Pergamon

Clinical Hemorheology, Vol. 14, No. 6, pp. 807–818, 1994

0271-5198/94 $6.00 + .00

0271-5198(94)00054-9

HEMORHEOLOGIC EFFECTS OF LIGHT PROLONGED EXERCISE

J.F.Brun, J.P Micallef(*), A. Orsetti
Service d'Exploration Physiologique des Hormones et des Métabolismes CHRU Lapeyronie 34059 Montpellier-cédex
(*) INSERM U103 (Biomechanics) F-34000 Montpellier, France

(Received 21.07.1994; accepted 09.08.1994)

ABSTRACT

Increases in blood viscosity during various kinds of strenuous exercise have been repeatedly described. In this study we investigated the rheologic effects of long duration low intensity exercise. Twelve volunteers (21-39 yr, 6 men and 6 women), after overnight fasting, performed a 60 min exercise on cycloergometer at 55% of the theoretical maximal heart rate. After an early increase at the 10th minute ($p<0.001$), blood lactate dicreases ($p<0.02$) and returns to normal. During exercise there is an increase in plasma viscosity ($p<0.001$) and hematocrit ($p<0.05$) at the 10th minute. Red cell rigidity index "Tk" increases at the 20 th minute ($p<0.05$). Whole blood viscosity ($p<0.01$) increases and hematocrit/viscosity ratio decreases ($p<0.01$). Thus, a light prolonged work load induces a transient hyperviscosity pattern very similar to that which is observed during strenuous exercise bouts.

Key words: Blood viscosity, hematocrit, exercise, hemorheology, erythrocyte deformability, blood lactate.

INTRODUCTION

Increased physical activity is now considered as an important tool against cardiovascular disease (1-3). While strenuous exercise is sometimes at risk for acute cardiovascular accidents (4), low intensity prolonged exercise is considered as a safe procedure for improving both lipidic and glucidic metabolism and reducing atherogenetic abnormalities (5). Such a 'metabolic fitness' may be clearly obtained by light muscular activity, such as walking at a brisk pace, which represents 50% of the maximal aerobic power (6). Many kinds of exercise have been shown to acutely impair blood rheology (7-12) but we were not aware of studies concerning light prolonged exercise and rheology. Therefore, we investigated the possible hemorheologic effects of 1 hr cycling at 55% of the maximal theoretical heart rate.

SUBJECTS AND METHODS

Exercise test

Twelve healthy volunteers (age: 21-39 yr, 6 men and 6 women) remained fasting until they performed at 9 a.m. the exercise test. An indwelling catheter was set in a superficial vein of the cubital fossa. Blood samples were drawn at -15, 0, 10, 20, 30, 40, 50, 60 min as well as 10 min after stoping exercise (time 70 min). Exercise-tests were performed on a bicycle ergometer (Bodyguard, Jonas Oglaend A.S., N 4301- Sandnes, Norway). Heart rate was continuously monitored with the impulses coming from three electrodes taped to the subject's chest. Subjects exercised 1 hr at 55% of the maximal theoretical heart rate given by the tables of the American Heart Association.

Hemorheological measurements

Blood samples for hemorheological measurements (7 ml) were obtained with a large bore needle (Luer adaptor Venoject, set into the catheter) to avoid shear damage to erythrocytes. A vacuum tube was used for blood withdrawal, with potassium EDTA as the anticoagulant. No tourniquet was used for sample drawing in order to minimize venous stasis. Viscometric measurements were performed at high shear rate (1000 s^{-1}) with a falling ball viscometer (MT 90 Medicatest, 37 rue de l'Ermitage F-86280 Saint Benoit) (13-15). Accuracy of the measurements was regularly controlled with the Carrimed Rheometer 'CS' (Rhéo, 19 rue Ambroise Croizat, 91120 Palaiseau, France). The coefficient of variation of this method ranges between 0.6 and 0.8% (10 repetitive measurementsof the same sample). We measured with this device apparent viscosity of whole blood at native hematocrit, plasma viscosity, and blood viscosity at corrected hematocrit (45%) according to the equation of Quemada (16):

