Preventive effect of pre-dive hydration on bubble formation in divers

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ABSTRACT

Objective: To investigate whether prehydration 90 min before a dive could decrease bubble formation, and to evaluate the consequent adjustments in plasma volume (PV), water balance and plasma surface tension (ST).

Methods: Eight military divers participated in a crossover trial of pre-dive hydration using saline–glucose beverage (protocol 1) and a control dive with no prehydration (protocol 2). Drink volume was 1300 ml (osmolality 324 mOsm/l) and drinking time was 50–60 min. The diving protocol consisted of an open sea field air dive at 30 msw depth for 30 min followed by a 9 min stop at 3 msw. Haemodynamic parameters, body weight measurements, urine volume and blood samples were taken before/after fluid intake and after the dive. Decompression bubbles were examined by a precordial pulsed Doppler.

Results: Bubble activity was significantly lower for protocol 1 than for protocol 2. PV increased after fluid ingestion by 3.5% and returned toward baseline after diving for protocol 1, whereas it decreased by 2.2% after diving for protocol 2. Differences in post-dive PV between the two conditions were highly significant. Body weight loss before/after diving and post-dive urine volume after diving were significant in both protocols, but the relative decline in weight remained lower for protocol 1 than for protocol 2, with reduction of negative water balance due to higher fluid retention. There were no differences in ST after fluid intake and after diving for the two protocols.

Conclusion: Pre-dive oral hydration decreases circulatory bubbles, thus offering a relatively easy means of reducing decompression sickness risk. The prehydration condition allowed attenuation of dehydration and prevention of hypovolaemia induced by the diving session. Hydration and diving did not change plasma surface tension in this study.

Divers are at risk of decompression sickness (DCS) caused by bubbles of inert gas that may evolve in the tissues or blood due to supersaturation during decompression. It is generally accepted that: (1) gas bubbles grow from pre-existing nuclei attached to the vessel walls or by hydrodynamic cavitation resulting mainly from musculoskeletal activity[1]; and (2) the incidence of DCS is low when few or no bubbles are present in the circulation.[2] Accordingly, circulating bubble detection with Doppler systems can be considered as a valuable indicator of decompression stress and used as a tool for validation of the safety of decompression procedures.[2]

Preventive measures to reduce the risk of DCS could involve several procedures reported in some experimental studies such as oxygen breathing,[3] exercise before diving,[4 5] addition of deep stops,[6] slower ascent rates[7] or intake of exogenous nitric oxide (NO).[8] The possibility that fluid balance significantly affects DCS risk is not established in humans, and animal data are limited and conflicting.[9–12] Anecdotal reports speculated that pre-dive dehydration and haemoconcentration could be predisposing factors for DCS by increasing blood viscosity and by altering microcirculatory perfusion when bubble formation occurs.[13 14] Experimental studies suggest that during immersion, divers experience central fluid shifts that lead to a pronounced diuresis, with dehydration and reduction of plasma volume on surfacing.[15 16] Moreover, it is suggested that a low plasma surface tension favours bubble formation[17] whereas the ingestion of normal saline solution before hypobaric exposure might afford protection against altitude DCS by raising temporarily surface tension.[18]

To date, no human clinical data are available on the influence of pre-dive hydration on venous gas emboli (VGE) formation after decompression. The purpose of this study was: to investigate whether prehydration with a saline–glucose beverage before a dive could decrease post-dive VGE; and to evaluate the consequent modifications in plasma volume, water balance and plasma surface tension.

METHODS

Study population

Eight healthy military divers, mean (SD) age 36 (6) years, gave their written consent to participate. All the subjects were trained divers and none of them had experienced DCS in the past. Their body mass index varied between 23.2–26.1 kg/m^2. All experimental procedures were conducted in accordance with the declaration of Helsinki.

