become lodged in small vessels. Consequently, local ischemia results. If the emboli are massive, vital areas may be deprived of circulation. Shock and possibly death ensue.(31, 35, 38, 40, 48, 51, 54, 67)

The Gas Laws

No discussion of decompression sickness would be

appropriate without mention of the Gas Laws in relation to altered pressure environments. These Laws help explain why the body tissues become "saturated" with ambient air during the exposure period.

Dalton's Law of Partial Pressures states that a given gas in a mixture of gases will exert its pressure independent of the other gases within the mixture.(2, 47) Therefore, if a container has a mixture of nitrogen, oxygen, and helium the pressure exerted by these gases is the sum of their individual pressures if they were present independently in separate containers, assuming constant temperature and volume.(2, 7, 8, 30) The individual pressures are most often referred to as the partial pressure of the gas.(33, 42, 63) Placing this Law in a more realistic situation assume that an individual is under 80 psig of compressed air. Since air is composed primarily of nitrogen (80%) and oxygen (20%) we can assume that the partial pressure of nitrogen and oxygen under an 80 psig environment would be 64 psig and 16 psig, respectively.(9, 14, 60, 68) Compared to 0 psig for both oxygen and nitrogen at sea level, increasing the pressure to 80 psig would yield a remarkable increase in the partial pressures of these two gases. This has profound consequences physiologically.(64, 65, 69)

For more practical use Dalton's Law of Partial Pressures must be combined with another law, Henry's

Law.[2, 47] Henry's Law states that the volume of gas absorbed by a liquid is directly proportional to the partial pressure of the gas. Referring to the preceding paragraph, assume the partial pressure of nitrogen (pN_2) to increase from 0 psig to 64 psig within an apparatus containing an individual. According to Henry's Law the volume of nitrogen absorbed or dissolved in the individual's tissue fluids would be enhanced markedly. If this individual's tissues equilibrated with the ambient pN_2 he would be "saturated" with nitrogen.[27, 45, 66, 70] The same situation would occur with oxygen, helium or any gas that is used in the medium.

Boyle's Law, which is very significant in decompression sickness, states that the volume of a gas is indirectly proportional to the pressure imposed on that gas, that is, $P_1V_1=P_2V_2$.[13, 47, 48] When the pressure (P_1) exerted on a gas is increased (P_2), the original volume (V_1) is decreased (V_2) in proportion to the pressure (P_2). This Law will have more significance later when bubble formation following rapid decompression is considered. Tissue saturation, as indicated previously, involves equilibrating the tissue fluids with the ambient gases in accordance with Henry's Law. This occurs after prolonged exposure to an environment of increased partial pressures of the gases in the medium. Naturally, the

greater the ambient pressure, the greater will be the volume of gas dissolved in the tissue fluids.(21, 25, 55, 62)

Tissue Saturation

Most investigators in the field of dysbarism are confused by the variation in susceptibility of individuals to decompression sickness. Why, for example, do two individuals of varying physical and physiological type differ in their susceptibility to decompression sickness? And what is more confusing, why does the same individual develop a certain degree of tolerance to the effects of rapid decompression? Similar questions may be posed in this regard. This section will attempt to explain some of these questions.

Tissue saturation involves equilibrating the gas dissolved in tissue with the ambient or exogenous gas pressure. Naturally, as the ambient pressure increases the corresponding volume of gas dissolved in the tissue will increase.

In addition, if any type of tissue has a higher coefficient of solubility for a certain gas, it will act as a reservoir for that gas. Fat, or lipid tissue, acts as such a reservoir for nitrogen.(55)

The importance of body fat as a reservoir for inert gases has long been recognized.(61) Calculations have

indicated that if a 70 Kg man was composed of 10% lipid in adipose tissue, he would have 490 ml of N_2 dissolved per atmosphere of nitrogen (pN_2=760 mm Hg).[5] In addition, if the blood flow to these areas was approximately 0.4 liters per minute, the half-time for the removal of the dissolved nitrogen would be two hours. An additional ten hours would be necessary to remove 99% of the dissolved nitrogen. This information is of paramount importance when decompression is performed on individuals with high lipid content.[73] Autopsy findings, radioisotopic scanning and roentgenagrams all show the presence of bubble and/or nitrogen accumulations in lipid areas following rapid decompression. These findings indicate the presence of nitrogen reservoirs as discussed above.[5, 12] We can conclude from the above information that an individual of lean or nonfat stature is less susceptible to decompression sickness.[4, 47, 73] It has been stated that nitrogen is five times more soluble in the lipid phase than the aqueous phase.[47] This factor favors susceptibility of obese individuals to decompression sickness.

