Haemoconcentration, not decreased blood temperature, increases blood viscosity during cold water immersion

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Abstract
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Introduction: Prolonged cold-water immersion (CWI) has the potential to cause significant hypothermia and haemoconcentration; both of which have previously been shown to independently increase blood viscosity *in vitro*. The purpose of this study was to determine the effect of CWI on blood viscosity and examine the relative contribution of decreased blood temperature and haemoconcentration.

Methods: Ten healthy volunteers were immersed to mid-sternum in 10°C water for 90 minutes. Gastrointestinal (GI) temperature, haematocrit (Hct), and blood viscosity were measured pre- and post-CWI.

Results: CWI caused mean (SD) GI temperature to decrease from 37.5 (0.3)°C to 36.2 (0.7)°C ($P < 0.05$). CWI also caused mean Hct to increase from 40.0 (3.5)% to 45.0 (2.9)% ($P < 0.05$). As a result of the haemoconcentration and decreased GI temperature during CWI the mean blood viscosity increased by 19% from 2.80 (0.28) mPa·s^{-1} to 3.33 (0.42) mPa·s^{-1} ($P < 0.05$). However, when the pre-CWI blood sample was measured at the post-CWI GI temperature (36.2°C) there was no significant difference in the blood viscosity when compared to the pre-CWI (37.5°C) blood sample (2.82 (0.20) mPa·s^{-1} and 2.80 (0.28) mPa·s^{-1} respectively). Furthermore, the changes in Hct and blood viscosity during CWI were significantly correlated with an r = 0.84.

Conclusion: The results of the current study show that prolonged, severe CWI causes a significant 19% increase in blood viscosity. In addition, the results strongly suggest that almost all of the increased blood viscosity seen following CWI is the result of haemoconcentration, not decreased blood temperature.

Introduction

Prolonged cold-water immersion (CWI) is frequently encountered by recreational athletes (e.g., open ocean swimmers and divers, triathletes), military personnel during training (e.g., Special Forces) and is routinely used during recovery from exercise to attenuate inflammation and delayed-onset muscle soreness. It is well known that CWI results in haemoconcentration[1] and hypothermia[2]; both of which have been shown to independently increase blood viscosity, *in vitro*.[3–5] This is clinically important as increased blood viscosity has been shown to be associated with an increased risk for thrombo-emboli formation and thus the incidence of ischemic stroke, myocardial infarction, and pulmonary embolism.[6–8] It has been shown that cold air exposure can increase blood viscosity,[7] however, the effect of CWI on blood viscosity in humans is unknown. Likewise, the relative importance of decreased blood temperature and haemoconcentration on increasing blood viscosity during CWI has not been explored. Thus, the purpose of the current study was to address these two questions. It was hypothesized that CWI would significantly increase blood viscosity and that both haemoconcentration and decreased blood temperature would contribute to the increase.

Methods

PARTICIPANTS AND ETHICAL APPROVAL

Six male and four female participants ($n = 10$) with mean (SD) age 26 (6) years, height 1.70 (0.12) m, weight 73.4 (13.3) kg, and body mass index (BMI) 24.9 (3.0) kg·m^2, volunteered for the investigation. All volunteers initially provided a urine sample on the day of testing and hydration status was assessed using urine specific gravity (USG) via a clinical refractometer (Model 5711-2021; Schuco®, Williston Park, NY). USG values below 1.018 indicated euhydration and were required prior to the CWI. Pregnant females were excluded from the study. The study was conducted in accordance with the ethical standards of the San Diego State University Institutional Review Board for the protection of human subjects and with the 1964 Helsinki declaration and its later amendments. Informed written consent was obtained from all individual participants.

Figure 1
Mean (SD) GI temperature (°C) during 90 min of CWI. * indicates significantly different than the pre-CWI value