Experimental design

Each subject participated at weekly intervals in a crossover trial of pre-dive hydration using a saline–glucose formulation started 90 min before a dive (protocol 1) and a control dive with no prehydration (protocol 2). They were instructed to avoid physical exertion and diving during the 2 days that preceded each trial, and none of the divers were informed about which experimental conditions they were to dive under that day. The dive protocol consisted of an open sea field dive to 30 msw (400 kPa) breathing air for 30 min (sea temperature 14°C) with a decompression rate of 15 msw/min and a 9 min stop at 3 msw (French Navy MN90 procedure). For protocol 1, drink volume and composition were 1300 ml of water that contained 157 meq/l Na^+ and 23 g/l carbohydrate (osmolality = 324 mOsm/l) while drinking time was 50–60 min. This concentration was

chosen because it has been demonstrated that water alone was relatively ineffective for controlling and maintaining plasma volume.[19] None of the divers were allowed to drink liberally after the dive session.

Haematological variables

Blood samples were collected by venepuncture for haematocrit (Hct), haemoglobin (Hb) and plasma surface tension (ST) after resting in a supine position for 20 min; measurements were determined at baseline, immediately after fluid ingestion, and at 60 min after the dive completion. Percentage changes in plasma volume (PV) were calculated using the Hb-Hct transformation equation[20] while ST was analysed after blood centrifugation with a bubble pressure method by using a tensiometer (Sitamesstechnik GmbH, Dresden, Germany). Each ST sample was measured three parallel times, and the value was given as mean (SD). All measurements were done between 25–27°C (room temperature).

Physiological measurements

Body weight (using model I5S, OHAUS Corp, USA) was recorded at the beginning of the experimental study, just before diving, and at the end of each protocol while urine volume was collected in graduated cylinders during the entire trial session with two separate periods: before and after the dive. Body weight loss after each protocol was expressed as percentage change from baseline while water balance (WB) was calculated from fluid intake (FI) – total urinary loss.

Heart rate (HR) and blood pressure (BP) were also obtained immediately before each venepuncture (before/after fluid ingestion and after diving) by using a portable monitoring system (Propaq 104 EL, Protocol Systems, Inc, Beaverton, Oregon, USA). Pulse pressure was defined as PP = SBP − DBP, where SBP and DBP were the systolic and diastolic arterial blood pressure, respectively. Mean arterial BP (MBP) was calculated as MBP = DBP + 1/3 (SBP − DBP).

Bubble analysis

Decompression bubbles were examined by a pulsed Doppler device equipped with a 2 MHz probe on the precordial area (EZ-Dop, Compumedics Germany GmbH, Singen, Germany). Monitoring was performed by the same blind operator every 30 min for 90 min after surfacing in supine position for 3 min at rest. The signal of bubbles was graded according to the Spencer scale[21] before being converted into Kissman Integrated Severity Score (KISS). This score takes into account the kinetics of the bubbles at the different recording times and was assumed to be a meaningful linearised measure of post-decompression intravascular bubble activity status that may be treated statistically.[2]

Statistical analysis

All data are presented as mean (SD). For statistical processing, we used the Sigmastat 3.0 software program (SPSS, Chicago, Illinois, USA). Data were analysed using non-parametric statistics because of the small sample size. Wilcoxon signed rank test was used for paired data, whereas comparisons in different times for difference in surface tension were evaluated by Friedman test (repeated measures analysis of variance (ANOVA) on ranks). The level of significance was set at $p<0.05$ and p values of $0.05<p<0.1$ were considered a trend.

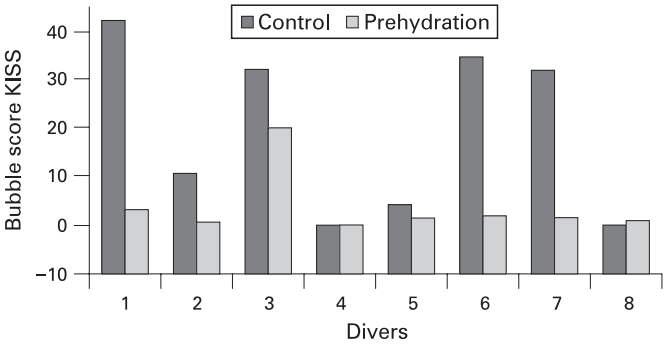

Figure 1 Individual bubble score after diving. Grey bars represent protocol 1 (prehydration) and black bars represent protocol 2 (control dive). KISS, Kissmen Integrated Severity Score.

RESULTS

Bubble production

Figure 1 shows the individual bubble score after the experimental dive with or without hydration before exposure. VGE activity was significantly lower for protocol 1 than for protocol 2 (mean KISS 3.5 *vs* 19.4, p = 0.031). One diver (number 8) showed a slight increase in venous bubble grade after drinking. None of the divers had any clinical symptoms in relation to the dives.

