Dehydration Effects on the Risk of Severe Decompression Sickness in a Swine Model

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Background: Several physiological factors have been suspected of affecting the risk of decompression sickness (DCS), but few have been thoroughly studied during controlled conditions. Dehydration is a potential factor that could increase the risk of DCS. It has been suggested that hydration may enhance inert gas removal or increase surface tension of the blood. ***Hypothesis:*** Dehydration increases DCS risk. ***Methods:*** Littermate pairs of male Yorkshire swine (n = 57, mean ± 1 SD 20.6 ± 1.7 kg) were randomized into two groups. The hydrated group received no medication and was allowed ad lib access to water during a simulated saturation dive. The dehydrated group received intravenous 2 mg · kg^{-1} Lasix (a diuretic medication) without access to water throughout the dive. Animals were then compressed on air to 110 ft of seawater (fsw, 4.33 ATA) for 22 h and brought directly to the surface at a rate of 30 fsw · min^{-1} (0.91 ATA · min^{-1}). Outcomes of death and non-fatal central nervous system (CNS) or cardiopulmonary DCS were recorded. ***Results:*** In the hydrated group (n = 31): DCS = 10, cardiopulmonary DCS = 9, CNS DCS = 2, Death = 4. In the dehydrated group (n = 26): DCS = 19, cardiopulmonary DCS = 19, CNS DCS = 6, Death = 9. Dehydration significantly increased the overall risk of severe DCS and death. Specifically, it increased the risk of cardiopulmonary DCS, and showed a trend toward increased CNS DCS. In addition, dehydrated subjects manifested cardiopulmonary DCS sooner and showed a trend toward more rapid death ($p < 0.1$). ***Conclusion:*** Hydration status at the time of decompression significantly influences the incidence and time to onset of DCS in this model.

Keywords: diving, swine, saturation diving, diuretics, hydration.

IT HAS BEEN KNOWN for more than a century that the risk of decompression sickness (DCS) is a function of the volumes of gases dissolved in tissues and the rate and magnitude of decompression from a higher pressure (1). For the diver, gas supersaturation occurs while breathing inert gas in hyperbaria and then reducing the pressure. The same phenomenon has also been reported in people rapidly exposed to hypobaria, as in high altitude flight or extravehicular activities in space (6). Safety has been significantly improved by empirically tested tables that limit the pressure exposure and decompression rate. However, some decompression events are associated with the development of DCS symptoms, even though the tables have been followed well within the limits (12).

It has been generally accepted that susceptibility to DCS is caused by both physiological and psychological factors. Despite this, it appears that a component of DCS risk remains approachable only by probabilistic mathematical treatment as a random event (23). However, physiological phenomena that appear to occur randomly may well contain physically or biologically definable variables that have not yet been identified as important factors, and, therefore, have not been experimentally controlled. Diving textbooks are filled with potential factors that may alter DCS risk, but many of these have not been tested under controlled conditions. Consequently, the challenge is to identify those that significantly alter DCS risk under a controlled experimental setting. Clearly defining risk factors, their effect(s), and magnitude would be of great benefit for a wide variety of people such as aviators, astronauts, commercial and military divers, caisson workers, sport divers, and hyperbaric chamber personnel.

Fluid balance is potentially one of these risk factors for DCS that has received little consideration. Many diving textbooks report the perception that DCS risk depends on hydration state. However, on closer scrutiny, the evidence to support these notions is often anecdotal, contradictory, or at best suggestive that more investigation is in order (5). One animal study in rats showed that the DCS incidence in dehydrated animals was 71% while in hydrated rats it was 55% (19). These differences were not statistically significant, but the author concluded that a trend was apparent, suggesting increased susceptibility in dehydrated animals. It is physiologically plausible that dehydration could alter inert gas removal by reducing blood flow to poorly perfused tissues, or that it may decrease surface tension and thereby facilitate bubble formation (5). However, hydration in humans did not lead to improved inert gas removal (7).

If it can be demonstrated that fluid balance significantly affects DCS risk, this may offer a relatively easy means of reducing susceptibility without resorting to prolonged decompression or subsequent recompression therapy. Therefore, in this trial, furosemide (Lasix; Aventis, Bridgewater, NJ) was used to induce experimental dehydration and assess its effect on DCS manifestations after direct ascent from saturation conditions.

