

Hemorheology, Sickle Cell Trait, and α -Thalassemia in Athletes: Effects of Exercise

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ABSTRACT

MONCHANIN, G., P. CONNES, D. WOUASSI, A. FRANCINA, B. DJODA, P. E. BANGA, F. X. OWONA, P. THIRIET, R. MASSARELLI, and C. MARTIN. Hemorheology, Sickle Cell Trait, and α -Thalassemia in Athletes: Effects of Exercise. *Med. Sci. Sports Exerc.*, Vol. 37, No. 7, pp. 1086–1092, 2005. **Purpose:** This study investigated hemorheological parameters in response to exercise in sickle cell trait (SCT) athletes with or without α -thalassemia. **Methods:** Six athletes with SCT (HbAS), 7 athletes with SCT and α -thalassemia (HbASAT), and 10 control athletes (HbAA) performed a progressive and maximal exercise test on cycloergometer. Blood viscosity (η_b), plasma viscosity (η_p), η_b at corrected hematocrit ($\eta_{b_{45}}$), hematocrit (Hct), and red blood cell (RBC) rigidity were assessed at rest, at maximal exercise and 24 h after exercise. **Results:** η_b and η_p were not different between the three groups at any time. Exercise induced changes in η_b in HbAA and HbASAT groups but not in HbAS group. $\eta_{b_{45}}$ was higher in HbAS group compared with the other groups ($P < 0.05$), at rest and 24 h after exercise and increased only in HbAA group in response to exercise. HbAS group had lower Hct than HbAA group at any time. Hct and η_p increased after exercise and declined under baseline values 24 h after exercise in all groups. RBC rigidity was higher in HbAS group compared with HbAA and HbASAT groups at any time, and was lower and higher at maximal exercise and 24 h after exercise, respectively, in all groups compared with resting values. **Conclusions:** These results demonstrate that HbAS group is prone to higher RBC rigidity, which might lead to hemorheological alterations that are thought to participate to microcirculation disorders. However, these alterations are limited by the coexistence of α -thalassemia. Moreover, hemorheological parameters were not further impaired in SCT athletes with or without α -thalassemia in response to exercise. Training status might be protective from physiological stresses usually leading to sickling process in SCT carriers. **Key Words:** HEMOGLOBINOPATHY, BLOOD VISCOSITY, RED BLOOD CELL DEFORMABILITY

Sickle cell disease (SCD) is caused by the mutation of hemoglobin (Hb) A into HbS and mainly affects people of African descent. This mutation is caused by the substitution of a single amino acid, valine for glutamic acid. Sickle cell trait (SCT) carriers are characterized by the heterozygous presence of both HbA and HbS (AS genotype) in their red blood cells (RBC). The prevalence of SCT is around 20–40% in some areas of Black Africa and reaches 15% in European metropolises. In normal environmental conditions, RBC from subjects with SCD or SCT are less

deformable than normal erythrocytes (AA genotype) (3). This hemorheological impairment is thought to participate to microcirculatory disorders reported in this population (21). If oxygen level is decreased, HbS polymerizes leading to a sickling process of erythrocytes that further alter their deformability (3,7). SCT has been associated with increased risk of sudden death during and several hours after intense exercise under normoxic and/or hypoxic conditions (20). Exercise induces physiological changes (acidosis, dehydration, regional hypoxemia, or hyperthermia) that might promote sickling of HbS and blood rheological changes that might be responsible of vaso-occlusive events. However, blood rheology alterations may also occur in healthy athletes during and several hours after exercise (10,30), leading to a drastically increased blood viscosity (η_b) and decreased RBC deformability (5,30). Hemorheological parameters have never been investigated in SCT carriers in response to exercise, and it is still unknown whether changes in η_b and RBC deformability are more marked during and after exercise in SCT athletes than in healthy athletes.

