Effects of hydration and dehydration on blood rheology in sickle cell trait carriers during exercise

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The present study examined the hemorheological responses of a group of sickle cell trait (SCT) carriers and a control (Cont) group during submaximal exercise (2, 37) or medical complications (31) have revived the debate.

Several mechanisms have been proposed to explain these adverse events. Notably, many authors suggested that hemorheological abnormalities (19, 40, 48) and inflammation-vascular adhesion mechanisms (41, 42, 47) might be involved in microcirculatory alterations leading to sickle cell anemia-like vasoocclusive crisis. The hyperthermia and dehydration induced by physical exercise are thought to play a role in the occurrence of exercise-related sudden death in this population (29, 35). Accordingly, it has been recommended that SCT carriers should rehydrate adequately during exercise (2, 20, 31, 34, 35) to reduce the risk for adverse events potentially related to the red blood cell sickling process observed by Bergeron et al. (8). However, the effects of hydration/dehydration on whole blood rheology have never been investigated. In the study of Bergeron et al. (8), the percentage of venous sickled red blood cells in the two exercising SCT carriers deprived of water did not reach the values usually observed in patients with sickle cell anemia experiencing vasoocclusive crisis (5, 43). This observation suggests that other parameters, such as blood rheology (20), could be modulated by hydration/dehydration and potentially play a contributing role in microcirculatory and vascular alterations.

The present study examined the hemorheological responses of SCT carriers and a control (Cont) group during submaximal exercise in two conditions: one with water offered ad libitum, i.e., the hydration (Hyd) condition, and one without water, i.e., the dehydration (Dehyd) condition. Blood and plasma viscosities, as well as red blood cell rigidity, were determined at rest, at the end of exercise, and at 2 h recovery with a cone plate viscometer at high shear rate and 37°C. The SCT and Cont groups lost 1.0 ± 0.7 and 1.6 ± 0.6 kg of body weight, respectively, in the Dehyd condition, indicating a significant effect of water deprivation compared with the Hyd condition, in which body weight remained unchanged. Plasma viscosity increased with exercise and returned to baseline during recovery independently of the group and condition. As previously demonstrated, resting blood viscosity was greater in the SCT carriers than in the Cont group. Blood viscosity increased by the end of exercise and returned to baseline at 2 h recovery in the Cont group in both conditions. The blood viscosity of SCT carriers did not change in response to exercise in the Dehyd condition and remained elevated at 2 h recovery. This extended hyperviscosity, in association with other biological changes induced by exercise, could be considered as a risk factor for exercise-related events in SCT carriers, similar to vasoocclusive crises, notably during the recovery. In contrast, the Hyd condition normalized the hyperviscosity and red blood cell rigidity of the SCT carriers, with blood viscosity values reaching the same lower values as those found in the Cont group during the recovery. Adequate hydration of SCT carriers should be strongly promoted to reduce the clinical risk associated with potential hyperviscosity complications.

red blood cell rigidity; endurance effort; water ingestion; hemoglobinopathy generally considered as a benign condition. However, epidemiological data from the United States Army demonstrated that exercise-related sudden death was 30 times more common in black recruits with SCT than in black recruits without SCT (29, 35). Since then, the benign nature of SCT during exercise has been questioned (7, 17, 36), and recent case studies of exercise-related sudden death (2, 37) or medical complications (31) have revived the debate.

Sickle cell trait (SCT; i.e., the heterozygous form of sickle cell anemia) is common in people of African origin and is
adequate hydration in SCT carriers during exercise as a means to prevent microcirculatory and vascular disorders.

**MATERIALS AND METHODS**

**Subjects.** Eleven SCT carriers (SCT group) and twelve subjects with normal hemoglobin (Cont group) participated in the study. All subjects were athletes (>10 h of team sports/wk) and students at the National Institute for Popular Education and Sport of the Cheikh Anta Diop University in Dakar, Senegal. Subject characteristics are presented in Table 1. The major exclusion criteria were anemia and/or α-thalassemia, hypertension, and malaria.