Various authors have indicated that rats, guinea pigs, dogs, and even man have the capability of acclimatizing to high pressure environments without developing "bends" upon subsequent decompression.[5, 6, 46] No answer has been presented as to why this occurs. Some

think that the cardiovascular system shunts these bubbles produced from rapid decompression to other body areas which are not adversely affected by the presence of bubbles.(44)

Rapid Decompression

It is necessary for investigators studying decompression sickness to induce the syndrome by decompressing the experimental animal at a very rapid rate.(58) Rapid decompression will result in the formation of nitrogen bubbles.(12, 13, 22) The reason for the formation of nitrogen bubbles is that decompression decreases the pressure exerted on the dissolved gas and as a result the body is "supersaturated" with nitrogen. Moments later the nitrogen will precipitate in the form of bubbles.(67) From this step on an entire chain of events supposedly occurs.

Induced, or experimental, decompression sickness has long been utilized by researchers studying in vivo decompression sickness.(12, 17, 22) Experimental subjects ranging from rats, guinea pigs, cats, and dogs, all the way to man have been employed.(51) Some investigators have actually been able to control the severity of decompression sickness by observing the formation rate of intravascular bubbles during the decompression phase.(36) This fact has important implications in terms of speeding up the lengthy, and costly, process of decompressing humans

following a saturation exposure to hyperbaric pressure.(5)

The largest problem coincidental to rapid decompression is decompression sickness. This syndrome appears to involve one primary organ system which eventually leads to the involvement of other organ systems. This situation resembles a line of dominos stacked on end. When one domino is knocked over the entire line reacts accordingly. The initiating domino in decompression sickness is the cardiopulmonary system.(6)

Many investigators agree that the bubbles liberated from the tissues upon decompression appear in the cardiovascular system before occurring extravascularly.(6, 19, 26) A tremendous amount of time has been devoted to developing in vivo techniques for observing bubble necleation, or genesis, and growth.(12, 13, 22) These investigators have also witnessed extravascular bubbles, but they seem to appear later than their intravascular counterparts.

Bubbles, once formed, become emboli of various sizes and generally move with blood flow. As the emboli approach vessels very small in diameter the air bubbles become lodged and local ischemia occurs. The severity of ischemia depends on how much blood flow is obstructed from the tissues.

It appears, however, that bubbles may be transported to the heart and subsequently enter the pulmonary system.(5)

The lungs apparently act as a reservoir or buffer to prevent these bubbles from reentering the systemic circulatory system. Behnke, for example, has shown that dogs could be injected intravenously with over 3900 cc of air within an 80 hour period without injuring the animals.(6) He attributed the lungs as being the organ which trapped the bubbles and subsequently removed them through normal respiratory functions.

If the bubbles do enter the systemic circulatory system the individual would be quite susceptible to cerebral embolism and numerous other occlusive maladies. Naturally, rapid death would ensue if certain areas of the brain were deprived of adequate blood flow. It seems, however, that this latter case is not the rule in massive vascular emboli caused by decompression sickness. (36)

Rapid decompression can give rise to a host of abnormal situations in relation to decompression sickness. The liberation of air bubbles is only one. Other factors include increase in arterial pCO_2, respiratory rate, blood pressure, lymph flow and a decrease in oxyhemoglobin saturation. See Table I for additional factors.

A number of factors have been studied in regard to the events occurring after the formation of air emboli in the cardiopulmonary system. See Table II for additional information relevant to this topic. Initially, due to the

trauma or distortion of the blood vessels by the air bubbles, the catecholamines, epinephrine, and norepinephrine, are released. These substances result in vasoconstriction, tachycardia, and dyspnea. In addition, histamine, bradykinin, and serotonin are also released. These substances result in addition vasoconstriction, pulmonary arterial hypertension, fall in peripheral blood pressure, and blood stasis. Subsequently, platelet aggregation, reduced plasma lipid, hemoconcentration, hypercarbia, plasma loss and eventually shock result.(10, 19, 26)

The previous information presents many of the facets either directly, or indirectly, involved in decompression sickness. Most researchers agree that regardless of the decompression sickness type, i.e. altitude, tunnel, or deep-sea, the pathological consequences are very similar.(5) One very important aspect of decompression sickness, however, is that investigators have not agreed upon the etiology of this syndrome.