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Harkness (18) reported that the severity of SCT could be limited by the coexistence of SCT with α -thalassemia. The latter is caused by the deletion of a part of the gene encoding for the α -globin chain leading to a decreased production of the protein. The study of the coexistence of SCT and α -thalassemia is particularly interesting because of the high prevalence of both genetic defects in the black population (13). Le Gallais et al. (22) have hypothesized that coexistence of SCT with α -thalassemia could be protective in decreasing the severity of health complications caused by SCT alone because of blood rheology improvement in SCT carriers. It is now well admitted that there is a profound influence of the concentration of HbS on both the kinetics and the extent of HbS polymerization and thus on the clinical expression of SCD and SCT (19). Coexistence of α -thalassemia with SCT should be protective against the sickling process (18) because of decreased HbS intra-erythrocyte concentration. Indeed, hemorheological changes induced by acute exercise should be reduced in athletes with both SCT and α -thalassemia compared with athletes with SCT alone.

To test these hypotheses, we compared hemorheological parameters at rest, immediately at the end of a progressive and maximal exercise, and several hours after this exercise (24 h) between healthy trained subjects and well-trained athletes with SCT with or without α -thalassemia.

METHODS

Subjects. Six subjects with SCT (HbAS group), 7 subjects with both SCT and α -thalassemia (HbASAT group), and 10 controls (HbAA group) participated in the study after giving their informed consent. All subjects were competitive athletes and trained regularly for 10–15 yr in endurance sports (soccer and running). Athletes were recruited at the National Institute of Youth and Sport in Yaoundé (Cameroon) after a complete medical examination. Exclusion criteria included hypertension, stroke and malaria.

Protocol. The protocol was approved by the National Ethics Committee of Cameroon (registration number: FWA IRB00001954) and was in accordance with the guidelines set by the Declaration of Helsinki. On the experimental day, all athletes performed an incremental maximal exercise on a cycloergometer until they reach maximal power (Ppeak). Venous blood samples were drawn from the antecubital vein of the nondominant arm at rest and after exercise to perform sickle cell trait and α -thalassemia detections, hemorheological measurements, and lactate concentration analysis. The laboratory was air-conditioned (room temperature 25–27°C, hygrometry 55%). No additional drink was allowed before and during exercise.

Sickle cell trait and α -thalassemia detections. Blood samples were collected at rest in EDTA tubes. The various Hb were isolated and quantified by an ion-exchange high-performance liquid chromatography (HPLC) (Variant I, Beta Thal Short Program; Bio-Rad Laboratories, Hercules, CA). Positive test results for SCT were determined by

the presence of HbS but with less than 50%. To study the presence of α -thalassemia, we used the technique from Chong et al. (8) with single-tube multiplex-PCR assay that is capable of detecting any combination of the six common single and double gene deletions in α -thalassemia. Mean values for HbS were $38.3 \pm 0.2\%$ and $33.4 \pm 0.1\%$ in HbAS group and HbASAT group, respectively. The heterozygous form of α -thalassemia found in HbASAT group was marked by a deletion of 3.7 kb of DNA, containing one of the two linked α -globin genes ($\alpha\alpha/\alpha^{3,7}$).

Exercise test. Each subject performed a progressive and maximal exercise test on a mechanically braked ergometer (Ergomeca, France). 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 (Ppeak).

Blood and plasma analysis. Blood for hemorheological measurements was sampled at rest; immediately at the end of exercise (Ppeak), that is, when subjects reached Ppeak and stopped exercise; and 24 h after the end of the test in EDTA tubes. To avoid RBC damage, hemorheological parameters were immediately measured after blood sampling. Measurements of η_b and η_p were performed with a cone plane viscometer (Brookfield DVII+, with CPE40 spindle), at high shear rate (375 s^{-1}). Under the present experimental conditions, the flow instability of the sample in the gap between the cone and the plate should not happen in this kind of viscometer (23). Hct was measured by a micro-method after blood microcentrifugation (Jouan Guetin-SA type 316).

Blood viscosity at corrected Hct (45%) ($\eta_{b_{45}}$) was calculated according to the equation of Quemada (26):

$$\eta_{b_{45}} = \eta_p \cdot (1 - 1/2 \cdot k \cdot \text{Hct})^{-2}$$

where k is a parameter that describes the rigidity of RBC at high shear rate that was calculated with the following equation:

$$k = 2 \cdot (1 - \eta_r^{-0.5}) \cdot \text{Hct}^{-1}$$

where η_r is relative blood viscosity (η_b/η_p).