**Protocol.** The subjects were informed of the procedures and purposes of the study, which was approved by the National Ethics Committee of Senegal, and gave written informed consent to participate. The protocol was in accordance with the Declaration of Helsinki.

On the first experimental day, each subject performed a progressive and maximal exercise test on a mechanically braked ergometer (Monark 824E, Stockholm, Sweden). The test began with a 5-min warm-up at 30 W. Explicit standardized instructions were given before each test. Pedaling speed remained constant (70 rpm) throughout the test, and a 30-W load was increased stepwise every minute until exhaustion (peak power output \( P_{\text{peak}} \)). Heart rate (HR) was measured continuously (S810, Polar Electo, Kempele, Finland), and maximal HR was noted.

Two weeks later, the same subjects participated in two randomized sessions (3–5 days apart), consisting of pedaling for 40 min at 55% \( P_{\text{peak}} \) (i.e., prolonged submaximal exercise). Subjects did not perform any other exercise for 2 days before each session. In one session, subjects could not drink water (Dehyd), whereas in the other they could drink water ad libitum (Hyd) during exercise. The volume of water ingested during the Hyd condition was measured accurately.

Rectal temperature (YSI thermistor thermometer, Yellow Springs Instrument, Yellow Springs, OH) and HR were measured continuously. Whatever the experimental conditions, the subjects consumed 40–50% of their body weight in the two groups remained constant between the two groups (Table 1).

**Hemorheological measurements.** Blood for hemorheological measurements was sampled in EDTA tubes. Hemorheological parameters were measured within 3 h of sampling. Measurements of blood viscosity \( (\eta_b) \) and plasma viscosity \( (\eta_p) \) were performed with a cone plate viscometer (Brookfield DVII+, with CPE40 spindle) at 37°C. Blood viscosity was determined at a shear rate of 225 s⁻¹. To avoid as much as possible the “surfactant layer effect” (6) on the measurements of \( \eta_b \), the analysis was done at a very high shear rate (i.e., 750 s⁻¹). Under the present experimental conditions, the flow instability of the sample in the gap between the cone and the plate should not occur in this type of viscometer (30). Hematocrit (Hct) was measured after blood microcentrifugation (Jouan-Hema-C, Saint Herblain, France), and hemorheological measurements were performed according to the recent guidelines for hemorheological laboratory techniques (6).

In addition, the index of red blood cell rigidity used by Dintenfass (25) was calculated according to the following equation: \( Tk = (\eta_b^{0.4} - 1)/ (\eta_p^{0.4} \times \text{Hct}) \), with \( \eta_b \) corresponding to the relative blood viscosity, i.e., the ratio of \( \eta_b \) to \( \eta_p \).

**Statistics.** All results are expressed as means ± SD. Subject characteristics, \( P_{\text{peak}} \), maximum HR, and the volume of water ingested were compared between the two groups using an unpaired Student’s \( t \)-test. The time courses of hemorheological parameters, rectal temperature, weights, and HR during the prolonged exercise (Hyd and Dehyd conditions) were compared between the two groups using a two-way analysis of variance with repeated measures. Pairwise comparisons (Fisher least significant difference post hoc tests) were used when necessary to locate where significant differences had occurred. The significance level was defined as \( P < 0.05 \). Analyses were conducted using Statistica (v. 5.5, Statsoft, Tulsa, OK).

**RESULTS**

**Subject characteristics and maximal exercise responses.** As shown in Table 1, no significant difference between the two groups was observed for height, weight, or age. The mean concentration of HbS for the SCT group was 38.0 ± 0.9%. In addition, the \( P_{\text{peak}} \) and maximum HR determined during the progressive and maximal exercise tests were not different between the SCT and Cont groups (Table 1).

**Exercise responses during the prolonged submaximal exercise.** Body weight in the two groups remained constant during the Hyd condition but decreased under resting value during the exercise conducted in the Dehyd condition (Table 2). The percentage of weight loss during the Dehyd condition was not significantly different between the two groups and was 2.0 ± 0.2 and 1.5 ± 0.3% in the Cont and SCT groups, respectively.

