Effects of fluid loss on the physiology of closed-circuit rebreather divers after 100- and 45-metre dives

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Abstract
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Introduction: Diving induced immersion diuresis predisposes divers to dehydration. Dehydration is considered a risk factor for decompression sickness (DCS) but there is very little evidence to prove it. Dehydration also potentially modifies venous gas emboli (VGE) formation and impairs endothelial function. The purpose of this study was to report the effects of fluid loss during a dive on the diver's physiology.
Methods: Nine divers performed a 45 metre fresh water (mfw) and a 100 mfw dive with predetermined dive profiles. Body weight was measured before and after the dive. Post-dive detection of VGE was performed according to the extended Eftedal-Brubakk scale. We also measured haematocrit and flow mediated dilation before and after the 100 mfw dives.
Results: After a 68-minute dive to 45 mfw, median weight loss was -1.1 kg, (IQR -1.2, -1.0; range -2.0, -0.6), $P = 0.009$ and VGE were detected in all divers. After a 170-minute dive to 100 mfw, median weight loss was -1.5 kg (IQR -1.8, -1.1; range -2.2, -0.8), $P = 0.009$ and VGE were detected in seven divers. Weight loss after the dive was statistically significant and there was a negative correlation between weight loss and bubbling after the 45 mfw dives. None of the divers suffered any symptoms of DCS.
Conclusions: We found significant weight loss after both decompression dives but there were no clinical DCS symptoms in any of the divers. This study does not offer new evidence supporting the notion that dehydration increases decompression stress in divers.

Introduction

Scuba dives, and especially deeper and longer technical dives, pose stress on the diver's body. Due to increased ambient pressure underwater, inert gasses dissolve in tissues and during ascent, inert gases are eliminated from the tissues. During the off-gassing, venous gas bubbles tend to form and these bubbles initiate a complex cascade of events that can lead to decompression sickness (DCS).[1] The deeper the dive, the more inert gas dissolves and divers need to decelerate ascent speed by adding decompression stops to allow sufficient time for off-gassing. Hence, great depths result in long dive times and increased decompression stress and risk of DCS.

Increased hydrostatic pressure also causes redistribution of venous blood from the caudal portions of the body to the intrathoracic circulation. This is augmented by cold-induced vasoconstriction that reduces heat loss by constricting peripheral thermoregulatory shunts yet increasing centralisation of blood flow. As a result, central blood volume and cardiac preload increase which triggers humoral responses leading to immersion diuresis and

decreased plasma volume. The effect of immersion on haemodynamic and fluid rearrangements is well established in the literature.[2–4] In addition to immersion-induced diuresis, divers lose fluids via respiration and evaporation, although diving with closed-circuit rebreathers (CCRs) reduces fluid loss due to humid breathing gas.

Increasing diuresis predisposes divers to dehydration. Dehydration is considered to be a risk factor for DCS but there is very little evidence to prove it.[1] There are also conflicting results regarding the role of fluid balance in the DCS risk in animals.[5–7] It is speculated that the risk of DCS increases because of dehydration and subsequent haemoconcentration due to higher blood viscosity and reduced perfusion of peripheral tissue such as skin and skeletal muscle which would reduce inert gas washout during decompression.[6]

Dehydration might also impair endothelial function.[8] An increase in blood flow-associated shear stress in blood vessels induces the release of vasodilators e.g., nitric oxide (NO) from the endothelium. This phenomenon is called flow-mediated vasodilatation (FMD). Flow-mediated vasodilatation is an important response regulating homeostasis of the peripheral circulation.[9,10]

Previous studies suggest dehydration modifies venous gas emboli (VGE) production. Gempp et al. found pre-dive fluid intake reduces VGE formation in divers.[11] On the other hand, Skogland et al. did not find dehydration causing more VGE in rats.[6] Though VGE potentially contribute to development of DCS, VGE grades are considered an imperfect outcome measure in DCS studies. Yet, the number of bubbles does have a weak positive predictive value, in correlation to the symptoms of decompression sickness.[12]

Dehydration is only one of the factors considered to predispose divers to DCS. However, the knowledge regarding dehydration and the risk of DCS is scarce, there are no human studies, using DCS as an endpoint, addressing this topic. The purpose of this study was to report the physiological effects of fluid loss in a group of divers that performed two different decompression dives.