Another index of RBC rigidity (Tk) was calculated at high shear rate (375 s^{-1}) according to the equation of Dintenfass (12):

$$\text{Tk} = (\eta_r^{0.4} - 1)/(\eta_r^{0.4} \cdot \text{Hct}).$$

Fingertip arterialized blood microsamples were taken for lactate analysis before beginning of pedaling and 3 min after the end of the test using a portable lactate analyzer (Lactate Pro LT-1710, Roche Bioelectronics, Switzerland, Basel).

Statistical analysis. Results are presented as mean \pm SEM. Data related to subject characteristics and Ppeak were compared between the three groups using a one-way analysis of variance (ANOVA). Hemorheological parameters and blood lactate concentrations at rest, at the end of exercise, and 24 h after the end of the test were compared between all groups using a two-way ANOVA with repeated measurements on the intra factor. Pairwise contrasts were used when necessary to determine where significant differ-

TABLE 1. Anthropometric characteristics and maximal exercise power in HbAA, HbAS, and HbASAT groups.

	HbAA (N = 10)	HbAS (N = 6)	HbASAT (N = 7)
Age (yr)	29.5 ± 0.7	29.5 ± 2.8	29.4 ± 1.9
Weight (kg)	74.6 ± 1.2	71.5 ± 6.2	71.7 ± 4.2
Height (cm)	176.5 ± 1.9	175.5 ± 3.3	175.6 ± 2.1
Ppeak (W)	252.6 ± 14.0	239.2 ± 5.8	260.0 ± 12.9

Values are means ± SEM.

ences occurred. The relationships between the percentage of HbS and hemorheological parameters in HbAS and HbASAT groups were evaluated by using a Pearson correlation. Statistical significance was established at $\alpha = 0.05$. Analyses were conducted using Statistica (v. 5.5, Statsoft, U.S.).

RESULTS

Anthropometric characteristics and Ppeak. As shown in Table 1, anthropometric data (age, weight, and height) and Ppeak achieved during the incremental exercise test were not different between the HbAA, HbAS, and in HbASAT groups.

Blood and plasma analysis. η_b was not statistically different among the three groups at any time (Table 2). η_b increased between rest and the end of exercise in the HbAA and HbASAT groups but did not change in the HbAS group. η_b was significantly decreased 24 h after exercise compared with resting values in all groups ($P < 0.05$)

η_p was not statistically different between the HbAA, HbAS, and HbASAT groups at any time (Table 2). Hct was lower in the HbAS group compared with the HbAA group at any time ($P < 0.05$; Table 2). Compared with resting values, Hct and η_p increased at the end of exercise ($P < 0.05$) and decreased 24 h after the exercise ($P < 0.05$) in the three groups.

When corrected to a 45% Hct, η_b (i.e., $\eta_{b_{45}}$) was found to be statistically higher in the HbAS group than in the HbAA and HbASAT groups at rest and 24 h after exercise ($P < 0.05$; Fig. 1). No significant difference in $\eta_{b_{45}}$ was found between the three groups at the end of exercise. Only the HbAA group exhibited a significant increase in $\eta_{b_{45}}$ at the end of exercise ($P < 0.05$). No statistical difference was found between resting $\eta_{b_{45}}$ and 24 h after exercise $\eta_{b_{45}}$ in all groups.

Tk and k were higher in the HbAS group compared with the HbAA and HbASAT groups at rest, at the end of exercise, and 24 h after exercise ($P < 0.05$; Fig. 2 and 3). Changes in Tk and k in response to exercise, that is, immediately at the end of exercise and 24 h after the test, did not differ among the three groups. Tk and k were lower and higher at the end of exercise and 24 h after exercise compared with resting values, respectively ($P < 0.05$).

No difference in blood lactate concentration was found at rest (Fig. 4), but it increased significantly during exercise in all groups ($P < 0.05$). Three minutes after the end of exercise, the HbAS group had a higher value than the HbAA group.

A positive correlation between $\eta_{b_{45}}$ and the percentage of HbS was found in the HbAS and HbASAT groups ($r = 0.61$;

$P < 0.05$; Fig. 5a) while the percentage of HbS was negatively correlated to Hct ($r = -0.636$; $P < 0.05$; Fig. 5b). No significant correlation was found between the percentage of HbS and either η_b , η_p , Tk, or k.