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METHODS

The experiments reported herein were conducted according to the guidelines on laboratory animal use (18). Before commencing, the Institutional Animal Care and Use Committee reviewed and approved all aspects of this protocol. The institutional animal care facility is fully AAALAC certified.

Subjects: Neutered male Yorkshire swine littermates from a closed breeding colony (n = 57, mean ± 1 SD 20.6 ± 1.7 kg) were examined by a veterinarian on arrival and housed in individual runs where water was freely available. Their daily feedings consisted of 2% by bodyweight of laboratory animal feed (Harlan Teklad, Madison, WI). Animals remained in the care facility for a minimum of 72 h before experiments.

Predive preparation: Animals were transported to the laboratory in plastic transport kennels (22″ × 32″ × 22″; Vari-Kennel, R.C. Steele, Brockport, NY), placed in a Panepinto sling (Charles River, Wilmington, MA) and anesthetized by intramuscular injection of ketamine (20 $mg \cdot kg^{-1}$) and xylazine (1 $mg \cdot kg^{-1}$). Using sterile technique, 5 cm of a customized Tygon catheter (model #RPC-040, Braintree Scientific, Braintree, MA) was inserted into the left external jugular vein and externalized in the posterior midline at the T1 level. After closing the incision, the catheter was connected to an injection port. Animals then received 500 mg chloramphenicol to reduce infection risk and 2 ml heparinized saline (2 $U \cdot ml^{-1}$) to maintain catheter patency overnight. The catheter was sutured to the skin and taped down using waterproof surgical tape. After a complete recovery from anesthesia, animals were returned to the care facility.

Procedure: The morning after catheterization, the animals were brought to the laboratory, weighed, and placed in a Panepinto sling. The animals were then randomized to either ad lib access to water during the hyperbaric exposure (hydrated group), or i.v. infusion of Lasix (2 $mg \cdot kg^{-1}$) delivered over 10 min follow by no access to water during the hyperbaric exposure (dehydrated group). All animals were placed in a modified transport kennel that allowed direct visualization throughout the dive.

Subject pairs were placed in a manually controlled hyperbaric chamber (656 ft^3 internal volume, WSF Industries, Buffalo, NY) and compressed to a 110 fsw (4.33 ATA) using air. Compression progressed in phases, beginning with 5 $fsw \cdot min^{-1}$ (0.15 $ATA \cdot min^{-1}$) to a depth of 33 fsw (2 ATA). If the animal showed no distress or other evidence of middle ear barotraumas, the compression rate was increased to 10 $fsw \cdot min^{-1}$ (0.30 $ATA \cdot min^{-1}$). The compression rate was further increased to 20 $fsw \cdot min^{-1}$ (0.45 $ATA \cdot min^{-1}$) beyond 99 fsw (4 ATA) if the animals tolerated the descent well. Animal comfort was the limiting factor in all descent rates. Temperature was maintained between 26.7–29.4°C, humidity between 50–75%, and CO_2 concentration < 0.3%. The animals were constantly monitored via closed-circuit television cameras through observation ports. After 22 h, the animals were returned to surface (1 ATA) at a nominal rate of 30 $fsw \cdot min^{-1}$ (0.91 ATA $\cdot min^{-1}$) with no decompression stops. In practice that rate was closely followed until a depth of about 33 fsw (2 ATA). Due to piping restrictions, the remainder of the decompression required 1.5–2 min.