Methods

The study adhered to the Declaration of Helsinki. Ethical approval was granted by the Ethical Committee of Helsinki University Hospital (HUS/976/2019). Research permission was received from Helsinki University Hospital (HUS/151/2022 and 124/2023).

STUDY DESIGN

Nine experienced, healthy and non-smoking subjects, male ($n = 8$) and female ($n = 1$), took part in the tests. The subjects were recruited from the Finnish recreational technical diving community. Two of the divers had experienced DCS in the past. None of the divers had any other medical conditions. Each diver performed one dive to 45 metres of fresh water (mfw) and one dive to 100 mfw. Each subject filled out a health survey, and a physician performed a fit-to-dive examination on the morning of the dive.

PREPARATIONS AND DIVING PROTOCOL

No alcohol was allowed for 24 hours before the dive. During the 45 mfw diving day, subjects were instructed to hydrate according to their regular routines until two hours before the dive. Thereafter, only 5 dL of sports drinks (Gatorade, PepsiCo, Nordic Finland Ltd, Helsinki, Finland) were consumed. For the 100 mfw dive, the divers were allowed to hydrate as they normally do. Preparations for the dives were made in a room with constant air temperature (19°C).

The 45 mfw test dives were conducted at an old water-filled mine in Ojamo (Lohja, Finland) during winter in January. Diving conditions were normal for this time of year: the water was covered with a thin sheet of ice; water temperature was 0–2°C near the surface and 4°C at a depth of 45 mfw. The divers made an identical dive to the depth of 45 mfw by following a preset line. The divers were instructed to start the ascent at 30 min runtime resulting 15 min time at the bottom depth. During the ascent, divers followed an earlier defined decompression profile: Suunto Fused™ RGBM 2 (Suunto Ltd, Vantaa, Finland) with personal adjustment +2 (Suunto EON Core and Suunto D5 dive computers). The median total dive time was 68 min (interquartile range [IQR] 63–71 min).

The 100 mfw test dives were conducted at an old water-filled mine in Montola (Pieksämäki, Finland) during one weekend in October. The water temperature was 4°C at depths below 25 mfw and 8°C above 25 mfw. The divers also followed an identical route to the depth of 100 mfw and spent five min at the bottom depth before starting the ascent. Decompression was performed according to Shearwater computers (Shearwater Research Inc, Richmond, BC, Canada) using gradient factor (GF) 20/70. The median total dive time was 170 minutes (IQR 155–178 min).

Divers used their own diving equipment during the test dives. These included their usual undergarments, heating vest and dry suits. The divers were allowed to use pee-valves during the dive, the amount of urine output was not measured. All subjects used their own CCR unit (JJ-CCR [$n = 8$], rEVO [$n = 1$]). All CCR devices used standardised diluent; trimix 20/40 for the 45 mfw dive and 10/70 for the 100 mfw dive. The oxygen controllers maintained constant oxygen partial pressure in the breathing loop (PO_2 = 70 kPa at the beginning of the dive and PO_2 = 120 kPa after reaching 21 mfw throughout the bottom time and ascent).

MEASUREMENTS

Preparations and measurements were made in a room with constant air temperature (19°C). Subjects' weight was measured with an InBody 720 composition analyzer (Biospace Ltd, Seoul, South-Korea) approximately two hours before the dive. After the dive, subjects were instructed to empty the bladder and be weighed as soon as possible before any fluid intake. Weighing was done wearing only underwear (no diving undergarments were allowed) before and after the dive.