DISCUSSION

The aim of this study was to determine whether hemorheological parameters of HbAS-trained athletes were affected at rest, at the end of a progressive and maximal exercise bout, and 24 h after exercise compared with HbAA-trained athletes and whether hemorheological alterations were less marked in HbASAT-trained subjects compared with HbAS-trained subjects. The main results of this study showed that 1) $\eta_{b_{45}}$ was higher in the HbAS group compared with the HbAA and HbASAT groups at rest and 24 h after exercise; 2) Hct was lower in the HbAS group compared with the HbAA group at any time; 3) RBC rigidity indexes were higher in the HbAS group compared with the HbASAT and HbAA groups at any time; and 4) changes in hemorheological parameters between rest, the end of maximal exercise, and 24 h postexercise were different between the HbAS and the other groups.

In the present study, maximal exercise performance (Ppeak) was not affected by the presence of the sickle cell trait (Table 1). Ppeak from the HbAS group was comprised in the range of Ppeak measured by Gozal et al. (15) and confirms the findings of previous studies that showed similar maximal exercise power, during cycling exercise test, between HbAS athletes and HbAA athletes (27,28). Our results also demonstrated that Ppeak from athletes with both SCT and α -thalassemia was not different from Ppeak of athletes with normal Hb and athletes with SCT. These results are in contrast with those from Le Gallais et al. (22), who have hypothesized that endurance performance is improved in subjects with both SCT and α -thalassemia when compared with subjects with SCT alone. However, the exercise protocol used in this study might have been too short to really assess endurance capacity of the subjects. Further studies are needed to analyze cardiorespiratory responses of these subjects during prolonged and intense exercise bouts.

Blood lactate concentration was higher 3 min after the end of the exercise in the HbAS group compared with the two other groups. This finding is in accordance with the results from Freund et al. (14) but contrasts with those from Bilé et al. (2), Gozal et al. (15), and Sara et al. (27), who observed lower lactate levels in SCT carriers compared with HbAA subjects for a given intensity during a progressive and maximal exercise. These discrepancies are not well understood, but it is often reported that when HbAS and HbAA subjects are correctly matched in term of both physical performance and physical activity—as done in the studies from Bilé et al. (2) and Sara et al. (27)—HbAS subjects exhibit lower lactate levels during exercise. The results reported in the present study did not confirm these conclusions. Freund et al. (14) reported that SCT carriers were likely to produce more lactate than control subjects reaching exhaustion and/or to have an impaired ability to clear cir-

TABLE 2. Hemorheological parameters at rest, at the end of maximal exercise, and 24 h after exercise in HbAA, HbAS, and HbASAT groups.

	HbAA (N = 10)			HbAS (N = 6)			HbASAT (N = 7)		
	Rest	At the End of Maximal Exercise	24 h after Exercise	Rest	At the End of Maximal Exercise	24 h after Exercise	Rest	At the End of Maximal Exercise	24 h after Exercise
η_b (mPa·s) at 375 s^{-1}	4.78 ± 0.14	$5.38 \pm 0.19^*$	$4.39 \pm 0.12^*$	5.01 ± 0.29	5.05 ± 0.4	$4.55 \pm 0.16^*$	5.24 ± 0.15	$5.59 \pm 0.21^*$	$4.49 \pm 0.18^*$
η_p (mPa·s)	1.56 ± 0.02	$1.69 \pm 0.08^*$	$1.45 \pm 0.03^*$	1.59 ± 0.06	$1.66 \pm 0.07^*$	$1.50 \pm 0.03^*$	1.57 ± 0.03	$1.67 \pm 0.05^*$	$1.51 \pm 0.06^*$
Hct (%)	46.02 ± 1.26	$47.16 \pm 0.98^*$	$41.75 \pm 0.61^*$	$43.66 \pm 2.03\text{§}$	$45.50 \pm 1.51^*\text{§}$	$40.90 \pm 1.93^*\text{§}$	46.11 ± 0.94	$48.75 \pm 1.00^*$	$43.90 \pm 0.96^*$

Values are means \pm SEM. η_b , blood viscosity; η_p , plasma viscosity; Hct, hematocrit; * significant difference between rest, end of maximal exercise, and 24 h after exercise ($P < 0.05$); † significant difference between HbAA and HbASAT groups ($P < 0.05$); § significant difference between HbAA and HbAS groups ($P < 0.05$).

culating lactate. Although, the HbASAT group tended to have higher blood lactate concentration than the HbAA group, there was no statistically significant difference between these groups that might indicate the efficacy of α -thalassemia in limiting the blood lactate increase during exercise in SCT subjects. Further studies are required to better understand lactate metabolism in SCT carriers exhibiting or not α -thalassemia.