$$\mu b = \mu pl \, . \, (1 - 1/2 \, k.h)^{-2}$$

where μs is blood viscosity, μpl plasma viscosity, h the hematocrit and k a shear dependent intrinsic viscosity of the red cells according to Quemada.
Two indices of erythrocyte rigidity (Dintenfass' 'Tk' and Quemada's 'k') were calculated from blood viscosity, hematocrit and plasma viscosity measured at time 0 with equations derived from those given above:

$$k = 2.(1 - \mu r\text{-}0.5).h^{-1}$$

and:

$$Tk = (\mu r^{0.4} - 1).(\mu r^{0.4}.h)^{-1} \qquad (17)$$

Where μr is relative blood viscosity $\mu b/\mu pl$. The hematocrit/viscosity ratio, an index of oxygen supply to tissues, was calculated according to Chien (18) and Stoltz (19), with h (as percentage) divided by μb value at high shear rate which was determined as described above.

Biochemical analyses

The sampled blood was centrifuged and the plasma assayed for diverse parameters by well standardized and routine techniques, on an automatic clinical analizer (DuPont de Nemours). Both lactate and ammonia were assayed with the kits from DuPont specially adapted to this analyzer. Blood lactate assay was based on NADH production by rabbit lactate dehydrogenase. Coefficients of variation range beween 0,7 and 5.6 %.

Changes in plasma volume

A formula for calculating plasma volume changes (% ΔPV) during exercise from hematocrit changes has been published by investigators of the NASA-Ames Research Center (20,21,22) who demonstrated its validity in moderate as well as maximal exercise. We applied this formula to our data. The equation is:

$$\% \, \Delta \, PV = 100/(100\text{-}Ho) \times 100 \, [(Ho\text{-}H)/Ho]$$

where Ho is resting hematocrit and H hematocrit during exercise.

Statistics.

Results are presented as mean $\pm$ the SE of the mean. Correlations were performed using the method of least squares. Variables were compared using the nonparametric test of Mann-Whitney and Wilcoxon. Significance was defined as $p<0.05$. The choice of nonparametric tests was done in order to

adhere the guidelines of the ICSH expert pannel for blood rheology (23), since hemorheological parameters usually appear to exhibit a nonnormal distribution.

RESULTS

For reasons discussed elsewhere (15), viscosity values obtained with the MT90 viscometer are 1.3 fold lower than those leasured at the same shear rate with the Carri-Med Rheometer. In this study, as well as our other ones, we did not apply this correction factor. Comparison of our values with values that would be given by a rotational viscometer can be made by multiplying the results by 1.3.

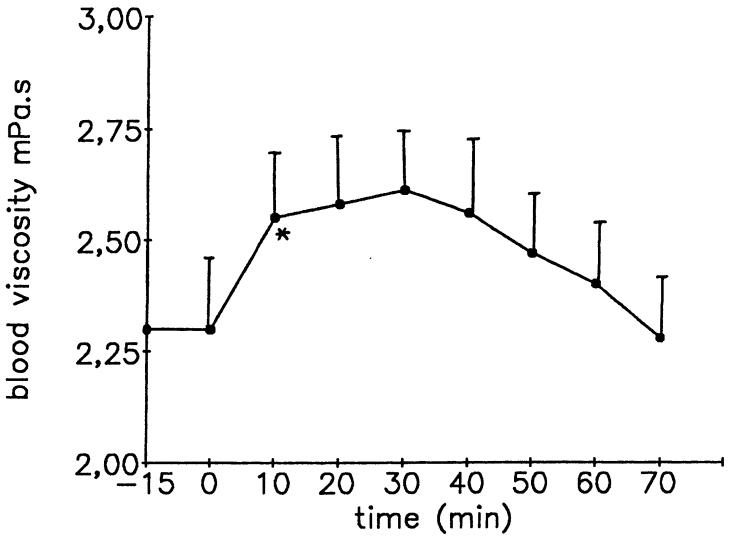

FIG.1

*Effects of light prolonged exercise on whole blood viscosity at native hematocrit: * $p < 0.05$ vs baseline.*