The presence of VGE in the cardiac chambers was determined with a 2D echocardiographic probe using an apical four-chamber view with a transthoracic approach (GE Vivid i, GEMS ultrasound, Tirat Carmel, Israel, transducer 2D 3S-RS). Subjects were placed in the supine left lateral decubitus position. Monitoring was performed at 30, 60, 90 and 120 minutes after surfacing, at rest and after performing Valsalva and leg and arm flexion. The observation was recorded and verified with at least one additional observer. Obtained images were graded from 0 to 5 according to the method described by Eftedal and Brubakk.[13]

The venous blood samples for measuring haematocrit (Hct) were taken from the antecubital vein 1–2 hours before and maximum one hour after the 100 mfw dives. Haematocrit was measured using the capillary (microhaematocrit) method. Blood samples were collected in heparinised microcapillary tubes, sealed at one end, and centrifuged at 10,000–12,000 rpm for five minutes to separate red cells, buffy coat, and plasma. The haematocrit value was then determined as the ratio of packed red cell column length to the total blood column length, expressed as a percentage.

Using a digital diagnostic ultrasound system (V-Scan Air, General Electric-Netherlands), FMD, an established measure of the endothelium-dependent vasodilation mediated by NO,[10] was used to assess the effect of diving on main conduit arteries after the 100 mfw dives. Brachial artery diameter was measured immediately before and one minute after a five-minute ischaemia induced by inflating a cuff placed on the forearm.[10] All ultrasound FMD assessments were obtained 60 min after surfacing while participants stayed at rest in the supine position for at least 15 min. During image analysis, the brachial artery boundaries were identified manually with an electronic caliper (provided by the ultrasonography software) in a threefold repetition pattern. The artery diameter was averaged over these three measurements. FMD was calculated as the percent increase in arterial diameter from the resting state to maximal dilation.

STATISTICS

We present the numerical data using medians, interquartile ranges (IQRs) and ranges, and the categorical data as counts and percentages. Comparisons were done using Mann-Whitney U tests and Spearman correlation (ρ) with 95% confidence intervals (CI). We visually assessed the normality of the variables and decided to use non-parametric tests. We considered *P*-values below 0.05 significant. All analyses were done using R software version 4.5.0 and the ggplot2 package was used for creating the figures.[14]

Taking the baseline measures as 100%, FMD changes were calculated for each diving protocol, allowing an appreciation of the magnitude of change rather than the absolute values.

Results

Median age of the divers was 45 years (IQR 42–50 years) and they had a long experience in diving, median experience was 16 years (IQR 14–19 years). All nine divers (one female, eight male) completed both of the decompression dives as planned. After diving and on the following day controlled, none of the divers presented any symptoms suggesting a diving incident. Subjects' demographics are presented in Table 1.

After a 68 min dive to 45 mfw, the median weight loss was -1.1 kg (IQR -1.2, -1.0; range -2.0, -0.6; $P = 0.009$) and after a 170 min dive to 100 mfw, the median weight loss was -1.5 kg (IQR -1.8, -1.1; range -2.2, -0.8; $P = 0.009$). Weight loss after the dives is presented in Figure 1. Weight loss relative to diver's body mass was 1.39% (IQR 1.04, 2.04; range 0.8, 3.1) after the 45 mfw dive and 2.05% (IQR 1.26, 2.4; range 0.9, 2.6) after the 100 mfw dive.

After the 45 mfw dives venous inert gas bubbles were detected in all nine divers during the 120-minute follow-up. After the 100 mfw dives, venous inert gas bubbles were detected in seven divers. Two of the divers did not produce any visible bubbles, not even when provoked with Valsalva manoeuvres and/or limb flexions. One diver with no history of DCS expressed a few occasional bubbles in the left heart also after the 45 mfw dive. There was a statistically significant negative correlation between weight loss and

Table 1
Subject demographics before the 100 mfw dive; data are median (interquartile range) unless otherwise indicated; BMI – body mass index; DCS – decompression sickness

Parameter	*n* = 9
Age (years)	45 (42–50)
Weight (kg)	82.9 (80.5–87.1)
BMI (kg·m^{-2})	26.5 (25.3–28.4)
Diving experience (years)	16 (14–19)
Diving experience (dives)	850 (700–1500)
Previous DCS (*n*)	2