Haematological variables

Figure 2 presents an overview on the PV adjustments for both protocols. PV increased significantly after fluid ingestion by 3.5% (mean p = 0.016) and returned towards baseline after diving for protocol 1 (0.9%, p = 0.024), whereas it decreased by 2.2% after diving for protocol 2 (p = 0.014). Differences in post-dive PV between the two conditions were highly significant (p = 0.007).

There were no significant differences in ST before and after fluid intake and after the dive for protocol 1 (71.3 (0.9), 70.4 (0.7) and 70.5 (0.8) dyn/cm, respectively). Comparison of baseline ST between the two conditions showed that ST values for protocol 1 were lower than for protocol 2 (71.3 (0.9) dyn/cm *vs* 73.5 (1.2) dyn/cm, p = 0.008) (fig 3).

Physiological measurements

Body weight loss and urinary loss before and after diving were significant in protocol 1 (−1020 (315) g, p = 0.008 and −810 (225) ml, p = 0.032, respectively) and in protocol 2 (−610 (350) g, p = 0.008 and −459 (170) ml, p = 0.019, respectively), but the relative decline in weight after diving from baseline remained significantly lower for protocol 1 than for protocol 2 (mean −0.5% *vs* −2.4%, respectively, p = 0.016). Negative water balance was significantly larger for protocol 2 than protocol 1 (−859 (375) g *vs* −140 (210) g, respectively) (fig 4).

HR and PP increased significantly after hydration (57 (5) beats/min *vs* 63 (6) beats/min, p = 0.016 and 52 (5) mm Hg *vs* 58 (6) mm Hg, p = 0.008, respectively) but returned to the baseline values after the dive (59 (10) beats/min and 53 (5) mm Hg) for protocol 1, whereas haemodynamic variables remained unchanged before and after diving for protocol 2 (61 (5) beats/min *vs* 58 (10) beats/min and 53 (6) mm Hg *vs* 55 (5) mm Hg, respectively). There was also a trend toward an increase in MBP after fluid intake (88 (6) mm Hg *vs* 92 (8) mm Hg, p = 0.07).

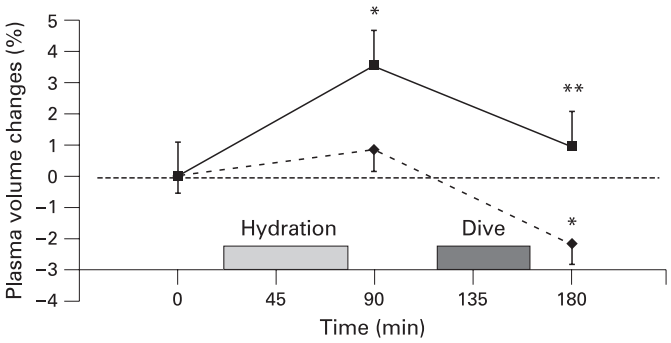

Figure 2 Experimental procedure with percentage changes in plasma volume from baseline values (time zero) between the protocol 1 (solid line) and the protocol 2 (dash line). *p<0.05 from baseline; **p<0.05 from the corresponding value in protocol 2. Values are mean (SD).

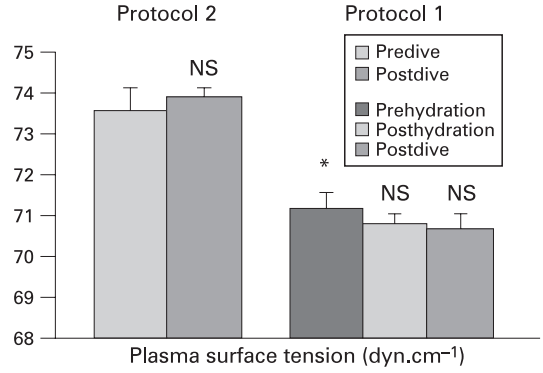

Figure 3 Measurements of plasma surface tension between the protocol 1 (prehydration) and the protocol 2 (control dive). NS, not significant compared with baseline values. *p<0.05 from the corresponding value in protocol 2. Values are mean (SD).