The volume of water ingested during the 40-min exercise period conducted in the Hyd condition did not differ between the two groups and was 604 ± 125 and 700 ± 73 ml in the Cont and SCT groups, respectively. HR and rectal temperature increased above baseline in the two groups and in the two conditions (Table 2). No significant difference was observed between the two groups.

Hct and \( \eta_p \) parameters are presented in Table 3. Hct did not differ between the two groups at any time and did not change with exercise. Plasma viscosity increased above baseline in response to exercise in the SCT group (Hyd and Dehyd conditions) and the Cont group (Hyd condition only) and then returned to baseline during the recovery. The change observed for \( \eta_p \) in the Cont group during the Dehyd condition was not statistically significant.

Resting \( \eta_b \) was higher in the SCT group than in the Cont group in the two conditions (Fig. 1). Exercise increased \( \eta_b \) in
the Cont group in the two conditions and \( \eta_b \) then returned to baseline during the recovery. The \( \eta_b \) did not change with exercise during the Dehyd and Hyd conditions in the SCT group. During the recovery, \( \eta_b \) decreased under the resting level in the Hyd condition only, reaching a value close to the values found in the Cont group (Hyd and Dehyd conditions) at that time. Blood viscosity in the SCT group did not change during the recovery of the Dehyd condition and, therefore, remained elevated. The Cont and SCT groups exhibited significantly lower values during the recovery of the Hyd condition compared with the Dehyd condition of the SCT group. The difference between the SCT group and Cont group during the recovery of the Dehyd condition did not reach statistical significance \((P < 0.1)\).

On the whole, baseline red blood cell rigidity was greater in the SCT group than in the Cont group in the two conditions (Fig. 2). Exercise did not significantly affect red blood rigidity in the Cont group. Red blood cell rigidity in the SCT group in the HYD condition was decreased at the end of exercise and during the recovery as compared with baseline. In contrast, it exhibited no change at the end of exercise compared with baseline in the Dehyd condition and then increased above baseline during the recovery. The Cont group exhibited significantly lower values during the recovery in both conditions compared with the Dehyd condition of the SCT group. The difference between the Dehyd and Hyd conditions of the SCT group during the recovery did not reach statistical difference \((P < 0.1)\).

**DISCUSSION**

**Main study outcome.** The main finding of the present study was the marked effect of ad libitum hydration on the \( \eta_b \) and red blood cell rigidity of exercising SCT carriers, with \( \eta_b \) and cell rigidity decreasing to the same values of the Cont group within 2 h of recovery.

**Exercise responses.** Our study confirmed previous findings showing that SCT carriers are able to perform progressive and maximal exercise or prolonged submaximal exercise at the same mechanical level as subjects with no hemoglobinopathy (see Ref. 20 for review). In addition, there were no unusual symptoms of general or local discomfort reported by either SCT carriers or Cont subjects. The magnitude of physiological stress was the same in the two groups, whatever the condition tested, as indicated by the outcome measures \(\text{i.e., HR and rectal temperature}\). Although the Dehyd condition induced a weight loss in both groups, HR and rectal temperature did not differ between the Hyd and Dehyd conditions at the end of exercise. This unexpected finding cannot be explained by environmental differences between the Hyd and Dehyd conditions since humidity and external temperature were the same in the two conditions. In addition, the exercise intensity was exactly the same for each subject in the two conditions. Although the preexercise body weight difference between the Hyd and Dehyd conditions was \(<1\%\) in both groups, the preexercise hydration level of the subjects was not evaluated \(\text{i.e., plasma osmolality or urine specific gravity}\) as recommended \((38, 39)\), and this is clearly a shortcoming of the study. The body weight loss during the Dehyd condition remained within an acceptable range \(\text{i.e., ~1.5 to 2\% (1)}\) and was apparently of limited effect on HR and rectal temperature. Recent studies comparing the effects of neutral and hot temperature exposures on cycling responses reported that HR and rectal temperature did not differ between the two environmental conditions \((32, 33)\). However, subjects exhibited higher skin temperature changes (not measured in our study) in the hot