Fig. 1 shows that blood viscosity increases during the test ($p<0.05$ at time 10 min). This increase is explained by an increase in hematocrit (fig. 2), plasma viscosity (fig.3), and RBC rigidity (fig. 4). Blood viscosity at corrected hematocrit 45% is also increased (+ 9.5% $p<0.01$). The hematocrit/viscosity ratio is decreased at t 10 (-5.8 % $p<0.01$). Blood lactate increases from baseline values of 1.83 ± 0.22 mmol/l up to 2.57 ± 0.29 mmol/l at 10 min and then gradually decreases (fig. 5) to return within the resting range after 40 min. Plasma volume changes calculated with the equation of Greenleaf showed a

reduction of this volume (-6 to -7%) during all the session, followed by a normalization at the 10th minute of recovery (fig. 6).

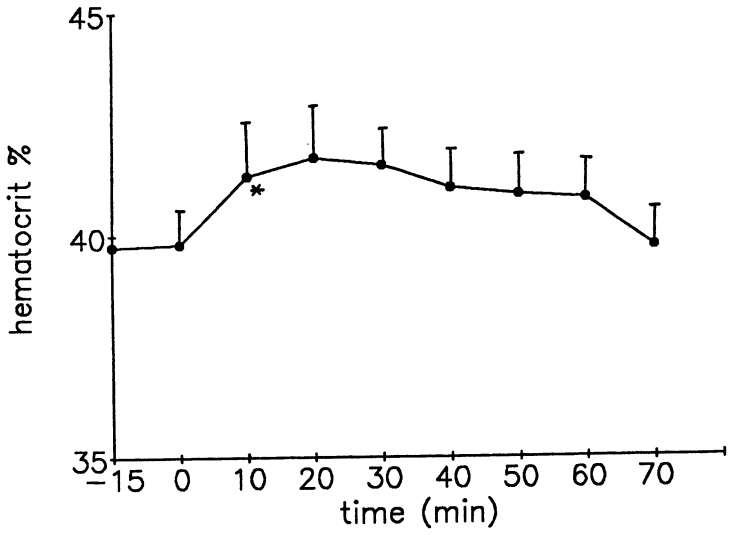

FIG.2

*Effects of light prolonged exercise on hematocrit. * $p < 0.05$ vs baseline.*

There is a correlation between Tk and blood lactate ($r = 0.227$ $p < 0.03$) as shown on fig. 7.

DISCUSSION

This study shows that very light exercise induces the same hemorheologic changes which have been reported during strenuous work loads. A rise in hematocrit, which indicates an outward movement of fluid from the vascular bed to the interstitial space during muscular activity (22) is a classical finding, even with a low intensity exercise (21). It was more surprising to notice that when viscosity was corrected for hematocrit, its increased remained highly significant, due to a rise in both plasma viscosity and red cell rigidity.

Since the red cell rigidity index "Tk" is correlated with blood lactate measured thorought the test, a role for blood lactate in this increase in red cell rigidity can be hypothesized, as previously published for stronger exercise protocols (12, 24, 25). Since both lactate (26) and acidosis (27) reduce red cell deformability, such a mechanism is theoretically possible. However, the increase in lactate observed during this protocol was moderate, far below the onset of metabolic