DISCUSSION

The main finding of this study is that oral pre-dive hydration with a hyperosmotic beverage is accompanied with a reduction of vascular bubble produced by decompression. To our knowledge, this study is the first to report this result in field conditions.

Although dehydration is commonly proposed as a risk factor for DCS in divers, there are no data that support this assertion in humans. Conversely, we have recently shown that moderate dehydration induced by a pre-dive exercise and declining stroke volume related to post-exercise hypovolaemia might influence inert gas load, and consequently decrease circulating bubbles.[22] Nevertheless, previous experiments in the past with aviators have demonstrated that there was some connection between water metabolism and susceptibility to altitude DCS: a high rate of water turnover estimated by measurement of fluid imbibed and urine excreted seems to be protective against DCS.[23] Animal studies are few and give contradictory results. Indeed, pre-dive intravenous infusion of crystalloid does not appear to reduce the risk of neurological decompression illness in pigs. Conversely, dehydration is not associated with an increased risk of DCS.[10] In water deprived rats, a trend has also been observed towards fewer venous circulating bubble production with a Doppler technique than in the control animals.[9] On the other hand, one experiment in a rabbit model with analysis of electrophysiological parameters showed that pre-existing extracellular dehydration appeared to be a major factor of spinal cord injury during severe decompression.[12] Recently, another trial supports the idea that normally hydrated pigs have a lower risk of severe DCS and death than those subjected to fluid intake restriction and diuretic administration.[11]

In our study, several hypotheses can be considered to explain that bubble reduction is a consequence of prehydration.

Prevention of diving induced hypovolaemia

It is generally accepted that haemoconcentration observed with DCS in both animals[24 25] and man[26] is associated with capillary leakiness and microcirculatory alterations due to interaction between intravascular bubbles and blood vessels. Moreover, some authors have argued that haemoconcentration resulting from post-dive dehydration could also precede and promote the development of DCS by reducing tissue blood perfusion rate, and finally inert gas elimination.[15] This assumption puts forward the idea that oral hydration should be particularly important after surfacing, notably in the case of repeated dives.[16] Recently, a significant decrease in cardiac preload 1 h after an open sea scuba dive, using Doppler echocardiography, has been observed, and this change could be attributed to a reduction in plasma volume secondary to immersion.[27]

In our study, the prehydration status before diving allowed us to prevent post-dive hypovolaemia while reducing the negative water balance. Thus, it is conceivable that fluid ingestion and resulting hypervolaemia in protocol 1 may impede the lowering of cardiac preload induced by the diving session. These findings are in agreement with recent work reporting that the consumption of 400 ml of an isotonic beverage could prevent dehydration and an increase in blood viscosity during prolonged sitting in a dry environment by attenuating negative water balance and hypovolaemia.[28] Previous observations have also shown that supine body position and other such interventions that increase central blood volume and cardiac preload significantly increased the rate of inert gas washout.[29 30] Consequently, we can speculate that large volume oral hydration might result in more rapid elimination of excess inert gas dissolved in body tissues during decompression, thus reducing circulating bubbles.

Peripheral vasoconstriction

It has been reported that water ingestion would enhance orthostatic tolerance in normal healthy adults by causing an increase in peripheral vascular resistance.[31] Moreover, some

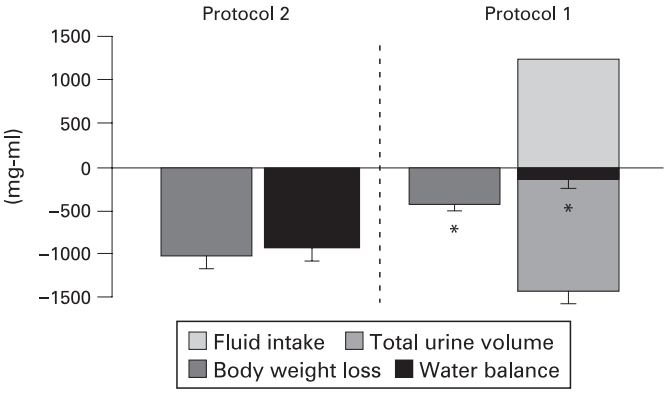

Figure 4 Fluid intake, water balance, total urinary volume and body weight loss recorded after the protocol from baseline between the two conditions (for protocol 2, water balance corresponds to total urinary volume). *p<0.05 from protocol 2 (control dive). Values are mean (SD).