Theories of Decompression Sickness

Prior to the mid 1950's investigators were quite certain that the factor precipitating decompression sickness was the formation of air bubbles following rapid decompression.(3, 6, 11) However, today new experimental findings are emerging which tend to exclude the bubble

theory as the primary causative factor of decompression sickness.(5, 28, 38)

There are basically four theories that most investigators are relying on to explain the etiological factors of decompression sickness. See Table III for further enumeration on these factors. All four theories are interrelated, that is, each contains essential features of the others. Thus, a real problem exists in deciding which theory explains the overall series of events involved with decompression sickness.

The first theory, the classical one, is the Bubble Theory. This concept has been the most popular and has survived the scrutiny of many investigators in explaining the etiology of decompression sickness. Even to this day investigators still agree upon the significance of the bubble.(12, 13, 22, 25)

Nitrogen precipitates in the form of bubbles in the tissue fluids following fulminating or rapid decompression according to the Bubble Theory. Subsequent events involve additional growth and vascular mobilization of the bubbles.(59, 66) The gas emboli, once formed, seem to attract additional dissolved gas and the bubbles continue to enlarge.(13) Once mobilized, the bubbles follow the direction of blood flow intravascularly until they become lodged in a vessel smaller than the diameter of the

individual bubbles. At this point the gas emboli become thrombi and cause extensive damage.(23)

The big question in the minds of researchers who do not accept the Bubble Theory is whether the air bubble is capable of initiating the series of responses as presented previously. Behnke, for example, has shown that the intravenous injection of 3900 cc of air into a dog over an 80 hour period has not resulted in adverse effects to the experimental animal.(4) Perhaps, then there is some other factor which causes the sickness, some factor which can be stimulated to act only upon rapid decompression.

One of the primary reasons for many people abiding by the Bubble Theory is that recompression therapy seems to alleviate the problems associated with decompression sickness.(30, 33) Therefore, it is assumed that recompression forces the bubbles back into solution and, subsequently, since the bubbles disappear the signs and symptoms will also subside. This does occur in a large percentage of the individuals that are cured using recompression therapy. However, there are many cases where recompression is not successful in treating decompression sickness.(38, 48, 50) It is for this reason that researchers are searching for new ways of explaining what initiates the decompression sickness syndrome.

The second theory, and a relatively new one which attempts to define decompression sickness more accurately,

is one suggesting that a hormone is released following rapid decompression. This hormone, as suggested by Chryssanthou who isolated it from lung homogenates of rapidly decompressed rats, causes bronchiole spasm with resultant bronchiole constriction.(19) Due to constriction of the air conduction pathways asphyxia results. This hormone is named Smooth Muscle Action Factor (SMAF) after its activity on smooth muscle. Chryssanthou's pioneer work in the area of hormone isolation from tissue following rapid decompression will no doubt stimulate additional research directed toward more such isolations.

The third theory, also a relatively new idea, is the theory of lipid embolization. The essence of this theory suggests that large amounts of unstable lipids are released from certain tissues of the body and enter the vascular system. This stimuli for lipid release is supposedly due to tissue trauma initiated by air bubbles. The unstable lipids, upon entering the vascular system, tend to coalesce into globules. These globules then function as emboli in the blood vessels.(48) From this step on a situation similar to what occurs when air emboli are present ensues. That is, the fat emboli become trapped in the blood vessels and become thrombi. Next, ischemia develops and hypoxia and hypercarbia set in. Subsequently, circulatory shock may develop.

The lipids are evidently mobilized or released from the liver, bone marrow and adipose tissue.[50] Studies have indicated that the globules have a tendency to accumulate in the pulmonary capillaries. Massive pulmonary thrombi occur and insufficient gas exchange results.

Others have shown that rapid decompression does not increase the amount of lipids in the pulmonary vessels.[38] This fact suggests that additional research is needed in order to further elucidate this area. Lipid mobilization has been known for its occurrence during multiple fracture of large bones and other circumstances resulting from trauma.[53, 57] Since air bubbles also cause tissue trauma it is not surprising that at least a certain degree of lipid embolization is found in decompression sickness.

The fourth theory, and one that has been gaining prominence, is disseminated intravascular coagulation (DIC). This describes what occurs in the microcirculation to produce decompression sickness following rapid or fulminating decompression.

DIC has derived its impetus due to investigators such as Holland, Swindle, and Philip.[36, 50] They noted that blood stasis occurred following rapid decompression. They also observed that the stasis was due to numerous microclots occurring in small circulatory vessels. They concluded that plasma exvasation, hemoconcentration, and

circulatory shock were due to the small clots' occluding function.