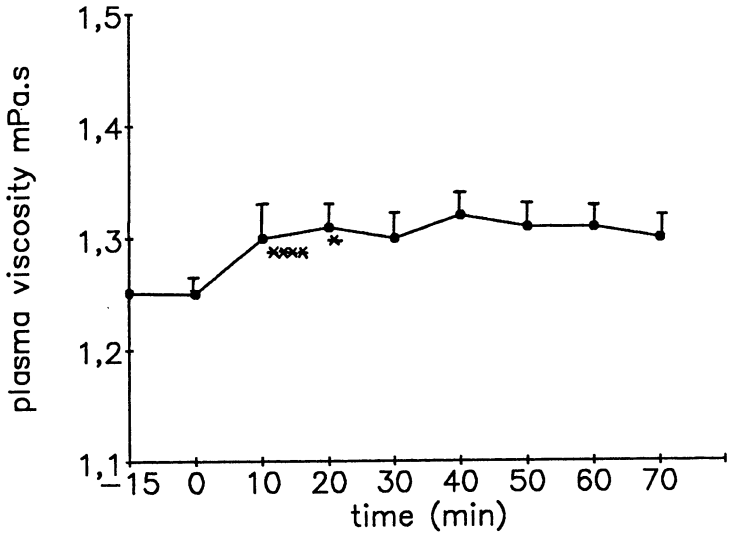

FIG.3

*Effects of light prolonged exercise on plasma viscosity . * p< 0.05 *** p< 0.01 vs baseline.*

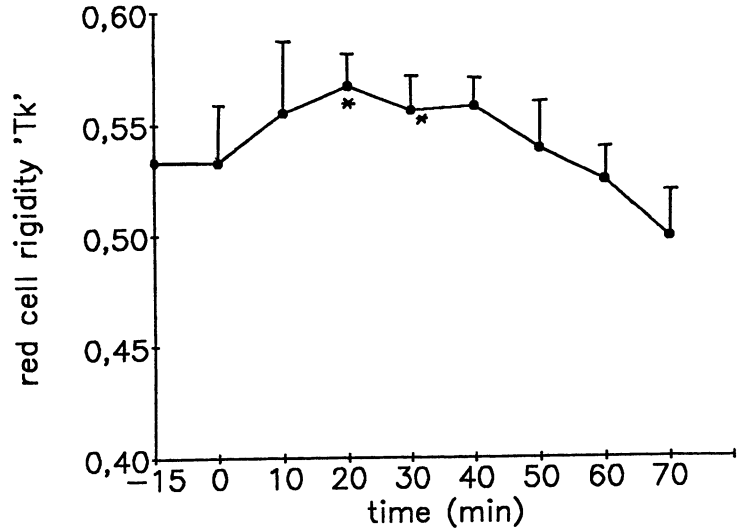

FIG.4

*Effects of light prolonged exercise on RBC rigidity index 'Tk' . * p< 0.05 vs baseline.*

acidosis (28). This kind of exercise is considered as an 'areobic' one in which fatigue is more related to glycogenic depletion and hypoglycemia, while lactate synthesis from pyruvate is not strongly stimulated (29). An early peak in blood lactate above 2 mmol/l is probably due to a rapid glucose oxidation to pyruvate, while the Krebs cycle becomes more slowly efficient (29). When the metabolism reaches a steady state, lactate is no longer released, as indicated by its progressive decline.