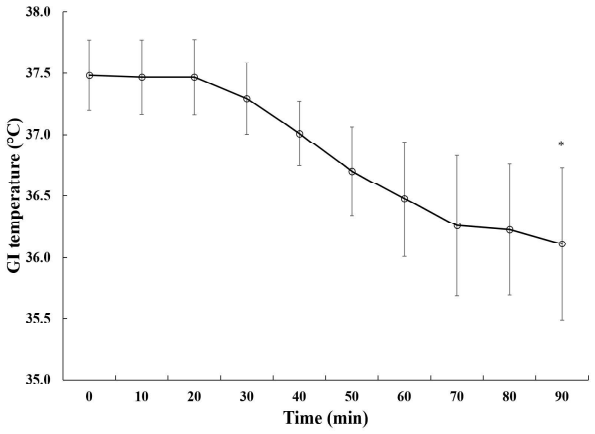
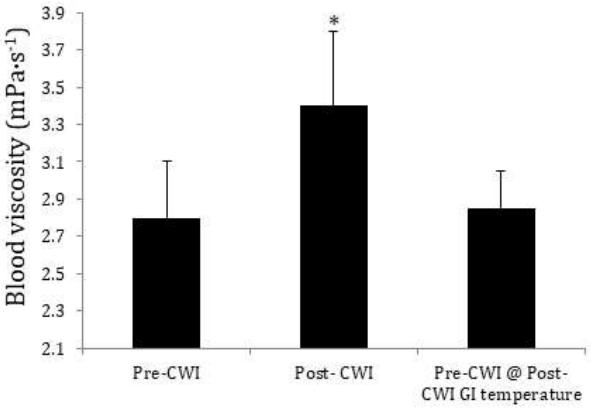


Figure 2
Blood viscosity values (mPa·s^{-1}) pre- and post-CWI. First bar represents the pre-CWI blood sample (40%, 37.5°C). Second bar represents the post-CWI blood sample (45%, 36.2°C). Third bar represents the pre-CWI blood sample measured at the post-CWI GI temperature (40%, 36.2°C). * indicates significantly different than the pre-CWI value



EXPERIMENTAL DESIGN

Each trial included 90 minutes of CWI (10°C) up to the participant's mid-sternum with the subject in the seated position in a large plastic tank with the right forearm and hand resting outside the tank not immersed. The subjects were clothed in shorts or a one-piece swimsuit. The water was not stirred during the CWI. Data were collected every five minutes throughout the trial and included gastro-intestinal (GI) temperature via an ingested pill (HQ Inc., Palmetto FL, USA) taken 3–6 hours before immersion and heart rate (Model RS400; Polar®, Lake Success NY, USA). The ambient laboratory temperature was 23°C.

Blood samples were collected, with the subject in the seated position, pre- and immediately post-CWI in order to determine haematocrit and blood viscosity. Approximately 300 µl of capillary blood was collected into a microtainer EDTA tube via a free-flowing fingertip stick from the non-immersed right hand. The tube was capped and inverted several times to prevent clotting. Blood was transferred to capillary tubes, centrifuged at 10,000 rpm, and measured in duplicate in order to determine pre- and post-CWI haematocrit (Hct). The mean was the recorded value.

BLOOD VISCOSITY ANALYSIS

To measure blood viscosity, 100 µl of the blood sample was transferred into a glass capillary tube along with a steel ball. The capillary tube was then capped and secured in the rotating arm of a Lovis 2000M microviscometer (Anton Paar, Graz, Austria) that was temperature controlled. Blood viscosity was determined three times: 1) pre-CWI blood sample at the pre-CWI GI temperature (37.5°C); 2) post-CWI blood sample at the post CWI GI temperature (36.2°C); and 3) pre-CWI blood sample at the post-CWI GI temperature (36.2°C). In order to determine the third blood viscosity measure, 100 µl of the pre-CWI blood sample was refrigerated until the CWI trial was completed when the post-CWI GI temperature was known. Blood viscosity was measured in mPa·s^{-1} and the shear rate was approximately 200 s^{-1}, which is well above the threshold (i.e., 100 s^{-1}) where whole blood is considered a Newtonian fluid.[9]

STATISTICAL ANALYSIS

Dependent *t*-tests were used to measure the changes between pre- and post-CWI GI temperature, Hct, and blood viscosity. The Bonferoni correction was performed to account for the multiple *t*-tests. Significance was set at the $P < 0.05$ level. Kolmogorov-Smirnov tests were used to ensure that the data did not differ significantly from normality. Pearson correlations were calculated between the change in blood viscosity and the change in Hct as well as with the change in GI temperature in order to determine if there was a relationship between the two variables. Statistical analysis was performed using VassarStats (http://vassarstats.net/).