Figure 1
Weight change (kg) after 45 mfw/68 min and 100 mfw/170 min dives; the median weight loss was -1.1 kg ($P = 0.009$) and -1.5 kg ($P = 0.009$) respectively

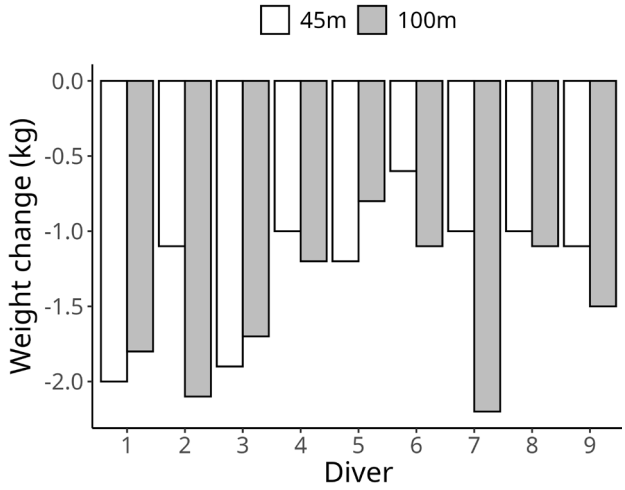
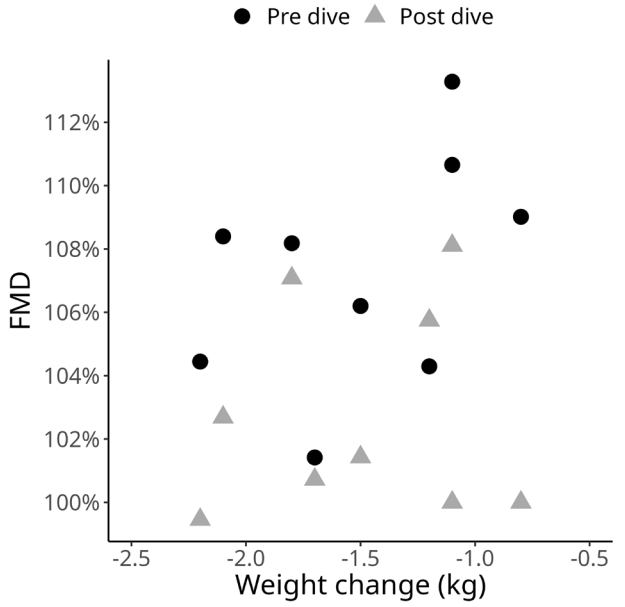


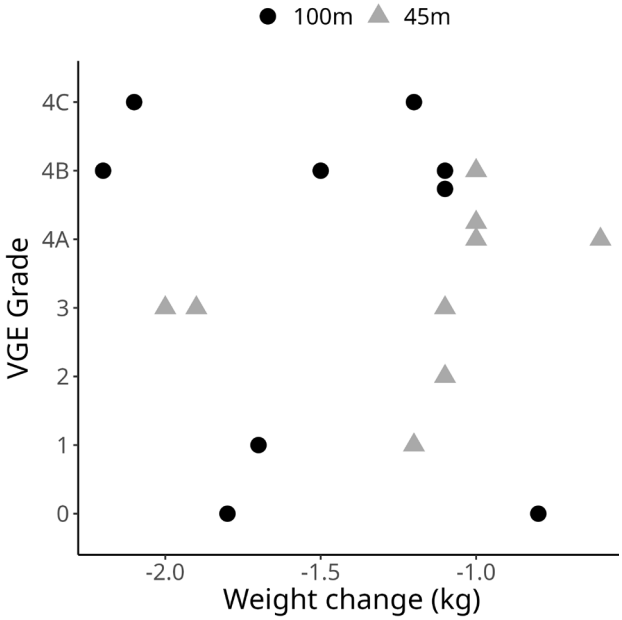
Figure 3
The flow-mediated dilation (FMD) measured before and after the 100 mfw dives and its relation to the divers' weight change. There was no correlation between FMD and weight change



bubbling after the 45 mfw dives (ρ -0.72; 95% CI -0.95, -0.01; $P = 0.028$) but no correlation after the 100 mfw dives (ρ 0.20; 95% CI -0.54, 0.77; $P = 0.601$). The relation between VGE and weight change is presented in Figure 2.