Holland concluded that the initiator of the DIC phenomenon was some tissue factor.[36] This factor was supposedly released from tissue cells following trauma. The entire sequence of events involved in DIC include elaborate biochemical, physical, and physiological occurrences. Initially trauma, resulting from rapid decompression and bubble formation, occurs with an ensuing rise in catecholamines. These, in turn, produce arteriolar constriction and opening of the A-V shunts. Once the A-V shunts open a large volume of blood bypasses the capillary beds and the tissue cells which were subserved by these capillaries become hypoxic. The deprived tissue cells then secrete histamine which serves to increase capillary permeability. Subsequently, histamine elicits increased volume of the capillary beds by capillary vasodilation. Unfortunately, blood volume is not increased with the increase in the vascular volume. Thus capillary blood pooling occurs. Blood pooling depresses capillary flow and hypercarbia due to lactic acid accumulation ensues. Slow flowing acidic blood is hypercoaguable so intracapillary coagulation occurs. Hypercoagulation consumes the clotting factors. As a result, diffuse capillary hemorrhage occurs and tissue

perfusion halts. Cellular death and microinfarction follow.(36)

METHODS AND MATERIALS

Experimental animals utilized were mongrel dogs, which varied in age, weight, and sex. Their age range was approximately 9-29 months and weight varied from a low of 24 pounds to a high of 50 pounds with a mean of 40 pounds (Table IV). They were maintained on commercial dog food* except 6-8 hours prior to placing them into the recompression chamber. This procedure served to control dietary intake prior to experimentation.

The pressure chamber employed in these investigations was a portable recompression chamber** located at the Oahe Reservoir Powerhouse, Pierre, South Dakota (See Figure 1). The chamber met all specifications of the U.S. Navy. It was a two compartment (medical lock) type with the primary compartment measuring six feet in diameter and five feet in length. Both compartments were fitted with marine-type lamps and compressed air supply lines equipped with valves for both internal and external pressure control. The compressed air supply line contained check and safety valves with a regulator providing ample air circulation within the chamber at all times.

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* Supersweet Feeds, A division of International Milling Company, Manufactured at: Minneapolis, Minnesota.

** Vacudyne Corporation, Chicago Heights, Illinois.

Both compartments contained a total of six observation ports permitting surveillance of occupants. In addition, both compartments were equipped with a caisson-type pressure gauge, inside and outside, for recording the air pressure within each chamber. Each compartment was equipped with a one and one-half inch safety valve set to open at 90 psig to prevent chamber over-pressurization and subsequent structural damage.

The animals were compressed to 80 psig (180 feet salt water equivalent) at 20 feet per minute. The subjects were then maintained at 80 psig for a period of 120 minutes allowing a sufficient period of time for tissue saturation. Animal activities were noted continuously while in the chamber. Following the 120 minute saturation period the animals were rapidly decompressed at 20 feet per minute (See Figure 2). Those showing signs of decompression sickness after the removal of the post-decompression blood sample were recompressed rapidly to 80 psig according to a therapeutic decompression table (Table VIII) modified from Workman and Reeves.(53) Recompression was quite successful for treating those with decompression sickness. Those that did not survive expired before recompression therapy could be employed. Autopsies were performed on those animals that did not survive with data shown in Table IX. Therapeutic recompression involves

recompressing the subject to a specified pressure followed by decompressing in small increments until ambient pressure is obtained. The slow rate of decompression supposedly allows sufficient time for tissue "desaturation".

Three repetitions, or phases, were performed on five animals for a total of fifteen individual trials. Each repetition was approximately thirty days apart allowing ample time for the subjects to recover from any effects that were experimentally produced during rapid decompression.

Control blood samples were withdrawn from each dog prior to introduction into the chamber. A test sample of blood was withdrawn fifteen minutes post-decompression or when decompression sickness signs appeared, whichever occurred first. The blood samples were centrifuged for five minutes at 750 rpm and the plasma was placed in six-ounce plastic bags for "quick-freezing" in liquid nitrogen.

After removal of the test sample the dogs were observed carefully for a period of 1-2 hours to observe any changes that may have occurred incident to the rapid decompression. Those animals exhibiting confirmatory signs of decompression sickness were recompressed for therapeutic decompression as mentioned previously.

The plasma samples were analyzed for their phospholipid content according to procedures by Connerty, et. al.