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**Table 2.** *Effects of prolonged submaximal exercise and hydration/dehydration on weight, rectal temperature, and HR in the two groups*

<table>
<thead>
<tr>
<th></th>
<th>Weight, kg</th>
<th>Rectal Temperature, °C</th>
<th>HR, beats/min</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>End of Exercise</td>
<td>Rest</td>
</tr>
<tr>
<td><strong>Cont</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehyd</td>
<td>70.2 ± 6.6</td>
<td>68.6 ± 6.6*</td>
<td>37.2 ± 0.3</td>
</tr>
<tr>
<td>Hyd</td>
<td>69.9 ± 6.2</td>
<td>69.8 ± 6.6</td>
<td>37.2 ± 0.3</td>
</tr>
<tr>
<td><strong>SCT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehyd</td>
<td>65.1 ± 7.0</td>
<td>64.1 ± 7.0*</td>
<td>37.1 ± 0.7</td>
</tr>
<tr>
<td>Hyd</td>
<td>64.6 ± 7.0</td>
<td>64.8 ± 6.0</td>
<td>37.3 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SD. Dehyd, dehydration condition; Hyd, hydration condition. *\(P < 0.05\), difference between rest and end of exercise.

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**Table 3.** *Hematocrit and plasma viscosity at rest, at the end of the prolonged submaximal exercise, and at 2 h recovery in the two groups and in the two conditions (Hyd/Dehyd)*

<table>
<thead>
<tr>
<th></th>
<th>Hematocrit, %</th>
<th>Plasma Viscosity, mPa/s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>End of Exercise</td>
</tr>
<tr>
<td><strong>Cont</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehyd</td>
<td>46.8 ± 3.6</td>
<td>47.4 ± 3.1</td>
</tr>
<tr>
<td>Hyd</td>
<td>45.3 ± 3.1</td>
<td>46.1 ± 3.7</td>
</tr>
<tr>
<td><strong>SCT</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehyd</td>
<td>46.5 ± 3.0</td>
<td>46.5 ± 3.1</td>
</tr>
<tr>
<td>Hyd</td>
<td>47.0 ± 2.6</td>
<td>47.6 ± 2.6</td>
</tr>
</tbody>
</table>

Values are means ± SD. *\(P < 0.05\), difference between rest and end of exercise; †\(P < 0.05\), difference between end of exercise and recovery.
condition, suggesting hyperthermic strain compared with the neutral condition (32, 33).

**Blood rheology at rest.** Blood viscosity was higher at rest in SCT carriers than in the Cont group. Since \( \eta_b \) and Hct were not different between the two groups, the difference in \( \eta_b \) was related to the reduced resting red blood cell deformability (i.e., higher Tk index reflecting higher red blood cell rigidity), as previously demonstrated (9, 18, 22). Although most of the hemorheological alterations usually found in SCT carriers may be considered as subclinical (45), \( \eta_b \) values can be as high as in patients with sickle cell hemoglobin C disease (45), who are known to develop frequent thromboembolic complications. The mean value of \( \eta_b \) in sickle cell hemoglobin C disease is about 6 mPa/s (45), and 5 of the 11 SCT carriers in the present study exhibited greater values at rest, indicating the presence of hyperviscosity. Two other SCT carriers exhibited viscosity values greater than the mean elevated blood viscosity value reported in the larger group of SCT carriers from the study of Tripette et al. (45). Thus 4 out of 11 SCT carriers exhibited normal values. The range of viscosity values for the 11 SCT subjects (4.76–7.48) was very closed to the range found in sickle cell hemoglobin C subjects (4.49–7.58) (45) but is different from the range found in the 11 control subjects of the present study (4.03–5.62). These results could explain why SCT carriers, like patients with sickle cell hemoglobin C, are at greater risk than normal subjects to develop veinous thromboembolism and pulmonary embolism (4). SCT carriers with elevated baseline \( \eta_b \) values could be considered as potentially at risk for microcirculatory disorders, and hydration levels should be carefully controlled in these individuals. Strategies to normalize \( \eta_b \) in SCT carriers would be beneficial for their health.

