HEMATOLOGIC RESPONSES TO SEVERE DECOMPRESSION STRESS

by

Michael J. Jacey,
Donald V. Tappan
and
Keith R. Ritzler

NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY
REPORT NUMBER 744

Bureau of Medicine and Surgery, Navy Department
Research Work Unit M4306. 02-5003BA9K. 01

Reviewed and Approved by:

Charles F. Gell

Charles F. Gell, M.D., D.Sc. (Med)
SCIENTIFIC DIRECTOR
NavSubMedRschLab

Approved and Released by:

J. H. Baker

J. H. Baker, CAPT MC USN
OFFICER IN CHARGE
NavSubMedRschLab

SUMMARY PAGE

THE PROBLEM

To evaluate the effects of decompression insult on the hematologic system in laboratory animals in order to better understand the effects of decompression injury in man.

FINDINGS

Red blood cell counts, hemoglobin and hematocrit levels all demonstrate a hemoconcentration occurring in rats within one hour after severe decompression but disappearing by one day post-decompression only to return within the following day accompanied by a microcytic hypochromic syndrome indicating changes in cell number and volume. Shifts in white blood cell populations which occur promptly after a decompression incident appear to be a direct result of adrenal cortical response to the stress. All leucocyte parameters return to control values within one day after decompression injury and remain at essentially normal levels.

APPLICATION

While a post-decompression hemoconcentration has been previously reported, the findings of a return of a plasma deficit accompanied by a decrease in erythrocyte volume and hypertonic plasma, after an apparent recovery period, has not to our knowledge been heretofore observed. Since decompression injury may have long-term hematologic effects in addition to the immediate painful symptoms of bends, it is recommended that divers subjected to possible decompression damage be allowed adequate time for recovery before further diving. Careful scrutiny of hematologic changes may serve as a useful monitor for otherwise undetected decompression injury.

ADMINISTRATIVE INFORMATION

This investigation was conducted as part of Bureau of Medicine and Surgery Research Unit M4306.02-5003BA9K. The present report is Number one on this work unit. It was submitted for review on 7 February 1973, approved for publication on 3 May 1973 and designated as NavSubMedRschLab Report No. 744.

PUBLISHED BY THE NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

ABSTRACT

Red and white blood cell parameters were measured in the blood of mature Sprague-Dawley rats at various times up to three days following compression and severe decompression. Acute decompression stress (one hour post-surfacing) produced an increase in red cell count, hematocrit and hemoglobin, without concomitant alterations in red cell indices. This was taken as evidence of hemoconcentration. The normalization of red cell parameters by one day post-decompression was indicative of a spontaneous resolution of the acute hypovolemia. During the late phase of the observation period (day two), red cell counts again rose without changes in hematocrit and hemoglobin levels. At day three, red cell counts further increased with accompanying hematocrit elevation but without alteration in hemoglobin. During this period (days two and three) mean corpuscular volume and mean corpuscular hemoglobin declined. Parallel decreases in these indices are indicative of microcytic hypochromic morphology and may be caused by the time-related hypertonic plasma. At no time was evidence of reticulocytosis or decrease in red cell counts, hemoglobin or hematocrit levels noted.

The increase in hematocrit and red cell count at the end of the experiment suggests another episode of hemoconcentration. These data demonstrate, then, that an apparent recovery from severe dysbaric stress may be followed by the development of a microcytic hypochromic condition, with an accompanying hemoconcentration.

Acute decompression stress caused a transient lymphocytic leucopenia together with a concomitant neutrophilia which was both relative and absolute. All white blood cell parameters returned to control values at one day post-decompression and remained at these levels for the duration of the observation period. The acute leucocytic changes following exposure to severe decompression fit the classical concept of adrenal cortical responses to stress.

HEMATOLOGIC RESPONSES TO SEVERE DECOMPRESSION STRESS

INTRODUCTION

Severe decompression sickness is usually accompanied by hemoconcentration [1, 4, 5, 11]. Plasma loss to the extravascular spaces has been shown to be the causative factor in the development of polycythemia following decompression stress and is attributed to increased capillary permeability [5, 11]. Treatment with plasma expanders is indicated in human cases of hemoconcentration resulting from inadequate decompression [2, 3, 4, 11].