The most interesting results found in this study concern the different hemorheological pictures between SCT carriers with and without α -thalassemia and athletes with normal Hb. Although η_b measured at high shear rate was not different among the three groups at any time, $\eta_{b_{45}}$ was higher in the HbAS group compared with the other groups at rest and after 24 h of recovery. This finding can be compared with those of Chien et al. (7), who reported that, even under conditions of complete oxygenation, the viscosity of HbSS blood exceeds the viscosity of HbAA blood at a same Hct. The positive correlation found between the percentage of HbS and $\eta_{b_{45}}$ suggests that the severity of hemorheological impairment could be linked to the percentage of HbS.

At high shear rate, η_b mainly depends on Hct, η_p , and RBC rigidity. η_p was not significantly different between the three groups at rest and in response to exercise. To determine η_p , a rotational viscometer was used. Even if falling

ball viscometer or capillary viscometer would have been more accurate to measure η_p (4), it has been shown that η_p could be determined with a rotational viscometer above a shear rate of 250 s^{-1} (16,23). Moreover, Haidekker et al. (16) recently demonstrated that η_p values obtained with a new accurate technique were close to values obtained with a mechanical or rotational viscometer, similarly to the present study. One may assume that *in vitro* measurement of η_p with a cone plane viscometer was sufficiently accurate to be interpreted. Because a comparable value of η_p was found in HbAA and HbAS groups, RBC rigidity might be a key factor to explain higher $\eta_{b_{45}}$ in subjects with SCT. The use of Dintenfass and Quemada's equations (12,26) confirmed our hypothesis because T_k and k were higher in HbAS group at any time. It might be advanced that altered membranes of HbAS erythrocytes and elevated internal viscosity of HbS have played a role (7).

The lack of difference in T_k , k , and $\eta_{b_{45}}$ between the HbAA and HbASAT groups suggests that the presence of α -thalassemia in SCT carriers might be considered as beneficial for blood rheology as initially proposed by Le Gallais et al. (22). This result might be explained by the reduction in intra-erythrocyte HbS concentration in subjects with the coexistence of SCT and α -thalassemia compared with SCT subjects, as proved by the difference in HbS percentage between these two groups.

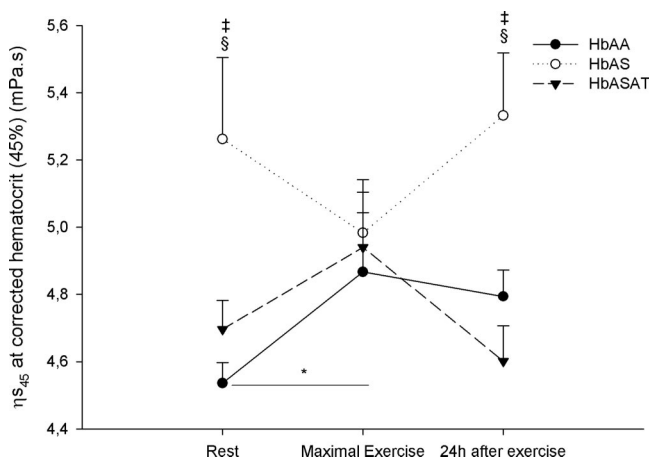


FIGURE 1—Blood viscosity at corrected Hct (45%) at rest, at the end of maximal exercise, and 24 h after exercise in HbAA, HbAS, and HbASAT groups. * Significant difference between rest, end of maximal exercise and 24 h after exercise ($P < 0.05$); † Significant difference between HbASAT and HbAS groups ($P < 0.05$); § Significant difference between HbAA and HbAS groups ($P < 0.05$). Values are mean \pm SEM.