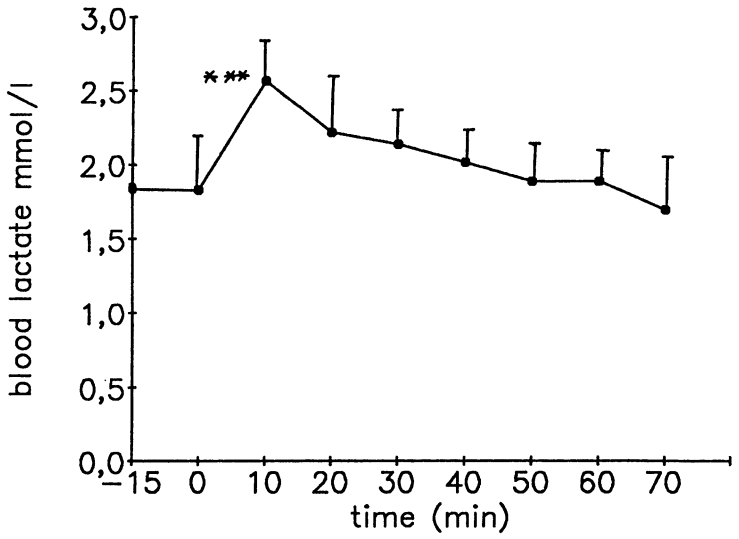

FIG.5

*Effects of light prolonged exercise on blood lactate. *** p< 0.01vs baseline.*

Another mechanism which may improve blood rheology during prolonged exercise is the oxidative stress induced by increased free radicals production (30). However, the kinetics of our modifications of 'Tk' which appear early and progressively return towards normal preexercise values, does not support this hypothesis since oxidative stress is more likely to progressively increase with the duration of exercise. Clearly, 'Tk' changes appear to be more parallel to the transient lactate peak and we believe that the two phenomenons are related.

An interesting hypothesis has been proposed by M. Gueguen-Delamaire (9) who suggested that impairment of blood rheology may be involved in the cardiovascular risk of maximal exercise, together with changes in hemocoagulatory parameters. Since we observe during this light, very safe exercise quite the same rheologic changes than during strong work loads, we think that these simple changes in hematocrit, red cell rigidity, and plasma viscosity are physiological adaptative modifications which occur during many

hematocrit/viscosity ratio, which measures the contribution of blood rheology to O_2 supply (18-19) is slightly decreased, but such a change can be easily overcome by vasodilatation. In our opinion, the risk of strong maximal or exhausting work loads is probably more related to modifications of hemostasis and to white cell activation.

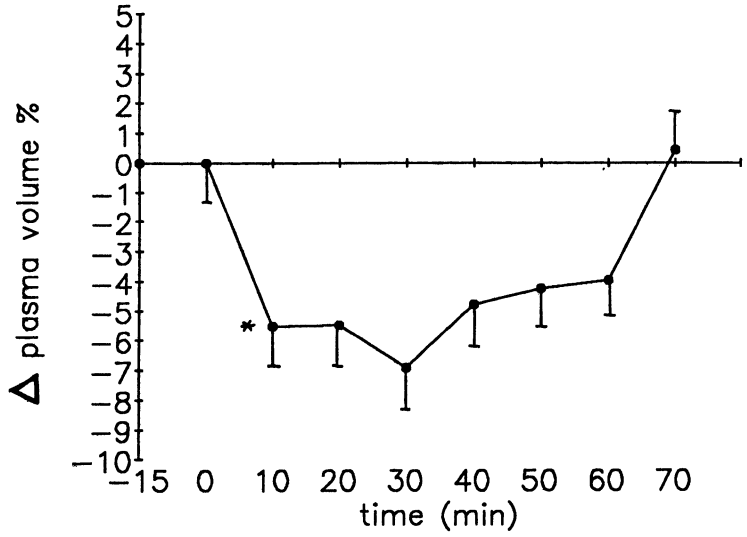

FIG.6

Changes in plasma volume caculated with the equations of Greenleaf during the prolonged light exercise session.

In conclusion, light prolonged exercise protocols at 55 % of the theoretical maximal heart rate induce the same hemorheologic modifications as strong short work loads, i.e. an increase in blood viscosity explained by a rise in hematocrit, plasma viscosity and erythrocyte rigidity. The latter event, which is transient and rapidly returns to normal, is correlated with blood lactate and may be related to its modifications during exercise.

ACKNOWLEDGEMENT

Our thanks are extended to Negma pharmaceuticals for their kind financial and technical support.