Animal studies by Cockett *et al*,[5] designed to investigate the loss of plasma volume after severe decompression, demonstrated increasing plasma deficit with time following surfacing. Associated histopathology included congestion of lung, liver, kidney, and brain.

While there is a consistent body of data available documenting acute changes in red cell parameters following decompression [1, 2, 3, 4, 5, 11], leucocyte counts after decompression in animals and man have been variously reported as depressed [13, 21], normal [13, 15] or elevated [3, 14, 15, 17].

By contrast, little has been recorded concerning the behavior of hematologic parameters during spontaneous recovery from an acute episode of severe decompression stress. With this in mind, a study was undertaken to investigate and identify potential hematologic alterations during a period of recovery from dysbaric stress.

For the purpose of this study, severe decompression stress is defined as that stress which is produced by decompression which is neither safe (100% survival) nor explosive (90-100% death rate within one hour surfacing). By these criteria, then, a 66% survival rate after one hour post-surfacing [10] would constitute severe decompression.

METHODS

Mature male rats of the Sprague-Dawley strain averaging 538 grams in weight were pressurized and decompressed according to a schedule previously determined by us [10] to give a proper incidence of survival. Briefly, the schedule entails pressurization on air to 300 feet of sea water at 60 feet/minute with a bottom time of 25 minutes. Decompression was accomplished in two stages: 300 feet of sea water to 60 feet of sea water at 20 feet/minute with a hold at 60 feet for 15 minutes and then to the surface at 4 feet/minute.

Following completion of the dive, animals were held in clean cages with food and water available for varying periods of time up to three days. Control animals were maintained in adjacent cages. A sufficient number of control animals was sacrificed at each time point. All animals were weighed daily. At the appropriate time period, the animals, control and experimental, were injected intraperitoneally with 40 mg. sodium pentobarbital/kg body weight. When the desired stage of anaesthesia was reached, blood was collected

anaerobically in siliconized syringes from the abdominal aorta. Immediately thereafter, 2 ml. aliquots were transferred to separate tubes containing .2 ml of a 5% sodium citrate solution and 20 microliters heparin (1000 units/cc), respectively. Blood and anticoagulant were mixed by several gentle inversions. In a separate but identical experiment, without prior bleeding, the vasculature of the spleen was ligated, the organ excised and the spleen weight/body weight ratio determined.

Red and white cell counts using citrated blood were done on a Coulter Model F electronic counter (Coulter Electronic Co.). The remaining parameters were measured utilizing heparinized blood. Hemoglobin concentrations were determined with the "Diagnostest" hemoglobin reagent set (Dow Chemical Co.). Microhematocrit was determined by using the high speed centrifugation method. Reticulocyte and white cell differential counts were performed according to standard methods [12, 22].

The red cell indices, mean corpuscular volume, mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration were calculated from the red cell count, hemoglobin concentration and the hematocrit.

Plasma osmotic pressure determinations were performed utilizing the Fiske Osmometer, Model G-62. (Fiske Associates, Inc.).

RESULTS

The effects of severe decompression on red cell count, hemoglobin, hematocrit and reticulocyte count over a three-day period are shown in Table 1. Red cell count significantly increased by one hour post-decompression and decreased to control level by one day. This transient decline was followed by another dramatic increase in red cell count at day two which continued to the end of the experiment. Hemoglobin rose markedly at one hour, fell to control by one day, and remained at this level for the remainder of the experiment.

The hematocrit exhibited a bimodal response. Acute decompression produced a significant elevation which declined at day one, stayed at this level for another day and rose again at three day post-decompression. Reticulocyte count was unchanged during the three-day period of observation.

The responses of the red cell indices to acute decompression followed by a three day recovery period are shown in Figure 1. Mean corpuscular volume was unaltered by the stress of acute decompression. However, a significant decrease in this index occurred during the late phase of the observation period. A parallel decline was seen in mean corpuscular hemoglobin. No changes occurred in mean corpuscular hemoglobin concentration.