(See Table X). This procedure involved comparing a standard phosphorus solution (40 micrograms per ml) against each sample which were read at 700 millimicrons as optical density utilizing a Beckman DB-G grating spectrophotometer.* The phospholipid values were statistically analyzed using Student's "t" test for small samples.

Duplicate red and white blood cell counts were obtained, utilizing hemocytometers. Duplicate packed cell volumes were determined with the microhematocrit technique.**

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* Beckman Grating Spectrophotometer, Model DB-G, Beckman Instruments, Incorporated, Fullerton, California 92634

** Clay Adams, Incorporated, New York, New York 10010

RESULTS

Considering all of the trials from the three repetitions as one group, laboratory analysis indicated that there was a phospholipid increase in thirteen trials out of the fifteen trials that were performed. Statistical analysis utilizing Student's "t" test for small samples showed that the overall increase in phospholipids for the fifteen trials was significant at the .01 level.

The phospholipid alteration elicited a mean increase of 12.5 mg. One trial had a decrease in phospholipid concentration, another showed no change, while thirteen increased.

TABLE V. PRE- AND POST-DECOMPRESSION PLASMA PHOSPHOLIPID VALUES*

	PHASE I		PHASE II		PHASE III	
Subject	Pre**	Post***	Pre	Post	Pre	Post
1	303	304	318	336	389	401
2	309	336	410	412	378	360
3	379	388	400	410	358	363
4	345	358	445	460	305	305
5	402	413	402	433	318	336

*Plasma phospholipid values in mg/100 ml
**Pre-decompression
***Post-decompression

Total white blood cell count, red blood cell count, and hematocrit were the hematological parameters measured to ascertain any alteration due to decompression. This data is delineated in Table XI.

The total white blood cell counts for the fifteen trials indicated increased total counts in nine trials and decreased counts in six trials. Changes varied from a low of about 1% to a high of 50% with a mean decrease of 9%.

Red blood cell counts indicated decreased values in twelve trials. Two of the three that decreased were from different repetitions of the same dog. Therefore, approximately four out of the five dogs, or 80%, showed depressed red blood cell counts in all three repetitions. Percent changes ranged from 1% to 55% in terms of red blood cell concentration with a mean decrease of about 15%.

The packed cell volume, or hematocrit, was elevated in eight trials, depressed in five trials, and demonstrated no alteration in two trials. The range of hematocrit changes was from 0% to 16% with a mean of about 3.8%.

Overall, total white blood cell counts showed an increasing trend (9 out of 15 or 60%). In addition, red blood cell counts indicated a decreasing trend in concentration (12 out of 15 or 80%). However, the

blood cell counts. However, the hematocrit was somewhat more evenly distributed with eight showing an increase (53%) while five showed a decrease (33%) and two demonstrated no change.

Subjects displayed increased respiratory depth and rate, occasional gasping, and extensive appendage involvement following rapid decompression. Figure 3 illustrates a typical response to rapid decompression. Therapeutical recompression alleviated these signs.

DISCUSSION

The elevated phospholipid levels following rapid decompression may provide additional information relative to etiological factors involving decompression sickness. If the tissue factor which initiates coagulation is a phospholipid or a number of phospholipids,[28, 36] the experimental findings presented here support the DIC Theory of decompression sickness.

Guyton has indicated the phospholipid, phosphatidyl ethanolamine, as being a primary factor initiating blood coagulation.[28] A quantitative test for this specific phospholipid utilizing paper chromatography, spectrophotometry, or another method may provide more information as to its blood levels following fulminating decompression relative to pre-decompression blood levels. Perhaps, then, a truer picture of phosphatidyl ethanolamine's role in decompression sickness would be known.

Assuming that the observed rise in phospholipids was composed of sufficient phospholipid coagulation factor (i.e. phosphatidyl ethanolamine) to elicit diffuse capillary coagulation, consideration then must be given to methodology detecting intracapillary coagulation. Holland and Philip have performed in vivo research concerning the role of blood clots in decompression sickness.[36, 49]

They actually observed blood clot formation due to platelet aggregation. However, they did not obtain data relative to phospholipid changes during their projects.[36, 49] Perhaps a more definitive picture of phosphatidyl ethanolamine's role in decompression sickness could be obtained if simultaneous measures of blood clot formation and phosphatidyl ethanolamine concentration were obtained.

Hematological data obtained from this project showed alterations from the control sample in all three parameters measured (Table XI). However, the overall importance of these changes is not certain.