On reaching surface the animals were fitted with individual monitors that measured heart rate and hemoglobin saturation (VetOx 4404, Heska, Ft. Collins, CO) and then transferred to individual 36″ × 23″ × 22″ clear Plexiglas observation pens without access to food or water. Onset of severe DCS (neurological or cardiopulmonary dysfunction) was recorded to the nearest minute by dedicated observers. Disease and symptom onset times are referenced to the time the animals reached surface. Neurological/central nervous system (CNS) DCS was defined as motor weakness (diminished limb strength, repeated motor incoordination, or inability to stand after being righted by the investigator), paralysis (complete limb dysfunction, areflexia, hypotonia), or cranial nerve dysfunction (10). An animal was diagnosed with cardiopulmonary (CP) DCS if it sustained the following parameters for 1 min or more: observed respiratory rate (f_R) > 60 breaths $\cdot min^{-1}$, heart rate (f_H) > 150 bpm, and arterial O_2 saturation (S_{pO_2}) < 80%. This condition was usually accompanied by respiratory distress, as evidenced by open-mouthed, labored breathing, central cyanosis, inversion of the normal inspiratory/expiratory ratio, and production of frothy white sputum (10). All subjects with signs of severe DCS were removed to a Panepinto sling and given 2.5 mg diazepam i.v. as necessary to alleviate their distress. Skin DCS and behavioral features (e.g., limb lifting) indicative of milder DCS were noted but not classified as positive cases for this study. After the 1-h observation period the subjects were weighed. Close observation continued until 4 h post-surface, at which time they were euthanized by cardioplegia with bolus i.v. injection of 40 ml of 4-Molar potassium chloride solution. Previous experiments using this model have shown that all cases of DCS presented within this 4-h period (8). All animals that developed severe DCS or expired from their disease were immediately sent for necropsy as previously detailed to clinically verify the diagnosis based on observed symptoms (10).

Analysis: A priori calculations using the Chi-square test indicated that sample sizes of 26 subjects per group would detect a 50% change in incidence with $p = 0.05$ and 95% power. Independent variables included age, pre-dive weight, weight change during the dive, and treatment group. Differences in independent variables between groups were determined by *t*-tests or Mann-Whitney in the case of unequal variances. A survival analysis, using a log-rank test, was used to compare the time to symptom onset for each of the two groups. The influence of specific independent variables on outcome was determined using logistic regression and likelihood ratio testing in the manner described by Hosmer and Lemeshow (14). The logistic regression analysis was performed incorporating four independent variables; pre-weight, weight loss, age, and group (hydrated or dehydrated). Initially, a univariate analysis on each independent variable was performed; only those variables with a p-value > 0.20 (Wald test) were included in a multivariate analysis. Exclusion of a variable from the

TABLE I. SUMMARY OF OUTCOMES BY GROUP.

		DCS Cases				Death	Time of Onset (min)			
Group	N	CNS	CP	Both	Total		All DCS	CNS	CP	Death
Hydrated	31	2	9	1	10	4	22.6 ± 3.2	6.5 ± 1.5	23.4 ± 2.6	28.5 ± 5.4
Dehydrated	26	6	19	6	19	9	15.7 ± 2.3	17.3 ± 7.0	16.4 ± 2.4	20.3 ± 2.7
p-value		> 0.9	< 0.1	< 0.05	< 0.01	< 0.1	< 0.01	< 0.1	< 0.01	< 0.1

CNS = central nervous system; CP = cardiopulmonary DCS; Both = animals manifested both CNS and CP DCS; Death = animal expired from their disease and mean time of severe DCS for all animals (All DCS), for those with CNS or CP DCS, and the mean time of death after surfacing. The p-values represent differences in DCS outcome (Yates Chi-square) or mean values among groups (log-rank test for time of onset).

multivariate analysis was based on the log-likelihood ratio test. Statistical significance was set at the $p < 0.05$ level and p-values $0.05 < p < 0.1$ were considered a trend.

RESULTS

There were no significant differences in age between groups (mean ± SE; hydrated: 71 ± 1 d; dehydrated: 70 ± 1 d). Statistically significant pre-dive weight differences existed between the two groups ($p < 0.05$, ANOVA, mean ± SE; hydrated: 20.1 ± 0.3 kg; dehydrated: 21.0 ± 0.3 kg), but was only a significant predictor of CNS DCS. Dehydrated animals lost significantly more weight during the dive than the hydrated group ($p < 0.01$, ANOVA, mean ± SE; hydrated: 0.8 ± 0.1 kg; dehydrated: 1.7 ± 0.1 kg). The weight loss, expressed as a percentage of the pre-dive weight, was 4.0 ± 0.6% for the hydrated group and 8.1 ± 0.4% for the dehydrated animals.