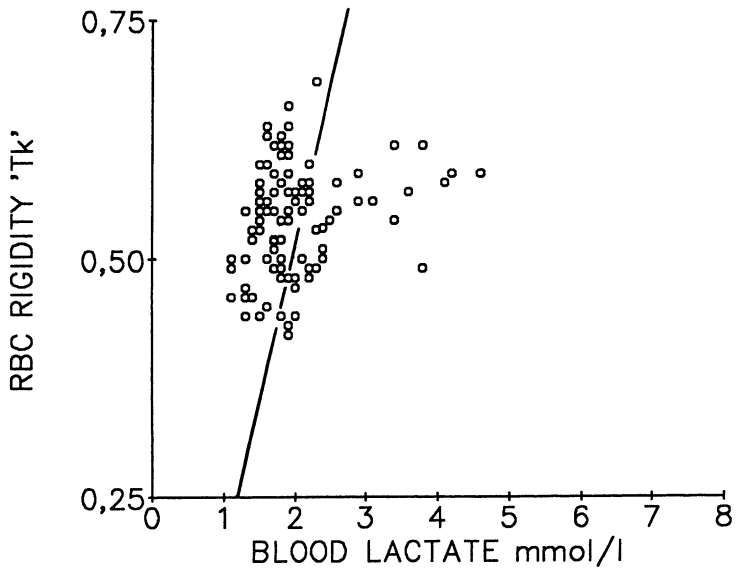

FIG.7

Correlation between Tk and blood lactate during the prolonged light exercise session. r=0.227 p< 0.03.

REFERENCES

1. SOPKO G, OBARZANEK E, STONE E. Overview of the National Heart, Lung and Blood Institute workshop on physical activity and cardiovascular health. Med Sci Sports Exerc 24, S192-S195, 1992.

2. BERLIN JA, COLDITZ GA. A metaanalysis of physical activity in the prevention of coronary heart disease. Am J Epidemiol 132, 612-628, 1990.

3. WOOD P. Effects of exercise on obesity, serum lipoproteins and blood pressure. In: Y Oomura, S. Tarui, S Inoue, and T Shimazu (Eds): Progress in Obesity Research. Proceedings of the 6th International Congress on Obesity. John Libbey, London, 1991: 385-389.

4. MARON BJ, EPSTEIN SE, ROBERTS WC. Causes of sudden death in competitive athletes. J Am Coll Cardiol 7, 204-214, 1986

5. TREMBLAY A, DESPRES JP, MAHEUX J, POULIOT MC, NADEAU A, MOORJANI S, LUPIEN PJ, BOUCHARD C. Normalization of the metabolic

profile in obese women by exercise at a low fat diet. Med Sci Sports Exerc 23, 1326-1331, 1991.

6. DESPRES JP, PRUD'HOMME D, TREMBLAY A, BOUCHARD C. Contribution of low intensity exercise training to the treatment of abdominal obesity: importance of "metabolic fitness". Int J Obes 15, suppl 1, 24, 1991.

7. LETCHER RL, PICKERING TG, CHIEN S, LARAGH JH Effects of exercise on plasma viscosity in athletes and sedentary normal subjects. Clin Cardiol 4, 172-179, 1981

8. ERNST E Changes in blood rheology produced by exercise. J Am Med Ass 253, 2962-2963, 1985

9. GUEGUEN-DUCHESNE M, DDURAND F, BEILLOT J, DEZIER JF, ROCHCONGAR P, LE GOFF MC, POMMEREUIL M, GENETET B Could maximal exercise be a hemorheological risk factor? Clin Hemorheol 7, 418, 1987

10. BRUN JF, LAGOUEYTE C, RAUTURIER M, FEDOU C, ORSETTI A. Hématocrite, viscosité plasmatique, et activité musculaire chez l'enfant. Science & Sports 4, 53-63, 1989

11. VANDEWALLE H, LACOMBE C, LELIEVRE JC, POIROT C Blood viscosity after a 1-h submaximal exercise with and without drinking. Int J Sports Med 9, 104-107, 1988.