**Blood rheology in response to exercise in Cont group.** The increase of \( \eta_b \) in the Cont group during the cycling exercise conducted in both conditions is a classical finding (10, 14, 23, 26) and is related to the changes in \( \eta_p \). Although not significant, the changes in red blood cell rigidity during the Dehyd condition also contributed to the increase in \( \eta_b \). The increase in \( \eta_b \) during the Dehyd condition resulted in values in the Cont group close to the values found in the SCT group, in both conditions. The \( \eta_b \) of the Cont group then returned to baseline during the recovery, partly in relation with the return to baseline of \( \eta_p \) and the nonsignificant reduction in red blood cell rigidity. The lack of difference between the Hyd and Dehyd conditions in the Cont group was very surprising but indicated that dehydration did not exert any adverse effects on blood rheology during a 40-min submaximal exercise. The plasma volume change in both conditions was minimal and can be evaluated (50) at 2 to 3% after exercise (and less during recovery) compared with the resting value. The plasma shifts between intravascular and extravascular spaces may play a role in this equilibrium (27).

**Blood rheology in response to exercise in SCT carriers.** In contrast, it seems that water hydration played a very important role in the exercising SCT carriers. When the SCT carriers performed the exercise protocol in Dehyd condition, \( \eta_b \) remained at the same elevated value as observed at baseline, and this effect was persistent at 2 h recovery. Given the mean blood viscosity (±SD) values published by Tripette et al. (45) for healthy individuals (5.10 ± 0.60) and subjects with either SCT (5.58 ± 0.70) or sickle cell disease (5.99 ± 1.30), the difference between the Dehyd (5.69 ± 0.97) and Hyd (5.08 ± 0.37)
conditions observed in the SCT group during recovery is of clinical relevance. We suggest that the persistent hyperviscosity during the recovery period, in association with the other physiological/biological changes previously observed in the hours following an exercise bout in SCT carriers, could increase the risks for clinical complications in this population at that time. Trippette et al. (47), for example, reported that exercise may activate white blood cells to a greater level in SCT carriers than in a control group, as evidenced by the increases in the plasma L-selectin level during the recovery of an exercise bout. Moreover, in a study investigating heart rate variability, Hedreville et al. (31) observed impaired autonomic nervous system activity in SCT carriers during the recovery of a strenuous exercise. Altogether, these biological and physiological changes (i.e., hyperviscosity, decreased red blood deformability, increased adhesion molecules level, and autonomic imbalance) could indicate an increased risk for adverse events during the recovery of an exercise.

Nevertheless, as at rest, hyperviscosity is not always observed in SCT carriers in response to exercise (40). Although not addressed in this study, this heterogeneity could result from hemorheological compensation, such as lowered Hct to compensate for the reduction in red blood cell deformability (40). Improving physical fitness might potentially be a way to improve the hemorheological profile of SCT carriers, as demonstrated in noncarriers (10, 11). Moreover, the athletes with the SCT from the study of Monchanin et al. (40) were fitter than the athletes with SCT from the present study, as indicated by the difference in $P_{\text{peak}}$ reached during the maximal incremental exercise test. The recent findings of Aufradet et al. (3) support the hypothesis that training and physical fitness can modulate vascular adhesion phenomena in SCT carriers, but this hypothesis needs to be rigorously tested in hemorheological studies. In addition, it should be noted that hemorheological responses to exercise are dependent on the type of exercise (16), with some being more stressful for SCT carriers (40, 48) than others (14, 22).