White cell and differential counts are depicted in Table 2. A significant decrease in total leucocyte count was detected concomitant with an increase in percent neutrophils and a decrease in percent lymphocytes within one hour after severe decompression stress. These counts returned to control levels

Table 1. Effects of Severe Decompression on Rat Red Cell Count, Hemoglobin, Hematocrit and Reticulocyte Count

		POST DECOMPRESSION TIMES				
		CONTROL	1 HOUR	1 DAY	2 DAYS	3 DAYS
RED CELL COUNT $\frac{\text{Cells} \times 10^6}{\text{mm}^3}$	Mean	5.135	6.121*	5.069	6.276*	6.659*
	SE	.265	.280	.275	.251	.351
	N	30	28	26	28	30
HEMOGLOBIN Grams/100 ml Blood	Mean	14.96	16.19 *	15.39	14.59	15.21
	SE	.17	.24	.15	.25	.17
	N	22	24	20	23	24
HEMATOCRIT % Packed Cells	Mean	44.3	46.6 *	43.9	44.4	46.5 *
	SE	.3	.4	.6	.6	.6
	N	32	27	28	28	24
RETICULOCYTE COUNT % of Red Cells	Mean	2.67	2.70	2.19	3.26	3.24
	SE	.32	.37	.29	.33	.28
	N	31	30	32	29	30

*Statistically significant at the 5% level or better

by one day after surfacing and remained at these values for the duration of the experiment.

Data on body weights and spleen weight/body weight ratios are reported in Table 3. No differences were observed in body weights of the animals in each group. Severe decompression had no effect on spleen weight/body weight ratio for the three-day period of observation. Osmotic pressure measurements in plasma are given in Table 4. A hypertonic plasma was noted at one and three days post-decompression.

