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Hematologic changes after daily asymptomatic dives

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Diercks, K. J., and P. T. Eisman. 1977. Hematologic changes after daily asymptomatic dives. Undersea Biomed. Res. 4(4): 325–331.—Hematologic alterations after daily exposure to compression/decompression in open water are described. A standard dive to 100 fsw for 25 min was employed. Erythrocytes decreased postdive, reaching an apparent minimum 4 to 6 h after exposure and recovering to normal or supranormal value by 20 h postdive. Measurements of plasma volume after repeated dives showed a significant increase in volume at 4 h postdive. It is speculated that a phenomenon akin to cardiovascular deconditioning, caused by loss of the hydrostatic blood column in the lower extremities during immersion, results in a transitory fluid recruitment that may persist following several daily dives. Pre- and postdive partial thromboplastin times measured after repeated exposures failed to show a consistent response to compression/decompression.

immersion
erythrocytes
hemoglobin
plasma volume
fluid recruitment

The hematologic and hemostatic alterations occurring after severe decompression stress are well documented (Philp 1974; Ackles 1973), but conditions that precede or predispose to decompression injury are less well known or understood. It has been shown in guinea pigs that a second exposure to compression/decompression causes bubbles presumably entrapped in the capillaries after the first exposure to be released into the venous circulation (Gait, Miller, Paton, Smith, and Welch 1975). If a similar phenomenon occurs in man, it would seem that repetitive diving would predispose divers to decompression injury. Under actual dive conditions, however, the opposite has been the case: there is a lower rate of injury after repeated dives than after isolated ones (Biersner 1975). Diving schedule is probably a factor. Marked hematologic and hemostatic changes following repeated dives at 3-day intervals have been reported (Jacey, Tappan, and Gonzales 1975; Jacey, Gonzales, and Tappan 1976). Daily exposure may be cause for additional concern. This study was an attempt to examine hematologic changes as indicators of increased or decreased potential for decompression injury during daily repetitive diving.

METHODS

Two divers were studied. Diver One was 44 yr old and had been diving regularly for 16 yr; Diver Two was 26 yr old and had been diving regularly for approximately 2 yr. Both divers exhibited the same morphology; they were about 1.79 m tall and weighed approximately 66 kg.

A standard dive of 4 ATA for 25 min was established. The diver's task was to swim along a line between two anchor points at approximately 1.24 km/h. A repetitive schedule of one standard dive per day for one period of five and another of three consecutive days was examined.

We obtained blood samples by venipuncture, using vacutainers appropriate for the tests described. This was not a funded study; therefore, the number and extent of the hematological analyses performed were limited. Samples were taken 30 min predive and 4 h postdive, with additional samples obtained at shorter and longer postdive times on some days for better definition of the response curve. Leucocytes, erythrocytes (RBC) hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and MCH concentration (MCHC) were evaluated with a Coulter Counter Model S. We performed parallel evaluations on different counters using samples drawn at the same time to establish machine reliability. Differences between the machines were usually less than the specified tolerances for a single machine. Pre- and postdive prothrombin times (PTs) and partial thromboplastin times (PTTs) were measured on the last day of each of the repetitive-dive trials. Blood-volume estimates, from radioactive iodinated human serum albumin (RISA) plasma-volume determinations, were made before and after the last dive of the second trial period. We obtained control samples at the same times of day as the repetitive-diving samples during periods between trials.

RESULTS

We drew blood samples from both divers during periods of diving inactivity throughout the 8 mo preceding the first repetitive-dive trial and during the interim between trials to establish normal (base-line) values for the various blood tests. These values, including control-sample data, are listed in Table 1.

TABLE 1

RESULTS OF ANALYSES OF BLOOD SAMPLES FROM TWO DIVERS UNDER NORMAL NONDIVE CONDITIONS

	Leucocytes, 10^{-3}	RBC, 10^{-6}	Hb, gm	HCT, %	MCV, $10^{-3}\mu^3$	MCH, %	MCHC, %
Diver One $n = 31$	6.1 (1.2)	4.57 (0.15)	14.0 (0.5)	41.3 (1.4)	91 (1.0)	30.6 (0.6)	34.1 (0.6)
Diver Two $n = 23$	6.8 (0.6)	4.91 (0.17)	14.5 (0.5)	42.2 (1.3)	86 (1.0)	29.4 (0.5)	34.4 (0.6)

Values are means of samples obtained between April 1, 1975 and March 31, 1976; numbers in parentheses are standard deviations; n is number of samples; RBC is erythrocytes, Hb is hemoglobin; HCT is hematocrit; MCV is mean corpuscular volume; MCHC is mean corpuscular volume concentration.