The animals had DCS manifestations, case presentations, and histopathology similar to that previously described (9). Overall, 28/57 animals sustained severe DCS, and 13/57 succumbed to the disease. Hydrated animals had significantly less DCS (32.3%) compared with dehydrated animals (73.0%, $p < 0.01$) and there was a trend for a lower death rate (hydrated 12.9%, dehydrated 34.6%, $p < 0.1$, Yates Chi-square). The overall DCS onset time and CP DCS onset time was significantly shorter in the dehydrated group than the hydrated group ($p < 0.01$, log-rank test) and there was a trend toward a more rapid time to onset for CNS DCS and death. **Table I** summarizes results by group.

Neurological DCS was observed in 8/57 animals in this study and appeared < 1 h after surfacing, developing rapidly over the course of a few minutes. It manifested as progressive weakness of one or more limbs, most commonly involving the hind limbs. Most animals with evidence of CNS DCS began to show recovery within the 4-h observation period. CP DCS occurred in 28/57 animals, presenting as progressive tachypnea and tachycardia, with respiratory rates often exceeding 100 breaths · min^{-1} (> 300% of baseline) and sustained heart rates near 200 bpm (200% of baseline), combined with declining hemoglobin saturation often accompanied by production of frothy white sputum. If the animal did not recover, it manifested central cyanosis, increasing respiratory distress, eventually declined into agonal respiration, and died.

The logistic regression analysis suggested that dehydration with Lasix significantly increased the overall risk of severe DCS and death as compared with the hydrated animals ($p < 0.01$). Specifically, dehydration increased the risk of CP DCS ($p < 0.01$), and showed a trend toward increased CNS DCS ($p = 0.069$) in this model. In no case was pre-weight an important covariate, suggesting that the significant differences in pre-weight did not affect the DCS outcome.

DISCUSSION

Some studies have tried to find a correlation between DCS risk and variables such as body temperature, bodyweight, exercise, gender, adiposity, age, serum cholesterol, sensitivity to complement activation, Doppler bubble grades, patent foramen ovale, and hydration status, but most of these have had contradictory results (see the references in 13). The only physiological variable that has been undisputedly correlated with DCS risk is bodyweight in rats (17). Fluid status, on the other hand, is considered an important factor that can alter DCS risk, but only a few controlled studies have been conducted to verify this connection. A study in rats concluded that there appears to be a connection between increased DCS risk and dehydration (19). Previous work demonstrated that swine receiving i.v. injections of normal saline before a 22-h hyperbaric exposure to 110 fsw had a lower incidence of severe DCS as compared with a historical control group (11). However, comparing historical data complicates evaluation as minor differences in the experimental design may significantly alter outcome. Therefore, the purpose of the current investigation was to compare the DCS incidence in hydrated and dehydrated animals in a paired experimental design.

Swine have well recognized anatomical and physiologic similarities to humans, and volumes have been written about their use as biomedical research models (21). In recent years they have been successfully used to study a variety of diving-related conditions (2,3,20) and methods to improve decompression safety (8,16). The pathological findings of livid skin DCS, multiple punctate spinal and cerebral hemorrhages, and profuse pulmonary congestion after no-stop decompression are consistent with previous observations in other animal models of DCS, as well as human studies (2,4,22).

Outcome criteria in this study were necessarily severe, in part because the more subtle manifestations of the disease often cannot reliably be detected in an animal model. More invasive and, therefore, more sensitive testing methods were precluded by the need to observe the untreated natural history of the disease.

TABLE II. LOGISTIC REGRESSION RESULTS FOR ANIMAL AGE, PRE-DIVE WEIGHT, WEIGHT LOSS DURING THE DIVE, AND TREATMENT (DF = 1 FOR ALL) FOR ANIMALS WITH ACCESS TO WATER (HYDRATED) OR WITHOUT ACCESS TO WATER THROUGHOUT THE HYPERBARIC EXPOSURE AND GIVEN 2 MG · KG^{-1} LASIX (DEHYDRATED).