The red blood cell count decrease indicates hemodilution following rapid decompression. In five of fifteen cases, the hematocrit also decreased. This factor also indicates hemodilution. Review of literature constantly states that hemoconcentration results from fulminating decompression.[16, 19, 32, 47] However, such was not the case as shown experimentally in these investigations.

Eight of the fifteen, or 53%, did have post-decompression elevated hematocrits. This fact suggests hemoconcentration did occur even though the red blood cell count decreased. Hemoconcentration is evidently due to alterations in capillary dynamics with plasma exvasation following the altered capillary permeability or changes in osmotic pressure gradients during decompression

sickness.

Hemoconcentration causes increased workload on the heart due to increased viscosity of the blood. The total resistance of the capillary beds due to hemoconcentration has a deleterious effect on the efficiency of tissue perfusion and cardiac function. Eventually the tissue subserved by the cardiovascular system become hypoxic and cease to function.(28)

The overall mean decrease of 9% in the white blood cell count may be due to one of two factors. The first is hemodilution. Red blood cell count and hematocrit both indicate an overall decrease. Therefore, hemodilution may be occurring. Hemodilution would decrease the white blood cell concentration and a depressed white blood cell count results. The second factor is exvasation of phagocytic white blood cells. Decompression sickness probably causes extensive tissue trauma. Trauma quite often causes the release of a number of substances from the injured cells. These substances, in part, cause the margination and diapedesis of white blood cells to the injured tissue. If the extent of the trauma is widespread an initial mass exvasation of white blood cells would occur. Subsequently, the white blood cell count would be depressed.

The signs of decompression sickness encountered in

these investigations are enumerated in Table VII. By far the most common occurring sign involved the rear appendages. Figure 3 illustrates this sign. The large dog in Figure 3 has his left, rear leg extended. Other typical responses involved full extension of the right, rear leg; contraction of the right, rear leg; contraction of the left, rear leg and full contraction of both rear legs. Front leg involvement was limited. However, Figure 3 shows front leg extension by the dog on the right. The appendage involvement exhibited itself approximately five minutes after fulminating decompression.

Evidently the trauma of rapid decompression affected the appendage's blood flow, innervation, or both, to illicit such a response. Temporary spinal cord derangement due to rapid decompression may be another contributing factor.

The effects of rapid decompression on the respiratory system have been observed by other investigators.(52, 56) It is believed that the air emboli created by rapid decompression cause stasis of pulmonary blood flow by occluding the pulmonary capillaries. Ineffective gaseous exchange occurs and respiratory rate and depth is increased due to increased carbon dioxide in the blood. Eventually asphyxiation due to respiratory embarrassment occurs.

Figure 1. Experimental recompression chamber located at the Oahe Reservoir Powerhouse, Pierre, South Dakota.

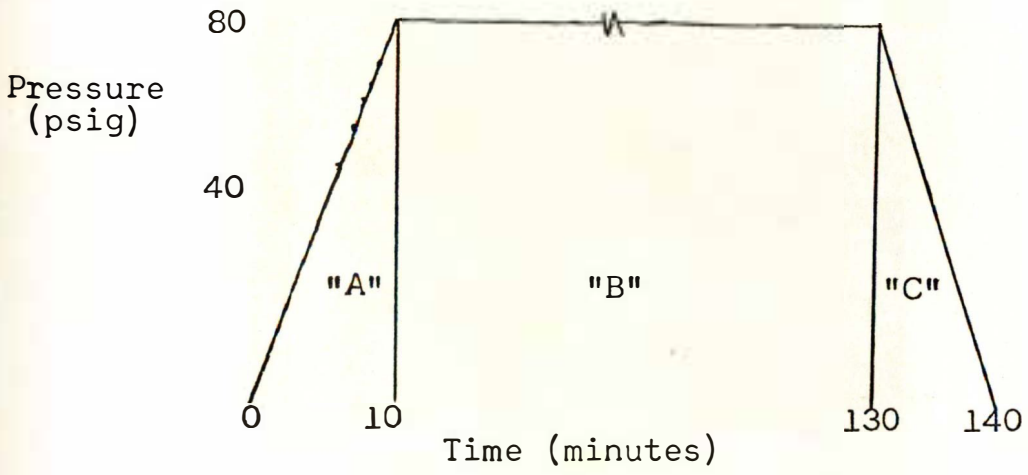

Figure 2

Figure 2 demonstrates the time-pressure periods to which the subjects were exposed. "A" represents the compression period. "B" represents the saturation period. "C" represents the rapid decompression period.

Figure 3. Illustrating effects of rapid decompression.