The first repetitive-dive trial occurred in December 1975. Water temperature was 17.5°C. A standard dive had been made 7 days preceding the trial so that we could obtain recent blood-test values and a recent indication of hematological response to compression/decompression. The repetitive-dive trial was one standard dive per day on five consecutive days, beginning on day 8. Postdive changes were observed in four values: leucocytes, RBC, Hb, and HCT. Changes in leucocyte count occurred unpredictably and, therefore, we presumed they were not caused by compression/decompression-related stresses. Since HCT is calculated from RBC and MCV in the Coulter Counter, it is not explicitly discussed in this presentation. Changes in Hb paralleled changes in RBC in all instances; thus, display of changes in both variables is redundant. Accordingly, only changes in RBC are described.

RBC values before and after the daily exposures to compression/decompression and base-line RBC values from Table 1 for both divers are plotted in Fig. 1. The diving schedule is diagrammed in the lower part of the figure. We drew a broken line from the last postdive value for each day to the following day's predive value to indicate that the shape of the recovery curve is unknown. A second, nonstandard dive was made on the fifth day of the trial (as diagrammed in the figure), and a blood sample was obtained 75 min postdive. PTTs were measured before and after the first dive on the fifth day, and these results are shown in the bar graph in the figure. Recovery RBC values were obtained on the morning of day 15, 69 h after completion of the repetitive-diving trial. Control RBC values were obtained 3 wk after the trial (during January 1976).

Diver One responded to the isolated dives on days 1 and 8, and to the three succeeding repeated dives on days 9, 10, and 11 with a decrement in RBC. The maximum pre- to postdive change was − 8% on day 11. He did not evidence recovery on day 12 and showed further

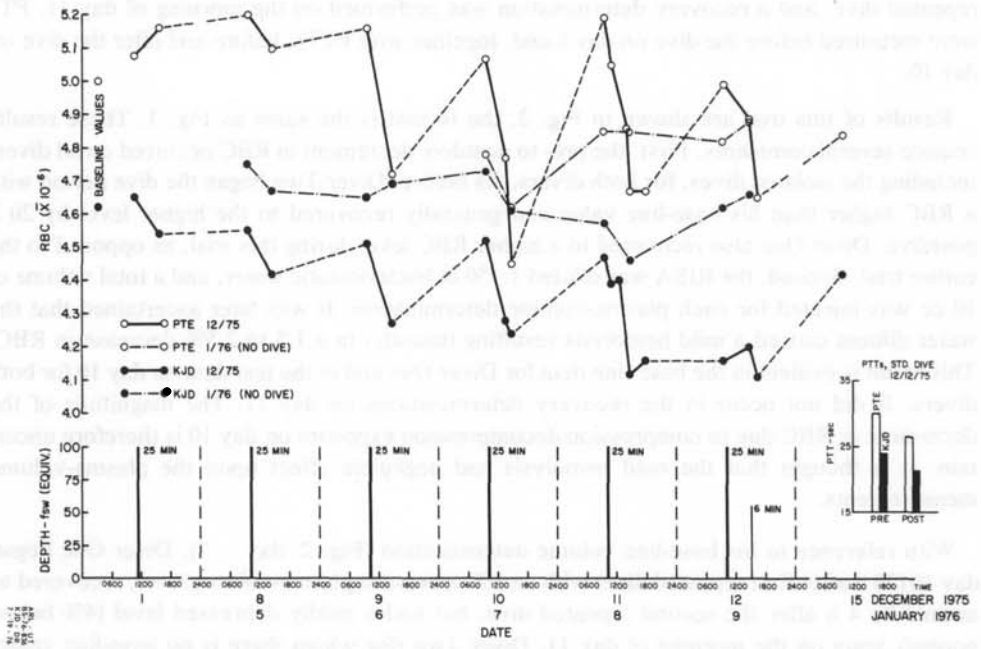

Fig. 1. Erythrocyte (RBC) decrements after repeated exposures of two divers to asymptomatic compression/decompression in open water during December 1975. Water temperature was 17.5°C. Control values were obtained during January 1976. Base-line values are from Table 1. Predive and postdive partial thromboplastin times (PTTs) for day 12 are shown in the bar graph.

decrement following the second dive on that day. Diver Two exhibited a slight increase in RBC following the isolated dive on day 1, a comparable decrease after the isolated dive on day 8, and then experienced significant decrements on all subsequent repetitive dives. The maximum pre- to postdive change was − 12% on day 10. Neither diver's RBC had recovered to its pretrial or base-line level by the morning of day 15. Diver Two began the trial period with a RBC higher than his base-line value and recovered daily (generally) to the higher level. His control values, however, lay below his base line.