After the 100 mfw dives, the haematocrit decreased by 0.8% (IQR -2.8, 2.0; range -3.7,11.0; $P = 1.0$). FMD decreased by 5.0% (IQR -5.7, 1.1; range -10.7, 1.5; $P = 0.020$). There was no correlation between haematocrit and weight change (ρ 0.16; 95% CI -0.57, 0.75; $P = 0.30$) or FMD and weight

Figure 2
The relation between maximum venous gas embolism grade and weight change; there was a statistically significant negative correlation after the 45 mfw dives but no correlation after the 100 mfw dives



change (ρ -0.36; 95% CI -0.83, 0.42; $P = 0.34$). The relation between FMD measured before and after the 100 mfw dives and weight change is presented in Figure 3.

Discussion

In this study, we focused on the effects of fluid loss and decompression stress, and furthermore on DCS risk factors in a group of divers after two decompression dives to different depths. The same nine divers did a short 45 mfw dive and a longer and deeper 100 mfw dive in cold water. Though the 100 mfw/170 min dive in cold water poses great decompression stress on divers' bodies, we did not observe any symptoms of DCS. Weight loss was statistically significant after both diving depths.

The divers lost weight up to 2.2 kg, corresponding to a volume depletion of 2.2 liters. Overall, weight loss was greater after the 100 mfw dive, though three of the divers lost more weight during the 45 mfw dive. The difference in weight loss between the 45 mfw/68 min and 100 mfw/170 min dives was not clinically very significant, being 1.1 kg and 1.5 kg respectively. These results are in line with previous studies that report weight loss after diving indicating fluid loss due to immersion.[11,15,16] There are also data showing that during hyperbaric conditions, divers also become dehydrated without water immersion.[17]

We found a statistically significant, negative correlation between weight change and VGE after the 45 mfw dives.

This result suggests dehydration might decrease VGE formation. However, due to the small size of our study population, which may result in an imprecise correlation estimate, causal interpretations should be made with caution. This finding is in concordance with Skogland et al. study, that found no difference in VGE formation between dehydrated and normally hydrated rats.[6] Yet the only human study Gempp et al. found pre-dive fluid intake reducing VGE formation in eight divers.[11] Given the limited number of divers in both studies, the findings may be influenced by random variability. In our study, the divers were allowed eat and drink in the morning according to their usual routines (approximately 4–5 hours before the dive). The controlled fluid intake was only two hours before the dive and the amount was less than in the study by Gempp et al,[11] being 500 ml vs 1,300 ml.

Weight loss was significant after the 100 mfw dive, but we did not find a significant difference in haematocrit, showing that the compensatory mechanism shifting fluid from the extravascular compartment to the vascular one was not overwhelmed. Therefore, despite the fluid loss, divers seemed to retain intravascular volume and we found no correlation between weight change and VGE formation after the 100 mfw dives. For safety reasons, the fluid intake was not controlled before the 100 mfw dives, most probably resulting greater fluid intake than before the 45 mfw dives (observed at the dive site but not quantified). The deeper dives were also done in warmer decompression water temperature and with a different decompression algorithm that produces longer decompression stops at the shallow depth. Hence, inert gas washout should be better during 100 mfw dives. Overall, after the 100 mfw dives, the divers had more VGE as predicted after deeper dive and greater helium content in the breathing gas. Yet, there were two divers with no visible bubbles even after limb movement after the 100 mfw dives. The intra- and interpersonal variability in bubbling in our study is also is in line with previous studies.[12,18]