12. BRUN JF, FONS C, RAYNAUD E, FEDOU C, ORSETTI A. Influence of circulating lactate on blood rheology during exercise in professional football players. Rev Port Hemorreol 5, 219-229, 1991

13. DOFFIN J, PERRAULT R, GARNAUD G Blood viscosity measurements in both extensional and shear flow by a falling ball viscometer. Biorheology (suppl.1), 89-93, 1984.

14. AILLAUD MF, POISSON C, BUONOCORE M, BILLEREY M, LEFEVRE P, JUHAN-VAGUE I. Etude du viscosimètre médical à chute de bille. Le pharmacien biologiste, 159, 291-294, 1985.

15. FONS C, BRUN JF, SUPPARO I, MALLARD C, BARDET C. Evaluation of blood viscosity at high shear rate with a falling ball viscometer. Clin. Hemorheol 13, 651-659, 1993

16. QUEMADA D. Rheology of concentrated disperse systems. II. A model of non newtonian shear viscosity in steady flows. Rheol Acta 17, 632-642, 1978.

17. DINTENFASS L. Red cell rigidity, "Tk", and filtration. Clin Hemorheol 5, 241-244, 1985.

18. CHIEN S, SUNG LA. Physicochemical basis and clinical implications of red cell aggregation. Clin Hemorheol 7, 71-91, 1987

19. STOLTZ JF, DONNER M, MULLER S. Syndromes d'hyperviscosité et transport d'oxygène: notion de profil hémorhéologique. 7e Réunion Conjointe de la Société d'Hémorhéologie de l'Ouest et de la Société de Biorhéologie de Langue Française. Rennes (France) May 18th, 1990.

20. GREENLEAF JE, CONVERTINO VA, MANGSETH GR. Plasma volume during stress in man: osmolality and red cell volume. J Appl Physiol 47, 1031-1038, 1979

21. Van BEAUMONT W, GREENLEAF JE JUHOS L. Disproportional changes in hematocrit, plasma volume and proteins during exercise and bed rest. J Appl Physiol 33, 55-61, 1972.

22. CONVERTINO VA, KEIL LC, BERNAUER EM, GREENLEAF JE Plasma volume, osmolality, vasopressin, and renin activity during graded exercise in man. J Appl Physiol 50, 123-128, 1981.

23. ICSH expert panel on blood rheology. Guidelines for measurement of blood viscosity and erythrocyte deformability. Clin Hemorheol 6, 439-453, 1986.

24. BRUN JF, FONS C, SUPPARO I, ORSETTI A. Low values of blood viscosity are associated with lower increases in blood lactate during submaximal exercise. 7th European Conference on Clinical Haemorheology 16th-19th July 1991, Southampton (United Kingdom) abstract book p 75.

25. BUCHERER C, LACOMBE C, LELIEVRE JC, WANDEVALLE H, VAUTHIER JF. Effet d'un apport hydrique sur les propriétés rhéologiques du sang de sujets effectuant un exercice submaximal. XIe Congrès National Scientifique de la Société Française de Médecine du Sport. Nantes (France) 13-15 Juin 1991 (abstract).

26. LIPOVAC V, GAVELLA M, TURK Z, SKRABALO Z. Influence of lactate on the insulin action on red blood cell filterability. Clin Hemorheol 5, 421-428, 1985.

28. KINDERMANN W, SIMON M, KEUL J. The significance of the aerobic-anaerobic transition for the determination of work load intensities during endurance training. Eur J Appl Physiol 42, 25-34, 1979.

29. RIEU M. Lactatémie et exercice musculaire. Signification et analyse critique du "seuil aérobie-anaérobie". Science & Sports 1, 1-23, 1986.

30. JACKSON MJ. Damage to skeletal muscle during exercise: relative roles of free radicals and other processes. In : Muscle fatigue in exercise training. P. Marconnet, PV Komi, B Saltin B and OM Sejersted (Eds). Basel, München, Paris, London and New York: Karger., 1992, pp131-139.