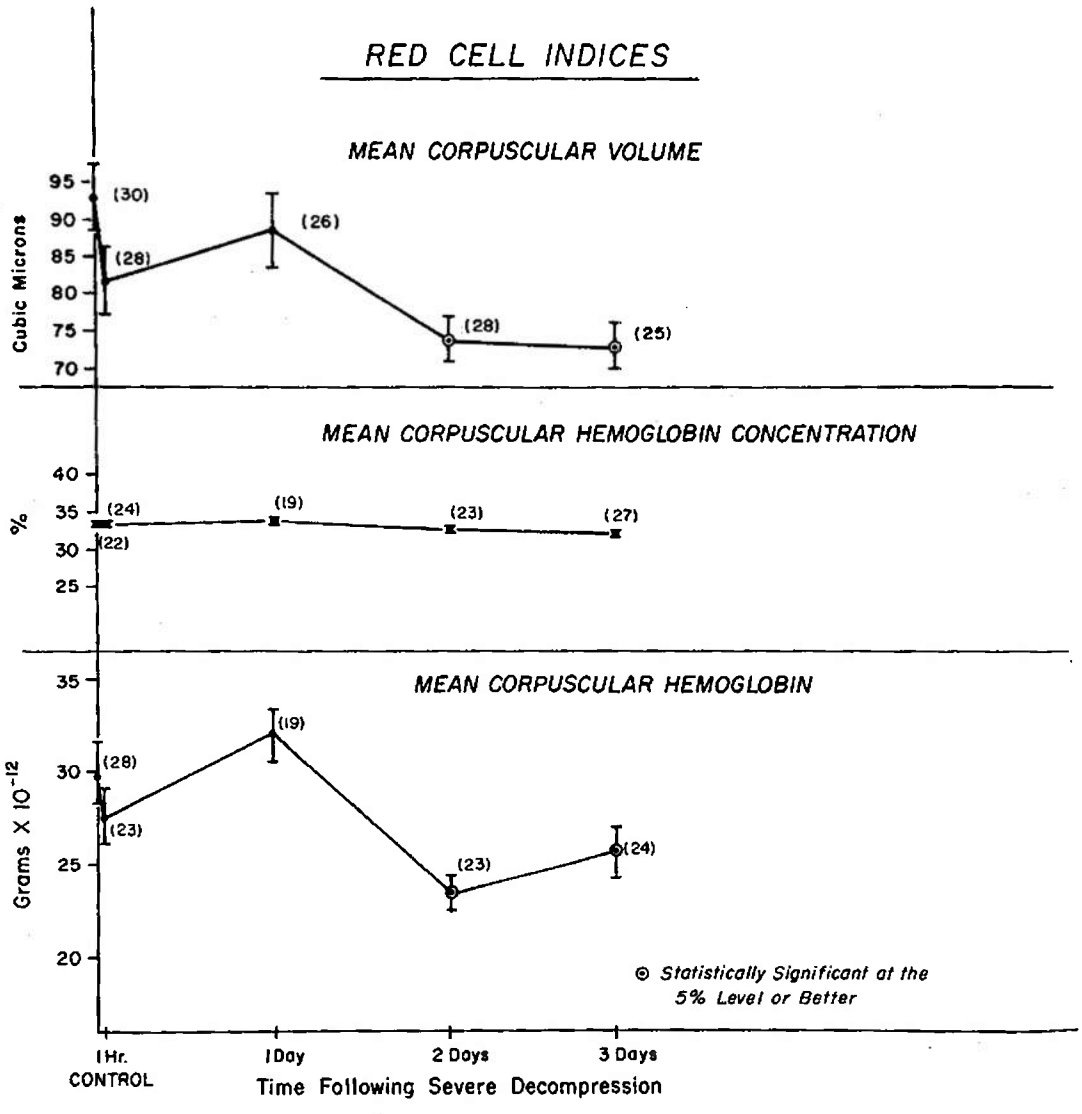

Fig. 1. Erythrocyte responses during recovery from decompression stress in rats. Numbers within the parentheses equal number of animals in each group.

Table 2. Response of Leucocyte and Differential Counts Following Severe Decompression

	WHITE BLOOD CELLS $\frac{\text{Cells} \times 10^3}{\text{mm}^3}$	NEUTROPHILS %	LYMPHOCYTES %
CONTROL	7.47 ±.35 (31)	18.8 ±2.1 (30)	78.7 ±2.3 (31)
POST DECOMPRESSION PERIODS			
1 HOUR	5.86 ±.29* (28)	30.1 ±3.3* (28)	68.3 ±3.4* (28)
1 DAY	6.91 ±.48 (29)	22.4 ±2.4 (26)	74.7 ±2.6 (26)
2 DAYS	7.56 ±.46 (29)	17.3 ±1.8 (28)	80.9 ±2.0 (28)
3 DAYS	7.21 ±.35 (30)	15.0 ±1.4 (29)	84.0 ±1.5 (29)

Values are means ±SEM. Numbers in ( ) equal number of animals.

* Statistically significant at the 5% level or better.

Table 3. Body Weight and Spleen Weight/Body Weight Ratios of Rats Following Severe Decompression

	CONTROL	POST SURFACING			
		1 HOUR	1 DAY	2 DAYS	3 DAYS
Body Weight Grams	546±14.2 (34)	516±11.2 (30)	539.8±12.1 (32)	538.8±11.8 (30)	531.8±14.8 (30)
Spleen Weight/ Body Weight Ratio x 10^{-2}	178±.009 (11)	171±.014 (12)	155±.009 (11)	163±.005 (12)	171±.007 (12)

Values are means ±SEM. Numbers in ( ) equal number of animals.

Table 4. Osmotic Pressure Measurements In Plasma Of Rats Following Severe Decompression

	CONTROL	POST SURFACING			
		1 HOUR	1 DAY	2 DAYS	3 DAYS
Osmotic Pressure mosmols/1	296.3±1.5 (12)	294.2±3.2 (17)	307.8±2.3* (17)	295.6±2.8 (24)	305.0±2.2* (19)

Values are means ±SEM. Numbers in ( ) equal number of animals.