Note extension of left, rear leg by dog on left. Also note extension of left, front leg by dog on right.

The autopsy findings (Table IX) were indicative of massive pulmonary occlusion. Froth located in the trachea and bronchioles indicated that plasma exvasation through the pulmonary capillaries into the alveoli occurred before death. In addition, dilation of the right ventricle suggests that blood was not permitted into the pulmonary vasculature due to some obstruction in the lungs.

The rest of the organs appeared normal. Occasional subcutaneous hemorrhage was noted but this was probably a normal post-mortem response.

SUMMARY

Subjects were compressed to 80 psig in 10 minutes and maintained at that pressure for 120 minutes. They were decompressed in 10 minutes to induce decompression sickness. This procedure was repeated three times on five dogs for a total of fifteen individual trials.

Pre- and post-decompression blood samples were obtained. Total phospholipids and three hemogram parameters were measured from the blood samples.

The overall increase in phospholipids was significant at the .01 level utilizing Student's paired "t" test. This finding may contribute substantially to the Disseminated Intravascular Coagulation Theory in relation to etiological factors of decompression sickness.

Hemogram measures performed included hematocrit, red blood cell count, and white blood cell count. All three parameters showed alterations from the pre-decompression samples.

Red blood cell counts decreased in twelve of fifteen samples. Of the three samples that did increase, two were from the same subject of different repetitions. Percent decreases ranged from 1% to 55% with a mean decrease of 15%.

Hematocrit values increased in eight trials, decreased in five trials, and were not altered in two trials. This information tends to contradict current literature in relation to hematological changes following rapid decompression where a rise in hematocrit is almost always expected.(16, 19, 39)

The importance of the white blood cell changes following fulminating decompression is difficult to assess. Nine of fifteen trials did increase and six decreased.

Overall, the white blood cell count decreased by 9%. The white blood cell decrease may have been due to hemodilution or exvasation of the phagocytic white blood cells.

Perhaps, in addition to total white blood cell count, a differential count may provide considerable information in relation to changes in the granulocytes and agranulocytes as effected by rapid decompression.

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APPENDIX

TABLE I. EFFECTS OF RAPID DECOMPRESSION (5-6 seconds) FROM 65 PSI ON BLOOD GASES AND BLOOD PRESSURE (dog)*

	Art. PCO_2 mm Hg	Resp. Rate	%Sat. HbO_2	Blood Pressure	Art.-Venous O_2 Diff. vol. %	O_2 Capacity vol. %
Pre-treatment	45	20	90	116	3.6	22.8
Post-treatment	59	142	24	140 to 30	6.9	26.1
O_2 Recompression (30 psi 2 hrs)	56	40	88	90	11.7	27.3
1 Ata (Air)	59	125	26	100	19.6	29.8

*From Behnke, et. al.(3)

TABLE II. AGENTS AND MECHANISMS INVOLVED IN CARDIO-PULMONARY DERANGEMENTS CHARACTERISTIC OF DECOMPRESSION SICKNESS*

PULMONARY EMBOLIZATION		
Catecholamines Histamine	Embolic Distortion of Blood Vessels	Dyspnea
Bradykinin	Interruption Pulsatile Flow	Bradycardia
Serotonin	Vasoconstriction	Fall Peripheral Blood Pressure
Reduced Plasma Lipid	Pulmonary Arterial Hypertension	
Cell Clumping Platelet Aggregation	Anoxemia Hypercarbia-Fall in pH (Blood, fixed tissues) Hemoconcentration Circulatory Shock	

*From Behnke, et. al.(3)

TABLE III. ETIOLOGICAL THEORIES OF DECOMPRESSION SICKNESS

1.	AIR EMBOLISM	Classical theory. Air bubbles cause all maladies associated with decompression sickness.
2.	DISSEMINATED INTRA-VASCULAR COAGULATION (DIC)	Air Bubbles initiate coagulation process.
3.	LIPID EMBOLIZATION	Trauma to body tissue causes release of lipids into blood-stream.