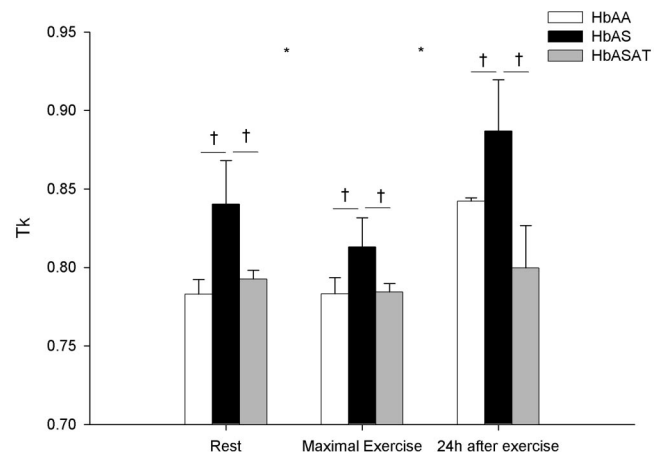


FIGURE 2—Red blood cell deformability (T_k index) at rest, at the end of maximal exercise, and 24 h after exercise in HbAA, HbAS, and HbASAT groups. * Significant difference between rest, end of maximal exercise, and 24 h after exercise ($P < 0.05$); † Significant difference between HbAA group and the other groups ($P < 0.05$); values are mean \pm SEM.

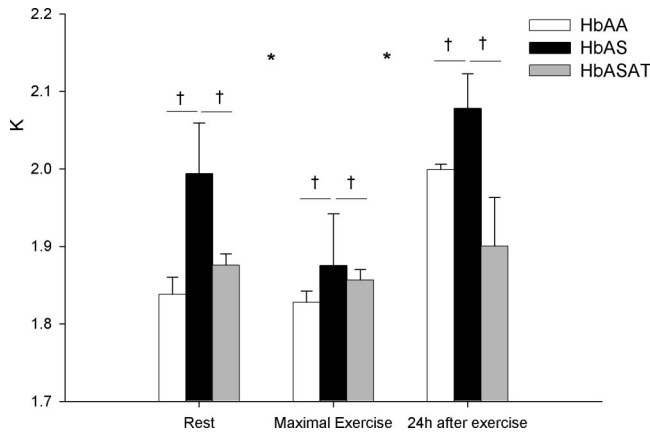


FIGURE 3—Red blood cell deformability (K index) at rest, at the end of maximal exercise and 24 h after exercise in HbAA, HbAS, and HbASAT groups. *Significant difference between rest, end of maximal exercise, and 24 h after exercise ($P < 0.05$); †significant difference between HbAA group and the other groups ($P < 0.05$); values are mean \pm SEM.

RBC deformability impairment in the HbAS group could exert detrimental effects on blood flow structuring in microcirculation (1,11,24,25). In the narrowest capillaries, whose luminal diameter is less than the undeformed size of RBC, erythrocytes advance in a greatly deformed state and in a single file flow. A thin blood plasma layer is always preserved during flow between the outer membranes of RBC and the luminal membranes of endothelial cells (25) and plays a lubricating role in preserving the blood flow in the lumina of microvessels. But, if RBC are less deformable, the plasma layer between RBC and endothelial membranes is reduced, leading to increased flow resistance (25). Moreover, less deformable RBC cannot pass through the narrowest capillaries or plug capillaries that favor plasma skimming (plasma flow without RBC) and lead to tissues ischemia (24). Indeed, SCT carriers could be prone to microvascular disorders because of the presence in their blood of low deformable RBC. However, the lower Hct observed in the HbAS group has partly compensated for the higher RBC rigidity found in this group leading to closed η_b in the

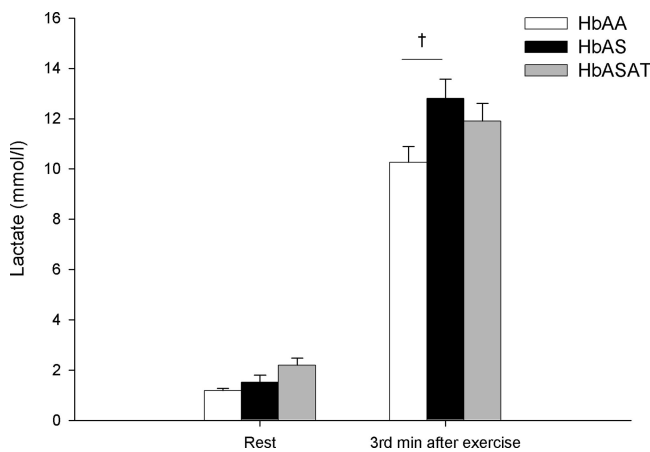


FIGURE 4—Evolution of blood lactate concentration between rest and the third minute after the end of exercise in HbAA, HbAS, and HbASAT groups. † Significant difference between HbAA and HbAS groups ($P < 0.05$); values are mean \pm SEM.