*Statistically significant at the 5% level or better.

Although in some of the experiments reported by other workers studying decompression stress, animals have been graded according to degree of symptoms, it should be pointed out that no attempt was made to grade our experimental animals on the basis of severity of symptoms. The data presented here are a strict presentation of results obtained on all animals in a particular time group.

DISCUSSION

Erythrocyte Factors

In a review of 35 cases of dysbarism, 12 of which included hematocrit values, Malette *et al.*[11] reported that hemoconcentration was a consistent and striking abnormal finding. Barnard and co-workers[2], Brunner *et al.*[3] and Cockett and Nakamura[4] demonstrated large plasma volume deficits in humans with acute decompression sickness. Philp and co-investigators[15] and Cockett *et al.*[5] showed marked hemoconcentration in rats and dogs, respectively, exposed to severe decompression schedules.

Our observation of acute post-decompression elevations of hemoglobin, hematocrit and red cell count without concomitant alterations in the red cell indices together with a normal reticulocyte count agrees with the above findings and may be taken as an indication of plasma volume deficit.

In a study of the mechanism of acute hypovolemia in dogs severely decompressed, Cockett and co-workers[4] detected no changes in red cell volumes while increasing plasma volume deficits were noted with time. Brunner *et al.*[3] also documented two cases of human hypovolemia following incomplete decompression. Both cases were treated with plasma expanders with subsequent recovery. Germaine to this point is the fact that red cell volumes did not change while plasma volume increased during the treatment period of several days.

In addition to hemoconcentration due to plasma deficit, there exists the possibility that spleen stimulation may account for some of the increases in red cell count and hematocrit seen early and late in the observation period. Although the spleen is known to function as a blood store[19] which in animals can accomodate over 10% of the blood volume, spleen weight/body weight ratios determined by us in control and experimental animals indicated no significant differences. In the face of increases in red cell counts of over 20% acutely and in the late phases of the observation period, the contribution of the spleen would be negligible.

Moreover the possibility of a major participation by the spleen seems unlikely when the experiments of Cockett and his group[5] are taken into consideration. These investigators found hemoconcentration and plasma volume loss without alterations in red cell volume in both intact and spleenectomized dogs suffering from dysbarism. In addition, the constant reticulocyte counts observed in our experiments would further indicate that an increase in red cell volume due to increased red cell production is unlikely.

A possible explanation of fluid loss, diaphoresis due to excessive chamber temperature, has been envoked by Philp and co-investigators [14] as a partial answer to the hemoconcentration noted by them in human subjects. Temperature profiles for our dive schedule [10], however, indicated only a peak at the end of compression, five minutes from the surface, which was promptly corrected by venting and subsequent decompression.

Since all red cell parameters were at control levels by one day post-decompression, it is evident that the hypovolemia noted acutely after severe decompression was spontaneously resolved at least temporarily.

Two days after surfacing, red cell count again rose significantly without concomitant changes in hemoglobin or hematocrit. Day three produced a further rise in red cell count together with an elevated hematocrit but without any alteration in hemoglobin level. The end of the observation period was marked by a conspicuous decline in both mean corpuscular volume and mean corpuscular hemoglobin.

Parallel decreases in these red cell indices are indicative of the existence of microcytic hypochromic morphology. The iron deficient anemia are characteristically microcytic and hypochromic. A physiologic deficiency of iron may be the result of low intake, inadequate absorption, inadequate utilization, increased demands, excessive loss or a combination of any or all of these factors [12, 22]. Furthermore according to Hays and Swenson [9] in iron deficiency states, the number of cells is not affected but the cells are microcytic and usually hypochromic. The lack of any changes in body weight would seem to discount the first two possibilities. Moreover, Garcia [7] has shown that it is highly unlikely that a nutritional anemia could develop in starved rats in so short a time. In regards to the possibility of excessive loss of iron due to hemorrhage, at no time did any red cell parameters especially hemoglobin decline below the control level.

An examination of the red cell indices themselves is in order.