TABLE IV. VARIOUS DATA ON EXPERIMENTAL DOGS

				Visible Signs of DCS*					
				Phase I		Phase II		Phase III	
Subject	Age(Mos.)	Weight(Lbs.)	Sex	Yes	No	Yes	No	Yes	No
1	12	30	M		X	X		X	
2	10	24	M		X	X		X	
3	24	47	M		X	X		X	
4	28	49	F	X		X		X	
5	36	50	M	X		X		X	

*DCS=Decompression Sickness

TABLE VI. LIST OF PRESSURE EQUIVALENTS

PSI	Ft. H_2O	Atm.	mm Hg	pN_2	pO_2
80	180	5.44	4134	3317	816
75	170	5.10	3870	3096	775
70	157	4.80	3618	2893	724
65	146	4.42	3359	2687	672
60	135	4.08	3100	2480	620
55	123	3.74	2842	2273	569
50	113	3.41	2591	2072	519
45	103	3.06	2325	1860	465
40	95	2.72	2052	1641	411
35	83	2.36	1824	1459	365
30	67	2.04	1520	1216	304
25	57	1.70	1292	1023	269
20	45	1.30	988	788	200
15	36	1.00	760	608	152
10	24	0.70	532	425	107
5	12	0.30	228	182	46

Note: All of the above values are approximate and based on gauge pressure (14.7 PSI=33 Ft. H_2O=1 Atmosphere of pressure).

TABLE VII. SIGNS OF DECOMPRESSION SICKNESS

1. High pitched whining
2. Apprehension
3. Rapid Respiration
4. Occasional gasping (chokes)
5. Congested lungs
6. Increase in respiratory depth
7. Intense contraction of right rear leg to lateral side of subject
8. Contraction of right rear leg to lateral side with full extension of left rear leg
9. Full extension of both rear legs
10. Intense contraction of both rear legs to lateral side
11. Minor effects on front legs
12. Gasping reflex in terminal cases with bluing of tongue

TABLE VIII. THERAPEUTIC DECOMPRESSION TABLE*

Feet (Sea Water Equivalent)	Minutes
180	10
150	15
130	10
110	10
90	20
70	20
50	20
35	30
15	30
0	-

*Modified from Reeves and Workman.[53]

TABLE IX. AUTOPSY FINDINGS*

Trachea	Froth accumulations at tracheal bifurcation No hemorrhage
Bronchi	Froth accumulations blocking bronchi passageways
Heart	Right chamber very dilated Left chamber occasionally dilated
Lungs	Massive accumulations of blood and clear fluid
Other	Remainder of organs, i.e. stomach, brain spleen, intestines, kidneys, liver, appeared normal

*Based on findings from three terminal decompression sickness subjects.

TABLE X. PROCEDURE FOR TOTAL PHOSPHOLIPID DETERMINATION*

1. Mix 0.2 ml plasma with 5 ml trichloracetic acid
2. Centrifuge 7 minutes at 750 rpm
3. Add 1 ml digestion mixture (conc. sulfuric acid + perchloric acid) to precipitate
4. Heat at 37°C for 30 minutes
5. Add 1 ml distilled water
6. Boil for 15 seconds
7. Add 1 ml sodium acetate
8. Add enough distilled water to make total volume 10 ml
9. Add 1 ml ammonium molybdate
10. Add 1 ml Elon
11. Let stand 15 minutes and read on spectrophotometer at 700 millimicrons against a blank
12. Prepare standard and read same as 11
13. Calculate phospholipid concentration by using:

$$\frac{\text{Optical Density of Unknown}}{\text{Optical Density of Standard}} \times 250 = \text{mg phospholipid per 100 ml of plasma}$$

*Procedure obtained from Connerty, Harold V., et. al.: Simplified determination of the lipid components of blood serum. J. Clin. Chem. 7:37-53, 1961.

TABLE XI. HEMATOLOGICAL DATA

	Hematocrit (%)		WBC Count (X1000)		RBC Count (X1,000,000)	
Subject	Pre*	Post**	Pre	Post	Pre	Post
			PHASE I			
1	35	35	10.45	5.00	5.25	5.06
2	38	38	4.10	4.85	3.39	3.37
3	54	38	5.20	5.45	5.08	4.60
4	38	41	4.65	5.20	4.39	3.62
5	42	37	5.05	6.10	4.68	4.43
			PHASE II			
1	25	34	14.45	12.90	3.42	3.36
2	25	30	8.75	10.90	3.78	4.30
3	43	38	6.95	10.35	4.66	3.52
4	29	30	12.40	10.20	4.17	3.74
5	38	35	10.80	10.60	3.75	5.60
			PHASE III			
1	40	47	14.05	14.85	6.54	4.61
2	44	41	8.90	9.60	6.74	6.38
3	49	50	7.50	6.45	7.54	3.34
4	53	58	12.85	8.20	6.28	7.20
5	35	38	8.66	9.56	4.48	4.91

*Pre-decompression
**Post-decompression