The divers in this study experienced fluid deficit (percentage reduction in body mass due to fluid loss) up to 3.1% and 2.6% after the 45 mfw and the 100 mfw dives respectively. The state, when fluid deficit exceeds 2% of body mass, is called hypohydration. Hypohydration results in water redistribution largely from the intra- and extracellular fluid spaces of muscle, gut and skin.[19] Divers dehydrate via immersion induced diuresis, respiration and evaporation. In order to preserve homoeostasis, the body immediately counterbalances by shifting extravascular water into the intravascular space. Fluid shift effects on blood volume and hemoconcentration. For example, haematocrit level depends on the difference between intracellular and extracellular dehydration, and if the loss of extracellular water is lower than the loss of intracellular water, we observed a decrease in the haematocrit level and vice versa. This might explain why we did not see a statistically significant change in haematocrit level in our study, though divers lost significant amount of weight indicating fluid loss.

Dehydration of up to 2% should not pose a threat to health or even be noticeable in normal life.[20] But hypohydration has been shown to possibly affect cognition; the loss of body mass of greater than 2% can lead to reductions in the subjective perception of alertness and ability to concentrate.[21,22] It has been suggested that for every 1% of total water loss, the body's physical capacity decreases by approximately 10%.[17] Hypohydration also leads to vasoconstriction and therefore causes a reduction in blood flow to the skin.[23] Blood flow also declines in the exercising muscles due to a lowering in perfusion pressure and systemic blood flow,[24] thus potentially compromising inert gas washout from tissues when diving.[1] Hence, for safety reasons, divers were not instructed to hydrate according to a certain protocol before the 100 mfw dive: The divers were allowed to consume drinks according to their usual procedures. All of the divers started focusing on hydration at least the day before the dive and all of them drank more than 500 ml in the morning of the dive.

Hydrating well with water or isotonic drinks before the dive is common among divers, especially technical divers. Increasing total body water above the normal, referred to as pre-exercise hyperhydration, provides a strategy to delay or reduce the adverse effects of hypohydration caused by exercise. But hydrating with large amounts of fluid alone is not reasonable as it inhibits the release of anti-diuretic hormone (ADH), leading to increased diuresis.[25] Also, hyperhydration may increase the risk of immersion pulmonary oedema among other risk factors, however the research to support this risk in scuba divers is limited.[26] Recent review of nutritional recommendations for scuba divers suggests all divers should take special care to hydrate themselves with an absolute minimum of 500 ml of fluid per hour when diving for more than 3 hours.[27]

FMD decrease reached 5% compared to the pre-dive value; this statistically significant change is very common in diving and does not seem to change in relation to the depth; it seems to be more related to the increase in inspire PO_2.[28,29] Previous studies suggest even mild levels of hypohydration impair endothelial function as assessed by FMD,[8] but we did not find any correlation between weight loss and FMD; thus, our results show a rather 'constant' acute vascular function reduction after diving.

LIMITATIONS

Our study was performed in a real diving environment with challenging conditions. The number of participants was limited by the depth of the dives and type of study, and therefore the results comparisons between the groups should be interpreted with caution. With this limited number of

divers in our study, the observed results may fluctuate due to underlying stochastic variability. A larger study group could have given a more precise understanding, and the correlations in various parameter could have been stronger.

For safety reasons, the fluid intake before the 100 mfw dive was not controlled as well as the decompression model used was not the same making comparison between the dive depths difficult. Another limitation of the study was the unavailability of FMD and Hct measurements in the 45 mfw dive. This did not allow comparison of these parameters in two different dive profiles and environmental conditions.

Conclusions

In conclusion, we found significant weight loss after both decompression dives and there was a negative correlation between weight loss and VGE after the 45 mfw dives. Because our study population is relatively small, leading to less precise correlation estimates, caution is needed when interpreting causal relationships. There were no clinical DCS symptoms in any of the divers. There was also no correlation between weight loss and haematocrit or FMD after the 100 mfw dives. This study does not offer new evidence supporting the notion that dehydration increases decompression stress in divers. Further studies with a greater number of participants are needed to potentially support the widely accepted concept that dehydration predisposes divers to DCS.

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