Mean corpuscular volume is a measure of the average volume of each erythrocyte. Mean corpuscular hemoglobin indicates the average weight of hemoglobin per erythrocyte. Mean corpuscular hemoglobin concentration reflects the average weight of hemoglobin per 100 ml. of packed cells. In our experiments mean corpuscular hemoglobin declined during the late portion of the observation period while mean corpuscular hemoglobin concentration was unchanged. The apparent hypochromic condition was not caused by decreases in hemoglobin content of whole blood but rather by an increase in red cell count.

Fluctuations in erythrocyte volume may be caused by alterations in the osmotic mileau of the plasma. It has been amply demonstrated [20] that the erythrocyte is a perfect osmometer over a wide range of osmotic concentrations (210 to 520 mosmol); gaining, i.e; swelling, or losing, i.e., shrinking, sufficient water to stay in osmotic equilibrium with its

surroundings. Stated in other terms, the activity of water inside the red cell is the same as that outside the cell over a wide range of osmotic conditions.

The hypertonic plasma detected at the third day post-decompression coupled with the second episode of hemoconcentration would offer a partial explanation for the presence of this microcytic hypochromic morphology following exposure to severe decompression stress.

While the increases in red cell count and microhematocrit together with normal reticulocyte count are certainly suggestive of a second episode of hemoconcentration, systematic red cell and plasma volume studies will be required to determine whether the phenomenon follows a unique pattern.

It would appear then, that a spontaneous recovery from severe dysbaric stress is followed by the development of a microcytic, hypochromic morphological condition with an accompanying hemoconcentration.

Leucocyte Factors

In addition to erythrocyte and plasma volume effects, acute decompression stress also caused a transient lymphocytic leucopenia together with a concomitant neutrophilia which was both relative and absolute. Twenty-four hours after surfacing, however, all white cell parameters were at normal levels.

The endocrine system is an important regulator of the number of leucocytes in the blood and probably controls most of the changes observed in our rats subjected to decompression insult. Hormones affect the production of leucocytes in the blood-forming organs, their storage and release from tissues and their disintegration[12]. The classical studies of Selye[18] on the alarm reaction demonstrated that, as a result of stress, animals showed a typical hematological triad of lymphopenia, eosinopenia and neutrophilia. Later, Gordon[8] showed the same effects were produced by the administration of ACTH. The lymphopenic response to ACTH is abolished by adrenalectomy. Selye[18] also reported that within a few hours after the alarm reaction, there is a marked disintegration of lymphocytes which is preventable by adrenalectomy. In contrast, stress and adrenal pituitary factors produces an enhancement of the number of circulating neutrophils. Neutrophilic leucocytosis is probably produced by myelopoiesis in the bone marrow and is further augmented by increased release of neutrophils from the bone marrow, lung, and other organs[12].

Williamson[21] in an extended series of experiments involving saturation of mice for two days at various pressures, studied white cell and differential counts after safe decompression. He noted a lymphocytic leucopenia with accompanying neutrophilia in intact animals following safe decompression from depths equivalent to 300 and 500 psig. A normal white cell and differential picture was seen when adrenalectomized mice were subjected to the same pressure profile. The classical studies of Dougherty and White[6] had previously demonstrated a lymphocytic leucopenia with parallel

neutrophilia within one hour after injecting mice with 1 mg of a pituitary adrenotropic substance and a complete remission of these conditions after twenty-four hours.

Emphasis must be put upon the fact that while the rat and mouse have similar differential pictures in terms of per cent neutrophils and lymphocytes, that picture seen in humans is reversed. While leucopenia due to a reduction in lymphocyte levels is rarely encounted in pathological states in humans, it follows the administration of adrenocorticotropic hormone or adrenal cortical hormone[12].

It is well known that physiological leucocytosis may be caused by exercise, emotional disturbances and exposure to cold, besides other factors while pathological leucocytosis may result from acidosis.