Outcome	Intercept	Age (d)	Pre-Weight (kg)	Weight Loss (kg)	Treatment Hydrated	LL	p-value
Severe DCS	—	—	—	—	—	−39.50	NULL
	1.47 ± 3.01	−0.02 ± 0.04	—	—	—	−39.39	> 0.6
	−3.45 ± 3.42	—	0.17 ± 0.17	—	—	−38.95	> 0.3
	−1.49 ± 0.64	—	—	−1.22 ± 0.45	—	−34.90	< 0.01
	0.13 ± 0.29	—	—	—	−0.87 ± 0.29	−34.63	< 0.01
	−0.81 ± 0.74	—	—	−0.73 ± 0.52	−0.57 ± 0.35	−33.58	> 0.1
CNS	—	—	—	—	—	−23.11	NULL
	−2.17 ± 4.31	0.01 ± 0.06	—	—	—	−23.10	> 0.9
	−14.37 ± 6.99	—	0.60 ± 0.33	—	—	−20.93	< 0.05
	−2.97 ± 1.01	—	—	−0.83 ± 0.61	—	22.06	> 0.1
	−1.94 ± 0.43	—	—	—	−0.74 ± 0.43	−21.46	< 0.1
	−13.19 ± 7.26	—	0.54 ± 0.34	—	−0.59 ± 0.46	−19.94	> 0.1
CP	—	—	—	—	—	−39.40	NULL
	2.20 ± 3.04	−0.03 ± 0.04	—	—	—	−39.12	> 0.4
	−3.17 ± 3.42	—	0.15 ± 0.17	—	—	−38.98	> 0.3
	−1.70 ± 0.67	—	—	−1.32 ± 0.47	—	−34.33	< 0.01
	0.05 ± 0.30	—	—	—	−0.95 ± 0.30	−33.82	< 0.01
	−0.94 ± 0.77	—	—	−0.64 ± 0.54	−0.77 ± 0.54	32.71	> 0.1
Death	—	—	—	—	—	−30.60	NULL
	3.39 ± 3.69	−0.07 ± 0.05	—	—	—	−29.79	> 0.2
	−1.97 ± 4.03	—	0.04 ± 0.19	—	—	30.51	> 0.8
	−1.79 ± 0.70	—	—	−0.44 ± 0.46	—	−30.10	> 0.3
	−1.27 ± 0.34	—	—	—	−0.64 ± 0.34	−28.61	< 0.05

Parameter estimates (± SE), log likelihood (LL), and p-value for the log likelihood ratio test compared to intercept only (NULL) models.

This was prompted by the very real possibility that a recompression chamber may not be available at a disabled submarine rescue site. The question to be answered was not how many subjects could benefit from recompression; theoretically they all would. Instead, we characterized how many subjects would require a chamber to prevent CNS morbidity or life-threatening DCS.

Bodyweight is frequently used as an easily measured, highly sensitive indicator of overall fluid loss. This is used both in a clinical situation (e.g., congestive heart rate failure patients) and as a standard technique in sport events (e.g., triathlon races, wrestling and tennis tournaments, and long-distance cycling events). In this study, without access to food or water, the weight loss is attributable to water losses from both normal dehydration, compression diuresis, and from the Lasix.

Regression analysis revealed that weight loss met or approached statistical significance. Addition of weight loss did not improve the log likelihood measurement when compared with the dehydrated group alone (**Table II**, severe DCS and CP DCS). The reason for this is that the two variables are highly correlated, as is also evident by the changing parameter estimates when both are included. Thus, in the final analysis, dehydration is the most significant predictor of severe and CP DCS and death in this study. In addition, hydrated animals had a longer time to symptom onset of severe DCS and a trend toward a prolonged time to death. This has important implications in the event of a disabled submarine (DISSUB) rescue operation, as hydrating survivors prior to decompression is a simple means of reducing DCS incidence and severity. For example, severe dehydration and hypothermia are common in DISSUB victims (15). It has long been suggested that dehydration may increase the risk of DCS and would impede the rescue effort. Thus, our results are suggestive of a simple and effective means to reduce DCS risk during a DISSUB rescue. In addition, the substantial effect of dehydration in the current study warrants further consideration for military, commercial, and recreational divers as well as astronauts and aviators as an important factor that affects DCS risk.

In conclusion, after direct ascent from saturation conditions, dehydrated animals manifest severe CP DCS sooner and more often than their hydrated counterparts. They also show a clear trend toward a higher rate of CNS DCS and more rapid death in this model.

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