Of greater importance, when ad libitum hydration was offered to the SCT carriers, their $\eta_b$ decreased during recovery and reached the very low values found in the Cont group. Similar to the findings in subjects with no hemoglobinopathy, the Hct of the SCT carriers was unchanged by exercise (plasma volume change was minimal) and $\eta_b$ increased slightly at the end of exercise before returning to baseline during the recovery. Therefore, the decrease in $\eta_b$ in the SCT carriers during the recovery of the Hyd condition ($-13\%$ from baseline) was related to a decrease in red blood cell rigidity below baseline. This reduction in $\eta_b$ and red blood cell rigidity might limit the risks for microvascular alterations, which was advanced to explain postexercise complications in SCT carriers (19, 20). This novel observation strengthens the findings of Bergeron et al. (8), showing beneficial effects of hydration on sickling in two walking SCT carriers. The reduction in red blood cell rigidity in the exercising SCT carriers was unexpected, but it had already been described during a short ramp exercise test (40). As extensively discussed (22, 24, 40), the increases in oxidative stress (44) and lactate (15) during exercise may have different effects on the red blood cells from trained and sedentary individuals, with the former being better protected against a reduction in red blood cell deformability, as observed in the present study. Although the effects of hydration and dehydration on the exercise physiological responses of SCT carriers appear to be the same, ad libitum hydration was able to normalize during the recovery the hemorheological alterations observed at rest. Hydration probably plays a key role in the hydration status of SCT carriers’ red blood cells, which are known to be moderately dehydrated at times (21), leading to a normalization of their deformability. In contrast, in the Dehyd condition, the red blood cell rigidity of the SCT carriers increased during recovery above the values found at the end of exercise and returned to the elevated baseline level; this may explain why $\eta_b$ remained elevated during recovery in that condition. Although the present study did not specifically investigate the reason for this late reincrease in red blood rigidity in the Dehyd condition, recent findings suggest that lipid peroxidation of red blood cells during the recovery of a submaximal exercise is specific to SCT carriers and that this could affect the red blood cell membrane (46). We also examined blood smears from two SCT carriers during the Dehyd and Hyd conditions. We observed few sickled red blood cells at rest or at the end of exercise (< 0.5%) and no change induced by exercise whatever the experimental conditions. The red blood cell alterations and sickling rate were probably of low magnitude during recovery since the cell rigidity values did not differ between baseline and recovery periods.

**Alternative view and conclusion.** The interpretation of hyperviscosity in SCT carriers is difficult since recent findings support the viewpoint that hyperviscosity might be beneficial for vascular adaptation (13, 23). The increase in $\eta_b$ may increase shear stress and stimulate vasodilation through a nitric oxide-dependent mechanism, thereby promoting adequate oxygen delivery to tissues (49). Unfortunately, hemodynamic parameters like vascular resistance were not measured in the present study and this hypothesis needs to be tested. Vincent et al. (51) observed that SCT carriers are characterized by lower capillary tortuosity, reduced counts of microvessels with diameters < 5 μm, and a higher percentage of broader microvessels, i.e., with diameters > 10 μm, compared with individuals without hemoglobinopathy. This suggests that SCT carriers undergo chronic vascular remodeling to compensate as much as possible for the hemorheological disorders.

As noted above, the mean $\eta_b$ values of the SCT carriers were close to the values found in sickle cell hemoglobin C disease (45), and Austin et al. (4) demonstrated that SCT carriers are prone to thromboembolic complications. We therefore suggest that the chronic hyperviscosity (found in approximately one-third of SCT carriers), which is associated with other exercise-induced biological changes previously described, should be considered as a risk factor for exercise-related sudden death and exercise-related medical complications in SCT carriers, especially during the recovery. However, the main finding of the present study is that ad libitum hydration is able to normalize $\eta_b$ in SCT carriers. This normalization, in association with the chronic structural adaptation of microvessels (i.e., larger microvessels), may favor normal blood flow and adequate tissue perfusion in this population. Physicians and sports trainers should vigorously promote adequate hydration in SCT carriers before, during, and after exercise to spare them as much as possible from clinical problems.
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DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

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