Contrasting results in leucocyte counts following pressurization and subsequent decompression with and without symptoms of decompression sickness have been reported by various investigators. Osborne and fellow workers[13] demonstrated a decrease in white cell count due to a lymphopenia in rats exposed to high pressure oxygen at 4 ATA. No neutrophilia was noted. Pressurization with air under the same experimental conditions resulted in no changes in leucocyte parameters. From this the authors concluded that 100% oxygen at 60 psia presents a greater physiological stress than does air at the same pressure. Normal and elevated leucocyte counts after severe decompression were reported by Philp *et al.*[15] in rats preconditioned with various diets and drugs. Their decompression profile required treadmill exercise at altitude. In a separate study Philp and co-workers[14] found normal white cell counts in humans with and without symptoms of decompression sickness following a dive. Brunner *et al.*[3] published data on a case of post-decompression shock due to plasma loss in which a high leucocyte count existed in the face of a profound hemoconcentration.

Schaefer and co-investigators in two separate experiments documented leucocytosis during prolonged saturation at 7 ATA of sea water[16] and after decompression from 1000 feet of sea water[17]. Retention of CO_2 was reported for the saturation study while hyperexcitable state was detected during decompression from 1000 feet of sea water.

Whether these contrasting findings are a function of the severity of stress, different sampling times, or other factors, remains to be seen.

In any event, the acute transient lymphocytic leucopenia with accompanying relative and absolute neutrophilia observed in our experiments is more striking when viewed against the background of the time-related hemoconcentration.

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UNCLASSIFIED
Security Classification

DOCUMENT CONTROL DATA - R & D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY *(Corporate author)*
NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY
Naval Submarine Medical Center

2a. REPORT SECURITY CLASSIFICATION
UNCLASSIFIED

2b. GROUP

3. REPORT TITLE
HEMATOLOGIC RESPONSES TO SEVERE DECOMPRESSION STRESS

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*
Interim report

5. AUTHOR(S) *(First name, middle initial, last name)*
Michael J. Jacey, Donald V. Tappan and Keith R. Ritzler

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
3 May 1973	12	22

8a. CONTRACT OR GRANT NO.	9a. ORIGINATOR'S REPORT NUMBER(S)
b. PROJECT NO.	NSMRL Report Number 744
c. M4306.02-5003BA9K	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*
d.	

10. DISTRIBUTION STATEMENT
Approved for public release; distribution unlimited

11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	Naval Submarine Medical Center Box 600, Naval Submarine Base Groton, Connecticut 06340

13. ABSTRACT

Red and white cell parameters were measured in the blood of mature Sprague-Dawley rats at various time points up to three days following compression and severe decompression. Acute decompression stress (one hour post-surfacing) produced an increase in red cell count, hematocrit and hemoglobin without concomitant alterations in red cell indices. This was taken as evidence of hemoconcentration. The normalization of red cell parameters by one day post-decompression was indicative of a spontaneous resolution of the acute hypovolemia. During the late phase of the observation period (day two), red cell count again rose without changes in hematocrit and hemoglobin. At day three, red cell count further increased with accompanying hematocrit elevation but without alteration in hemoglobin. During this period (days two and three) mean corpuscular volume and mean corpuscular hemoglobin declined. Parallel decreases in these indices are indicative of a microcytic hypochromic morphology and may be related to the time-related hypertonic plasma. At no time was evidence of reticulocytosis or decreases in red cell count, hemoglobin, or hematocrit noted. The increase in hematocrit and red cell count at the end of the experiment suggest another episode of hemoconcentration. These data demonstrate, then, that an apparent recovery from severe dysbaric stress may be followed by the development of a microcytic hypochromic condition, with an accompanying hemoconcentration. Acute decompression stress caused a transient lymphocytic leucopenia together with a concomitant neutrophilia which was both relative and absolute. All white cell parameters returned to control values at one day post-decompression and remained at these values for the duration of the observation period. The acute leucocyte changes following exposure to severe decompression fit the classical concept of adrenal cortical responses to stress.

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Decompression Effects						
Dysbaric Stress						
Hemoconcentration in Decompression						
Hypovolemia in Decompression						
Microcytic Morphology						
Hypochromic Morphology						