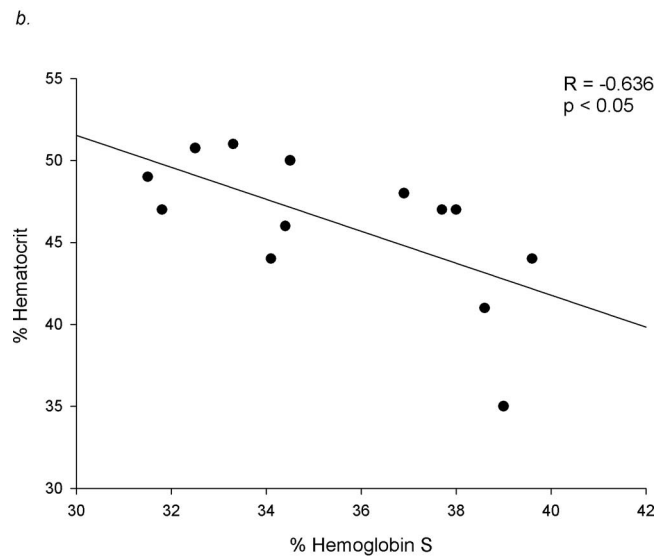
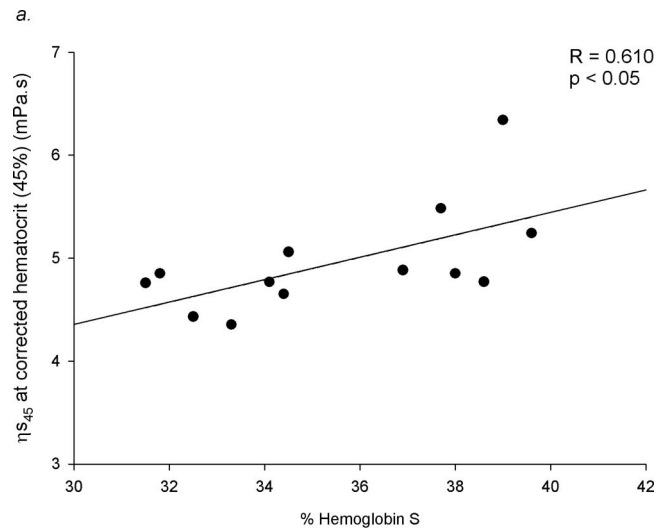


FIGURE 5—Relationship between percentage of Hb S and (a) baseline blood viscosity at corrected Hct (45%) and (b) Hct.

three groups. Hct was negatively correlated with the percentage of HbS in the HbAS and HbASAT groups. It is well known that the risk for hemolysis is increased in subjects with poor deformable RBC, as it was the case in the HbAS group. Indeed, the lower Hct found in HbAS athletes could be due to the lack of RBC deformability, which might be partly caused by the presence of high percentage of HbS. If Hct from the HbAS group had been around values found in the HbAA and HbASAT groups, η_b increase would have been dramatic because hyperviscosity syndrome has been found to strongly participate to cardiovascular complications in other diseases (1,11). The possibility of one hemorheological parameter compensating for the abnormality of another is frequently reported in the literature and is considered as beneficial for cardiovascular health and for blood flow structuring in macrocirculation (6).