Results

As shown in Figure 1, CWI caused mean GI temperature to decrease from 37.5 (SD 0.3)°C to 36.2 (0.7)°C ($P < 0.05$). CWI also caused mean haematocrit to increase from 40.0 (3.5)% to 45.0 (2.9)% ($P < 0.05$). As a result of the haemoconcentration and decreased blood temperature during CWI the mean blood viscosity increased by 19% from 2.80 (0.28) mPa·s^{-1} to 3.33 (0.42) mPa·s^{-1} ($P < 0.05$) (Figure 2). To determine the relative importance of decreased blood temperature and haemoconcentration on increasing blood viscosity during CWI, the pre-CWI blood sample was measured at both the pre-CWI and post-CWI GI temperature. This allowed for the determination of the effect of decreased blood temperature on blood viscosity, independent of

haemoconcentration. As seen in Figure 2, when the pre-CWI blood sample was measured at the post-CWI GI temperature (36.2°C) there was no significant difference in the blood viscosity when compared to the pre-CWI (37.5°C) blood sample (2.82 (0.20) mPa·s^{-1} and 2.80 (0.28) mPa·s^{-1} respectively). The change in GI temperature and change in blood viscosity during CWI were not significantly correlated with an r = 0.31. However, change in Hct and change in blood viscosity during CWI were significantly correlated with an r = 0.84.

Discussion

The most important new finding of the current study was the significant 19% increase in blood viscosity following 90 min of CWI. To our knowledge this is the first time such a finding has been reported in the literature. Although not previously reported, such a finding was not totally unexpected. Several previous studies[10–12] have reported a significant decrease in plasma volume following CWI. For example, Gordon et al.[10] using the Evans Blue direct-tracer dilution technique, reported that plasma volume decreased by 16% during one hour of CWI. As it has been shown that CWI does not alter red blood cell mass,[12] decreases in plasma volume must increase Hct. This is consistent with several past studies[1,13] that have reported increases in Hct of 4.3 to 4.6 units following CWI and are in agreement with the current finding of a 5.0 unit increase following CWI. It has been hypothesized that CWI decreases plasma volume via several mechanisms including increased blood pressure from cold induced vasoconstriction and hormonal induced diuresis.[10–12]

Numerous studies have examined the relationship between blood viscosity and Hct both *in vitro*[4,14] and *in vivo*.[15] In the normal physiologic Hct range (30–50%) the relationship is fairly linear, and the two variables are strongly correlated (r = 0.84). Within this range, blood viscosity increases about 4% for each one-unit increase in Hct.[16] Therefore, the five unit increase in Hct in our subjects following CWI would be predicted to increase blood viscosity by 20%, which is in close agreement to the 19% increase reported in Figure 2.

The relative contribution of decreased blood temperature and haemoconcentration was determined by measuring the pre-CWI blood sample at both the pre-and post CWI GI temperature. This allowed for the determination of the effect of decreased blood temperature on blood viscosity, independent of haemoconcentration. As seen in Figure 2, when the pre-CWI blood sample was measured at the post-CWI GI temperature (36.2°C) there was no significant difference in the blood viscosity when compared to the pre-CWI (37.5°C) blood sample (2.82 (0.20) and 2.80 (0.28) mPa·s^{-1} respectively). Furthermore, the change in blood viscosity was not significantly correlated (r = 0.31) with the change in GI temperature during CWI. However, the change in Hct and the change in blood viscosity during CWI were significantly correlated (r = 0.84). Such results strongly suggest that the increase in blood viscosity during CWI is primarily the result of haemoconcentration and not decreased blood temperature. Such a conclusion is supported by the findings of Azzopardi et al.[17] who used surface cooling to purposely cause hypothermia in newborn infants with birth asphyxia. The cooling resulted in an increase in blood viscosity that was not related to body temperature but was significantly correlated to Hct.

The major limitations of the current study are twofold. First, it was assumed that blood temperature during CWI would be equal to GI temperature. Thus, the effect of temperature on blood viscosity in the central circulation during CWI may be underestimated in the current study. Second, we did not re-warm the post-CWI blood samples back to 37.5°C as a positive control.

Conclusion

In conclusion, the results of the current study show that prolonged, severe CWI causes a significant 19% increase in blood viscosity. In addition, the results strongly suggest that almost all the increased blood viscosity seen following CWI is the result of haemoconcentration, not decreased blood temperature.

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