These results fostered much speculation about the nature and causes of the observed decrements in RBC. Was the phenomenon truly a cellular loss? If so, what was becoming of the cells? (No abnormal fragmentation was observed in the samples.) Or, were the measured changes in RBC artifacts of intravasation of tissue fluids? If so, was the causative agent immersion and/or cold stress resulting in circulatory shifts and increased diuresis, or was it some unknown phenomenon of compression or of decompression, or perhaps a combination of some or all of these or some other agent, such as increased P_{O_2}.

In this study we could only test the hypothesis of fluid recruitment. A second repetitive-dive trial was carried out in March 1976 and changes in blood volume in addition to change in RBC after a repeated dive were measured. Water temperature during this trial was 12°C. A base-line blood-volume determination on Diver One was performed 14 days prior to the dive trial. As before, a standard dive was made 7 days preceding the trial to obtain recent blood-test values and a recent indication of hematological response to compression/decompression. Three consecutive standard dives, one each day, were made beginning on day 8; that is, the trial was continued only long enough to establish the previously observed decrement in RBC. Pre- and postdive plasma volumes of both divers were measured on day 10, the second repeated dive, and a recovery determination was performed on the morning of day 11. PTs were measured before the dive on day 8 and, together with PTTs, before and after the dive on day 10.

Results of this trial are shown in Fig. 2; the format is the same as Fig. 1. These results require several comments. First, the pre- to postdive decrement in RBC occurred on all dives, including the isolated dives, for both divers. As before, Diver Two began the dive period with a RBC higher than his base-line value and generally recovered to the higher level by 20 h postdive. Diver One also recovered to a higher RBC level during this trial, as opposed to the earlier trial. Second, the RISA was diluted 1:150 in bacteriostatic water, and a total volume of 10 cc was injected for each plasma-volume determination. It was later ascertained that the water diluent caused a mild hemolysis resulting (usually) in a 1.5 to 2.5% decrease in RBC. This result is evident in the base-line data for Diver One and in the test data on day 10 for both divers. It did not occur in the recovery determinations on day 11. The magnitude of the decrement in RBC due to compression/decompression exposure on day 10 is therefore uncertain. It is thought that the mild hemolysis had negligible effect upon the plasma-volume measurements.

With reference to his base-line volume determination (Fig. 2, day − 7), Diver One began day 10 (20 h after first repeated dive) with an 8% depressed plasma-volume level, recovered to normal by 4 h after the second repeated dive, but had a mildly depressed level (4% below normal) again on the morning of day 11. Diver Two (for whom there is no base-line value) showed similar results except for a severely depressed level (13%) on the morning of day 11.

Pre- and postdive PTs for both divers, measured during the March trial, were 10.7 sec, which indicated no measurable change due to compression/decompression. The PTT determinations were inconsistent, although both divers showed a reduced postdive PTT during the

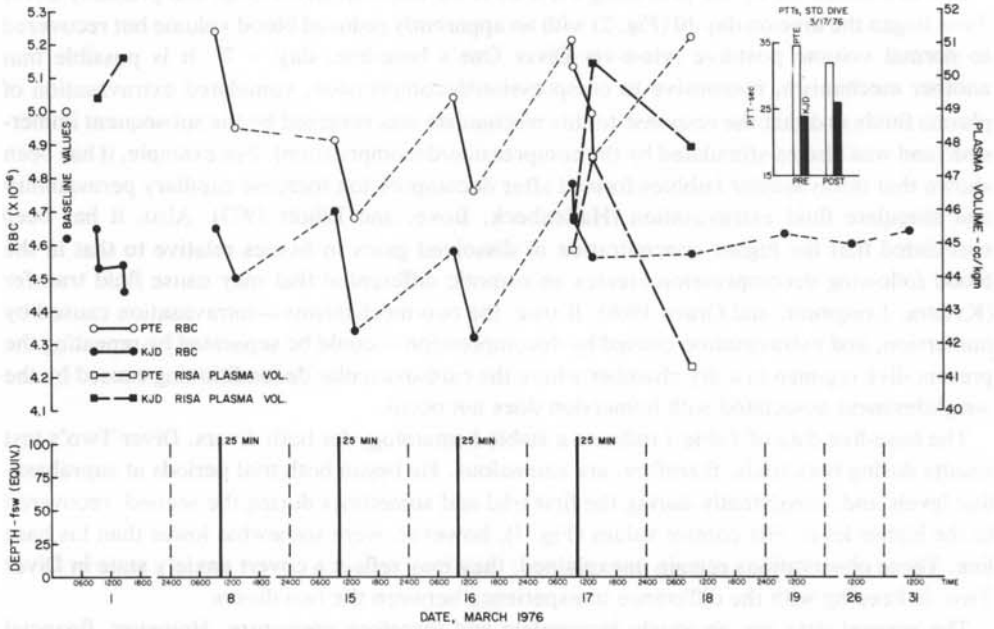

Fig. 2. Erythrocyte (RBC) decrements after repeated exposures of two divers to asymptomatic compression/decompression in open water during March 1976. Water temperature was 12°C. Normal plasma volume was determined using ^{129}I-tagged serum albumin (RISA) for Diver One on day − 7. Pre- to postdive changes in plasma volume were determined for both divers on days 10 and 11 (*see* text). Pre- and postdive partial thromboplastin times (PTTs) were measured on day 10 and are shown in the bar graph.