Changes in η_p and Hct between rest and the end of maximal exercise were similar in the three groups of athletes. Change in Hct during exercise has already been described in healthy athletes (5,9) and can be explained by

several mechanisms including fluid shift, water loss, and RBC released from spleen (5). The increase in ηp is also frequently observed during exercise (5) and is thought to be due to a rise in plasma protein content, like $\alpha 1$ -globulins, $\alpha 2$ -globulins, β -globulins, and γ -globulins (29). Changes in Hct and ηp should both contribute to the rise in ηb during exercise. However, ηb increased in the HbAA and HbASAT groups but not in the HbAS group. Whereas ηb_{45} significantly increased between rest and the end of exercise in the HbAA group, no statistical ηb_{45} variation was measured in the two other groups. Calculations of ηb_{45} do not take into account the variations in Hct values. Indeed, one may suggest that Hct change in response to exercise exerted a great influence on ηb change in the HbASAT group. This was not the case for the HbAS group because ηb and ηb_{45} were unchanged by exercise. This finding can be explained by the surprising decrease in Tk and k during exercise that was more pronounced in the HbAS group compared with other groups. This marked decrease in RBC rigidity in the HbAS group has compensated both the rise in Hct and ηp . This finding implies that exercise did not lead to further hemorheological alterations in SCT carriers compared with other groups.

Results concerning the values of Tk and k contrast with previous studies that reported impairment in RBC deformability at the end of exercise bout in healthy subjects (5,30) and with results from Connes et al. (9), who observed no changes in Tk in some endurance athletes exhibiting repeated episodes of exercise induced hypoxemia. Nevertheless, the present findings are in accordance with other results from Connes et al. (9) and Hardeman et al. (17), who reported RBC deformability improvement during exercise in trained athletes. Among factors that alter RBC deformability, lactate is a powerful candidate. However, in this study, although the increase in lactate concentration was more pronounced in the HbAS group during exercise, RBC deformability was improved, as well as in the two other groups. Even if the decrease of Tk has already been reported in healthy athletes during exercise (9), it is very surprising that exercise did not alter RBC deformability in SCT carriers with or without α -thalassemia because lactic acidosis is known to lead HbS to polymerize that, usually, decrease RBC deformability (3,7). In healthy subjects, it has recently been demonstrated that relationships between lactate and RBC deformability could depend on training status (10). In addition, it is now recognized that in healthy athletes or in diabetics and cardiac patients (5), training reduces hemorheological variations caused by exercise regardless of whether blood rheology was normal or abnormal at baseline (5). In our study, because the HbAS and HbASAT groups were composed of well trained athletes, it is possible that

regular training lead RBC from SCT carriers to better tolerate lactate (and lactic acidosis).

Twenty four hours after exercise, ηb_{45} tended to return to resting values in all groups, and the HbAS group had higher values than the HbAA and HbASAT groups. The decrease in ηb_{45} in the HbAA and HbASAT groups may be explained by the decrease of ηp , which has partly compensated the increase in RBC rigidity. These findings are in accordance with previous studies that reported auto-hemodilution phenomenon and plasma volume expansion some hours after acute exercise (5) and alteration in RBC deformability 12 h after one exercise bout (30). The increase in RBC rigidity between rest and 24 h after exercise was not different between the HbAA and HbAS groups (8.9% and 6.1%, respectively). However, the decrease in ηp between rest and 24 h after exercise was lower in the HbAS group than in the HbAA group (5.6% and 7.0%, respectively). Indeed, the drastically increase in RBC rigidity in the HbAS group was not entirely compensated by the small decrease in ηp . It seems that during recovery, SCT carriers are prone to higher hemorheological alterations than other groups. The reappearance and magnification of hemorheological alterations in SCT carriers some hours after one exercise bout might be implied in injuries and incidents frequently observed in this population (20,21).

In conclusion, blood rheological impairment was found at rest and 24 h after exercise in athletes with SCT alone. Thus, α -thalassemia seems to protect SCT carriers against hemorheological impairment caused by the presence of SCT as previously suggested by Le Gallais et al. (22).

As already demonstrated by Brandao et al. (3), SCT carriers are prone to a reduced RBC deformability. These hemorheological alterations are thought to increase blood viscosity and participate to microcirculation disorders raising the risk for vaso-occlusion events. Surprisingly, in SCT carriers with or without α -thalassemia, hemorheological parameters in response to exercise were not further altered compared with resting values. All subjects were well trained and it is well known that training modifies and improves hemorheological behavior at rest and in response to exercise (9,10). Indeed, the surprising lack of hemorheological impairment amplification in HbAS and HbASAT groups in response to exercise may simply be related to their training status.

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