studies have shown evidence for increased muscle sympathetic activity after drinking a volume of water of 480 ml, leading to peripheral vasoconstriction.[32] In elderly subjects or in patients with autonomic failure, drinking provokes a pronounced pressor response, whereas in young healthy persons it is generally accepted that water ingestion has no effect on BP and notably no change, or even a slight reduction, in HR.[32 33] It has been suggested that this paradoxical mechanism may be due to a concomitant increase in cardiac vagal activation, which prevents a rise in arterial pressure by reducing cardiac output.[32]

Based on physiological responses in animal and human experiments, the underlying mechanism of autonomic responses to water ingestion has been argued to involve gastrointestinal distension[34] and water hypo-osmotic properties.[33] However, in contrast to water drinking, stomach distension also increases HR and BP proportional to intragastric pressure level,[34] while saline drink has no effect on cardiovascular autonomic regulation.[33]

In the present study, ingestion of saline–glucose fluid over a period of 1 h induced a significant increase in HR and PP with a trend toward higher MBP. These findings are consistent with the involvement of a gastrovascular reflex. Indeed, it is possible that the large volume of fluid intake contributed to provoking a gradual increase in gastric distension, and consequently an increase in HR and BP parameters with concomitant activation of sympathetic vasomotor discharge to skeletal muscles, as described above. It is admitted that if the rate of blood flow is lower during the dive, inert gas load in body tissues would be less and consequently bubble formation would be reduced. This raises the possibility that peripheral vasoconstriction induced by the drink ingestion before the dive may also have implications for the reduction of generated bubbles during decompression.

Plasma surface tension

Based on theoretical considerations, the growth and stability of a gas bubble is affected by the surface tension of the fluid; a low surface tension favours all aspects of bubble development.[18] It has been demonstrated experimentally that bubble formation after decompression in pigs[17] was inversely proportional to serum surface tension, and it seems plausible that plasma surface tension might be elevated in well hydrated divers.[18] Moreover, there is evidence that serum surface tension among healthy individuals varies over time, suggesting possible intra-individual differences in vascular bubble formation after decompression.[35]

In this study, no changes were observed in ST after drinking or diving, indicating a lesser influence of fluid ingestion on plasma surface tension than was previously thought. Concerning the significant differences of ST at the first measurement between the two trials, we are in accordance with previous data.[35] This finding could reflect the difference in the amount and kind of food they had eaten before the blood was collected, especially fat content.[35] However, it was surprising to note that the variations in ST within each individual over a period of 1 week were not inversely correlated with the bubble grades observed after the two dives, as opposed to the findings of Hjelde *et al*.[17] These results highlight the lack of human data concerning the impact of ST on the course of bubble formation, and it remains to be established whether this parameter can be a decisive factor in DCS development. Possible ways of influencing changes in ST are numerous and not yet fully understood.

What is already known on this topic

Dehydration is generally considered as a risk factor for decompression sickness but human data are anecdotal and animal studies are conflicting. The suggested mechanisms to support this assumption include tissulary inert gas removal alterations induced by haemoconcentration and decrease in blood surface tension facilitating bubble formation.

To date, the influence of pre-dive hydration on venous gas emboli formation in divers is not established and plasma surface tension in pigs might be inversely correlated to bubble formation after decompression.

What this study adds

- Pre-dive oral hydration with a hyperosmotic fluid reduced circulating bubble formation.
- The prehydration condition allowed attenuation of negative water balance and hypovolaemia induced by the diving session.
- Fluid intake and diving did not change plasma surface tension in this study.

Conclusion

Our study supports the idea that pre-dive oral hydration of 1300 ml of a hyperosmotic fluid decreases circulatory VGE, thus offering a relatively easy means of reducing DCS risk. The prehydration condition allowed attenuation of the dehydration resulting from higher fluid retention and prevention of hypovolaemia induced by the diving session. Conversely, hydration did not appear to increase plasma surface tension in this study, and consequently may have less influence on the risk of DCS than was previously thought. Further investigations are required to elucidate the mechanisms underlying this prehydration induced reduction in bubble formation.

Competing interests: None.

Patient consent: Obtained.

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