December trial, and Diver Two showed a similar reduction during the March trial. Diver One displayed an increase in postdive PTT in March.

DISCUSSION

It is reasonable to speculate that the observed postdive decrements in RBC were artifacts of fluid recruitment, a manifestation of a phenomenon akin to cardiovascular deconditioning caused by the loss of the hydrostatic blood column in the lower extremities during immersion (Pilmanis, Given, and Pilmanis 1972). The unit exposure time is exceptionally brief; however, there may be a cumulative effect. The latter would help to account for the observation that occasionally, no decrement occurred on isolated dives, e.g., for Diver Two on day 1 of the December trial (Fig. 1) and on other isolated dives performed by both divers which are not reported here.

The data (Fig. 1, day 11) indicate that recruitment continues for 4 to 6 h after exposure when the maximum decrement is observed, followed by a response reversal, i.e., extravasation of plasma fluids or increased diuresis (although the latter, if extant, was not exceptional). In perhaps the most significant single event of this study, Diver One did not evidence a reversal after the third repetitive dive of the December trial (Fig. 1, day 11). The import of this event for continued exposures is moot. It could indicate a developing severe hematological disruption or a lessening of predisposition to decompression injury, i.e., acclimatization. Recovery to normal RBC level (or normal blood volume) was evidenced by day 15, 69 h postdive, and may have occurred sooner.

Not accounted for by the preceding theories is the fact that Diver One, and probably Diver Two, began the dive on day 10 (Fig. 2) with an apparently reduced blood volume but recovered to normal volume postdive (vis-a-vis Diver One's base-line, day − 7). It is possible that another mechanism, responsive to compression/decompression, stimulated extravasation of plasma fluids and that the response to this mechanism was reversed by the subsequent immersion (and was also re-stimulated by the compression/decompression). For example, it has been shown that intravascular bubbles formed after decompression increase capillary permeability and stimulate fluid extravasation (Hallenbeck, Bove, and Elliott 1973). Also, it has been speculated that the higher concentration of dissolved gases in tissues relative to that in the blood following decompression creates an osmotic differential that may cause fluid transfer (Kylstra, Longmuir, and Grace 1968). If true, the two mechanisms—intravasation caused by immersion, and extravasation caused by decompression—could be separated by repeating the present dive regimen in a dry chamber where the cardiovascular deconditioning caused by the weightlessness associated with immersion does not occur.

The base-line data of Table 1 indicate a stable hematology for both divers. Diver Two's test results during both trials, therefore, are anomalous. He began both trial periods at suprabase-line levels and, consistently during the first trial and sometimes during the second, recovered to the higher level. His control values (Fig. 1), however, were somewhat lower than his base line. These observations remain unexplained; they may reflect a covert anxiety state in Diver Two, in keeping with the difference in experience between the two divers.

The present data are obviously incomplete and therefore premature. However, financial limitations precluded a more comprehensive investigation of the phenomena reported here.

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Diercks, K. J. and P. T. Eisman. 1977. Modifications hématologiques après de plongées quotidiennes asymptomatiques. Undersea Biomed. Res. 4(4): 325–331.—Des modifications des paramètres sanguins normaux après exposition quotidienne à la compression/décompression en l'eau sont décrites. (Plongée standard à 100 fsw pendant 25 minutes.) Le numéro érythocytaire tombe après la plongée pour atteindre un minimum apparent 4–6 heures après l'exposition; les valeurs normales sont rétablies ou même dépassées avant 20 h postplongée. Les mesures de volume plasmatique à la suite de plongées itératives mettent en évidence une augmentation significative 4 h après la plongée. Il se peut qu'un phenomène semblable au déconditionnement cardiovasculaire, dû à la perte du colonne sanguin hydrostatique des jambes pendant l'immersion, provoque un recrutement transitoire des fluides qui persisterait à la suite de plusieurs plongées quotidiennes. Nous n'avons pas pu trouver de correlation consistente entre les temps pré- et post-plongée de thromboplastine partielle après plusieurs séries compression-décompression.

immersion
numéro érythrocytaire
volume plasmatique
hémoglobine
recrutement des fluides

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